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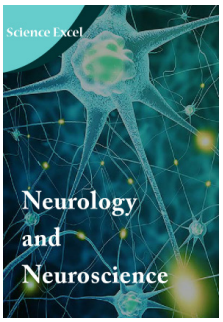

The Role of Brain Mapping and EEG in Chronic Pain and Neurofeedback Therapy: The BrainView System

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Abstract

Chronic pain and traumatic brain injury (TBI), particularly mild TBI (mTBI), are prevalent neurological conditions that remain difficult to detect and manage using conventional imaging methods. Electroencephalography (EEG), with its portability and high temporal resolution, offers a promising alternative for assessing neural dysfunction—though historically limited by noise, artifacts, and complex interpretation.

Recent advances in artificial intelligence (AI)-driven EEG analysis have demonstrated high accuracy in distinguishing chronic pain conditions and show potential for detecting subtle abnormalities associated with mTBI. Additionally, neurofeedback interventions targeting specific EEG frequency bands (e.g., alpha, theta) have been shown to reduce pain intensity and improve functional outcomes in conditions such as spinal cord injury and fibromyalgia. The increasing availability of portable EEG systems also expands access to care for underserved populations.

This paper reviews the integration of EEG, quantitative EEG (qEEG), AI, and neurofeedback as emerging tools for the diagnosis and treatment of chronic pain and TBI. We highlight the BrainView system, a non-invasive, portable platform that combines these technologies to provide AI-enhanced EEG analysis, source localization, and neurofunctional assessment. BrainView generates comprehensive neurofunctional response test reports, including brain maps, EEG frequency analysis, evoked potentials (EPs), neurobehavioral assessments, self-report questionnaires, and physician summaries. We also examine recent technical advances, current limitations, and future directions for EEG-based neuromodulation. As a combined system, BrainView and related EEG-based technologies offer a powerful, non-invasive approach to improving diagnosis and treatment, particularly in remote and military healthcare settings.

Introduction

Chronic pain is a pervasive global health issue affecting an estimated 25% of adults and a substantial proportion of youth [1-7]. In Europe, around 19% of adults report experiencing chronic pain, while prevalence in the United States (U.S.) exceeds 25% [7-10]. Recent estimates indicate more than 50 million U.S. adults — roughly 20.5% of the population — live with chronic pain nearly every day, often in areas such as the back, hip, knee, and foot [11]. Chronic pain is a leading cause of disability worldwide, limiting daily activities and contributing significantly to lost productivity and missed workdays [10,11].

A French study found that approximately 30% of adults suffer from chronic pain, which not only reduces the quality of life but also places a heavy burden on society due to increased healthcare utilization and workforce attrition [12-18]. Beyond its physical toll, chronic pain is strongly associated with psychological challenges, including depression, anxiety, sleep disturbances, and reduced life satisfaction [18-20].

The impact of chronic pain is especially severe in certain populations. Among post-9/11 military veterans, prevalence rates range from 44% to 50%, substantially higher than in the general population [21-23]. In these individuals, chronic pain often co-occurs with traumatic brain injury (TBI) and post-traumatic stress disorder (PTSD), forming what is known as the "polytrauma clinical triad" [24-29]. This triad profoundly affects physical, emotional, and social well-being and has been linked to an elevated risk of suicide.

Chronic pain is also closely intertwined with mental health disorders, particularly depression and anxiety [30-35]. This complex relationship increases the risk of suicidal ideation and behaviors [36,37]. Studies report that up to 41% of individuals with chronic pain experience suicidal thoughts, with particularly high risk observed among those suffering from migraines, arthritis, and back pain [36-38].

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According to the International Association for the Study of Pain (IASP), chronic pain is defined as pain that persists for more than three months and causes significant emotional distress or functional impairment [17,39]. It may be caused by injury, underlying medical conditions, or occur idiopathically. Unlike acute pain, which serves as a warning signal of injury or illness, chronic pain continues beyond the typical healing period and is increasingly understood as a condition in its own right. In 2010, the economic cost of chronic pain in the United States was estimated between $560 billion and $635 billion ─ exceeding the combined costs of heart disease and cancer [17].

Emerging research supports the understanding of chronic pain as a disease of the central nervous system, characterized by long-term neuroplastic or brain changes [30-35,40]. Brain circuits involved in pain perception overlap significantly with those responsible for emotion regulation and motivation, particularly those related to sensory processing, mood, and reward [41-43]. Disruptions in these systems may contribute not only to persistent pain but also to increased vulnerability to mood disorders and suicidality.

One important mechanism in chronic pain is central sensitization, a condition in which the nervous system becomes hyper-responsive to even mild stimuli [44]. Neuropathic pain, caused by damage to the somatosensory system, is especially difficult to diagnose and treat [45-47]. Despite the high burden of chronic pain, assessment and diagnosis still rely primarily on subjective tools such as the Numeric Rating Scale (NRS) and Visual Analog Scale (VAS), which are prone to variability and are less effective for non-verbal or cognitively impaired individuals [48,49]. Traditional methods like sensory testing and behavioral observation are similarly limited, especially in detecting spontaneous or ongoing pain [50,51].

In response to these challenges, neuroimaging technologies such as functional magnetic resonance imaging (fMRI) and electroencephalography (EEG) are being explored as objective tools for pain assessment [52]. EEG, in particular, offers advantages over other imaging modalities: it is non-invasive, cost-effective, and well-suited for real-time monitoring and machine learning applications [49,51]. Quantitative EEG (qEEG) has shown promise in classifying neurological conditions, including chronic pain, through advanced data analysis techniques.

EEG measures brain electrical activity via scalp electrodes, providing a direct and accessible means of evaluating assessing neural function [53-57]. QEEG enhances this by quantifying power and coherence across specific brainwave frequencies ─ delta, theta, alpha, beta, and gamma ─ each associated with different cognitive and physiological processes [58-61]. Coherence analysis offers insights into functional connectivity between brain regions [62], while qEEG brain mapping visually highlights abnormal patterns of activity. Though traditionally used in epilepsy diagnosis, qEEG is now gaining traction in pain research [63].

One emerging tool in this space is BrainView, an FDA-cleared Class II medical device developed by Medeia Inc. that integrates EEG and event-related potentials (ERP) to assess brain structure and function (Figure 1). The BrainView system offers:

- 3D Brain Maps: Averaged EEG data showing brainwave power deficits and surpluses.
- LORETA Source Localization: Identifies deeper brain activity sources for targeted treatment planning.
- Functional Assessment: Evaluates cognitive function, memory storage, and learning.

Figure 1. *An image of the BrainView Neural Scan System developed by Medeia Inc. The BrainView system is portable, easy-to-use, and non-invasive. The BrainView system is a 21-channel EEG/ERP amplifier with a dedicated laptop and testing supplies. The system utilizes high-quality circuit boards and components to allow for high-quality brain measurements, as well as essential heart rate variability data.*

BrainView's Neuro Functional Response Test incorporates Artificial Intelligence (AI) and deep-learning analytics to support personalized, data-driven care.

Another promising application is neurofeedback, or EEG biofeedback, a non-invasive therapy in which individuals learn to modulate their brain activity in real-time [64-66]. Patients receive auditory or visual feedback based on their EEG patterns, gradually training the brain toward more optimal functioning through operant conditioning [67-69]. Neurofeedback has shown encouraging results in chronic pain treatment by targeting brainwave activity associated with pain perception [70]. While questions remain about whether its effects surpass placebo, early clinical findings are promising [65,71,72]. Neurofeedback protocols typically involve 15–50 sessions of 20–40 minutes, tailored using individual qEEG data [67-69].

As the understanding of chronic pain evolves, integrating objective, brain-based tools like EEG and qEEG into clinical care presents a promising shift toward more personalized management strategies. Technologies such as BrainView represent this new frontier, combining neuroimaging, AI, and neurofeedback to offer comprehensive, real-time insights into brain function.

This paper explores the application of EEG biomarkers in brain mapping and their role in neurofeedback therapy. It also presents BrainView's Neuro Functional Response Test Report, which incorporates advanced neuroimaging and machine learning to assist in diagnosing conditions such as TBI — highlighting the potential of big data and AI to improve chronic pain diagnosis and treatment.

The Neuroscience of Pain

Although pain is inherently subjective, neuroscience research increasingly demonstrates that chronic pain is associated with long-term changes in brain function and structure, often arising from physical injury or psychological stress [73-77]. Chronic pain is now widely recognized as a disorder of the central nervous system, shaped by the complex interaction of biological, psychological, and social factors [78].

Neuroimaging studies have consistently shown that individuals with chronic pain exhibit both structural and functional brain abnormalities, including altered patterns of electrical activity detectable through EEG [79-82]. Encouragingly, some of these changes appear reversible with effective treatment, underscoring the potential of brain-targeted interventions [66,79,83].

Initial studies, using functional imaging techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) identified involvement of prefrontal, parietal, and motor areas in pain responses [84,85]. However, subsequent research clarified that these regions are primarily engaged in attention and motor planning related to pain, rather than its direct perception [84-87]. For example, prefrontal and parietal areas are part of the attentional networks that activate when individuals focus on painful stimuli, while motor regions, including the primary motor cortex and cerebellum, are involved in preparing defensive responses [84,85,88-91].

These insights contributed to the identification of the so-called "pain matrix", a distributed network of brain regions consistently activated by nociceptive input [54,57]. This matrix encompasses areas involved in sensory processing, emotional experience, memory, and anticipation of pain [92-97]. Notably, this network can also be activated by empathic experiences, such as observing others in pain [98-100]. However, in these cases, sensory-specific regions like the insula, primary somatosensory cortex (SI), secondary somatosensory cortex (SII), and caudal anterior cingulate cortex (ACC) are typically less active — differentiating real pain from vicarious pain responses [90,98-101].

Since the early 1990s, when brain imaging was first used to study pain responses in humans, our understanding of neuropathic pain, a chronic pain condition caused by nerve damage, has advanced substantially [102,103]. Imaging has revealed abnormalities in the insula, SI, SII, and thalamus among individuals with neuropathic pain. In rare cases, a second lesion in these regions has even led to pain relief, highlighting their critical role in generating pain [104,105]. Functional connectivity studies further show that thalamocortical feedback loops are often disrupted in chronic pain conditions such as diabetic neuropathy, suggesting the that network-level dysfunction plays a key role in pain persistence [106].

Meta-analyses have identified the insula and SII as the most consistently activated regions across chronic pain studies, challenging earlier emphasis on SI, the thalamus, ACC, and periaqueductal gray (PAG) [54,57,90]. This shift has prompted a re-evaluation of classical pain processing models [90].

Neurostimulation techniques, such as motor cortex stimulation, have demonstrated effectiveness in approximately 60% of patients with neuropathic pain [90]. Though these interventions primarily target motor areas, they also influence deeper pain-processing structures, including the thalamus, rostral ACC, and subgenual prefrontal cortex [107-109].

EEG research complements these findings. Individuals with chronic pain often show elevated theta oscillations, particularly in fronto-central and cingulated regions [110-115]. These abnormal patterns tend to normalize following successful treatments such as thalamotomy, further supporting their association with persistent pain [113-115]. The thalamocortical dysrhythmia model offers a compelling explanation for these findings: injury or sensory deprivation disrupts normal thalamic input, producing pathological theta-frequency bursts that interfere with cortical processing [116,117]. Although not all studies agree on theta increases, elevated theta activity has also been linked to impaired descending pain inhibition, suggesting its potential as a biomarker for dysfunctional central pain modulation [118-120].

Contemporary research has moved beyond the static concept of a discrete "pain neuromatrix" toward a more dynamic framework involving multiple overlapping brain networks that contribute to pain persistence, perception, and emotional response [30,44,121]. Despite these advances, reliable neuroimaging biomarkers for clinical diagnosis and personalized treatment personalization remain limited [122].

Brain Mapping Techniques in Chronic Neuropathic Pain

Diagnosing and treating chronic neuropathic pain remains a significant clinical challenge, largely due to the absence of reliable and objective biomarkers. This issue is particularly evident among individuals with spinal cord injury (SCI), many of whom suffer from persistent, treatment-resistant pain [123,124].

Recent research highlights resting-state EEG as a promising technique for identifying neurophysiological markers associated with chronic pain [51]. EEG-based brain mapping typically focuses on alterations in specific frequency bands, particularly theta, alpha, and beta, to detect patterns indicative of disrupted neural function.

In patients with SCI and chronic pain, studies consistently report [114,125]:

- Increased slow-wave activity (theta and delta bands)
- Elevated beta activity
- Reduced alpha activity compared to both healthy individuals and SCI patients without pain

For instance, Sarnthein et al. observed abnormal EEG patterns in neuropathic pain patients, which partially normalized following pain-relieving surgery, although the recordings were conducted one-year post-treatment, limiting causal conclusions [114]. Similarly, Boord et al. found reduced alpha and slowed peak frequencies in SCI patients with chronic pain [125]. These EEG alterations may serve as potential biomarkers, offering targets neuromodulation interventions such as neurofeedback [126]. However, more research is needed to establish causality and refine these patterns for clinical use.

While gamma oscillations are reliably evoked by acute nociceptive stimuli, their relevance to chronic neuropathic pain is inconsistent [127]. Some studies have reported increased

gamma activity in chronic pain states, but findings are limited and may reflect anxiety-related processes rather than pain perception itself [113,128]. Moreover, gamma frequencies are highly susceptible to contamination from muscle artifacts, complicating their use as reliable cortical markers [129-133].

By contrast, one of the most consistent EEG findings in chronic neuropathic pain is a dominant shift toward lower frequencies, marked by [51]:

- Increased theta power (4-7 Hz)
- Decreased alpha activity (8-12 Hz)
- A shift in the dominant peak frequency (DPF) into the theta-alpha range

This pattern aligns with the framework of Thalamocortical Dysrhythmia (TCD), which posits that impaired thalamic input disrupts normal cortical oscillatory rhythms [77,118,125,134-141]. Supporting this theory, Jensen et al. found distinct EEG profiles among SCI patients with chronic pain — specifically reduced alpha and increased theta power — with regional variability across the scalp [141].

