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Integrating Psychological Care in Multimodal Management of Whiplash-Associated Disorder After Motor Vehicle Accidents

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Purpose Statement

This white paper is intended to educate healthcare and legal professionals on the psychological conditions associated with Whiplash Associated Disorder WAD following Motor Vehicle Accidents MVA. It provides evidence-based guidance for identifying, understanding, and treating the psychological sequelae of WAD in order to improve patient outcomes and support informed medico-legal decision-making.

Executive Summary

WAD is more complex than a simple cervical strain and frequently includes psychological dysfunction that can significantly affect pain, disability, recovery, and quality of life. For many patients, symptoms may persist for months or years after the initial injury, and early psychological factors can be predictive of chronicity. This white paper reviews the psychological components of WAD and the neurophysiological mechanisms by which these factors may contribute to ongoing symptom burden. It also outlines evidence-based interventions including Cognitive Behavioral
Therapy, Pain Neuroscience Education, and Mindfulness-Based Stress Reduction, all of which have demonstrated benefit in appropriate patients. Early identification and timely treatment are recommended to optimize recovery and reduce the risk of long-term disability.

Key Findings

- A substantial proportion of patients with WAD develop depressive symptoms early after injury and a meaningful subset continue to experience long-term symptoms
- A notable percentage of whiplash patients meet diagnostic criteria for post-traumatic stress disorder following MVA
- Poor recovery expectations, post-traumatic stress symptoms, and passive coping strategies are consistent predictors of chronic neck pain and disability
- Catastrophizing and fear-avoidance behaviors are associated with a significantly greater risk of persistent pain and functional limitation
- Higher initial pain intensity is associated with increased risk of psychological sequelae and delayed recovery
- Persistent WAD-related stress may contribute to maladaptive neurophysiological changes including hyperalgesia and symptom amplification
- Psychological interventions have demonstrated benefit not only for emotional symptoms but also for pain, disability, and quality of life
- Delayed psychological treatment is associated with worse long-term functional outcomes compared with earlier intervention

Implications

Early recognition and treatment of the psychological components of WAD should be considered a core element of comprehensive care. Evidence-based interventions such as Cognitive Behavioral Therapy, Pain Neuroscience Education, and Mindfulness-Based Stress Reduction may improve recovery, reduce chronicity, and enhance overall function and well-being. Providers who can identify these issues early and coordinate appropriate referral patterns reflect a higher standard of care. From a medico-legal perspective, attorneys are well served by working with healthcare teams who understand both the physical and psychological dimensions of WAD and can document, treat, and explain these conditions effectively.

Introduction

Motor vehicle accidents MVA are traumatic events with well-documented effects on the musculoskeletal and nervous systems. One of the most common resulting conditions is Whiplash Associated Disorder WAD, a complex diagnosis characterized by a broad spectrum of symptoms that can be challenging to evaluate and treat.[1] WAD may involve multiple body regions including the cervical spine, shoulders, thoracic spine, lumbar spine, extremities, and can also include headache presentations.[2-7]

Prognosis following WAD is variable. While some patients recover within months, a significant proportion experience persistent pain and disability. As described by Gwendolen Jull et al., approximately 50 percent of individuals recover within 3 to 6 months, while others report symptoms for 1 to 2 years or longer. Among those who do not recover, 30 to 40 percent experience ongoing mild to moderate pain and 10 to 20 percent develop more severe chronic pain syndromes. [8]

In addition to physical symptoms, WAD is frequently associated with psychological sequelae that significantly influence recovery, disability, and quality of life. These factors are often under-recognized, particularly when patients present with substantial physical pain. Psychological symptoms including depression, sleep disturbance, anger, fear, anxiety, and post-traumatic stress disorder PTSD are commonly reported and may persist for years following the initial injury. [9-14] These symptoms have been documented to last up to 5 years in some cases.[15-16]

The interaction between psychological and physical symptoms further complicates management. Patients with persistent pain beyond six months frequently demonstrate elevated psychological distress and post-traumatic stress responses. [17] Higher pain and disability levels are closely associated with post-traumatic stress symptoms, and validated measures such as the Neck Disability Index and Tampa Scale of Kinesiophobia demonstrate meaningful correlations between disability and fear-based behaviors.[18-19]

Importantly, a systematic review of 31 studies identified poor recovery expectations, post-traumatic stress symptoms, and passive coping strategies as consistent predictors of chronic neck pain and disability following whiplash injury. [20] These findings highlight a bidirectional relationship in which psychological distress and physical pain reinforce one another, contributing to symptom persistence.

