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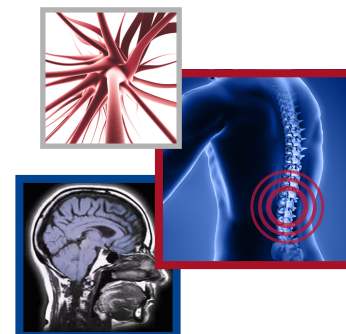
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# Emotional awareness and other emotional processes: implications for the assessment and treatment of chronic pain

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## Practice points

- Emotions and the processes of emotion regulation contribute to chronic pain onset and progression.
- Deficient emotional awareness is generally maladaptive, related to the presence and greater severity of chronic pain. In contrast, awareness of one's emotions is generally adaptive.
- Patients often report secondary emotional reactions (e.g., depression, anxiety, frustration), but accessing their primary emotions (e.g., anger, fear, sadness, love) is usually adaptive and has the potential to improve pain.
- Clinical assessment should include examining patients' awareness of their emotions and the link between their emotions and pain.
- Assessment for centrally mediated pain – the type of pain most influenced by psychosocial factors – suggests the use of emotional processing treatments.
- Psychological interventions that increase nonjudgmental awareness of one's emotions have small benefits on reducing pain severity and improving other pain-related outcomes.
- Broader emotional processing treatments, including Eye Movement Desensitization and reprocessing, Short-term Dynamic Psychotherapy and Emotional Awareness and Expression Therapy show promise for creating large magnitude reductions in pain.

Emotional awareness (EA) is a key emotional process that is related to the presence and severity of chronic pain (CP). In this report, we describe primary and secondary emotions, discuss the distinction between emotional states and emotional regulation/processing, and summarize theory and research highlighting the significance of EA for CP. We describe ways to assess EA and diagnose centrally-mediated CP, for which emotional processes appear most relevant. We review several psychological interventions designed to enhance EA as well as several broader emotional processing treatments developed to address trauma and psychosocial conflicts underlying many patients' pain. We conclude by offering our perspective on how future integration of emotional processing into pain care could promote recovery from CP.

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## Affective science & pain

Pain is both a sensory and emotional experience [1], but how emotion relates to pain needs clarification. It is widely recognized that persistent or chronic pain (CP) leads to emotional changes such as depression and anxiety. Emotions, however, are more than consequences of CP; emotions – and how they are regulated – are key drivers and maintainers of CP [2].

Research in affective science highlights two fundamental principles of emotions. First, emotions can be 'primary/adaptive' or 'secondary/reactive'. Primary emotions lead to healthy or adaptive activation in specific

contexts, including anger in response to violation or injustice, sadness when there is loss, fear of true danger, joy when experiencing gain, and pride of accomplishment. In contrast, secondary emotions inhibit or block getting one's needs met and usually stem from avoidance of adaptive primary emotions. Secondary emotions include anxiety, shame, tension, depression and frustration. As we elaborate below, processing of patients' primary emotions helps reduce secondary emotions and can reduce CP.

A second principle of affective science is the distinction between emotional states and emotional processes or regulation [3–5]. When asked by clinicians, patients typically report their emotional state – usually secondary emotions as noted above. Emotion regulation, however, refers to the processes governing the experience, expression, modification and use of emotions as well as the 'processing' or eventual cognitive-emotional resolution of trauma and conflicts. Although, emotion regulation often implies the 'downregulation' or reduction of negative feelings and the enhancement of positive ones, research shows that 'upregulating' patients' primary emotions – enhancing their awareness, internal experience, external expression and processing of emotions that are related to trauma and conflict – can be very beneficial for CP [2].

Of the various emotion regulation processes, emotional awareness (EA) is a foundational ability upon which subsequent processes depend and are facilitated. EA involves the capacity to identify and label one's emotions and to differentiate among emotions and between emotions and somatic states such as pain. In the sections below, we first present research and theory showing that deficits in EA are linked to CP. We then describe approaches to clinical assessment and intervention that target EA, and we subsequently expand the presentation to broader psychological interventions for CP that incorporate EA as part of emotional processing.

### Theory & research linking EA & CP

Deficits in EA and related problems such as lack of insight have long been linked to CP [6], but recent years have seen theoretical development and empirical support for the importance of EA. Research on EA has assessed this construct with self-rated questionnaires, responses to hypothetical scenarios, daily diary or experiential sampling, and clinical interviews. We will briefly examine research that uses the first three of these approaches and then discuss the role of the clinical interview in more depth in the next section. All these approaches, however, converge on the conclusion that deficient EA is linked to the presence and severity of CP.

