

Treating affective illness in patients with chronic pain

They resist psychiatric assessment, but you
can change 'patients with pain' to 'clients managing pain'



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Thomas K. Nelson, MD

Instructor in psychiatry, Mayo College of Medicine
Consultant in psychiatry
Mayo Clinic Scottsdale, Arizona

Lois E. Krahn, MD

Associate professor of psychiatry
Mayo College of Medicine
Chair, department of psychiatry and psychology
Mayo Clinic Scottsdale, Arizona

Ms. A, age 44, fell from a 3-foot stool while reaching for a high kitchen shelf and suffered severe neck flexion. Her initial pain persisted for weeks and then months, resulting in chronic neck pain aggravated by movement.

Over the past year, her doctor has prescribed numerous analgesics and muscle relaxants, including tramadol, hydrocodone, oxycodone, tizanidine, and nonsteroidal anti-inflammatory drugs (NSAIDs). Treatments at a pain clinic have included trigger-point injections, cervical epidural corticosteroid injection, left-sided cervical medial branch blocks, transcutaneous electrical nerve stimulation, and physical therapy. None provided sustained relief.

During a pain clinic visit, Ms. A wept and said she was tired of living with pain. She acknowledged depression and agreed to psychiatric consultation.

continued



As in Ms. A's case, physicians often refer patients with chronic pain and affective symptoms for psychiatric evaluation. These patients are often fearful, angry, and suspicious of any suggestion that their physical discomfort has a psychiatric component. They typically believe their pain had a clear onset and therefore should have an end point. Many have experienced unproductive specialty evaluations and failed treatments.

To help you overcome these obstacles when treating patients with chronic pain and depression, we discuss:

- strategies to gain patients' trust and build a therapeutic alliance
- how to assess their pain, depression, and suicide risk
- the role of psychotherapy in treating chronic pain
- and evidence for choosing effective, nonaddicting medications.

PSYCHIATRIC EVALUATION

Depression and pain are linked psychologically and biochemically, sharing neurotransmitters involved in both nociceptive pathways and mood, especially serotonin and norepinephrine.¹² One-third to one-half of patients with chronic pain report comorbid depression,³ and more than one-half of depressed patients presenting to primary care physicians report only somatic symptoms—various pain complaints among the most common.^{4,5}

Primary care doctors tend to refer chronic pain and depression cases to psychiatrists when:

- patients are preoccupied with medication, have not followed treatment recommendations, or do not respond to treatment as expected
- extensive medical evaluations reveal few or equivocal findings
- somatic complaints are vague and diffuse, or there is marked disparity between pain complaints/disability and objective findings.^{6,7}

Assessing pain. In the initial assessment, validate

the patient's pain experience by asking about the location, quality, and severity of pain. The visual analogue scale (VAS) is commonly used to measure pain severity. The patient marks a spot on a line from "no pain" to "worst possible pain," or—on a numbered VAS—from 0 (no pain) to 10 (extreme pain). The least and most severe pain over the preceding month can be ranked as baseline values.⁸

Be sensitive to the patient's fear that you will attribute the pain to psychosocial issues or imply that "the pain is in your head." Emphasize that you intend to evaluate the "whole person," not just the part that hurts. Focus on how the pain affects the patient's lifestyle—rather than its cause—and explore medication use patterns.

In about 5 minutes, Beck-II can assess vegetative symptoms that overlap in pain and depression

Assessing depression. The word "depression" is emotionally charged for chronic pain patients, who view affective symptoms—if they acknowledge them at all—as secondary to pain. They may strongly resist treatment for anything but pain. One way to defuse this defensiveness is to avoid attributing the pain to stress or depression.

Begin by assessing vegetative symptoms, which overlap in chronic pain and depression. The Beck Depression Inventory-II (Beck-II) may be a useful screening tool in a busy practice; the short form (13 questions) takes about 5 minutes to complete.⁹

Explore cognitive and behavioral symptoms such as concentration, pleasure and interest level, activity, and self-esteem. Review the chronology of pain onset, mood changes, and stressors (proximate, remote, and cumulative).