Sarnthein et al. also reported elevated power across all major bands (delta to beta), reduced DPF, and altered alpha-beta coupling in frontoparietal regions — consistent with TCD [114]. Following central lateral thalamotomy, both pain and theta power decreased, though potential confounds like medication withdrawal were noted. Discriminant analysis of EEG data successfully distinguished pain patients from controls, supporting its diagnostic potential.

That said, not all chronic pain patients exhibit dysrhythmic EEG patterns, suggesting individual variability and the influence of additional factors. For instance, disrupted sleep or drowsiness — both known to increase theta and suppress alpha activity — may partially explain some EEG alterations [141,142]. An unexpected finding by researchers revealed that higher frontal alpha activity was associated with increased pain, possibly reflecting reduced prefrontal inhibitory control over pain signals [141,143-147].

Findings on alpha power remain mixed. Some studies report decreases, while others find increases [115,140,148-150]. This inconsistency may stem from distinctions within the alpha band:

- Low-alpha (8-10 Hz) is associated with rest and relaxation
- High-alpha (10-12 Hz) is linked to sensorimotor activity

Despite this variability, the shift in DPF toward lower frequencies remains one of the most robust EEG features in chronic neuropathic pain [51].

The beta band (13–30 Hz) is typically subdivided as follows [151-154]:

- Low-beta (13–20 Hz): Associated with sensorimotor processing
- High-beta (20–30 Hz): Linked to emotional and cognitive functions

In chronic pain populations, decreased low-beta power is often correlated with higher pain intensity, while high-beta power tends to be elevated in posterior regions [113,115,118,134,148,155]. These patterns have also been observed in non-neuropathic pain, suggesting shared neurophysiological mechanisms across different pain types [111,135,156]. Support for the clinical utility of these EEG patterns comes from neurofeedback research, where interventions that aim to increase low-beta and decrease high-beta activity have shown promise in managing chronic neuropathic pain [157-161].

ERP and qEEG Findings in Chronic Pain

EEG measures — particularly ERPs and qEEG — have revealed important alterations in brain function associated with chronic pain. A review by Pinheiro et al. emphasizes how these neurophysiological markers reflect disruptions across sensory, cognitive, and emotional domains, with growing potential for clinical application in diagnosis and treatment monitoring [81].

Chronic pain is associated with abnormalities in both early (e.g., P50, N100) and late (e.g., P300) ERP components [162-164]. Early components reflect automatic, pre-attentive sensory responses, while late components are linked to cognitive appraisal and attentional engagement. Changes in these ERP components suggest impaired cognitive flexibility and dysfunctional attentional processing in pain states [165-169]. Common ERP findings in chronic pain populations include [165-169]:

- Increased N100 amplitudes and reduced habituation to repeated stimuli, indicating central sensitization.
- Stable or unchanged P300 amplitudes, even under increased task difficulty, suggesting preserved attention but reduced adaptability to cognitive demands.

These patterns are thought to stem from maladaptive neuroplasticity in pain-related networks [81]. ERP responses can vary depending on the type of stimulus and the specific pain condition. For example, while some studies report reduced ERP amplitudes due to impaired cortical inhibition, others show enhanced ERP responses to unpleasant or emotionally charged stimuli — especially in conditions like fibromyalgia — highlighting the role of emotional salience in shaping cortical responses [54,167,170-174]. Importantly, these EEG and ERP changes are not localized to a single region. Altered activity has been observed in frontal, parietal, occipital, and somatosensory cortices, consistent with neuroimaging findings and reinforcing the model of a distributed pain network that integrates sensory, emotional, and cognitive processing [54,81].

QEEG studies provide further insights, consistently reporting [81]:

- Increased theta and alpha power
- Reduced ERP amplitudes
- A Shift in DPF
- Altered beta dynamics, such as reduced sensorimotor beta power modulation after movement, especially in motor-related pain conditions [175-177].

These findings reflect possible cortical signatures of chronic pain and may help differentiate pain subtypes for targeted treatment. Notably, alpha activity has been associated with pain duration, suggesting its potential role as a marker of disease progression [77,178,179]. Moreover, combining qEEG with cognitive performance tasks has yielded additional insights into how chronic pain disrupts attention and executive function, while also providing tools to monitor chronification processes [77,180,181]. Taken together, ERP and qEEG techniques hold substantial promise for:

- Objective diagnosis of chronic pain
- Patient stratification based on neurophysiological profiles
- Tracking therapeutic responses over time
- Personalizing interventions that target central mechanisms of pain processing

In summary, EEG-based methods reveal consistent alterations

in sensory, attentional, and cognitive brain processes associated with chronic pain. As non-invasive and relatively cost-effective tools, ERP and qEEG approaches are well-positioned to enhance the clinical assessment and individualized management of chronic pain disorders.

Challenges in Interpreting EEG Findings

Although numerous studies have documented EEG differences between individuals with chronic pain and healthy controls, these findings do not necessarily imply causation or fully capture the multi-dimensional nature of pain [51]. EEG alterations may reflect correlates of pain rather than its underlying mechanisms [51]. Pain is not purely a sensory phenomenon — it involves cognitive, emotional, and behavioral components that are difficult to isolate using EEG alone.

A major limitation in the current literature is the lack of comprehensive clinical characterization. Most studies rely heavily on unidimensional pain intensity ratings, such as numerical scales, which offer limited insight into the complex and fluctuating experience of chronic pain [51]. Without richer clinical data — including measures of affect, sleep, medication, use, and functional impairment — interpretations of EEG findings remain speculative. Furthermore, results across studies are often inconsistent. Some report positive correlations between pain intensity and EEG power in specific frequency bands (theta, alpha, beta, or gamma), while others observe negative correlations, especially in the alpha and low-beta ranges [113,128,141,148]. For example, while increased alpha power has been linked to pain suppression in some studies, others associate it with reduced cortical inhibition and higher pain sensitivity [115,140,148-150]. These conflicting findings undermine the reliability of any single frequency band as a universal biomarker for chronic pain.

Jensen et al. have suggested that certain EEG profiles, such as increased theta or altered alpha activity, may reflect a general vulnerability to developing chronic pain, particularly in populations like individuals with SCI [141]. However, the direct relationship between EEG activity and pain severity remains unclear. Longitudinal studies are needed to determine whether interventions that normalize these EEG patterns, such as neurofeedback or neuromodulation, can produce sustained pain relief.

Despite these limitations, EEG-based brain mapping continues to offer valuable insights into the neurophysiological mechanisms underlying chronic neuropathic pain. Among the various oscillatory features, increased theta power, altered rhythms, and imbalances in beta sub-bands are among the most promising candidates for biomarker development. However, the field faces significant challenges including:

- Methodological variability across studies,
- Inconsistent findings in relation to clinical symptoms,
- Lack of standardized recording and analysis protocols, and
- Insufficient integration of EEG data with behavioral or functional outcomes.

Moving forward, standardized methodologies, larger and more diverse samples, and richer clinical phenotyping will be essential. Validation of EEG-derived biomarkers will also require studies that evaluate their predictive value, not just their correlation with current pain states. If these hurdles are overcome, EEG may eventually play a critical role in personalizing treatment for chronic pain by guiding patient stratification and monitoring therapeutic response.

BrainView: Advanced Brain Mapping for Neurological Assessment

BrainView is an advanced, non-invasive diagnostic platform designed to assess brain function in real-time. Built for use in clinical environments, it integrates multiple neurophysiological measures to identify and monitor neurological and cognitive conditions, including chronic neuropathic pain. By combining qEEG with other assessment modalities, BrainView offers a data-driven, personalized approach to brain health evaluation and intervention.

At the core of BrainView is a proprietary, evidence-based EEG protocol, which includes:

- Electroencephalography (EEG): Captures electrical brain activity.
- Electrocardiogram (ECG): Assesses heart-brain interactions.
- Evoked Potentials (EPs): Measures processing speed of auditory and visual stimuli.
- Neuropsychological Survey: Gathers subjective data on mood, cognition, and functioning.

The procedure is painless non-invasive, typically lasting 20–30 minutes. The patient wears a sensor-equipped cap during resting-state recording session (Figures 2A and 2B). Data are then analyzed and compared to a normative reference database — adjusted for age and gender — to detect underactive or overactive brain regions that may contribute to symptoms such as pain, cognitive fog, or emotional dysregulation.

The BrainView system generates a comprehensive clinical report that includes (Figure 3):

- EEG acquisition and artifact removal,
- Fast Fourier Transform (FFT) and spectral analysis,
- Normative comparisons to isolate deviations from healthy brain patterns, and
- Clinical interpretation for diagnosis and treatment planning.

***Figure 2A**: Illustration of a patient positioned for the BrainView procedure, a painless and non-invasive process typically lasting 20-30 minutes. During the resting-state recording session, the patient wears a sensor-equipped cap.*

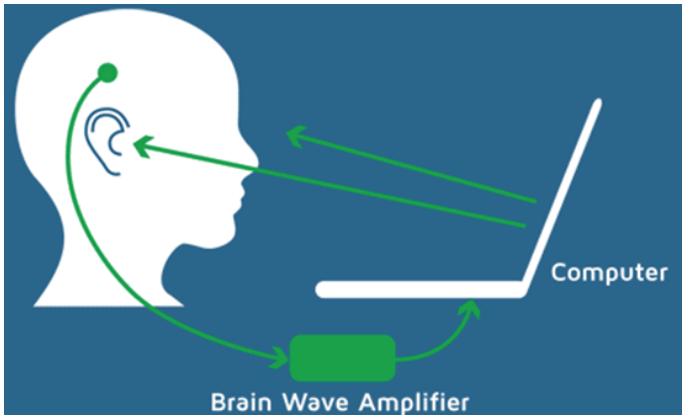

Figure 2B: BrainView utilizes a brain wave amplifier and advanced algorithms to analyze and extract detailed brain wave patterns.

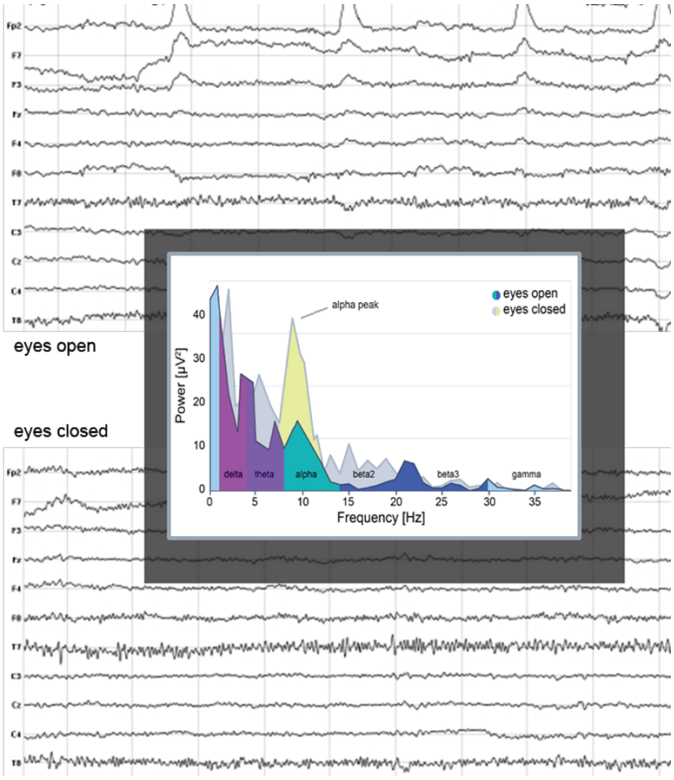

***Figure 3**: Example of BrainView's qEEG module identifying frequency and power domains during both eyes-open and eyes-closed sessions.*

BrainView supports precision medicine by establishing a baseline of brain function, informing personalized treatment strategies, and enabling serial assessments to track patient progress over time. Its integration into clinical workflows enhances diagnostic confidence and helps guide interventions, such as neurofeedback, pharmacotherapy, or neuromodulation.

As a leader in brain mapping technologies, BrainView supports advanced methods including:

- AI-based classification algorithms,
- Source localization via sLORETA (standardized Low-Resolution Electromagnetic Tomography), and
- Functional connectivity mapping.

These techniques enhance the clinical interpretation of EEG data by identifying both the frequency domains and cortical locations involved in chronic pain [182,183]. For example, studies sLORETA have revealed increased theta and low-alpha activity in regions such as the prefrontal cortex and operculo-insular region, especially in the left hemisphere [111,183,184]. Other studies report increased frontal lobe connectivity in the theta and gamma bands among individuals with chronic pain [110].

A foundational innovation in this field was the introduction of LORETA in 1994 by Pascual-Marqui, Michela, and Lehmann, which allowed for the estimation of deep-brain electrical sources using scalp-recorded EEG [185]. BrainView builds upon this framework, enhancing resolution and expanding clinical applicability.

BrainView's qEEG module uses sophisticated algorithms ─ such as wavelet transforms and machine learning classifiers ─ to extract detailed brainwave patterns. These are compared against large normative datasets to identify EEG phenotypes linked to a range of neurological and psychiatric conditions, including TBI, anxiety, depression, and chronic neuropathic pain. By integrating multimodal data, BrainView empowers clinicians to:

- Identify dysregulated brain networks,
- Visualize dynamic changes in brain activity during rest or task conditions,
- Monitor treatment response through repeat assessments, and
- Tailor therapeutic strategies to each patient's unique neurophysiological profile.

In summary, BrainView represents a major advancement in the clinical application of EEG and brain mapping. By providing an objective, repeatable, and comprehensive assessment of brain function, it enhances both diagnostic precision and treatment personalization ─ key priorities in the management of chronic pain and other complex neurological conditions.

AI and Machine Learning in EEG-Based Pain Analysis

AI and machine learning (ML) are rapidly transforming the analysis of EEG data in the context of pain research. These approaches offer powerful tools for identifying subtle, complex patterns in brain activity that may serve as objective biomarkers of chronic pain [186]. In particular, ML classifiers have been shown to distinguish chronic pain patients from healthy controls with classification accuracies exceeding 85%, especially when focusing on activity in regions such as the somatosensory cortex and anterior cingulate cortex [51,187]. Combining EEG signal localization, connectivity metrics, and ML offers a promising path toward more reliable and precise diagnostic models for chronic neuropathic pain.