The construct of alexithymia (literally meaning 'no words for feelings') refers to difficulty identifying and describing one's feelings and a preference for externally-oriented thinking rather than introspection [7]. Alexithymia is routinely measured with self-rating scales [8], and numerous studies have examined alexithymia in CP. A recent meta-analysis reported higher levels of alexithymia among those with CP compared with both healthy controls and nonpain clinical controls, and the degree of alexithymia was positively associated with pain intensity and interference [9]. This review also noted, however, that controlling for depression or anxiety largely accounted for the alexithymia-CP relationships, which raises questions about whether self-rated alexithymia has incremental validity or, alternately, whether alexithymia's effects are mediated by increases in distress.

Lane and colleagues proposed a developmental framework to conceptualize EA as ranging from the inability to identify and label emotions at one end, to the ability to recognize complex and differentiated emotions at the other. They also developed a measure that captures emotion language differentiation and complexity in response to imaginary emotional scenarios [10,11], and this measure is largely independent of distress. Studies using this measure have found that patients with CP have lower EA than controls, and the severity of pain is inversely associated with EA [12–14]. Lane and colleagues proposed the Model of Biased Competition to explain the EA/CP link. This model posits that bodily pain is a learned response to intolerable emotional pain; early life adversity leads to numbing and distancing from one's emotional experience, and attending instead to somatic symptoms, leading to CP in adulthood [15].

The ability to differentiate among one's own emotions is an aspect of EA. Studies using daily diaries or experience sampling [16] support the Dynamic Model of Affect, which posits that CP interferes with people's ability to differentiate their negative from positive affect [17]. This relationship is likely bidirectional: poor affect differentiation augments CP, suggesting that developing the ability to differentiate among emotions might reduce pain.

Research on EA – and on emotional processes more generally – has focused on the brain as the key organ of pain. For example, imaging research shows that accurately labeling emotions reduces activity in pain- and emotion-related brain regions, such as the amygdala and other limbic circuits [18]. Research also demonstrates that emotional pain stemming from social rejection activates similar brain regions to those activated by pain from physical injury [19].

More broadly, a wealth of animal and human research connects various emotion-regulating brain pathways to experiences such as childhood adversity, negative mood inductions and other affective experiences [20]. Importantly, activation in brain centers that regulate emotion is highly correlated with subjectively reported pain, whereas findings from spinal imaging and other clinical or laboratory studies of peripheral tissue pathology are not [21,22]. Emotional processes appear to be particularly relevant to the subset of CP conditions that are centrally-mediated (i.e., ‘centralized’, ‘primary’ or ‘nociplastic’ pain), such as fibromyalgia (FM), irritable bowel syndrome (IBS), head pain, pelvic pain syndromes and nonspecific chronic back and other musculoskeletal pain [23]. Indeed, numerous studies link adverse childhood events, trauma and post-traumatic stress disorder to the presence and severity of centralized CP [24–28]. Moreover, centralized pain conditions appear to be driven by trauma and emotion regulation difficulties more than are nociceptive or neuropathic types of pain [29–31].

### Assessment of EA & centrally-mediated pain in clinical settings

Research on the role played by emotions in triggering and maintaining CP [32], especially centralized pain, suggests the importance of assessing and enhancing EA. We view the assessment of EA in CP as comprising two interrelated processes: assessment of patients’ EA *per se*, and assessment aimed at patients’ understanding of the contribution of psychosocial and CNS factors to their CP (i.e., making a presumptive diagnosis of centrally mediated pain). We provide a brief overview here but note that the clinical assessment of emotional processes and centrally mediated pain can be complex and challenging, and we recommend further reading [31,33]. The questionnaires, scenarios and diary methods for assessing EA noted above are rarely used in clinical practice because they are usually too burdensome for patients or practitioners or seem too disconnected from the usual activities of clinical practice [34]. Instead, we commonly use targeted interviews and attending to patients’ responses (verbal and behavioral).

In our practice, we encourage patients to experience emotions during the interview, usually by having them recall and talk about stressful events or conflictual experiences. Doing so usually activates emotions as well as defenses against them. We probe to evaluate awareness of three components of emotions – the cognitive label, physical sensations, and behavioral impulses or action tendencies. We pay particular attention to the patients’ awareness of the somatic sensations associated with specific emotions (e.g., for anger, rising heat and energy), inquire about changes in their pain and explore whether they can differentiate their emotions from their pain. We also inquire whether patients have noticed how their stress, difficult relationships or other emotional situations affect their pain.