Previous work has outlined the biomechanics, pathophysiology, and medical management of WAD, including the importance of early intervention for pain control. [8] This paper expands on those concepts by focusing on psychological dysfunction, its influence on pain processing, and the role of early identification and treatment. Recognition and management of these factors may improve adherence to care, enhance recovery, and favorably influence long-term prognosis. [21-23]

Psychological Sequelae of Whiplash-Associated Disorder

Following MVA trauma, patients may develop a range of psychological responses that warrant early identification and, when appropriate, referral for specialized care. These include acute stress reactions, pain-related fear and hypervigilance, anxiety, depression, post-traumatic stress symptoms, and catastrophization.

Acute stress reactions occur within 3 days to 4 weeks following trauma and are defined by a constellation of symptoms including intrusive memories, nightmares, flashbacks, negative mood, dissociation, avoidance behaviors, and heightened arousal such as sleep disturbance and irritability.[24-25] Assessment tools such as the Impact of Event Scale can assist in identifying patients at increased risk for persistent pain, disability, and reduced functional outcomes at one year.[25]

Pain-related fear and hypervigilance are also common. Pain-related fear reflects the perception that pain signals indicate ongoing harm or reinjury, while hypervigilance refers to heightened attention and sensitivity to pain-related stimuli. [26-27] These responses can contribute to avoidance behaviors and functional decline.

Anxiety is characterized by apprehension or anticipation of perceived danger. In WAD populations, higher pain intensity is associated with increased anxiety, which may amplify attention to symptoms and negatively affect recovery. [28-30]

Depression is frequently observed in WAD, particularly in patients with higher initial pain levels. Up to 40 percent of patients may experience depressive symptoms early after injury, with approximately 20 percent continuing to report symptoms long term. [31] Depression is associated with poorer recovery trajectories and serves as an important prognostic indicator.[32-33]

Post-traumatic stress symptoms PTSS may develop shortly after trauma and progress to PTSD if symptoms persist beyond 30 days. [34] These symptoms include avoidance, nightmares, irritability, cognitive changes, hypervigilance, and emotional detachment. PTSS and PTSD have been associated with prolonged pain, increased pain sensitivity, cervical muscle changes, and sustained disability. [13-35-41] These findings emphasize the importance of early recognition and intervention.

Catastrophization represents an exaggerated negative cognitive response to pain, including rumination, magnification of symptoms, and feelings of helplessness. [42] It has been described as one of the most influential factors in pain-related disability and is strongly associated with poor outcomes if not addressed early. [43-46]

Collectively, these psychological factors are modifiable and represent critical targets for early, multidisciplinary intervention. Timely identification and appropriate management may reduce the risk of chronic pain, improve function, and enhance overall patient outcomes. [9-47-48]

Pathophysiology of Psychological Contributors to Chronic Pain

The role of the central nervous system in chronic pain has been extensively studied, with strong evidence demonstrating that neurophysiological changes contribute to heightened pain sensitivity and symptom persistence.

Following a traumatic event such as an MVA, the body activates the sympathetic nervous system, initiating a fight-or-flight response. This response involves two key pathways: the sympathetic-adreno-medullary SAM axis and the hypothalamic-pituitary-adrenal HPA axis.[49] The SAM axis mediates the immediate stress response through catecholamine release, while the HPA axis produces a slower hormonal response.[49] Although these systems are essential for normal adaptation, dysregulation is commonly observed in patients with chronic pain.

A key hormone affected in this process is cortisol.[50] Acute stress may variably increase or decrease pain sensitivity, but chronic stress, as seen in persistent WAD, is associated with prolonged elevation of cortisol levels and maladaptive physiologic effects.[51-53] These include impaired immune function, increased adiposity, vascular changes, and importantly, hyperalgesia, an amplified pain state.[51-53]

This creates a reinforcing cycle in which persistent nociceptive input from WAD contributes to central nervous system activation, leading to hyperalgesia, which in turn amplifies pain perception and psychological stress.[50-54-57] This feedback loop is a key mechanism underlying prolonged pain and increased disability in WAD populations.