As outlined by Alouani et al., ML in EEG analysis typically falls into three categories [188]:

- **Supervised learning:** Uses labeled input─output data to train predictive models (e.g., support vector machines [SVM], decision trees).
- **Unsupervised learning:** Identifies intrinsic structure or clusters in data without labeled outcomes (e.g., K-means clustering).
- **Reinforcement learning**: Learns optimal behavior through trial and error interactions with the environment; often used brain–computer interface (BCI) applications.

In clinical neurophysiology, ML has gained traction particularly in EEG-based detection of TBI, where models are trained using features such as band power, total power, and coherence [189-191]. Algorithms commonly employed include SVM, decision trees, random forests, and k-nearest neighbors [188].

Insights from recent studies in EEG-based classification of pain and TBI include [192-195]:

- Feature selection critically impacts model performance.
- Raw EEG and artifact-cleaned data sometimes yield comparable accuracy, suggesting limitations in current artifact removal techniques.
- EEG features are highly noise-sensitive, due to the inherently low signal-to-noise ratio in EEG data.

A 2020 review concluded that the lack of a standardized qEEG feature set limits cross-study comparisons [196,197]. Moreover, in some cases, ML approaches did not outperform classical regression models for TBI outcome prediction. These challenges underscore the need for improved preprocessing, larger datasets, and clearer feature definitions, particularly for subtle conditions like mild TBI (mTBI) [194].

Deep learning (DL), a subset of ML that uses multi-layered neural networks, has shown substantial promise in EEG analysis. DL methods can learn complex patterns directly from raw EEG data, reducing the need for manual feature engineering [188]. Common DL architectures include:

- Artificial Neural Networks (ANNs)
- Convolutional Neural Networks (CNNs)
- Recurrent Neural Networks (RNNs)

Although computationally intensive and requiring large datasets, DL has been successfully applied in diverse domains, including seizure detection, medicine, computer vision, and energy systems [198-207]. For instance, transforming EEG signals into time–frequency images using continuous wavelet transform (CWT) or short-term Fourier transform (STFT) has enabled CNNs to classify neurological conditions with high accuracy. CWT often outperforms STFT for EEG-based tasks [205].

Despite their promise, DL and ML methods face several challenges in EEG-based pain and TBI research [188]:

- EEG artifacts (e.g., from eye movement, muscle activity) significantly degrade signal quality.
- Many processing algorithms assume signal stationarity and linear artifact addition — assumptions that do not hold true for EEG and may reduce feature validity.
- Standardization is lacking, especially in defining qEEG features for pain classification and TBI detection.

Addressing these limitations is essential to advance the clinical utility of AI-driven EEG analysis. Improved artifact removal techniques, standardized protocols, and larger, curated datasets will enhance reproducibility and generalizability.

AI and ML hold significant potential for transforming EEG into a more reliable, objective tool for the assessment and management of chronic neuropathic pain. As research progresses, especially with the incorporation of deep learning, these technologies could enable the development of personalized, brain-based biomarkers to guide diagnosis, monitor treatment response, and optimize therapeutic interventions.

Limitations of EEG in Pain Research

While EEG-based methods offer valuable insights into brain activity related to pain, important limitations remain. Zis et al. emphasizes that traditional spectral and topographic EEG analyses may not adequately capture the complexity of pain-related neural processes [50]. In contrast, dynamic brain network analyses, such as functional connectivity and coherence, especially when combined with ML, are better suited to detect the non-stationary and distributed nature of brain activity underlying pain [50,208].

To enhance the rigor and reproducibility of EEG-based pain research, several methodological factors must be addressed [50]:

1. Participant Characteristics
   - Age significantly influences EEG features, particularly alpha frequency. Studies should recruit homogeneous age groups or control for age-related variability [209].
2. Confounding Variables
   - Factors such as attention, pain expectation, and emotional states can distort EEG signals. Some studies have mitigated these confounds by integrating additional physiological recordings (e.g., heart rate variability, skin conductance).
3. Protocol Variability
   - Inconsistencies in procedures (e.g., eyes open vs. closed, participant handedness, or site of stimulation) introduce uncontrolled variance across studies.
4. Pain Intensity Measurement
   - Pain ratings obtained immediately post-stimulus vs. continuously during EEG recording can alter attentional engagement and EEG signal quality. Standardizing rating methods is essential.
5. Stimulus Type and Perception
   - Discrepancies between stimulus intensity and subjective pain perception, along with heterogeneity in pain modalities (e.g., thermal, mechanical, cold), may affect EEG outcomes [210-212]. Quantitative sensory testing is recommended for more precise stimulus calibration [210-212].

QEEG, though increasingly used in clinical settings, has its own constraints [54,81]:

- High susceptibility to external artifacts, including electromagnetic interference and muscle activity.
- Dependence on expert interpretation, requiring both theoretical and technical proficiency.
- Limited spatial resolution, particularly for detecting activity in deep brain structures or generating detailed anatomical information.

Many of these limitations can be mitigated through careful study design and technical optimization [81]:

- Creating an optimized recording environment (e.g., temperature control, noise reduction, and electromagnetic shielding).
- Providing intensive training for clinicians and researchers in EEG data acquisition and analysis.
- Employing advanced preprocessing algorithms and connectivity-based analyses to capture more nuanced brain dynamics.

Although EEG remains a promising tool for identifying biomarkers of chronic pain, its effectiveness is currently limited by methodological inconsistencies, technical constraints, and sensitivity to confounding variables. Future research should prioritize standardized protocols, rigorous artifact control, and the integration of advanced analytical techniques, including connectivity metrics and AI, to better reflect the dynamic, network-based architecture of pain processing in the human brain.

Neurofeedback for Chronic Pain Management

Neurofeedback is a non-invasive technique that enables individuals to self-regulate their brain activity through real-time EEG feedback [213]. Grounded in neuroplasticity and operant conditioning principles, neurofeedback aims to retrain dysfunctional neural patterns associated with chronic pain [214-216]. Chronic pain induces structural and functional changes in brain regions such as the prefrontal cortex and thalamus, which are crucial for pain modulation and perception [82]. These changes impact not only sensory processing but also emotional and cognitive domains.

Neurofeedback primarily targets modulation of brainwave activity—particularly the alpha band (8–13 Hz)—to reduce pain perception [215-218]. Increased alpha synchrony is associated with decreased pain sensitivity, whereas diminished alpha activity correlates with heightened pain responses [77,219]. Several studies support neurofeedback's effectiveness across diverse chronic pain populations:

- Al-Taleb et al. demonstrated reduced neuropathic pain in SCI patients following tailored neurofeedback interventions [220].
- Shimizu et al. reported that alpha-wave neurofeedback alleviated chronic low back pain [221].
- Patel et al. highlighted the importance of cognitive engagement and relaxation for optimizing alpha neurofeedback's analgesic effects [222].

A systematic review by Hesam-Shariati et al. found a medium effect size favoring neurofeedback over control treatments in managing chronic pain [223]. Other supportive studies include those by Terrasa et al., Barbosa-Torres & Delgado., and Jacobs & Jensen [224-226].

Typical neurofeedback treatment involves 20–40 sessions, each lasting 20–40 minutes, varying by protocol and condition being treated [227-229]. However, frequent clinic visits may pose accessibility challenges, particularly for veterans in rural areas [160,230-233]. EEG-based neurofeedback shows promise in managing central neuropathic pain (CNP). This approach allows individuals to gain voluntary control over brain activity through real-time feedback, leveraging neuroplasticity to adjust maladaptive neural patterns associated with persistent pain [158,161,223,234]. CNP arises from damage to central somatosensory pathways, such as after SCI, stroke, or multiple sclerosis [235]. In paraplegic SCI patients, neurofeedback has been used to modulate abnormal cortical activity, particularly during imagined movement of paralyzed limbs, resulting in reduced pain intensity [157,161]. While early findings are encouraging, larger, controlled clinical trials are needed to establish neurofeedback's efficacy, safety, and long-term benefits for the CNP populations [45,234].

Despite its potential, neurofeedback is not without limitations:

- Some patients report transient side effects such as increased fatigue or pain during treatment; however, these effects may not be directly related to the intervention [143,221,228,236-238].
- The precise mechanisms underlying neurofeedback-induced pain relief remain unclear, necessitating further research is needed to clarify how specific EEG changes mediate pain relief [65].
- Evidence for effectiveness in pediatric chronic pain populations is limited [72].

Neurofeedback represents a promising, non-pharmacological approach to chronic pain management by targeting maladaptive neural circuits involved in pain perception. Early results are particularly positive in conditions like SCI and fibromyalgia, with reductions in pain intensity and related symptoms. However, further research is essential to clarify mechanisms, optimize protocols, and expand accessibility, especially in underserved populations and children [220,223-226].

EEG Biomarkers to Guide Neuromodulation

Identifying and localizing specific EEG brain rhythms associated with chronic neuropathic pain is crucial for optimizing neuromodulation techniques such as neurofeedback and transcranial electrical stimulation [51]. EEG biomarkers can enhance neuromodulation by enabling:

- Personalized neurofeedback protocols tailored to individual brain activity.
- Prediction of treatment response, improving patient selection.
- Real-time therapy monitoring to enable adaptive modulation.

Initial neurofeedback studies targeted modulation of particular EEG frequency bands to restore healthier neural oscillatory patterns, including [157-161]:

- Reducing theta and high-beta activity, which tend to be elevated in chronic pain.
- Enhancing low-beta and alpha activity, often suppressed in chronic pain states.
- Adjusting frequency ratios, such as alpha/theta and low-beta/high-beta, to rebalance oscillatory activity [233,239].

Electrode placement has varied, but central sensorimotor regions (e.g., Cz, C3, C4) have shown promising results in reducing pain-related brain activity [239,240]. Notably, a study employing sLORETA source localization demonstrated that neurofeedback at electrode C4 effectively reduced theta activity across frontal brain regions, significantly alleviating pain [241].

While EEG provides excellent temporal resolution, its spatial resolution remains limited. Improving spatial precision is vital for more accurate neurofeedback targeting. Clinical EEG systems generally use 20–25 EEG channels, but expanding this number could enhance the localization of pain-related signals [188]. However, increasing channel count introduces challenges such as:

- Determining the optimal number and arrangement of electrodes for accurate source localization.
- Designing electrodes that maintain stable scalp contact and resist motion artifacts.
- Scaling hardware and computational resources for real-time data processing, especially for portable or standalone EEG systems used outside clinical settings.

EEG-based neurofeedback holds promise as a personalized, adaptive intervention for chronic and central neuropathic pain, particularly in complex conditions like SCI. Targeted modulation of brain rhythms guided by EEG biomarkers can reduce cortical hyperactivity and alleviate pain [242]. Future research priorities include:

- Identifying optimal frequency bands and electrode placements for targeted neuromodulation.
- Advancing EEG hardware to improve signal quality and user comfort.
- Developing scalable systems capable of real-time, mobile neurofeedback delivery for broader accessibility.

As the evidence base grows, EEG biomarkers will not only deepen our understanding of chronic pain mechanisms but also help tailor more effective, precise, and accessible neuromodulation therapies.

The Global and Economic Burden of Traumatic Brain Injury

TBI is a significant global health crisis, affecting an estimated 69 million individuals annually worldwide and incurring over $40 billion in economic burden from non-fatal cases in the United States in 2016 alone [243,244]. TBI rates are notably higher in military combat zones, emphasizing the urgency for early detection and effective intervention strategies [245].

mTBI represents 70–90% of all TBI cases, yet it is frequently undiagnosed due to subtle clinical signs [246]. Despite being classified as 'mild', undetected mTBI can lead to long-term cognitive, emotional, and functional impairments. While the Glasgow Coma Scale remains the gold standard for classifying TBI severity, it lacks sensitivity for deting mTBI [247].

Conventional TBI diagnostics such as CT and fMRI have limitations [248]:

- CT scans offer only static images and may miss subtle or diffuse injuries [249].
- fMRI provides excellent spatial resolution but poor temporal resolution, limiting its use in acute scenarios [249,250].
- Both modalities are expensive, non-portable, and not ideal for real-time, field-based use [250].

EEG, known for its high temporal resolution and portability, is emerging as a powerful tool for TBI assessment and monitoring [251-254]. However, raw EEG signals are highly sensitive to noise and artifacts, which limits their clinical utility [188]. To address these challenges, qEEG extracts features such as band power, coherence, and symmetry across standard frequency bands (delta to gamma), and feeds them into statistical and ML models for pattern classification [255-265]. Despite this, qEEG remains limited by the non-stationary nature of EEG signals and the lack of a universal diagnostic index for TBI [188].