Seek clues to endogenous factors by asking about past affective episodes, response to antidepressants, and family history of psychopathology. Substances that may induce depression include reserpine, interferon, and antiparkinsonian agents. Screen for potential organic mood disorders, such as depression secondary to hypothyroidism, corticoid-

teroid use, Parkinson's disease, lupus, HIV infection, or cerebrovascular disease. Where appropriate, obtain collateral information from family or friends.

Assessing suicide risk. Chronic pain patients may be at greater risk of suicide than the general population. Besides pain, other risk factors for suicide—such as major depression, anxiety disorders, alcohol/substance abuse, sleep disturbances, male gender, diminished social support, and recent loss—are common among these patients.^{10,11}

Screen chronic pain patients with suicidal ideation for these risk factors. Interventions include:

- aggressively treat associated depression, anxiety, or insomnia
- elicit support from family or other caregivers
- pay close attention to talk about suicide
- hospitalize when necessary
- and, of course, treat pain.

Case continued: No stranger to depression

Ms. A's psychiatric assessments revealed a pain severity ranking of 9 on a 1-to-10 scale, frequent crying, hopelessness, disrupted sleep, low energy, limited ability to concentrate, and fleeting suicidal thoughts. Her history included counseling during her first marriage and severe depression after separation from her second husband 3 years ago. An 8-week trial of fluoxetine, 20 mg/d, did not improve her depression then.

On examination, she displayed obvious pain behavior, constantly shifting her neck position and moving about the room. Her affect was tearful and her mood depressed. She was taking the NSAID celecoxib, 100 mg bid, and the skeletal muscle relaxant tizanidine, 4 mg tid. She was no longer using opioids and had no history of alcohol or illicit drug abuse.

Based on this assessment, the psychiatrist diagnosed Ms. A as having pain disorder with medical and psychological features, including symptom amplification and depression.

Table 1

4 treatment goals for patients with chronic pain and depression

- **Identify** and reduce suicide risk.
- **Simplify** medications by eliminating as many as possible, while keeping those that are helpful.
- **Break** the cycle of repetitive physician evaluations and testing.
- **Improve** the patient's attitude, activity level, and ability to focus on something other than pain.

EDUCATING THE PATIENT

As part of your assessment, explain the reciprocal effects of depression and pain. Acknowledge that:

- chronic pain is different from acute pain, although the patient's pain experience is the same
- treatment often becomes part of the problem in chronic pain.

Doctors tend to apply acute pain treatments chronically, risking long-term effects of polypharmacy to achieve short-term relief. Depressed patients may be more likely than nondepressed patients to receive opioids for chronic pain,¹² and opioids and benzodiazepines may have depressive effects, as reflected by DSM-IV-TR's inclusion of criteria for "opioid-induced mood disorder" and "sedative-, hypnotic-, or anxiolytic-induced mood disorder."

To reduce patients' resistance to antidepressants, reiterate any history of cumulative stressors and affective episodes unrelated to pain. Try using an analogy, such as "stress and pain are like waves on a rock" that eventually damage mood and coping mechanisms, or depression complicating pain is like having "too much on one's plate."

Finally, help patients understand that chronic pain is managed, not cured. Encourage them to set treatment goals beyond reducing pain (*Table 1*) and to make the transition from "patient with pain" to "client managing pain."