In addition to neurophysiological changes, learned pain behaviors contribute to symptom persistence. Patients often develop avoidance of movements associated with pain, which may initially reduce symptoms but ultimately leads to deconditioning and increased disability.[58-59]

As described by Vangronsveld et al., individuals with pain catastrophizing are more likely to develop fear-based avoidance and hypervigilance. While these behaviors may reduce pain in the short term, they contribute to long-term disability and reinforce negative pain perceptions. [59]

Patients demonstrating catastrophizing and fear-avoidance behaviors have a significantly higher risk of chronic pain and disability. [60-66] Central nervous system effects may also extend beyond pain perception, with studies demonstrating associated cognitive impairments in chronic WAD populations.[67]

Given these impacts, early identification of maladaptive psychological and behavioral patterns is critical. Clinicians should incorporate screening and consider referral to pain psychology specialists when appropriate to improve outcomes and reduce chronicity.

Evidence-Based Psychological Interventions in WAD

Evidence supports several psychological interventions that improve pain, function, and quality of life in patients with WAD.

Cognitive Behavioral Therapy CBT focuses on identifying and modifying maladaptive thought patterns that interfere with recovery. This includes addressing automatic thoughts, cognitive distortions, and core beliefs that influence perception of pain and injury. [68] Dysfunctional beliefs such as fear of reinjury or misinterpretation of pain signals can significantly hinder recovery.[68-69]

CBT interventions may include psychoeducation, exposure-based strategies, cognitive restructuring, anxiety management, and relapse prevention, all aimed at improving coping and facilitating recovery. [70-71]

Pain Neuroscience Education PNE is an educational approach that helps patients understand the neurobiological mechanisms underlying pain. [72] By reframing pain as a multifactorial and modifiable process, PNE can reduce fear, catastrophizing, and maladaptive beliefs.

PNE has demonstrated effectiveness across multiple chronic pain conditions and is particularly beneficial when combined with active rehabilitation. It has been associated with reduced activation in brain regions related to pain processing and improvements in anxiety, kinesiophobia, and catastrophizing. [72-74]

Mindfulness-Based Stress Reduction MBSR emphasizes present-moment awareness and non-judgmental acceptance of experiences. [75] Interventions may include meditation, group therapy, and body awareness practices.[76-77]

MBSR has demonstrated improvements in mood, coping, and overall quality of life, even in the presence of ongoing pain, making it a valuable adjunct in the management of chronic WAD. [76-78]

Clinical Evidence Supporting Psychological Care in WAD

A growing body of literature supports the use of psychological interventions such as Pain Neuroscience Education PNE, Mindfulness-Based Stress Reduction MBSR, and Cognitive Behavioral Therapy CBT in the management of WAD. These approaches utilize different strategies but consistently demonstrate benefit in addressing psychological dysfunction and improving patient outcomes.

Pain Neuroscience Education PNE has demonstrated measurable effects on central nervous system processing. A 2024 study by Corbo et al. reported that PNE altered neural activity in regions associated with pain perception and motor control, including decreased activation of the periaqueductal gray and cerebellum with increased activation of the motor cortex following treatment. [74]

Clinical outcomes further support these findings. A pilot study in patients with chronic WAD demonstrated improvements in kinesiophobia, pain coping, photophobia, disability, pain-pressure thresholds, and pain-free movement following PNE intervention. [79] Additional case-based evidence has reported similar functional improvements. [80]

Larger studies evaluating PNE across chronic spinal pain and PTSD populations have demonstrated consistent improvements in pain intensity, disability, illness perception, catastrophizing, kinesiophobia, and post-traumatic stress symptoms across a combined cohort of 978 patients. [81-83]

Importantly, a 2025 trial including over 100 patients with chronic WAD demonstrated that PNE improved neck-related disability both immediately post-treatment and at 12-month follow-up. Additional benefits included reductions in pain-related anxiety, central sensitization symptoms, and improved cost-effectiveness of care. [84]