Alouani et al. propose that AI and DL could improve the classification of EEG patterns linked to TBI severity, especially in subtle mTBI cases that elude traditional tools [188]. One clinical example is the BrainView Neuro Response Functional Test (Figure 4), which combines:

- EEG frequency analysis
- Evoked potentials
- Neurobehavioral assessments
- Self-report questionnaires
- Physician medical summary

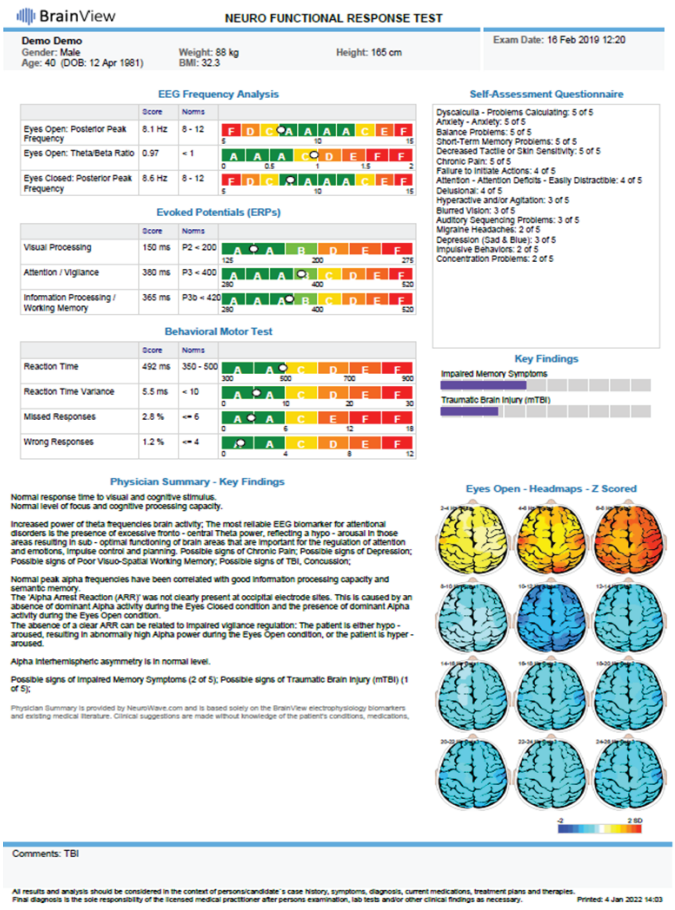

Figure 4: *An example of a Neuro Functional Response Test Report showing EEG frequency analysis, evoked potentials, behavioral motor test, self-assessment questionnaire, physician summary, and brain (or head) maps, with a potential diagnosis included as a comment at the end of the report.*

This system generates composite reports including theta/beta ratios and functional brain maps, aiding in diagnosis, treatment planning, and longitudinal tracking — especially when paired with neurofeedback.

Neurofeedback uses real-time EEG data to train individuals to self-regulate brainwave activity based on principles of neuroplasticity and operant conditioning [214,215]. Protocols often target [266-268]:

- Alpha/theta training to reduce pain perception
- Sensorimotor rhythm (SMR) for arousal and emotional regulation
- Theta/beta ratio and slow cortical potentials for disorders like ADHD and epilepsy, and post-concussion symptoms [266,267]

Though individual responses vary, neurofeedback has been effective in managing headaches, memory and concentration issues, and post-concussive symptoms [223,228,269-271].

Chronic pain is highly prevalent among military veterans, many of whom also suffer from TBI and PTSD. Neurofeedback offers a non-pharmacological, accessible alternative, particularly when conventional treatments are unavailable (Figure 5) [272]. Elbogen et al. demonstrated the feasibility of mobile neurofeedback, with veterans completing 33 at-home

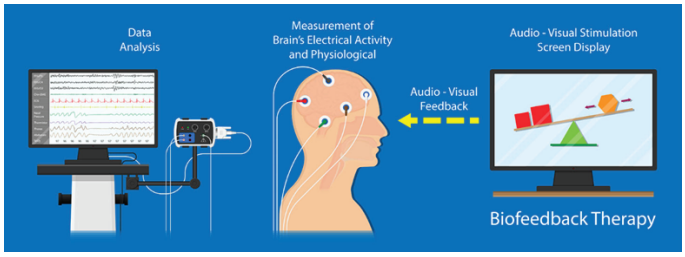

Figure 5: *An illustration of the setup for neurofeedback therapy [272].*

sessions and reporting significant pain reduction with no serious adverse effects [273]. Trullinger et al. found that combining neurofeedback with physical therapy improved treatment outcomes [274]. Advances in portable EEG systems are helping to lower access barriers for rural and underserved populations [275,276].

Despite its promise, EEG-based TBI detection faces several limitations [188]:

- Low signal-to-noise ratio hinders reliability in detecting subtle injuries.
- Current artifact removal techniques rely on flawed assumptions of linearity and stationarity.
- To improve TBI detection, future research should:
- Enhance artifact filtering and noise reduction techniques
- Improve real-time signal clarity
- Develop adaptive AI models tailored to EEG variability

The "golden hour" immediately following injury is critical for accurate diagnosis and intervention [277-279]. Improving the speed and accuracy of EEG-based diagnostics could transform clinical outcomes during this window..

TBI continues to impose a major personal, societal, and economic burden. While conventional tools like CT and MRI remain vital, their limitations underscore the need for complementary, real-time, and portable approaches. Technologies such as EEG, qEEG, neurofeedback, and AI-based analytics offer a path toward faster, more personalized, and more accessible TBI care. With continued innovation, especially in mobile systems, these tools could significantly impact military, emergency, and remote healthcare settings.

Conclusion

EEG is emerging as a vital tool in the diagnosis and management of chronic pain and traumatic brain injury (TBI), especially in cases where conventional imaging falls short. The development of qEEG and the integration of AI, including ML and DL, have significantly improved the capacity to detect subtle neurophysiological changes associated with mild TBI and chronic pain conditions.

Despite technical limitations, such as susceptibility to artifacts and limited spatial resolution, EEG offers high temporal resolution, portability, and adaptability for real-time applications. Neurofeedback, in particular, presents a promising non-invasive approach for pain relief and functional recovery by enabling individuals to regulate brain activity through operant conditioning. Tailored neurofeedback protocols guided by EEG biomarkers can modulate maladaptive neural patterns, with early evidence supporting their efficacy across various pain and TBI-related conditions.

However, challenges remain. Standardization of protocols, improved artifact removal techniques, and greater understanding of the neurophysiological mechanisms underlying treatment effects are essential for broader clinical adoption. Continued research should prioritize accessibility, especially for veterans, rural populations, and underserved communities, by developing portable, AI-enhanced EEG systems capable of delivering personalized, at-home care.

In summary, the convergence of EEG, AI, and neurofeedback in the BrainView system offers a transformative pathway for advancing the detection and treatment of chronic pain and TBI. As the BrainView system and its underlying technologies mature, they hold significant promise for reshaping neurorehabilitation and optimizing patient outcomes across diverse clinical settings.

References

1. Diotaiuti P, Corrado S, Tosti B, et al. Evaluating the effectiveness of neurofeedback in chronic pain management: A narrative review. Front Psychol. 2024;15:1369487. doi:10.3389/fpsyg.2024.1369487
2. King S, Chambers CT, Huguet A, et al. The epidemiology of chronic pain in children and adolescents revisited: A systematic review. Pain. 2011;152(12):2729-2738. doi:10.1016/j.pain.2011.07.016
3. Goldberg DS, McGee SJ. Pain as a global public health priority. BMC Public Health. 2011;11:770. doi:10.1186/1471-2458-11-770
4. Schopflocher D, Taenzer P, Jovey R. The prevalence of chronic pain in Canada. Pain Res Manag. 2011;16(6):445-450. doi:10.1155/2011/876306
5. van Hecke O, Torrance N, Smith BH. Chronic pain epidemiology and its clinical relevance. Br J Anaesth. 2013;111(1):13-18. doi:10.1093/bja/aet123
6. Huguet A, Miró J. The severity of chronic pediatric pain: An epidemiological study. J Pain. 2008;9(3):226-236. doi:10.1016/j.jpain.2007.10.015
7. Nahin RL. Estimates of pain prevalence and severity in adults: United States, 2012. J Pain. 2015;16(8):769-780. doi:10.1016/j.jpain.2015.05.002
8. Johannes CB, Le TK, Zhou X, Johnston JA, Dworkin RH. The prevalence of chronic pain in United States adults: Results of an internet-based survey. J Pain. 2010;11(12):1230-1239. doi:10.1016/j.jpain.2010.07.002
9. Kuehn BM. Chronic pain prevalence. JAMA. 2018;320(16):1632. doi:10.1001/jama.2018.16009
10. GBD 2016 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990–2016: A systematic analysis for the Global Burden of Disease Study 2016. Lancet. 2017;390(10100):1211-1259. doi:10.1016/S0140-6736(17)32154-2
11. Yong RJ, Mullins PM, Bhattacharyya N. Prevalence of chronic pain among adults in the United States. Pain. 2022;163(2):e328-e332. doi:10.1097/j.pain.0000000000002291
12. Chenaf C, Delorme J, Delage N, et al. Prevalence of chronic pain with or without neuropathic characteristics in France using the capture–recapture method: A population-based study. Pain. 2018;159(11):2394-2402. doi:10.1097/j.pain.0000000000001347
13. Institute of Medicine (US), Committee on a National Agenda for the Prevention of Disabilities. Disability in America: Toward a National Agenda for Prevention. Pope A, Tarlov A, eds. National Academy Press; 1991.
14. Groenewald CB, Essner BS, Wright D, Fesinmeyer MD, Palermo TM. The economic costs of chronic pain among a cohort of treatment-seeking adolescents in the United States. J Pain.

2014;15(9):925-933. doi:10.1016/j.jpain.2014.06.002
15. Finan PH, Goodin BR, Smith MT. The association of sleep and pain: An update and a path forward. J Pain. 2013;14(12):1539-1552. doi:10.1016/j.jpain.2013.08.007
16. De Ruddere L, Craig KD. Understanding stigma and chronic pain. Pain. 2016;157(8):1607-1610. doi:10.1097/j.pain.0000000000000512
17. Gaskin DJ, Richard P. The economic costs of pain in the United States. J Pain. 2012;13(8):715-724. doi:10.1016/j.jpain.2012.03.009
18. Bair MJ, Robinson RL, Katon W, Kroenke K. Depression and pain comorbidity: A literature review. Arch Intern Med. 2003;163(20):2433-2445. doi:10.1001/archinte.163.20.2433
19. Turk DC, Okifuji A. Psychological factors in chronic pain: Evolution and revolution. J Consult Clin Psychol. 2002;70(3):678-690. doi:10.1037/0022-006X.70.3.678
20. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: Scientific advances and future directions. Psychol Bull. 2007;133(4):581-624. doi:10.1037/0033-2909.133.4.581
21. Gironda RJ, Clark ME, Massengale JP, Walker RL. Pain among veterans of Operations Enduring Freedom and Iraqi Freedom. Pain Med. 2006;7(4):339-343. doi:10.1111/j.1526-4637.2006.00146.x
22. Toblin RL, Quartana PJ, Riviere LA, Walper K, Hoge CW. Chronic pain and opioid use in US soldiers after combat deployment. JAMA Intern Med. 2014;174(8):1400-1401. doi:10.1001/jamainternmed.2014.2726
23. Higgins DM, Kerns RD, Brandt CA, et al. Persistent pain and comorbidity among Operation Enduring Freedom/Operation Iraqi Freedom/Operation New Dawn veterans. Pain Med. 2014;15(5):782-790. doi:10.1111/pme.12388
24. Dobscha SK, Clark ME, Morasco BJ, et al. Systematic review of the literature on pain in patients with polytrauma including traumatic brain injury. Pain Med. 2009;10(7):1200-1217. doi:10.1111/j.1526-4637.2009.00721.x
25. Nampiaparampil DE. Prevalence of chronic pain after traumatic brain injury: A systematic review. JAMA. 2008;300(6):711-719. doi:10.1001/jama.300.6.711
26. Buckenmaier CC, Galloway KT, Polomano RC, et al. Preliminary validation of the Defense and Veterans Pain Rating Scale (DVPRS) in a military population. Pain Med. 2013;14(1):110-123. doi:10.1111/j.1526-4637.2012.01516.x
27. Lew HL, Otis JD, Tun C, et al. Prevalence of chronic pain, posttraumatic stress disorder, and persistent postconcussive symptoms in OIF/OEF veterans: Polytrauma clinical triad. J Rehabil Res Dev. 2009;46(6):697-702. doi:10.1682/jrrd.2009.01.0006
28. Stojanovic MP, Fonda J, Fortier CB, et al. Influence of mild traumatic brain injury (TBI) and posttraumatic stress disorder (PTSD) on pain intensity levels in OEF/OIF/OND veterans. Pain Med. 2016;17(11):2017-2025. doi:10.1093/pm/pnw042
29. Blakey SM, Wagner HR, Naylor J, et al. Chronic pain, TBI, and PTSD in military veterans: A link to suicidal ideation and violent impulses? J Pain. 2018;19(7):797-806. doi:10.1016/j.jpain.2018.02.012
30. Martucci KT, MacKey SC. Neuroimaging of pain: Human evidence and clinical relevance of central nervous system processes and modulation. Anesthesiology. 2018;128(6):1241-1254. doi:10.1097/ALN.0000000000002137
31. Treede R-D, Rief W, Barke A, et al. Chronic pain as a symptom or a disease: The IASP classification of chronic pain for the International Classification of Diseases (ICD-11). Pain. 2019;160(1):19-27. doi:10.1097/j.pain.0000000000001384
32. Simons LE, Elman I, Borsook D. Psychological processing in chronic pain: A neural systems approach. Neurosci Biobehav Rev. 2014;39:61-78. doi:10.1016/j.neubiorev.2013.12.006
33. Lenoir D, Willaert W, Coppieters I, et al. Electroencephalography during nociceptive stimulation in chronic pain patients: A systematic review. Pain Med. 2020;21(12):3413-3427. doi:10.1093/pm/pnaa131
34. Otti A, Guendel H, Wohlschläger A, Zimmer C, Noll-Hussong M. Frequency shifts in the anterior default mode network and the salience network in chronic pain disorder. BMC Psychiatry. 2013;13:84. doi:10.1186/1471-244X-13-84
35. Kim JA, Davis KD. Neural oscillations: Understanding a neural code of pain. Neuroscientist. 2021;27(5):544-570. doi:10.1177/1073858420958629
36. Racine M. Chronic pain and suicide risk: A comprehensive review. Prog Neuropsychopharmacol Biol Psychiatry. 2018;87(Pt B):269-280. doi:10.1016/j.pnpbp.2017.08.020
37. Hooley JM, Franklin JC, Nock MK. Chronic pain and suicide: Understanding the association. Curr Pain Headache Rep. 2014;18(8):435. doi:10.1007/s11916-014-0435-2
38. Calati R, Laglaoui Bakhiyi C, Artero S, Ilgen M, Courtet P. The impact of physical pain on suicidal thoughts and behaviors: Meta-analyses. J Psychiatr Res. 2015;71:16-32. doi:10.1016/j.jpsychires.2015.09.004
39. International Association for the Study of Pain. IASP terminology. Published 2021. Accessed June 15, 2025. https://www.iasp-pain.org/resources/terminology/
40. Bhatt RR, Haddad E, Zhu AH, et al. Mapping brain structure variability in chronic pain: The role of widespreadness and pain type and its mediating relationship with suicide attempt. Biol Psychiatry. 2024;95(5):473-481. doi:10.1016/j.biopsych.2023.07.016
41. Elman I, Borsook D, Volkow ND. Pain and suicidality: Insights from reward and addiction neuroscience. Prog Neurobiol. 2013;109:1-27. doi:10.1016/j.pneurobio.2013.06.003
42. Ren W, Centeno MV, Berger S, et al. The indirect pathway of the nucleus accumbens shell amplifies neuropathic pain. Nat Neurosci. 2016;19(2):220-222. doi:10.1038/nn.4199
43. Elman I, Borsook D. Common brain mechanisms of chronic pain and addiction. Neuron. 2016;89(1):11-36. doi:10.1016/j.neuron.2015.11.027
44. Fitzcharles MA, Cohen SP, Clauw DJ, et al. Nociplastic pain: Towards an understanding of prevalent pain conditions. Lancet. 2021;397(10289):2098-2110. doi:10.1016/S0140-6736(21)00392-5
45. Scholz J, Finnerup NB, Attal N, et al. The IASP classification of chronic pain for ICD-11: Chronic neuropathic pain. Pain. 2019;160(1):53-59. doi:10.1097/j.pain.0000000000001365
46. Moisset X, Bouhassira D, Avez Couturier J, et al. Pharmacological and non-pharmacological treatments for neuropathic pain: Systematic review and French recommendations. Rev Neurol (Paris). 2020;176(5):325-352. doi:10.1016/j.neurol.2020.01.361
47. Baron R, Binder A, Wasner G. Neuropathic pain: Diagnosis, pathophysiological mechanisms, and treatment. Lancet Neurol. 2010;9(8):807-819. doi:10.1016/S1474-4422(10)70143-5
48. Herr KC, Coyne PJ, McCaffery M, Manworren R, Merkel S. Pain assessment in the patient unable to self-report: Position statement with clinical practice recommendations. Pain Manag Nurs. 2011;12(4):230-250. doi:10.1016/j.pmn.2011.10.002
49. Miller J, Jacobs S, Koppes W, et al. Development of machine learning algorithms using EEG data to detect the presence of chronic pain. Preprint. 2024. doi:10.1101/2024.09.18.24313903
50. Zis P, Liampas A, Artemiadis A, et al. EEG recordings as