continued



Table 2

Dosing antidepressants and anticonvulsants for chronic pain and depression

Drug	Starting (mg/d)	Target (mg/d)	Administration tips
TCAs			
Amitriptyline	10 to 25	75 to 300	Check serum levels for dosages ≥ 150 mg/d (nortriptyline ≥ 100 mg/d) to assess rapid metabolism, adherence, or toxic levels
Clomipramine	10 to 25	75 to 250	
Desipramine	10 to 25	75 to 200	
Doxepin	10 to 25	75 to 300	
Imipramine	10 to 25	75 to 300	
Nortriptyline	10 to 25	40 to 200	
SNRI			
Venlafaxine	37.5 to 75	75 to 375	Use XR form to minimize side effects and for once-daily dosing
SSRIs			
Citalopram	10 to 20	40 to 60	May increase carbamazepine, TCA blood levels and inhibit efficacy of codeine, dihydrocodeine, and hydrocodone
Fluoxetine	10 to 20	20 to 80	
Paroxetine	10 to 20	20 to 60	
Anticonvulsants			
Carbamazepine	200	800 to 1,200	Check blood levels; may increase clomipramine levels, reduce acetaminophen, contraceptive levels
Clonazepam	0.5	1 to 2	Habituating potential with chronic use
Gabapentin	300 to 900	3,600 to 4,800	Blood monitoring not necessary
Valproate	250	750 to 2,500 (maximum dosage 60 mg/kg/d)	Check blood levels (trough plasma level 50 to 100 $\mu\text{g/mL}$)

TCA: tricyclic antidepressant

SNRI: serotonin-norepinephrine reuptake inhibitor

SSRI: selective serotonin reuptake inhibitor

PRESCRIBING PRINCIPLES

Before adding any new pain medications, consider reducing dosages or discontinuing opioids or benzodiazepines and other substances the patient may be taking. Opioid use is associated with risks of dependence, addiction, and side effects including somnolence, cognitive impairment, and reduced activity that amplify depressive symptoms.

Benzodiazepines can generally be tapered by 10% per day, although you may need to extend the final taper over 3 to 4 days or longer, depending upon chronicity of use. Opioids may be tapered by 20% over 5 to 7 days. Breakthrough doses may be needed for marked withdrawal symptoms. Converting to longer half-life agents—such as clonazepam for benzodiazepines or methadone for opi-

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oids—often aids tapering, although other agents and strategies exist.¹³

To gauge patient attempts at self-medication, monitor use of alcohol or illicit drugs with urine screening. For patients with a substantial history of substance abuse or positive toxicology screens, monitor randomly every 2 to 4 weeks.

On the other hand, undertreated pain also may impair mood and function.¹ If pain and mood improve and problematic drug-related behaviors resolve with increased opioid analgesia, consider maintaining opioids with regular re-evaluation of mood, coping, and medication adherence.¹¹ Transfer from immediate-release to controlled-release opioids to reduce dosing frequency, clock-watching, and the likelihood of inter-dose pain escalation. In general, maintain and optimize the dosage of nonaddictive analgesics such as NSAIDs, anticonvulsants, or antidepressants.

An antidepressant's so-called 'adverse' effects could have a corresponding clinical benefit

Case continued: Switching medication

The psychiatrist started Ms. A on nortriptyline, 25 mg at bedtime, to be increased after 3 nights to 50 mg at bedtime. Tizanidine, which had been ineffective, was discontinued to reduce the risk of xerostomia and oversedation in combination with nortriptyline. If tolerated, nortriptyline was to be further increased by 25 mg every 3 days to an initial target dosage of 100 mg at bedtime. The psychiatrist explained to Ms. A that it might take 4 to 6 weeks to gauge the medication's efficacy.

Psychoeducation addressed the importance of stress reduction, prioritizing commitments, and setting limits on other people's expectations. The door was left open to future psychotherapeutic exploration of past cumulative stressors.

Because antidepressants may provide an analgesic effect,^{6,14} they are often used to treat affective symptoms in chronic pain. Headache and neuralgia tend

to respond to antidepressants more robustly than do arthritis and low-back pain. Although some patients respond to low-dose antidepressants, a definitive trial requires full doses for 6 to 8 weeks (*Table 2*).

Matching a patient's symptoms with medication side effects is useful when choosing antidepressants (*Table 3*). So-called "adverse" effects may have a corresponding benefit, depending on the clinical presentation. For example, a more-activating antidepressant—such as the selective serotonin reuptake inhibitor (SSRI) fluoxetine—may help a patient with fatigue, whereas a more-

sedating agent—such as a tricyclic antidepressant (TCA) or mirtazapine—may improve sleep for a patient with insomnia.