Another goal of the interview is to diagnose whether the patient’s CP is centrally mediated, which is the type of pain that we specifically target for emotion-focused intervention. We obtain both a medical and psychosocial history, and several findings point toward centrally-mediated pain [23,35]. Clinicians should inquire not only about current mental health but also about premorbid stressors, conflicts and mental health diagnoses; stressors that precede or accompany the onset of pain should be particularly noted. A careful examination of the patient’s medical and pain history and presentation is also needed to diagnose centralized CP. Specifically, the clinician should assess how closely the patient’s pain location correlates with findings on physical examination, imaging or other studies (e.g., electromyography); if pain is located in regions without associated objective findings, then a presumptive diagnosis of centrally mediated pain can be made. Pain characteristics can provide confirmatory information: centrally mediated pain is often randomly intermittent, symmetric, shifting in location, inconsistent, does not conform to a physiologic region and can be triggered by emotionally charged events or by innocuous stimuli, such as light, sounds, smells, computer screens, hot or cold, wind or foods [33,36].

### EA interventions for CP

Enhancing patients’ EA is a foundation of many forms of psychotherapy, and there is a long-held view that improving awareness of emotions and psychological conflicts can reduce somatic symptoms including pain [37]. There likely are multiple mechanisms linking enhanced EA to pain reductions, including direct changes in emotion and pain-related neural circuitry, reduced distress that drives pain, changes in beliefs about the dangerousness of pain and changes in stress-inducing relationships. Despite uncertainty about mechanisms, however, various interventions have targeted increasing EA to reduce pain.

Sarno’s astute clinical observations and theoretical models have had a substantial impact on innumerable patients and more recently on many clinicians working with CP [38]. In brief, Sarno proposed that most CP is driven by unconscious emotions – particularly anger – that are being repressed [38,39]. Although Sarno’s proposed mechanisms linking emotional processes and pain (e.g., oxygen deprivation in the tissues, pain as a distraction from emotions)

have doubtful validity, his intervention – educating patients that their emotional processes rather than tissue damage are causing the pain – is credited by numerous patients for greatly reducing or eliminating their pain [40].

There are relatively few interventions that focus solely on enhancing EA to reduce CP. Some self-help books, such as those by Sarno [40], might fit this category, but most other books include recommendations that go beyond EA to include expressing emotions, modifying unhealthy beliefs, and changing problematic interpersonal patterns [41,42]. There are many clinical suggestions for enhancing EA, such as training in self-observational skills or expanding one's emotional vocabulary, but these techniques lack controlled testing for CP. Experimental studies of some novel interventions, however, suggest some support for the benefit of enhancing EA. One randomized controlled trial (RCT) of patients with IBS found that providing EA training – education about primary emotions and tracking emotions in daily life – led to greater pain reduction than treatment-as-usual [43]. Another RCT found that a pain psychology and neuroscience education and self-awareness exercise reduced musculoskeletal pain severity and interference compared with a matched control exercise [44].

Many controlled studies have been conducted on two other interventions that emphasize EA. Mindfulness-based interventions for CP enhance nonjudgmental awareness of emotions as well as various internal and external sensations and experiences. Although popular in research and practice, a meta-analysis of 30 controlled trials of mindfulness-based interventions found only small improvements in pain, depressive symptoms and quality of life [45]. The self-directed technique of written emotional disclosure (expressive or therapeutic writing) aims not only to increase EA but encourage expressing in language one's emotions related to stressful experiences [46]. Reviews of the many experiments conducted in various populations show very small and inconsistent benefits of this technique [47]. A review of emotional disclosure in CP concluded that this technique has rather small benefits overall, but may show greater effects for centrally-mediated conditions such as FM than for disease-based pain like rheumatoid arthritis [48], perhaps because of the elevated trauma and psychiatric problems found in FM [49].

We think that for a minority of people, developing awareness of one's emotions and conflicts and their links to CP may be sufficient to reduce pain. For most people with centrally mediated CP, however, interventions to change emotional processing more generally are needed.

### Emotional processing interventions for CP

The term 'emotional processing' refers to set of inter-related processes including the somatic and mental experiencing of one's activated emotions, verbal or behavioral expression of emotions, and cognitive reflection on the goals, meaning and implications of one's experience [50,51]. Enhancing EA increases the likelihood that adaptive emotional processing will occur, but many patients may need help increasing their awareness, experience, and expression; and making cognitive, behavioral and interpersonal changes. Several therapies for CP have been developed and tested that include EA as part of a broader emotional processing framework.

Eye movement desensitization and reprocessing (EMDR) was developed to treat post-traumatic stress disorder and has been studied extensively in CP. EMDR facilitates processing of trauma by recounting emotionally disturbing memories while engaging in bilateral stimulation, usually eye movements but sometimes taps or tones [52]. Systematic reviews of case reports, uncontrolled case series and RCTs show EMDR often has large magnitude benefits for patients with CP, including substantial pain reduction or elimination, especially for phantom limb pain [53,54].