biomarkers of pain perception: Where do we stand and where to go? Pain Ther. 2022;11(2):369-380. doi:10.1007/s40122-022-00372-2
51. Mussigmann T, Bardel B, Lefaucheur JP. Resting-state electroencephalography (EEG) biomarkers of chronic neuropathic pain: A systematic review. Neuroimage. 2022;258:119351. doi:10.1016/j.neuroimage.2022.119351
52. Tracey I. Neuroimaging enters the pain biomarker arena. Sci Transl Med. 2021;13(619):eabj7358. doi:10.1126/scitranslmed.abj7358
53. Wager TD, Atlas LY, Lindquist MA, Roy M, Woo CW, Kross E. An fMRI-based neurologic signature of physical pain. N Engl J Med. 2013;368(15):1388-1397. doi:10.1056/NEJMoa1204471
54. Apkarian AV, Bushnell MC, Treede RD, Zubieta JK. Human brain mechanisms of pain perception and regulation in health and disease. Eur J Pain. 2005;9(4):463-484. doi:10.1016/j.ejpain.2004.11.001
55. Duerden EG, Albanese MC. Localization of pain-related brain activation: A meta-analysis of neuroimaging data. Hum Brain Mapp. 2013;34(1):109-149. doi:10.1002/hbm.21416
56. Tracey I, Becerra L, Chang I, et al. Noxious hot and cold stimulation produce common patterns of brain activation in humans: A functional magnetic resonance imaging study. Neurosci Lett. 2000;288(2):159-162. doi:10.1016/s0304-3940(00)01224-6
57. Peyron R, Laurent B, Garcia-Larrea L. Functional imaging of brain responses to pain: A review and meta-analysis. Neurophysiol Clin. 2000;30(5):263-288. doi:10.1016/s0987-7053(00)00227-6
58. Sornmo L, Laguna P. Bioelectrical Signal Processing in Cardiac and Neurological Applications. Academic Press; 2005. doi:10.1016/B978-012437552-9/50000-3
59. Donoghue T, Haller M, Peterson EJ, et al. Parameterizing neural power spectra into periodic and aperiodic components. Nat Neurosci. 2020;23(12):1655-1665. doi:10.1038/s41593-020-00744-x
60. Cox R, Fell J. Analyzing human sleep EEG: A methodological primer with code implementation. Sleep Med Rev. 2020;54:101353. doi:10.1016/j.smrv.2020.101353
61. Gross J. Analytical methods and experimental approaches for electrophysiological studies of brain oscillations. J Neurosci Methods. 2014;228(100):57-66. doi:10.1016/j.jneumeth.2014.03.007
62. Zavaglia M, Astolfi L, Babiloni F, Ursino M. The effect of connectivity on EEG rhythms, power spectral density, and coherence among coupled neural populations: Analysis with a neural mass model. IEEE Trans Biomed Eng. 2008;55(1):69-77. doi:10.1109/TBME.2007.897814
63. da Silva FL. EEG and MEG: Relevance to neuroscience. Neuron. 2013;80(5):1112-1128. doi:10.1016/j.neuron.2013.10.017
64. Williams ACdeC, Fisher E, Hearn L, Eccleston C. Psychological therapies for the management of chronic pain (excluding headache) in adults. Cochrane Database Syst Rev. 2020;2020(8):CD007407. doi:10.1002/14651858.CD007407.pub4
65. Jensen MP, Day MA, Miró J. Neuromodulatory treatments for chronic pain: Efficacy and mechanisms. Nat Rev Neurol. 2014;10(3):167-178. doi:10.1038/nrneurol.2014.12
66. Roy R, de la Vega R, Jensen MP, Miró J. Neurofeedback for pain management: A systematic review. Front Neurosci. 2020;14:671. doi:10.3389/fnins.2020.00671
67. Hammond DC. What is neurofeedback: An update. J Neurother. 2011;15(4):305-336. doi:10.1080/10874208.2011.623090
68. Heinrich H, Gevensleben H, Strehl U. Annotation: Neurofeedback – Train your brain to train behavior. J Child Psychol Psychiatry. 2007;48(1):3-16. doi:10.1111/j.1469-7610.2006.01665.x
69. Sherlin LH, Arns M, Lubar J, et al. Neurofeedback and basic learning theory: Implications for research and practice. J Neurother. 2011;15(4):292-304. doi:10.1080/10874208.2011.623089
70. Ibric VL, Dragomirescu LG. Neurofeedback in pain management. In: Introduction to Quantitative EEG and Neurofeedback. Elsevier Inc; 2009:417-451. doi:10.1016/B978-0-12-374534-7.00016-2
71. Thibault RT, Lifshitz M, Raz A. The climate of neurofeedback: Scientific rigor and the perils of ideology. Brain. 2017;141(2):e11. doi:10.1093/brain/awx330
72. Miró J, Castarlenas E, de la Vega R, et al. Psychological neuromodulatory treatments for young people with chronic pain. Children (Basel). 2016;3(4):41. doi:10.3390/children3040041
73. Davis KD, Racine E, Collett B. Neuroethical issues related to the use of brain imaging: Can we and should we use brain imaging as a biomarker to diagnose chronic pain? Pain. 2012;153(8):1555-1559. doi:10.1016/j.pain.2012.02.037
74. Arendt-Nielsen L, Graven-Nielsen T, Svensson P, Jensen TS. Temporal summation in muscles and referred pain areas: An experimental human study. Muscle Nerve. 1997;20(10):1311-1313. doi:10.1002/(SICI)1097-4598(199710)20:10<1311::AID-MUS15>3.0.CO;2-5
75. Prichep LS, John ER, Howard B, Merkin H, Hiesiger EM. Evaluation of the Pain Matrix Using EEG Source Localization: A Feasibility Study. Pain Med. 2011;12(8):1241-1248. doi:10.1111/j.1526-4637.2011.01191.x
76. Baliki MN, Schnitzer TJ, Bauer WR, Apkarian AV. Brain morphological signatures for chronic pain. PLoS One. 2011;6(10):e26010. doi:10.1371/journal.pone.0026010
77. de Vries M, Wilder-Smith OHG, Jongsma MLA, et al. Altered resting state EEG in chronic pancreatitis patients: Toward a marker for chronic pain. J Pain Res. 2013;6:815-824. doi:10.2147/JPR.S50919
78. Chapin H, Bagarinao E, Mackey S. Real-time fMRI applied to pain management. Neurosci Lett. 2012;520(2):174-181. doi:10.1016/j.neulet.2012.02.076
79. May A. Chronic pain may change the structure of the brain. Pain. 2008;137(1):7-15. doi:10.1016/j.pain.2008.02.034
80. Davis KD, Moayedi M. Central mechanisms of pain revealed through functional and structural MRI. J Neuroimmune Pharmacol. 2013;8(3):518-534. doi:10.1007/s11481-012-9386-8
81. Pinheiro ESS, de Queirós FC, Montoya P, et al. Electroencephalographic patterns in chronic pain: A systematic review of the literature. PLoS One. 2016;11(2):e0149085. doi:10.1371/journal.pone.0149085
82. Apkarian AV, Hashmi JA, Baliki MN. Pain and the brain: Specificity and plasticity of the brain in clinical chronic pain. Pain. 2011;152(Suppl 3):S49-S64. doi:10.1016/j.pain.2010.11.010
83. Flor H. Psychological pain interventions and neurophysiology: Implications for a mechanism-based approach. Am Psychol. 2014;69(2):188-196. doi:10.1037/a0035254
84. Garcia-Larrea L, Peyron R, Laurent B, Mauguière F. Association and dissociation between laser-evoked potentials and pain perception. Neuroreport. 1997;8(17):3785-3789. doi:10.1097/00001756-199712010-00026
85. Peyron R, García-Larrea L, Grégoire MC, et al. Haemodynamic brain responses to acute pain in humans: Sensory and attentional networks. Brain. 1999;122(9):1765-1780. doi:10.1093/brain/122.9.1765
86. Frankenstein UN, Richter W, McIntyre MC, Remy F. Distraction modulates anterior cingulate gyrus activations during the cold pressor test. Neuroimage. 2001;14(4):827-836. doi:10.1006/nimg.2001.0883