Psychosocial therapies such as cognitive-behavioral therapy (CBT) or relaxation training (*Table 4*) may help patients with chronic pain to:

- process covert emotions such as fear and anger as well as guilt, loss, and disability
- reduce somatic preoccupation that is aggravating the pain
- adhere to treatment.

Evidence strongly supports using relaxation techniques to reduce chronic pain in many medical conditions and hypnosis to ameliorate cancer pain. CBT and biofeedback appear moderately effective in relieving chronic pain.¹⁵ CBT is significantly more effective than waiting list control conditions for relieving chronic nonheadache pain in measures of pain experience, mood/affect, cognitive coping and appraisal, pain behavior and activity level, and social role functioning.¹⁶

Pain and opioid medications can impair concentration and affective processing, so initial psychotherapy may need to be supportive while you provide other treatments and simplify medication regimens. Eventually the patient may be ready to address underlying issues that may be contributing to the pain syndrome, such as a history of abuse.

continued



However, it is important to address this potentially destabilizing subject only after carefully gauging a patient's defenses and readiness.

Case continued: A bump in the road

The psychiatrist saw Ms. A 18 months later. Interim history revealed that her pain and mood improved on nortriptyline, 100 mg at bedtime. When she stopped taking nortriptyline 5 months earlier, her neck pain increased and she experienced a "deep blue mood." Her physician restarted the nortriptyline.

At follow up, Ms. A reported no depressive symptoms and very little neck pain. The psychiatrist discussed with her depression's relapse rate and the importance of continuing antidepressant therapy. As Ms. A was feeling much better and functioning normally, the psychiatrist decided additional psychotherapeutic intervention was not necessary.

ANTIDEPRESSANT OPTIONS

TCAs provide analgesia via descending regulatory pathways by inhibiting serotonin and norepinephrine reuptake.¹⁷ When using TCAs for chronic pain, start with 10 to 25 mg at bedtime and increase by 10 to 25 mg every 3 to 7 days as tolerated. Increase incrementally until the pain responds or to the full antidepressant dosage (Table 2). Drug levels (when available) can help you provide an appropriate trial and monitor the patient's adherence.

If the pain does not respond after 6 to 8 weeks, consider switching to another dual-action agent such as venlafaxine or to an SSRI.

SNRIs. Venlafaxine is a serotonin and norepinephrine reuptake inhibitor (SNRI) with less-troublesome side effects than TCAs. It is structurally similar to tramadol¹⁸ and has combined serotonin and norepinephrine inhibition at dosages >75 mg/d. Although venlafaxine is not indicated for chronic pain, some studies have suggested possible benefits,

including long-term analgesia, reduced polyneuropathic pain, and migraine prophylaxis.¹⁹⁻²¹ Venlafaxine may be a reasonable first or second choice for treating depression in patients with chronic pain, especially headache.¹⁴

Duloxetine—another SNRI—awaits FDA approval. Some studies have suggested that duloxetine improves painful physical symptoms as well as mood and functioning in major depression.²²

SSRIs may be effective for certain types of pain, but the evidence is conflicting. Results of 41 controlled trials support TCAs' analgesic efficacy for neuropathic pain, headache, and central and post-stroke pain, whereas SSRIs' analgesic efficacy varies from study to study. Comparisons of

TCAs and SSRIs as analgesics uniformly show TCAs to be more effective, with the SSRIs often showing no analgesic effect.

Of three controlled trials of SSRIs for diabetic neuropathy, one showed fluoxetine similar to placebo, and two smaller studies showed paroxetine and citalopram more effective than placebo.

Fluoxetine has shown analgesic effect for fibromyalgia in one study, but no effect in another. Citalopram showed no analgesic effect for fibromyalgia in another study.²³

A prospective, double-blind study comparing fluoxetine, sertraline, paroxetine, and venlafaxine for migraines reported moderate to significant improvement in less than one-half of SSRI-treated patients vs two-thirds of venlafaxine-treated patients.²¹ SSRIs are no longer recognized by the International Headache Society as primary preventative medications for migraine.