Mindfulness-Based Stress Reduction MBSR has also demonstrated meaningful clinical and neurophysiological benefits. Reviews of chronic spinal pain populations identify MBSR as a commonly utilized and effective intervention, with evidence suggesting it may serve as a protective factor against trauma-related psychopathology. [77]

Neuroimaging research has shown that MBSR alters brain connectivity in regions associated with anxiety, with corresponding reductions in patient-reported anxiety following an 8-week intervention. [85] In patients with PTSD, MBSR has been associated with improved regulation of limbic system activity and symptom reduction.[86] Case-level evidence further supports improvements in catastrophizing and quality of life in WAD populations.[87]

Multimodal approaches incorporating MBSR have also shown benefit. A 2021 review recommended combining stress reduction, education, and exercise to address the multifactorial nature of WAD, emphasizing the importance of treating both physical and psychological contributors to pain. [88] These findings are supported by meta-analytic data demonstrating MBSR as an effective intervention for psychological dysfunction.[78]

Cognitive Behavioral Therapy CBT remains one of the most extensively studied interventions for chronic pain and WAD-related psychological dysfunction. Evidence supports its use in reducing disability associated with chronic spinal pain. [89]

Randomized controlled trials have demonstrated that CBT improves catastrophizing, fear of movement, self-efficacy, and pain-related disability compared to standard self-care approaches. [90] Notably, both in-person and internet-delivered CBT have been shown to be equally effective, increasing accessibility to care.[90]

A systematic review and meta-analysis of randomized controlled trials found moderately favorable evidence supporting combined physical therapy and CBT compared to advice alone for improving long-term disability in WAD patients. [91]

CBT has also demonstrated effectiveness in addressing PTSD associated with WAD. [92] Further research evaluating timing of intervention found that early CBT initiated within one week of identifying psychological risk factors resulted in improved pain and psychological outcomes at one year compared to delayed treatment at three months. Patients receiving delayed care demonstrated increased disability, suggesting that an early therapeutic window may exist for preventing chronicity following whiplash injury. [23]

Conclusion

Whiplash Associated Disorder WAD is not limited to cervical spine injury, but represents a complex, multi-system condition with significant psychological and neurophysiological components following MVA trauma. These factors play a critical role in pain persistence, disability, and overall quality of life.

Early identification and appropriate treatment of psychological dysfunction may be a key determinant in whether a patient recovers or progresses to chronic pain and long-term disability. Evidence supports the integration of psychological care into comprehensive treatment strategies to improve outcomes and reduce the risk of chronicity.

For attorneys and healthcare providers, a clear understanding of these mechanisms is essential for effective case management and patient advocacy. The principles outlined in this paper reflect current evidence and clinical practice patterns aimed at optimizing recovery while strengthening medico-legal support.

Psychological interventions can significantly influence pain perception, functional outcomes, and recovery trajectory in patients with WAD. Referral to experienced providers who can identify and address both physical and psychological contributors to injury may improve patient outcomes and help prevent the transition to chronic pain.