87. Valet M, Sprenger T, Boecker H, et al. Distraction modulates connectivity of the cingulo-frontal cortex and the midbrain during pain—An fMRI analysis. Pain. 2004;109(3):399-408. doi:10.1016/j.pain.2004.02.033
88. Legrain V, Perchet C, Garcia-Larrea L. Involuntary orienting of attention to nociceptive events: Neural and behavioral signatures. J Neurophysiol. 2009;102(4):2423-2434. doi:10.1152/jn.00372.2009
89. Peyron R, Kupers R, Jehl JL, et al. Central representation of the RIII flexion reflex associated with overt motor reaction: An fMRI study. Neurophysiol Clin. 2007;37(4):249-259. doi:10.1016/j.neucli.2007.07.002
90. Peyron R, Faillenot I. Functional brain imaging applied to the analysis of painful phenomena: Imagery and cognition (2) – Functional brain mapping of pain perception. Med Sci (Paris). 2011;27(1):82-87. doi:10.1051/medsci/201127182
91. Frot M, Magnin M, Mauguière F, Garcia-Larrea L. On SI responses to painful laser and non-painful electrical stimuli: An intracortical recording study [Poster presentation]. Presented at: 12th World Congress on Pain; 2008; Glasgow, United Kingdom. Poster PF134.
92. Ploghaus A, Narain C, Beckmann CF, et al. Exacerbation of pain by anxiety is associated with activity in a hippocampal network. J Neurosci. 2001;21(24):9896-9903. doi:10.1523/JNEUROSCI.21-24-09896.2001
93. Peyron R, Créac'h C, Vinson J, et al. Brain responses to laser stimulation in fMRI and anticipatory responses to this same stimulation. Rev Neurol (Paris). 2007;163(1):8-11. doi:10.1016/S0035-3787(07)90368-9
94. Bingel U, Schoell E, Herken W, Büchel C, May A. Habituation to painful stimulation involves the antinociceptive system. Pain. 2007;131(1-2):21-30. doi:10.1016/j.pain.2006.12.005
95. Godinho F, Magnin M, Frot M, et al. Emotional modulation of pain: Is it the sensation or what we recall? J Neurosci. 2006;26(44):11454-11461. doi:10.1523/JNEUROSCI.2260-06.2006
96. Craig AD, Reiman EM, Evans A, Bushnell MC. Functional imaging of an illusion of pain. Nature. 1996;384(6606):258-260. doi:10.1038/384258a0
97. Rainville P, Duncan GH, Price DD, Carrier B, Bushnell MC. Pain affect encoded in human anterior cingulate but not somatosensory cortex. Science. 1997;277(5328):968-971. doi:10.1126/science.277.5328.968
98. Botvinick M, Jha AP, Bylsma LM, et al. Viewing facial expressions of pain engages cortical areas involved in the direct experience of pain. Neuroimage. 2005;25(1):312-319. doi:10.1016/j.neuroimage.2004.11.043
99. Jackson PL, Brunet E, Meltzoff AN, Decety J. Empathy examined through the neural mechanisms involved in imagining how I feel versus how you feel pain. Neuropsychologia. 2006;44(5):752-761. doi:10.1016/j.neuropsychologia.2005.07.015
100. Saarela MV, Hlushchuk Y, Williams ACdC, et al. The compassionate brain: Humans detect intensity of pain from another's face. Cereb Cortex. 2007;17(1):230-237. doi:10.1093/cercor/bhj141
101. Singer T, Seymour B, O'Doherty J, et al. Empathy for pain involves the affective but not sensory components of pain. Science. 2004;303(5661):1157-1162. doi:10.1126/science.1093535
102. Jones AK, Brown WD, Friston KJ, et al. Cortical and subcortical localization of response to pain in man using positron emission tomography. Proc Biol Sci. 1991;244(1309):39-44. doi:10.1098/rspb.1991.0048
103. Talbot JD, Marrett S, Evans AC, et al. Multiple representations of pain in human cerebral cortex. Science. 1991;251(4999):1355-1358. doi:10.1126/science.2003220
104. Daniele O, Fierro B, Brighina F, Magaudda A, Natalè E. Disappearance of haemorrhagic stroke-induced thalamic (central) pain following a further (contralateral ischaemic) stroke. Funct Neurol. 2003;18(2):95-96.
105. Helmchen C, Lindig M, Petersen D, Tronnier V. Disappearance of central thalamic pain syndrome after contralateral parietal lobe lesion: Implications for therapeutic brain stimulation. Pain. 2002;98(3):325-330. doi:10.1016/S0304-3959(02)00139-2
106. Cauda F, Sacco K, D'Agata F, et al. Low-frequency BOLD fluctuations demonstrate altered thalamocortical connectivity in diabetic neuropathic pain. BMC Neurosci. 2009;10:138. doi:10.1186/1471-2202-10-138
107. Peyron R, Garcia-Larrea L, Deiber MP, et al. Electrical stimulation of precentral cortical area in the treatment of central pain: Electrophysiological and PET study. Pain. 1995;62(3):275-286. doi:10.1016/0304-3959(94)00211-V
108. García-Larrea L, Peyron R, Mertens P, et al. Electrical stimulation of motor cortex for pain control: A combined PET-scan and electrophysiological study. Pain. 1999;83(2):259-273. doi:10.1016/s0304-3959(99)00114-1
109. Peyron R, Faillenot I, Mertens P, et al. Motor cortex stimulation in neuropathic pain: Correlations between analgesic effect and hemodynamic changes in the brain. A PET study. Neuroimage. 2007;34(1):310-321. doi:10.1016/j.neuroimage.2006.08.037
110. Ta Dinh S, Nickel MM, Tiemann L, et al. Brain dysfunction in chronic pain patients assessed by resting-state electroencephalography. Pain. 2019;160(12):2751-2765. doi:10.1097/j.pain.0000000000001666
111. Case M, Shirinpour S, Zhang H, et al. Increased theta band EEG power in sickle cell disease patients. J Pain Res. 2018;11:67-76. doi:10.2147/JPR.S145581
112. Fallon N, Chiu Y, Nurmikko T, Stancak A. Altered theta oscillations in resting EEG of fibromyalgia syndrome patients. Eur J Pain. 2018;22(1):49-57. doi:10.1002/ejp.1076
113. Michels L, Moazami-Goudarzi M, Jeanmonod D. Correlations between EEG and clinical outcome in chronic neuropathic pain: Surgical effects and treatment resistance. Brain Imaging Behav. 2011;5(4):329-348. doi:10.1007/s11682-011-9135-2
114. Sarnthein J, Stern J, Aufenberg C, et al. Increased EEG power and slowed dominant frequency in patients with neurogenic pain. Brain. 2006;129(Pt 1):55-64. doi:10.1093/brain/awh631
115. Stern J, Jeanmonod D, Sarnthein J. Persistent EEG overactivation in the cortical pain matrix of neurogenic pain patients. Neuroimage. 2006;31(2):721-731. doi:10.1016/j.neuroimage.2005.12.042
116. Colgin LL. Mechanisms and functions of theta rhythms. Annu Rev Neurosci. 2013;36:295-312. doi:10.1146/annurev-neuro-062012-170330
117. Jeanmonod D, Magnin M, Morel A, et al. Thalamocortical dysrhythmia II: Clinical and surgical aspects. Thalamus Relat Syst. 2001;1(3):245-254. doi:10.1016/S1472-9288(01)00026-7
118. Simis M, Pacheco-Barrios K, Uygur-Kucukseymen E, et al. Specific electroencephalographic signatures for pain and descending pain inhibitory system in spinal cord injury. Pain Med. 2022;23(5):955-964. doi:10.1093/pm/pnab124
119. Schmidt S, Naranjo JR, Brenneisen C, et al. Pain ratings, psychological functioning, and quantitative EEG in a controlled study of chronic back pain patients. PLoS One. 2012;7(3):e31138. doi:10.1371/journal.pone.0031138
120. Levitt J, Edhi MM, Thorpe RV, et al. Pain phenotypes classified by machine learning using electroencephalography features. Neuroimage. 2020;223:117256. doi:10.1016/j.neuroimage.2020.117256

121. Canavero S, Bonicalzi V. Pain myths and the genesis of central pain. Pain Med. 2015;16(2):240-248. doi:10.1111/pme.12509
122. van der Miesen MM, Lindquist MA, Wager TD. Neuroimaging-based biomarkers for pain: State of the field and current directions. Pain Rep. 2019;4(4):e751. doi:10.1097/PR9.0000000000000751
123. Cardenas DD, Jensen MP. Treatments for chronic pain in persons with spinal cord injury: A survey study. J Spinal Cord Med. 2006;29(2):109-117. doi:10.1080/10790268.2006.11753864
124. Jensen MP, Hoffman AJ, Cardenas DD. Chronic pain in individuals with spinal cord injury: A survey of a longitudinal study. Spinal Cord. 2005;43(12):704-712. doi:10.1038/sj.sc.3101777
125. Boord P, Siddall PJ, Tran Y, Herbert D, Middleton J, Craig A. Electroencephalographic slowing and reduced reactivity in neuropathic pain following spinal cord injury. Spinal Cord. 2008;46(2):118-123. doi:10.1038/sj.sc.3102077
126. Jensen MP, Hakimian S, Sherlin LH, Fregni F. New insights into neuromodulatory approaches for the treatment of pain. J Pain. 2008;9(3):193-199. doi:10.1016/j.jpain.2007.11.003
127. May ES, Nickel MM, Ta Dinh S, et al. Prefrontal gamma oscillations reflect ongoing pain intensity in chronic back pain patients. Hum Brain Mapp. 2019;40(1):293-305. doi:10.1002/hbm.24373
128. Zhou R, Wang J, Qi W, et al. Elevated resting state gamma oscillatory activities in electroencephalogram of patients with post-herpetic neuralgia. Front Neurosci. 2018;12:750. doi:10.3389/fnins.2018.00750
129. Chouchou F, Perchet C, Garcia-Larrea L. EEG changes reflecting pain: Is alpha suppression better than gamma enhancement? Neurophysiol Clin. 2021;51(3):209-218. doi:10.1016/j.neucli.2021.03.001
130. Mussigmann T, Lefaucheur JP, McGonigal A. Gamma-band activities in the context of pain: A signal from brain or muscle? Neurophysiol Clin. 2021;51(3):287-289. doi:10.1016/j.neucli.2021.03.007
131. Liberati G, Algoet M, Klöcker A, et al. Habituation of phase-locked local field potentials and gamma-band oscillations recorded from the human insula. Sci Rep. 2018;8(1):8265. doi:10.1038/s41598-018-26604-0
132. Liberati G, Mulders D, Algoet M, et al. Insular responses to transient painful and non-painful thermal and mechanical spinothalamic stimuli recorded using intracerebral EEG. Sci Rep. 2020;10(1):22319. doi:10.1038/s41598-020-79371-2
133. Liberati G, Klöcker A, Algoet M, et al. Gamma-band oscillations preferential for nociception can be recorded in the human insula. Cereb Cortex. 2018;28(10):3650-3664. doi:10.1093/cercor/bhx237
134. Krupina NA, Churyukanov MV, Kukushkin ML, Yakhno NN. Central neuropathic pain and profiles of quantitative electroencephalography in multiple sclerosis patients. Front Neurol. 2020;10:1380. doi:10.3389/fneur.2019.01380
135. Vanneste S, Ost J, Havenbergh TV, Ridder DD. Resting state electrical brain activity and connectivity in fibromyalgia. PLoS One. 2017;12(6):e0178516. doi:10.1371/journal.pone.0178516
136. Kim JA, Bosma RL, Hemington KS, et al. Neuropathic pain and pain interference are linked to alpha-band slowing and reduced beta-band magnetoencephalography activity within the dynamic pain connectome in patients with multiple sclerosis. Pain. 2019;160(1):187-197. doi:10.1097/j.pain.0000000000001391
137. Villafaina S, Collado-Mateo D, Fuentes-García JP, Cano-Plasencia R, Gusi N. Impact of fibromyalgia on alpha-2 EEG power spectrum in the resting condition: A descriptive correlational study. Biomed Res Int. 2019;2019:7851047. doi:10.1155/2019/7851047
138. Lim M, Kim JS, Kim DJ, Chung CK. Increased low- and high-frequency oscillatory activity in the prefrontal cortex of fibromyalgia patients. Front Hum Neurosci. 2016;10:111. doi:10.3389/fnhum.2016.00111
139. Walton KD, Dubois M, Llinás RR. Abnormal thalamocortical activity in patients with Complex Regional Pain Syndrome (CRPS) Type I. Pain. 2010;150(1):41-51. doi:10.1016/j.pain.2010.02.023
140. Vuckovic A, Hasan MA, Fraser M, et al. Dynamic oscillatory signatures of central neuropathic pain in spinal cord injury. J Pain. 2014;15(6):645-655. doi:10.1016/j.jpain.2014.02.005
141. Jensen MP, Sherlin LH, Gertz KJ, et al. Brain EEG activity correlates of chronic pain in persons with spinal cord injury: Clinical implications. Spinal Cord. 2013;51(1):55-58. doi:10.1038/sc.2012.84
142. Santamaria J, Chiappa KH. The EEG of drowsiness in normal adults. J Clin Neurophysiol. 1987;4(4):327-382. doi:10.1097/00004691-198710000-00002
143. Jensen MP, Grierson C, Tracy-Smith V, et al. Neurofeedback treatment for pain associated with complex regional pain syndrome type I. J Neurother. 2007;11(1):45-53. doi:10.1300/J184v11n01_04
144. Mathew A, Mishra H, Kumaraiah V. Alpha feedback in the treatment of tension headache. J Pers Clin Stud. 1987;3(1):17-22.
145. McKenzie RF, Ehrisman WJ, Montgomery PS, Barnes RH. The treatment of headache by means of electroencephalographic biofeedback. Headache. 1974;13(4):164-172. doi:10.1111/j.1526-4610.1974.hed1304164.x
146. Fregni F, Boggio PS, Lima MC, et al. A sham-controlled, phase II trial of transcranial direct current stimulation for the treatment of central pain in traumatic spinal cord injury. Pain. 2006;122(1–2):197-209. doi:10.1016/j.pain.2006.02.023
147. Niedermeyer E. The normal EEG of the waking adult. In: Niedermeyer E, Da Silva FL, eds. Electroencephalography: Basic Principles, Clinical Applications, and Related Fields. 5th ed. Lippincott Williams & Wilkins; 2005:167-192.
148. Di Pietro F, Macey PM, Rae CD, et al. The relationship between thalamic GABA content and resting cortical rhythm in neuropathic pain. Hum Brain Mapp. 2018;39(5):1945-1956. doi:10.1002/hbm.23973
149. Micoulaud-Franchi JA, McGonigal A, Lopez R, et al. Electroencephalographic neurofeedback: Level of evidence in mental and brain disorders and suggestions for good clinical practice. Neurophysiol Clin. 2015;45(6):423-433. doi:10.1016/j.neucli.2015.10.077
150. Babiloni C, Del Percio C, Arendt-Nielsen L, et al. Cortical EEG alpha rhythms reflect task-specific somatosensory and motor interactions in humans. Clin Neurophysiol. 2014;125(10):1936-1945. doi:10.1016/j.clinph.2014.04.021
151. Barone J, Rossiter HE. Understanding the role of sensorimotor beta oscillations. Front Syst Neurosci. 2021;15:655886. doi:10.3389/fnsys.2021.655886
152. Kilavik BE, Zaepffel M, Brovelli A, MacKay WA, Riehle A. The ups and downs of β oscillations in sensorimotor cortex. Exp Neurol. 2013;245:15-26. doi:10.1016/j.expneurol.2012.09.014
153. Betti V, Della Penna S, de Pasquale F, Corbetta M. Spontaneous beta band rhythms in the predictive coding of natural stimuli. Neuroscientist. 2021;27(2):184-201. doi:10.1177/1073858420928988
154. Pomper U, Höfle M, Hauck M, et al. Crossmodal bias of visual input on pain perception and pain-induced beta activity. Neuroimage. 2013;66:469-478. doi:10.1016/j.neuroimage.2012.10.040
155. Teixeira M, Mancini C, Wicht CA, et al. Beta electroencephalographic oscillation is a potential GABAergic biomarker of chronic peripheral neuropathic pain. Front Neurosci.