Fluoxetine may help chronic daily headache, and paroxetine and citalopram may be useful for diabetic neuropathy. However, one cannot generalize that all SSRIs are similarly effective as analgesics.¹⁴

SSRIs have fewer side effects than TCAs and

SSRIs are effective for some types of pain but usually are tried after dual-action agents

Table 3

Antidepressant side effects: Limitations and potential benefits in chronic pain

Side effects/ agents	Problems	Conditions potentially benefited	Possible alternatives
Anticholinergic TCAs	Xerostomia, constipation, urinary slowing (esp. when combined with opioids)	Diarrhea-predominant irritable bowel syndrome	SSRIs, nefazodone, venlafaxine
Sedation TCAs, mirtazapine, nefazodone, trazodone	Excessive sedation, cognitive impairment, driving risk (esp. when combined with opioids, benzodiazepines)	Pain with insomnia	SSRIs, venlafaxine, bupropion
Insomnia SSRIs, venlafaxine	Pain with pre-existing insomnia; equivocal analgesic effects	Excess sedation related to depression, polypharmacy for pain	TCAs, mirtazapine, trazodone, nefazodone
Orthostasis TCAs (esp. with methadone), nefazodone	Falls, especially in elderly patients	-----	Nortriptyline, SSRIs bupropion, venlafaxine
Weight gain TCAs, mirtazapine	Pain patients are often sedentary, get limited exercise	Pain and depression with weight loss	Bupropion, fluoxetine
Hypertension Bupropion, venlafaxine	Pre-existing hypertension	? Hypotensive state	Citalopram (hypertensive side effects infrequent)
Cardiac TCAs	ECG abnormalities, conduction delays, arrhythmias aggravate pre-existing cardiac abnormalities; avoid if recent MI	-----	SSRIs, bupropion
Overdose lethality TCAs	Prominent suicidal ideation	-----	SSRIs, venlafaxine
Seizures Esp. maprotiline, clomipramine, bupropion	Lower seizure threshold, aggravation of seizure disorders	-----	SSRIs
Sexual dysfunction SSRIs	Pre-existing sexual dysfunction secondary to pain, medications, stress; equivocal analgesic effects	-----	Bupropion, nefazodone, mirtazapine



Table 4

How psychosocial therapies can help treat chronic pain and depression

Therapies	Purpose/benefits
Behavioral therapy	Increase activity and learn to balance activity with limitations Reduce pain behaviors and analgesic use Decrease dependency and secondary gain
Cognitive-behavioral therapy	Identify automatic thoughts Challenge negative cognitions, catastrophizing Substitute and rehearse positive thoughts, capabilities Transition from patient role to self-care
Couples' therapy	Assist adaptation to role changes Diminish spousal solicitousness or excessive caretaking Increase communication
Biofeedback, relaxation, imagery	Adjunctive role in pain management Reduce tension, comorbid anxiety
Hypnosis	Dissociate awareness of pain Substitute, displace, reinterpret pain sensations
Vocational rehabilitation	Increase activity, ability to distract Regain sense of control, identity, and productivity Increase socialization
Pain management program	Multiple treatment effects Useful for complex pain with affective states

are less dangerous in overdose. In general, however, SSRIs are a second-line treatment for pain, to be used when dual-action agents pose disadvantageous side effects (Table 3) or have been poorly tolerated or ineffective.

Monoamine oxidase inhibitors (MAOIs) may have some efficacy for neuropathy and headache, but the need for a tyramine-free diet and potential for drug-drug interactions limit their usefulness. Co-administering an MAOI and meperidine is always contraindicated, as this combination can produce fever, delirium, seizures, circulatory collapse, and death. Similarly, avoid using an MAOI with any other antidepressant.

Others. Evidence is very limited on using other antidepressants such as trazodone, nefazodone, bupropion, and mirtazapine in chronic pain:

- Trazodone may help pediatric migraine,

but it is not a consistent analgesic and may not be well-tolerated.

- Case reports suggest bupropion may help with headaches and chronic low-back pain.¹⁴
- Mirtazapine and trazodone may be useful adjuncts for treating insomnia in depressed patients with chronic pain.