References

1. Hendriks EJ, Scholten-Peeters GG, van der Windt DA, et al. Prognostic factors for poor recovery in acute whiplash patients. Pain. 2005;114:408-416.
2. Ghorayeb JH. The nosological classification of whiplash-associated disorder: a narrative review. J Can Chiropr Assoc. 2021;65:76.
3. Vállez García D, Dierckx RAJO, Otte A, et al. Whiplash: real or not real? A review and new concept. In: PET and SPECT in Neurology. 2014:947-963.
4. Bannister G, Amirfeyz R, Kelley S, et al. Whiplash injury. J Bone Joint Surg Br. 2009;91:845-850.
5. Fundaun J, Kolski M, Baskozos G, et al. Nerve pathology and neuropathic pain after whiplash injury: a systematic review and meta-analysis. Pain. 2022;163:e789-e811.
6. Särkilahti N, Leino S, Takatalo J, et al. Symptom profile of individuals with whiplash-associated disorder: a mixed-method systematic review. J Bodyw Mov Ther. 2024.
7. Alderink GJ, Ashby BM. Cervical spine: lateral whiplash, neck pain, and nerve palsy. In: Clinical Kinesiology and Biomechanics. 2023:145-177.
8. Jull GA, Söderlund A, Stemper BD, et al. Optimal early management after whiplash injury to reduce chronicity. Spine. 2011;36:S335-S342.
9. Al-Khazali HM, Ashina H, Iljazi A, et al. Psychiatric sequelae following whiplash injury: a systematic review. Front Psychiatry. 2022;13:814079.
10. Sterling M, Jull G, Vicenzino B, et al. Physical and psychological factors predicting outcome after whiplash injury. Pain. 2005;114:141-148.
11. Smith A, Andruski B, Deng G, et al. Sleep disturbance and health outcomes in chronic WAD. Clin J Pain. 2022;38:612-619.
12. Williamson E, Williams M, Gates S, et al. Psychological factors and late whiplash syndrome. Pain. 2008;135:20-30.
13. Andersen TE, Elklit A, Vase L. Chronic WAD and PTSD: attachment anxiety as a vulnerability factor. Eur J Psychotraumatol. 2011;2:5633.
14. Carroll LJ, Liu Y, Holm LW, et al. Pain-related emotions in early WAD recovery. Psychosom Med. 2011;73:708-715.
15. Craig A, Tran Y, Guest R, et al. Psychological impact of motor vehicle injuries: systematic review. BMJ Open. 2016;6:e011993.