Short-term psychodynamic psychotherapy (STPP) includes brief therapy methods that aim to develop insight into emotional conflicts and recurrent patterns, express blocked emotions and improve interpersonal functioning. A recent meta-analysis of 17 RCTs reported that STPP led to large effect size reductions in somatic symptoms, including pain, in functional somatic disorders, when compared with minimal or no treatment [55]. A specific type of STPP, Intensive Short-Term Dynamic Psychotherapy, focuses especially on activating emotions related to conflict or trauma and has been found to have particularly strong effects on pain and other medically unexplained symptoms [56,57].

Emotional awareness and expression therapy (EAET) was developed to target psychological trauma or conflict in patients who have centrally-mediated CP [58]. Drawing from pain neuroscience as well as exposure, psychodynamic and other emotion-focused therapies, EAET reduces pain by helping patients connect unresolved emotional traumas and conflicts to their pain, increase awareness and expression of primary emotions and communicate emotional needs in important relationships [58]. Two RCTs found that a single session of EAET reduced pain and other symptoms compared with waitlist controls [59,60], and 3-session EAET reduced somatic symptoms in IBS compared with waitlist [61]. A large-scale RCT for FM compared 8-session group EAET, cognitive-behavioral therapy (CBT) and FM education, finding that EAET was superior to FM education on most outcomes and better

than CBT on several pain-related secondary outcomes [62]. Finally, a recent RCT on older military veterans with CP found that group EAET produced substantially greater reductions in pain severity and marginally greater reduction in pain interference than CBT [63].

In summary, these treatments that enhance EA as part of a broader emotional processing approach appear to have larger effects on pain reduction than interventions that focus only on EA or that downregulate negative emotions rather than processing trauma or conflicts, such as most cognitive-behavioral approaches for CP. Substantial pain reduction, especially with patients who have trauma histories or more complicated psychosocial presentations, likely requires emotional processing and linking such processing to pain.

## Conclusion

Improved outcomes of psychological treatments are both needed and possible. Widely used and tested psychological treatments for CP, such as cognitive behavioral pain management, mindfulness-based interventions, and acceptance and commitment therapy, do not reliably produce clinically-meaningful improvements for CP: although recent meta-analyses of randomized trials show that these treatments improve pain-related outcomes when compared to treatment-as-usual or no intervention, the effect sizes are often small, especially with respect to pain reduction [45,64,65]. In contrast, we and others have shown in clinical practice and randomized trials that centrally mediated CP can be substantially reduced and sometimes eliminated by incorporating pain neuroscience-based education to help patients learn that the pain is more brain-based than a sign of tissue injury or danger, as well as including emotional processing into treatments, creating powerful cognitive, emotional, and interpersonal changes in our patients.

## Future perspective

We anticipate and encourage several directions in research and practice that will advance the psychological assessment and treatment of CP. First, a key opportunity for integrating emotional processing into psychological treatments for CP is to shift how one conceptualizes and works with negative emotions. Emotional states such as depression, anxiety and frustration have long been viewed as undesired consequences of CP needing downregulation. Such a focus on secondary emotional reactions, however, misses the potential value that the activation and processing of primary emotions have for motivating patients to overcome trauma, engage more adaptively in relationships and recover from CP. We encourage a more nuanced view of emotions, differentiating secondary from primary emotions and understanding when to activate versus reduce emotions [2].

Second, despite increased uptake of some psychosocial treatments in multidisciplinary pain programs, a latent mind-body dualism still often stymies clinicians and leaves too many patients in pain. Psychological treatments are nearly always relegated to use as adjunctive to body-based treatments such as surgery, injections, physical therapy and analgesic medications. In fact, pain management is often agnostic about the etiology of pain and treats most types of CP similarly. Moreover, pain management often views pain as ‘chronic’ and needing to be managed, coped with, or accepted, rather than as malleable and potentially able to be greatly reduced or eliminated. Yet, recent advances in pain neuroscience and diagnosis indicate that subtypes of CP can be differentiated by the extent of underlying peripheral nociceptive or neuropathic processes versus central mediation. Many types of CP are generated primarily by brain processes such as predictive coding [20,66], and the brain’s emotions and cognitions influence all experiences of pain. If implemented, such mechanism-based treatment could direct clinicians toward the most appropriate treatments for each patient’s condition. Such an approach would make psychological treatment primary in many cases and included in all. A paradigm shift toward recognizing the brain as ‘the modifiable organ of pain’ holds great promise to go beyond pain management toward recovery.

## Author contributions

All authors contributed to the conceptualization, background research, writing and editing of the manuscript and approve the final article.

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