OTHER OPTIONS

Anticonvulsants appear useful for neuropathic pain and are appropriate for chronic pain patients who cannot tolerate TCAs.²⁴ Like TCAs, anticonvulsants are not addictive. Unlike TCAs, anticonvulsants may help stabilize other affective illnesses that may coexist with chronic pain, including bipolar disorder, schizoaffective disorder, and impulsivity/aggression related to dementia or personality disorder.⁶ If the starting dosage is not effective within 1 week,

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increase gradually every 2 to 3 days to target dosages comparable to those for anticonvulsant efficacy.

Carbamazepine and gabapentin are recommended first-line medications for neuropathy. Carbamazepine is indicated for treating trigeminal neuralgia, although its cytochrome P-450 3A3/4 isoenzyme induction may reduce serum levels of acetaminophen, opioids, and oral contraceptives. Gabapentin, although not clearly beneficial for bipolar disorder, has anxiolytic effects and a benign side-effect profile, which may help patients with chronic pain.

Valproate can help prevent migraines. Clonazepam can help reduce anxiety and restless legs syndrome but may be habituating. Anticonvulsants' common adverse effects include sedation, GI upset, dizziness, and fatigue.

Lithium has known efficacy for mood stabilization in bipolar disorder and can ameliorate cluster headaches.

Antipsychotics. Evidence is sparse on whether antipsychotics have analgesic activity. Their side effects generally limit their usefulness to treating pain in patients with psychosis or delirium.⁶

Stimulants such as dextroamphetamine and methylphenidate can be helpful adjuncts for treating depression, especially for medical inpatients who require a rapid therapeutic response. Stimulants may reduce fatigue or excessive sedation and improve concentration in patients receiving opioids for chronic pain. They also may have analgesic effects when combined with opioids. Potential adverse effects include appetite suppression, anxiety or agitation, confusion, tics, and addiction.⁶

Precautions. The muscle relaxant carisoprodol is associated with potential dependence and withdrawal. Cyclobenzaprine, another muscle relaxant, has a TCA-like structure and can be lethal in overdose. Baclofen can be useful for chronic pain related to spasticity, although psychotic depression and mania have been reported with abrupt withdrawal.⁶

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continued

Because pain perception and mood regulation are mediated by serotonin and norepinephrine, first consider dual-action antidepressants for treating comorbid pain and depression. Some SSRIs may be effective for some pains, such as chronic headache. Anticonvulsants may reduce pain while promoting affective stability.

BottomLine



Chronic pain

Related resources

- ▶ American Academy of Pain Medicine. www.painmed.org/
- ▶ American Academy of Pain Management. www.aapainmanage.org
- ▶ Pain.com: continuing medical education for anesthesiology professionals. www.pain.com/index.cfm
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DRUG BRAND NAMES

- | | |
|-------------------------------|-----------------------------------|
| Amitriptyline • Elavil | Imipramine • Tofranil |
| Baclofen • Lioresal | Lithium • Eskalith CR, Lithobid |
| Bupropion • Wellbutrin | Maprotiline • Ludiomil |
| Carbamazepine • Tegretol | Meperidine • Demerol |
| Carisoprodol • Soma | Methadone • Dolophine |
| Celecoxib • Celebrex | Mirtazapine • Remeron |
| Citalopram • Celexa | Nefazodone • Serzone |
| Clomipramine • Anafranil | Nortriptyline • Pamelor |
| Clonazepam • Klonopin | Oxycodone • OxyContin |
| Cyclobenzaprine • Flexeril | Paroxetine • Paxil |
| Desipramine • Norpramin | Sertraline • Zoloft |
| Dihydrocodeine • Synalgos-DC | Tizanidine • Zanaflex |
| Doxepin • Sinequan | Tramadol • Ultram |
| Duloxetine • Cymbalta | Trazodone • Desyrel |
| Fluoxetine • Prozac | Valproate • Depakote |
| Gabapentin • Neurontin | Venlafaxine • Effexor, Effexor XR |
| Hydrocodone • Vicodin, Lortab | |

DISCLOSURE

The authors report no financial relationship with any company whose products are mentioned in this article or with manufacturers of competing products.

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