16. Styrke J, Sojka P, Björnstig U, et al. Symptoms and disability five years after whiplash. Scand J Pain. 2014;5:229-236.
17. Sterling M, Kenardy J, Jull G, et al. Development of psychological changes after whiplash. Pain. 2003;106:481-489.
18. Sterling M. Whiplash-associated disorder: clinical findings. J Man Manip Ther. 2011;19:194-200.
19. Vernon H, Guerriero R, Soave D, et al. Disability, fear-avoidance, and nonorganic signs in WAD. J Manipulative Physiol Ther. 2011;34:506-513.
20. Campbell L, Smith A, McGregor L, et al. Psychological factors in chronic WAD. Clin J Pain. 2018;34:755-768.
21. La Touche R, Pardo-Montero J, Grande-Alonso M, et al. Psychological and disability factors in musculoskeletal pain recovery. Healthcare. 2023;12.
22. Gasslander N, Alfonsson S, Jackalin A, et al. Predictors of adherence to internet-based CBT for chronic pain. BMC Psychol. 2021;9:156.
23. Andersen TE, Ravn SL, Mejldal A, et al. Values-based CBT for prevention of chronic WAD: RCT. Eur J Pain. 2022;26:1256-1268.
24. Fanai M, Khan MA. Acute stress disorder. 2020.
25. Kongsted A, Bendix T, Qerama E, et al. Acute stress response after whiplash. Eur J Pain. 2008;12:455-463.
26. Rogers AH, Farris SG. Fear-avoidance model and chronic pain outcomes: meta-analysis. Eur J Pain. 2022;26:1611-1635.
27. Zhang W, Löffler M, Usai K, et al. Hypervigilance and transition to chronic pain. J Pain Res. 2025;3141-3158.
28. Clark VL, Kruse JA. Clinical methods: history and examination. JAMA. 1990;264:2808-2809.
29. Pina D, Puente-López E, Ruiz-Hernández JA, et al. Biopsychosocial profiles in WAD forensic cases. Front Psychol. 2021;12:716513.
30. Elklit A, Jones A. Anxiety and chronic pain after whiplash. Clin J Pain. 2006;22:487-490.
31. Phillips LA, Carroll LJ, Cassidy JD, et al. Depression in WAD. Eur Spine J. 2010;19:945-956.
32. Wenzel HG, Haug TT, Mykletun A, et al. Anxiety and depression in whiplash trauma. J Psychosom Res. 2002;53:831-835.
33. Roditi D, Robinson ME. Psychological interventions in chronic pain. Psychol Res Behav Manag. 2011:41-49.
34. Sparks SW. Posttraumatic stress syndrome overview. J Trauma Nurs. 2018;25:60-65.
35. Ravn SL, Eskildsen NB, Johnsen AT, et al. PTSD and WAD: qualitative study. Pain Med. 2020;21:1676-1689.
36. Andersen TE, Ravn SL, Carstensen T, et al. PTSD symptoms and pain sensitization after whiplash. Front Pain Res. 2022;3:908048.
37. Buitenhuis J, de Jong PJ, Jaspers JP, et al. PTSD symptoms and WAD progression. J Psychosom Res. 2006;61:681-689.
38. Sterling M, Hendrikz J, Kenardy J. Predictors of disability and PTSD after whiplash. Pain. 2011;152:1272-1278.
39. Elliott J, Pedler A, Kenardy J, et al. Fatty infiltrates in neck muscles after whiplash. PLoS One. 2011;6:e21194.
40. Ravn SL, Sterling M, Lahav Y, et al. Pain and PTSD interaction after whiplash. Eur J Pain. 2018;22:926-934.
41. Ditlevsen DN, Elklit A. Gender differences in PTSD prevalence. Ann Gen Psychiatry. 2012;11:26.
42. Quartana PJ, Campbell CM, Edwards RR. Pain catastrophizing review. Expert Rev Neurother. 2009;9:745-758.
43. Sullivan MJ, Tripp DA. Pain catastrophizing: controversies and future directions. J Pain. 2024;25:575-587.
44. Martinez Ortega S. Pain catastrophizing and depression in adolescents. 2024.
45. Walton DM, MacDermid JC, Giorgianni AA, et al. Risk factors for persistent WAD. J Orthop Sports Phys Ther. 2013;43:31-43.
46. Paré C, Yamada K, Sullivan MJ. Pain catastrophizing and outcomes after whiplash. Clin J Pain. 2024;40:10-17.
47. Wallis BJ, Lord SM, Bogduk N. Psychological distress resolution after radiofrequency neurotomy. Pain. 1997;73:15-22.
48. Smith AD, Jull G, Schneider GM, et al. Cervical nociception modulation in chronic WAD. PM R. 2015;7:913-921.
49. Godoy LD, Rossignoli MT, Delfino-Pereira P, et al. Stress neurobiology overview. Front Behav Neurosci. 2018;12:127.
50. Timmers I, Quaedflieg CW, Hsu C, et al. Stress and chronic pain interaction. Neurosci Biobehav Rev. 2019;107:641-655.
51. McEwen BS. Stress and physiological response. J Psychiatry Neurosci. 2005;30:315-318.
52. Elliott JM, Noteboom JT, Flynn TW, et al. Acute vs chronic WAD characterization. J Orthop Sports Phys Ther. 2009;39:312-323.
53. Jennings EM, Okine BN, Roche M, et al. Stress-induced hyperalgesia. Prog Neurobiol. 2014;121:1-18.
54. Hannibal KE, Bishop MD. Chronic stress and pain. Phys Ther. 2014;94:1816-1825.
55. Eller-Smith OC, Nicol AL, Christianson JA. Centralized pain mechanisms. Front Cell Neurosci. 2018;12:35.
56. Aloisi AM, Buonocore M, Merlo L, et al. Chronic pain and HPA axis dysfunction. Psychoneuroendocrinology. 2011;36:1032-1039.
57. Curatolo M, Arendt-Nielsen L, Petersen-Felix S. Central hypersensitivity after whiplash. Clin J Pain. 2004;20:469-476.
58. Nieto R, Miró J, Huguet A. Fear-avoidance model in whiplash. Eur J Pain. 2009;13:518-523.
59. Vangronsveld K, Peters M, Goossens M, et al. Fear-avoidance in chronic WAD. Pain. 2007;131:258-261.
60. Vangronsveld KL, Peters M, Goossens M, et al. Fear of movement and disability in WAD. Pain. 2008;139:449-457.
61. Pedrero-Martin Y, Falla D, Rodriguez-Brazzarola P, et al. Prognostic factors in WAD recovery. Clin J Pain. 2024;40:165-173.
62. Andersen TE, Karstoft KI, Brink O, et al. Pain catastrophizing and PTSD in WAD. Eur J Pain. 2016;20:1241-1252.
63. Kamper SJ, Maher CG, Costa LdCM, et al. Fear of movement and disability after whiplash. Pain. 2012;153:113-119.