2021;15:594536. doi:10.3389/fnins.2021.594536
156. Wang W, Roy A, Misra G, et al. Altered neural oscillations within and between sensorimotor cortex and parietal cortex in chronic jaw pain. Neuroimage Clin. 2019;24:101964. doi:10.1016/j.nicl.2019.101964
157. Hasan MA, Fraser M, Conway BA, Allan DB, Vučković A. Reversed cortical over-activity during movement imagination following neurofeedback treatment for central neuropathic pain. Clin Neurophysiol. 2016;127(9):3118-3127. doi:10.1016/j.clinph.2016.06.012
158. Vuckovic A, Altaleb MKH, Fraser M, McGeady C, Purcell M. EEG correlates of self-managed neurofeedback treatment of central neuropathic pain in chronic spinal cord injury. Front Neurosci. 2019;13:762. doi:10.3389/fnins.2019.00762
159. Jensen MP, Gianas A, George HR, et al. Use of neurofeedback to enhance response to hypnotic analgesia in individuals with multiple sclerosis. Int J Clin Exp Hypn. 2016;64(1):1-23. doi:10.1080/00207144.2015.1099400
160. Jensen M, Gertz KJ, Kupper AE, et al. Steps toward developing an EEG biofeedback treatment for chronic pain. Appl Psychophysiol Biofeedback. 2013;38(2):101-108. doi:10.1007/s10484-013-9214-9
161. Hassan MA, Fraser M, Conway BA, Allan DB, Vuckovic A. The mechanism of neurofeedback training for treatment of central neuropathic pain in paraplegia: A pilot study. BMC Neurol. 2015;15:200. doi:10.1186/s12883-015-0445-7
162. Luck SJ, Woodman GF, Vogel EK. Event-related potential studies of attention. Trends Cogn Sci. 2000;4(11):432-440. doi:10.1016/s1364-6613(00)01545-x
163. Sitges C, Bornas X, Llabrés J, Noguera M, Montoya P. Linear and nonlinear analyses of EEG dynamics during non-painful somatosensory processing in chronic pain patients. Int J Psychophysiol. 2010;77(2):176-183. doi:10.1016/j.ijpsycho.2010.05.010
164. Bjork M, Hagen K, Stovner L, Sand T. Photic EEG-driving responses related to ictal phases and trigger sensitivity in migraine: a longitudinal, controlled study. Cephalalgia. 2011;31(4):444-455. doi:10.1177/0333102410385582
165. Angelakis E, Lubar JF, Stathopoulou S, Kounios J. Peak alpha frequency: An electroencephalographic measure of cognitive preparedness. Clin Neurophysiol. 2004;115(4):887-897. doi:10.1016/j.clinph.2003.11.034
166. Flor H, Knost B, Birbaumer N. Processing of pain- and body-related verbal material in chronic pain patients: Central and peripheral correlates. Pain. 1997;73(3):413-421. doi:10.1016/S0304-3959(97)00137-1
167. Montoya P, Sitges C, García-Herrera M, et al. Reduced brain habituation to somatosensory stimulation in patients with fibromyalgia. Arthritis Rheum. 2006;54(6):1995-2003. doi:10.1002/art.21910
168. Gonzalez-Roldan AM, Muñoz MA, Cifre I, Sitges C, Montoya P. Altered psychophysiological responses to the view of others' pain and anger faces in fibromyalgia patients. J Pain. 2013;14(7):709-719. doi:10.1016/j.jpain.2013.01.775
169. Veldhuijzen DS, Kenemans JL, van Wijck AJM, et al. Processing capacity in chronic pain patients: A visual event-related potentials study. Pain. 2006;121(1-2):60-68. doi:10.1016/j.pain.2005.12.004
170. Vlaeyen JW. Learning to predict and control harmful events: Chronic pain and conditioning. Pain. 2015;156(Suppl 1):S86-S93. doi:10.1097/j.pain.0000000000000107
171. Palermo A, Giglia G, Vigneri S, et al. Does habituation depend on cortical inhibition? Results of an rTMS study in healthy subjects. Exp Brain Res. 2011;212(1):101-107. doi:10.1007/s00221-011-2701-4
172. Flor H, Diers M, Birbaumer N. Peripheral and electrocortical responses to painful and non-painful stimulation in chronic pain patients, tension headache patients, and healthy controls. Neurosci Lett. 2004;361(1-3):147-150. doi:10.1016/j.neulet.2003.12.064
173. Vossen CJ, Vossen HG, Joosten EA, van Os J, Lousberg R. Does habituation differ in chronic low back pain subjects compared to pain-free controls? A cross-sectional pain rating ERP study reanalyzed with the ERFIA multilevel method. Medicine (Baltimore). 2015;94(19):e865. doi:10.1097/MD.0000000000000865
174. Sitges C, García-Herrera M, Pericás M, et al. Abnormal brain processing of affective and sensory pain descriptors in chronic pain patients. J Affect Disord. 2007;104(1-3):73-82. doi:10.1016/j.jad.2007.02.024
175. Silen T, Forss N, Jensen O, Hari R. Abnormal reactivity of the ~20-Hz motor cortex rhythm in Unverricht Lundborg type progressive myoclonus epilepsy. Neuroimage. 2000;12(6):707-712. doi:10.1006/nimg.2000.0660
176. Juottonen K, Gockel M, Silén T, et al. Altered central sensorimotor processing in patients with complex regional pain syndrome. Pain. 2002;98(3):315-323. doi:10.1016/S0304-3959(02)00119-7
177. Raij TT, Forss N, Stancák A, Hari R. Modulation of motor-cortex oscillatory activity by painful Aδ- and C-fiber stimuli. Neuroimage. 2004;23(2):569-573. doi:10.1016/j.neuroimage.2004.06.036
178. Mo J, Maizels M, Ding M, Ahn AH. Does throbbing pain have a brain signature? Pain. 2013;154(7):1150-1155. doi:10.1016/j.pain.2013.02.013
179. Chizh BA, Priestley T, Rowbotham M, Schaffler K. Predicting therapeutic efficacy—Experimental pain in human subjects. Brain Res Rev. 2009;60(1):243-254. doi:10.1016/j.brainresrev.2008.12.016
180. Lelic D, Olesen SS, Graversen C, et al. Electrophysiology as a tool to unravel the origin of pancreatic pain. World J Gastrointest Pathophysiol. 2014;5(1):33-39. doi:10.4291/wjgp.v5.i1.33
181. Lorenz J, Beck H, Bromm B. Differential changes of laser evoked potentials, late auditory evoked potentials, and P300 under morphine in chronic pain patients. Electroencephalogr Clin Neurophysiol. 1997;104(6):514-521. doi:10.1016/S0168-5597(97)00064-6
182. Volker JM, Arguissain FG, Andersen OK, Biurrun Manresa J. Variability and effect sizes of intracranial current source density estimations during pain: Systematic review, experimental findings, and future perspectives. Hum Brain Mapp. 2021;42(8):2461-2476. doi:10.1002/hbm.25380
183. Prichep LS, Shah J, Merkin H, Hiesiger EM. Exploration of the pathophysiology of chronic pain using quantitative EEG source localization. Clin EEG Neurosci. 2018;49(2):103-113. doi:10.1177/1550059417736444
184. Garcia-Larrea L, Peyron R. Pain matrices and neuropathic pain matrices: A review. Pain. 2013;154(Suppl 1):S29-S43. doi:10.1016/j.pain.2013.09.001
185. Pascual-Marqui RD, Michela M, Lehmann D. Low resolution electromagnetic tomography: A new method for localizing electrical activity in the brain. Int J Psychophysiol. 1994;18(1):49-65. doi:10.1016/0167-8760(84)90014-x
186. Saeidi M, Karwowski W, Farahani FV, et al. Neural decoding of EEG signals with machine learning: A systematic review. Brain Sci. 2021;11(11):1525. doi:10.3390/brainsci11111525
187. Vuckovic A, Ferrer Gallardo VJ, Jarjees M, Fraser M, Purcell M. Prediction of central neuropathic pain in spinal cord injury based on EEG classifier. Clin Neurophysiol. 2018;129(8):1605-1617. doi:10.1016/j.clinph.2018.04.750

188. Alouani AT, Elfouly T. Traumatic brain injury (TBI) detection: Past, present, and future. Biomedicines. 2022;10(10):2472. doi:10.3390/biomedicines10102472
189. Jordan MI, Mitchell TM. Machine learning: Trends, perspectives, and prospects. Science. 2015;349(6245):255-260. doi:10.1126/science.aaa8415
190. Zhou L, Pan S, Wang J, Vasilakos AV. Machine learning on big data: Opportunities and challenges. Neurocomputing. 2017;237:350-361. doi:10.1016/j.neucom.2017.01.026
191. L'heureux A, Grolinger K, Elyamany HF, Capretz MA. Machine learning with big data: Challenges and approaches. IEEE Access. 2017;5:7776-7797. doi:10.1109/ACCESS.2017.2696365
192. Cao C, Tutwiler RL, Slobounov S. Automatic classification of athletes with residual functional deficits following concussion by means of EEG signal using support vector machine. IEEE Trans Neural Syst Rehabil Eng. 2008;16(4):327-335. doi:10.1109/TNSRE.2008.918422
193. Lai CQ, Abdullah MZ, Abdullah JM, Azman A, Ibrahim H. Screening of moderate traumatic brain injury from power feature of resting state electroencephalography using support vector machine. In: Proc 2019 2nd Int Conf Electron Electr Eng Technol. IEEE; 2019:99-103. doi:10.1145/3362752.336275
194. Schmid W, Fan Y, Chi T, et al. Review of wearable technologies and machine learning methodologies for systematic detection of mild traumatic brain injuries. J Neural Eng. 2021;18(4). doi:10.1088/1741-2552/ac1982
195. Vivaldi N, Caiola M, Solarana K, Ye M. Evaluating performance of EEG data-driven machine learning for traumatic brain injury classification. IEEE Trans Biomed Eng. 2021;68(11):3205-3216. doi:10.1109/TBME.2021.3062502
196. Noor NSE, Ibrahim H. Machine learning algorithms and quantitative electroencephalography predictors for outcome prediction in traumatic brain injury: A systematic review. IEEE Access. 2020;8:102075-102092. doi:10.1109/ACCESS.2020.2998934
197. Gravesteijn BY, Nieboer D, Ercole A, et al. Machine learning algorithms performed no better than regression models for prognostication in traumatic brain injury. J Clin Epidemiol. 2020;122:95-107. doi:10.1016/j.jclinepi.2020.03.005
198. Nti IK, Quarcoo JA, Aning J, Fosu GK. A mini-review of machine learning in big data analytics: Applications, challenges, and prospects. Big Data Min Anal. 2022;5(2):81-97. doi:10.26599/BDMA.2021.9020028
199. Hussein S, Kandel P, Bolan CW, et al. Lung and pancreatic tumor characterization in the deep learning era: Novel supervised and unsupervised learning approaches. IEEE Trans Med Imaging. 2019;38(8):1777-1787. doi:10.1109/TMI.2019.2894349
200. Roy S, Menapace W, Oei S, et al. Deep learning for classification and localization of COVID-19 markers in point-of-care lung ultrasound. IEEE Trans Med Imaging. 2020;39(8):2676-2687. doi:10.1109/TMI.2020.2994459
201. Wei XS, Song YZ, Mac Aodha O, et al. Fine-grained image analysis with deep learning: A survey. IEEE Trans Pattern Anal Mach Intell. 2022;44(12):8927–8948. doi:10.1109/TPAMI.2021.3126648
202. Khodayar M, Liu G, Wang J, Khodayar ME. Deep learning in power systems research: A review. CSEE J Power Energy Syst. 2021;7(2):209–220. doi:10.17775/CSEEJPES.2020.02700
203. Tang C, Yu C, Gao Y, et al. Deep learning in the nuclear industry: A survey. Big Data Min Anal. 2022;5(2):140–160. doi:10.26599/BDMA.2021.9020027
204. White J, Kameneva T, McCarthy C. Vision processing for assistive vision: A deep reinforcement learning approach. IEEE Trans Hum-Mach Syst. 2022;52(1):123–133. doi:10.1109/THMS.2021.3121661
205. Gong S, Xing K, Cichocki A, Li J. Deep learning in EEG: Advances of the last ten-year critical period. IEEE Trans Cogn Dev Syst. 2022;14(2):348–365. doi:10.1109/TCDS.2021.3079712
206. Hussein R, Palangi H, Ward RK, Wang ZJ. Optimized deep neural network architecture for robust detection of epileptic seizures using EEG signals. Clin Neurophysiol. 2019;130(1):25–37. doi:10.1016/j.clinph.2018.10.010
207. Rashed-Al-Mahfuz M, Moni MA, Uddin S, et al. A deep convolutional neural network method to detect seizures and characteristic frequencies using epileptic electroencephalogram (EEG) data. IEEE J Transl Eng Health Med. 2021;9:2000112. doi:10.1109/JTEHM.2021.3050925
208. Kucyi A, Davis KD. The neural code for pain: From single-cell electrophysiology to the dynamic pain connectome. Neuroscientist. 2017;23(4):397–414. doi:10.1177/1073858416667716
209. Klimesch W. EEG alpha and theta oscillations reflect cognitive and memory performance: A review and analysis. Brain Res Rev. 1999;29(2–3):169–195. doi:10.1016/S0165-0173(98)00056-3
210. Zhang CH, Sohrabpour A, Lu Y, He B. Spectral and spatial changes of brain rhythmic activity in response to the sustained thermal pain stimulation. Hum Brain Mapp. 2016;37(8):2976–2991. doi:10.1002/hbm.23220
211. Nickel MM, May ES, Tiemann L, et al. Brain oscillations differentially encode noxious stimulus intensity and pain intensity. Neuroimage. 2017;148:141–147. doi:10.1016/j.neuroimage.2017.01.011
212. Bunk SF, Lautenbacher S, Rüsseler J, et al. Does EEG activity during painful stimulation mirror more closely the noxious stimulus intensity or the subjective pain sensation? Somatosens Mot Res. 2018;35(3–4):192–198. doi:10.1080/08990220.2018.1521790
213. Sherlin L, Arns M, Lubar J, Sokhadze E. A position paper on neurofeedback for the treatment of ADHD. J Neurother. 2010;14(2):66–78. doi:10.1080/10874201003773880
214. Flor H, Turk DC. Cognitive and Behavioral Practice [Book review]. Cogn Behav Pract. 2013;20(1):117–118. doi:10.1016/j.cbpra.2012.10.001
215. Mayaud L, Congedo M, Van Laghenhove A, et al. A comparison of recording modalities of P300 event-related potentials (ERP) for brain-computer interface (BCI) paradigm. Neurophysiol Clin. 2013;43(4):217–227. doi:10.1016/j.neucli.2013.06.002
216. Skinner BF. Operant behavior. Am Psychol. 1963;18(8):503–515. doi:10.1037/h0045185
217. Schwartz M, Andrasik F. Biofeedback: A Practitioner's Guide. 4th ed. Guilford Press; 2017.
218. Ros T, Baars B, Lanius RA, Vuilleumier P. Tuning pathological brain oscillations with neurofeedback: A systems neuroscience framework. Front Hum Neurosci. 2014;8:1008. doi:10.3389/fnhum.2014.01008
219. Babiloni C, Brancucci A, Babiloni F, et al. Anticipatory cortical responses during the expectancy of a predictable painful stimulation: A high-resolution electroencephalography study. Eur J Neurosci. 2003;18(6):1692–1700. doi:10.1046/j.1460-9568.2003.02851.x
220. Al-Taleb M, Purcell M, Fraser M, Petric-Gray N, Vuckovic A. Home-used, patient self-managed, brain-computer interface for the management of central neuropathic pain post spinal cord injury: Usability study. J Neuroeng Rehabil. 2019;16(1):128. doi:10.1186/s12984-019-0588-7
221. Shimizu K, Inage K, Morita M, et al. New treatment strategy for chronic low back pain with alpha wave neurofeedback. Sci Rep. 2022;12(1):14532. doi:10.1038/s41598-022-18931-0