64. Ritchie C, Sterling M. Recovery pathways after whiplash. J Orthop Sports Phys Ther. 2016;46:851-861.
65. Yuh Y, Haruki K, Tadatoshi I, et al. Predictors of delayed recovery in WAD. Cureus. 2024;16.
66. Anarte-Lazo E, Liew BX, Devecchi V, et al. Network analysis of WAD symptoms. Eur J Pain. 2024;28:322-334.
67. Samusyte G, Ciceliene J, Pajediene E, et al. CNS impairments in chronic WAD. J Clin Med. 2025;14:6222.
68. Chand SP, Kuckel DP, Huecker MR. Cognitive behavioral therapy. StatPearls. 2023.
69. Nakao M, Shirotsuki K, Sugaya N. Advances in CBT. BioPsychoSocial Med. 2021;15:16.
70. Wenzel A. Strategies of CBT. Psychiatr Clin. 2017;40:597-609.
71. Dunne RL, Kenardy J, Sterling M. CBT for PTSD in chronic WAD. Clin J Pain. 2012;28:755-765.
72. Louw A, Nijs J, Puentedura EJ. Pain neuroscience education perspective. J Man Manip Ther. 2017;25:160-168.
73. Salazar-Mendez J, Nunez-Cortes R, Suso-Marti L, et al. Optimal duration of pain neuroscience education. Neurosci Biobehav Rev. 2023;153:105328.
74. Corbo D. Pain neuroscience education and neuroimaging. Brain Sci. 2024;14:947.
75. Leung L, Han H, Martin M, et al. Mindfulness-based stress reduction for chronic pain. BMJ Open. 2015;5:e007650.
76. Mohammed WA, Pappous A, Sharma D. MBSR in injured athletes. Front Psychol. 2018;9:330227.
77. Boyd JE, Lanius RA, McKinnon MC. Mindfulness for PTSD. J Psychiatry Neurosci. 2018;43:7-25.
78. Veehof MM, Trompetter HR, Bohlmeijer ET, et al. Mindfulness-based interventions for chronic pain. Cogn Behav Ther. 2016;45:5-31.
79. Craps M, Van den Keybus N. Pain neuroscience education in WAD. J Rehabil Res Dev. 2011;48:43-58.
80. Ingram J. Therapeutic neuroscience education in chronic neck pain. 2017.
81. Malfliet A, Kregel J, Meeus M, et al. Blended pain neuroscience education. Phys Ther. 2018;98:357-368.
82. Benedict TM, Nitz AJ, Gambrel MK, et al. Pain neuroscience education and PTSD outcomes. Mil Psychol. 2024;36:376-392.
83. Medina-Viedma L, Cortés-Pérez I, Obrero-Gaitán E, et al. Pain neuroscience education meta-analysis. Med Sci. 2025;13:290.
84. Malfliet A, Lenoir D, Murillo C, et al. Pain science education in WAD. JAMA Netw Open. 2025;8:e2526674.
85. Hölzel BK, Hoge EA, Greve DN, et al. Neural mechanisms of mindfulness. Neuroimage Clin. 2013;2:448-458.
86. King AP, Block SR, Sripada RK, et al. Mindfulness-based exposure therapy in PTSD. Front Psychiatry. 2016;7:154.
87. Winas J, Scherer A, Kim P. Mindfulness and pain catastrophizing. Neuromodulation. 2023;26:S68.
88. Willaert W, Leysen L, Lenoir D, et al. Stress management and pain education in WAD. Phys Ther. 2021;101:pzab105.
89. Eccleston C, Morley S, Williams AC. Psychological approaches to chronic pain. Br J Anaesth. 2013;111:59-63.
90. Bring A, Åsenlöf P, Söderlund A. Behavioral treatment in acute WAD. Clin Rehabil. 2016;30:441-453.
91. Kondo Y, Miki T, Kurakata H, et al. CBT in chronic WAD: meta-analysis. J Rational Emot Cogn Behav Ther. 2023;41:856-879.
92. Andersen TE, Ravn SL, Armfield N, et al. Trauma-focused CBT and exercise in WAD. Pain. 2021;162:1221-1232.