222. Patel K, Henshaw J, Sutherland H, et al. Using EEG alpha states to understand learning during alpha neurofeedback training for chronic pain. Front Neurosci. 2021;14:620666. doi:10.3389/fnins.2020.620666
223. Hesam-Shariati N, Chang WJ, Wewege MA, et al. The analgesic effect of electroencephalographic neurofeedback for people with chronic pain: A systematic review and meta-analysis. Eur J Neurol. 2022;29(3):921–936. doi:10.1111/ene.15189
224. Jacobs E, Jensen M. EEG neurofeedback in the treatment of chronic pain: A case series. Neuroregulation. 2015;2(2):86–102. doi:10.15540/nr.2.2.86
225. Barbosa-Torres C, Delgado S. Clinical findings in SMR neurofeedback protocol training in women with fibromyalgia syndrome. Brain Sci. 2021;11(8):1069. doi:10.3390/brainsci11081069
226. Terrasa JL, Barros-Loscertales A, Montoya P, Muñoz MA. Self-regulation of SMR power led to an enhancement of functional connectivity of somatomotor cortices in fibromyalgia patients. Front Neurosci. 2020;14:236. doi:10.3389/fnins.2020.00236
227. Zweerings J, Pflieger EM, Mathiak KA, et al. Impaired voluntary control in PTSD: Probing self-regulation of the ACC with real-time fMRI. Front Psychiatry. 2018;9:219. doi:10.3389/fpsyt.2018.00219
228. Patel K, Sivan M, Henshaw J, Jones A. Neurofeedback for chronic pain. In: Liao EDY, ed. Smart Biofeedback: Perspectives and Applications. IntechOpen; 2020. doi:10.5772/intechopen.93826
229. Son D, Does W, Band G, Putman P. EEG theta/beta ratio neurofeedback training in healthy females. Appl Psychophysiol Biofeedback. 2020;45(3):195–210. doi:10.1007/s10484-020-09472-1
230. Bair MJ, Matthias MS, Nyland KA, et al. Barriers and facilitators to chronic pain self-management: A qualitative study of primary care patients with comorbid musculoskeletal pain and depression. Pain Med. 2009;10(7):1280–1290. doi:10.1111/j.1526-4637.2009.00707.x
231. Sime A. Case study of trigeminal neuralgia using neurofeedback and peripheral biofeedback. J Neurother. 2004;8(1):59–71. doi:10.1300/J184v08n01_05
232. Prinsloo S, Novy D, Driver L, et al. The long-term impact of neurofeedback on symptom burden and interference in patients with chronic chemotherapy-induced neuropathy: Analysis of a randomized controlled trial. J Pain Symptom Manage. 2018;55(5):1276–1285. doi:10.1016/j.jpainsymman.2018.01.010
233. Caro XJ, Winter EF. EEG biofeedback treatment improves certain attention and somatic symptoms in fibromyalgia: A pilot study. Appl Psychophysiol Biofeedback. 2011;36(3):193–200. doi:10.1007/s10484-011-9159-9
234. Anil K, Demain S, Burridge J, et al. The importance of self-efficacy and negative affect for neurofeedback success for central neuropathic pain after a spinal cord injury. Sci Rep. 2022;12(1):10949. doi:10.1038/s41598-022-15213-7
235. Liu M, Li K, Wang Y, Zhao G, Jiang J. Stem cells in the treatment of neuropathic pain: Research progress of mechanism. Stem Cells Int. 2020;2020:8861251. doi:10.1155/2020/8861251
236. Hammond D. The need for individualization in neurofeedback: Heterogeneity in QEEG patterns associated with diagnoses and symptoms. Appl Psychophysiol Biofeedback. 2010;35(1):31–36. doi:10.1007/s10484-009-9106-1
237. Rogel A, Guez J, Getter N, et al. Transient adverse side effects during neurofeedback training: A randomized, sham-controlled, double blind study. Appl Psychophysiol Biofeedback. 2015;40(3):209–218. doi:10.1007/s10484-015-9289-6
238. Luctkar-Flude M, Groll D. A systematic review of the safety and effect of neurofeedback on fatigue and cognition. Integr Cancer Ther. 2015;14(4):318–340. doi:10.1177/1534735415572886
239. Kayiran S, Dursun E, Dursun N, Ermutlu N, Karamürsel S. Neurofeedback intervention in fibromyalgia syndrome: A randomized, controlled, rater-blind clinical trial. Appl Psychophysiol Biofeedback. 2010;35(4):293–302. doi:10.1007/s10484-010-9135-9
240. Bismuth J, Vialatte F, Lefaucheur JP. Relieving peripheral neuropathic pain by increasing the power-ratio of low-β over high-β activities in the central cortical region with EEG-based neurofeedback: Study protocol for a controlled pilot trial (SMRPain study). Neurophysiol Clin. 2020;50(1):5–20. doi:10.1016/j.neucli.2019.12.002
241. Hasan MA, Vuckovic A, Qazi SA, et al. Immediate effect of neurofeedback training on the pain matrix and cortical areas involved in processing neuropsychological functions. Neurol Sci. 2021;42(11):4551-4561. doi:10.1007/s10072-021-05125-1
242. Micoulaud Franchi J-A, Jeunet C, Lotte F. Neurofeedback: A challenge for integrative clinical neurophysiological studies. Neurophysiol Clin. 2020;50(1):1-3. doi:10.1016/j.neucli.2020.01.001
243. Dewan MC, Rattani A, Gupta S, et al. Estimating the global incidence of traumatic brain injury. J Neurosurg. 2019;130(4):1080-1097. doi:10.3171/2017.10.JNS17352
244. Miller GF, DePadilla L, Xu L. Costs of nonfatal traumatic brain injury in the United States, 2016. Med Care. 2021;59(5):451-455. doi:10.1097/MLR.0000000000001511
245. Agimi Y, Regasa LE, Stout KC. Incidence of traumatic brain injury in the US military, 2010–2014. Mil Med. 2019;184(5-6):e233-e241. doi:10.1093/milmed/usy313
246. Shan R, Szmydynger-Chodobska J, Warren OU, et al. A new panel of blood biomarkers for the diagnosis of mild traumatic brain injury/concussion in adults. J Neurotrauma. 2016;33(1):49-57. doi:10.1089/neu.2014.3811
247. Reith F, Van den Brande R, Synnot A, Gruen R, Maas AI. The reliability of the Glasgow Coma Scale: A systematic review. Intensive Care Med. 2016;42(1):3-15. doi:10.1007/s00134-015-4124-3
248. Panteliadis CP. Historical overview of electroencephalography: From antiquity to the beginning of the 21st century. J Brain Neurol Disord. 2021;3(1). https://escientificpublishers.com/historical-overview-of-electroencephalography-from-antiquity-to-the-beginning-of-the-21st-century-JBND-03-0020
249. Gudigar A, Raghavendra U, Hegde A, et al. Automated detection and screening of traumatic brain injury (TBI) using computed tomography images: A comprehensive review and future perspectives. Int J Environ Res Public Health. 2021;18(12):6499. doi:10.3390/ijerph18126499
250. Bardin JC, Fins JJ, Katz DI, et al. Dissociations between behavioural and functional magnetic resonance imaging-based evaluations of cognitive function after brain injury. Brain. 2011;134(Pt 3):769-782. doi:10.1093/brain/awr005
251. Arciniegas DB. Clinical electrophysiologic assessments and mild traumatic brain injury: State-of-the-science and implications for clinical practice. Int J Psychophysiol. 2011;82(1):41-52. doi:10.1016/j.ijpsycho.2011.03.004
252. Li D, Yang Z, Hou F, et al. EEG-based emotion recognition with haptic vibration by a feature fusion method. IEEE Trans Instrum Meas. 2022;71:2504111. doi:10.1109/TIM.2022.3147882
253. Wu D, Xu Y, Lu BL. Transfer learning for EEG-based brain–computer interfaces: A review of progress made since 2016. IEEE Trans Cogn Dev Syst. 2022;14(1):4-19. doi:10.1109/TCDS.2020.3007453

254. Khurana V, Gahalawat M, Kumar P, et al. A survey on neuromarketing using EEG signals. IEEE Trans Cogn Dev Syst. 2021;13(4):732-749. doi:10.1109/TCDS.2021.3065200
255. Penttonen M, Buzsáki G. Natural logarithmic relationship between brain oscillators. Thalamus Relat Syst. 2003;2(2):145-152. doi:10.1016/S1472-9288(03)00007-4
256. Sjaaheim H, Albert B, Setchi R, Noyvirt A, Strisland F. A portable medical system for the early diagnosis and treatment of traumatic brain injury. In: 2014 IEEE Int Conf Syst Man Cybern. 2014:2529-2534. doi:10.1109/SMC.2014.6974307
257. Rapp PE, Keyser DO, Albano A, et al. Traumatic brain injury detection using electrophysiological methods. Front Hum Neurosci. 2015;9:11. doi:10.3389/fnhum.2015.00011
258. Dingle AA, Jones RD, Carroll GJ, Fright WR. A multistage system to detect epileptiform activity in the EEG. IEEE Trans Biomed Eng. 1993;40(12):1260-1268. doi:10.1109/10.250582
259. Fisher JA, Huang S, Ye M, et al. Real-time detection and monitoring of acute brain injury utilizing evoked electroencephalographic potentials. IEEE Trans Neural Syst Rehabil Eng. 2016;24(9):1003-1012. doi:10.1109/TNSRE.2016.2529663
260. Chamanzar A, George S, Venkatesh P, et al. An algorithm for automated, noninvasive detection of cortical spreading depolarizations based on EEG simulations. IEEE Trans Biomed Eng. 2018;66(4):1115-1126. doi:10.1109/TBME.2018.2867112
261. Nuwer MR, Hovda DA, Schrader LM, Vespa PM. Routine and quantitative EEG in mild traumatic brain injury. Clin Neurophysiol. 2005;116(9):2001-2025. doi:10.1016/j.clinph.2005.05.008
262. Islam MK, Rastegarnia A, Yang Z. Methods for artifact detection and removal from scalp EEG: A review. Neurophysiol Clin. 2016;46(4-5):287-305. doi:10.1016/j.neucli.2016.07.002
263. Albert B, Zhang J, Noyvirt A, et al. Automatic EEG processing for the early diagnosis of traumatic brain injury. Procedia Comput Sci. 2016;96:703-712. doi:10.1016/j.procs.2016.08.253
264. Lewine JD, Plis S, Ulloa A, et al. Quantitative EEG biomarkers for mild traumatic brain injury. J Clin Neurophysiol. 2019;36(4):298-305. doi:10.1097/WNP.0000000000000588
265. Kostarelos F, MacNamee C, Mullane B. A hardware implementation of a qEEG-based discriminant function for brain injury detection. In: 2021 IEEE Biomed Circuits Syst Conf (BioCAS). 2021:1-6. doi:10.1109/BioCAS49922.2021.9645039
266. Marzbani H, Marateb HR, Mansourian M. Neurofeedback: A comprehensive review on system design, methodology and clinical applications. Basic Clin Neurosci. 2016;7(2):143-158. doi:10.15412/J.BCN.03070208
267. Enriquez-Geppert S, Smit D, Pimenta M, Arns M. Neurofeedback as a treatment intervention in ADHD: Current evidence and practice. Curr Psychiatry Rep. 2019;21(6):46. doi:10.1007/s11920-019-1021-4
268. Coben R, Hammond D, Arns M. 19-channel z-score and LORETA neurofeedback: Does the evidence support the hype? Appl Psychophysiol Biofeedback. 2018;44(1):1-8. doi:10.1007/s10484-018-9420-6
269. Sakurada T, Matsumoto M, Yamamoto S. Individual sensory modality dominance as an influential factor in the prefrontal neurofeedback training for spatial processing: A functional near-infrared spectroscopy study. Front Syst Neurosci. 2022;16:774475. doi:10.3389/fnsys.2022.774475
270. Tosti B, Corrado S, Mancone S, et al. Integrated use of biofeedback and neurofeedback techniques in treating pathological conditions and improving performance: A narrative review. Front Neurosci. 2024;18:1358481. doi:10.3389/fnins.2024.1358481
271. Fotuhi M. A comprehensive "Concussion Recovery Program" with emphasis on brain coaching and EEG-based neurofeedback therapy: A retrospective analysis. Neurology. 2018;91(23_Supplement_1):S19. doi:10.1212/01.wnl.0000550606.03730.0c
272. Neuro-Wave Brain Performance Center. What is neurofeedback therapy? https://neuro-wave.com/what-is-neurofeedback-therapy/
273. Elbogen EB, Alsobrooks A, Battles S, et al. Mobile neurofeedback for pain management in veterans with TBI and PTSD. Pain Med. 2021;22(2):329-337. doi:10.1093/pm/pnz269
274. Trullinger M, Pradhan D, Bruns TA, Clark BB. Benefits of combining neurofeedback and physical therapy for chronic pain: A case study. Surg Rehabil. 2017;1:Article 1000101. doi:10.15761/SRJ.1000101
275. Stopczynski A, Stahlhut C, Petersen MK, et al. Smartphones as pocketable labs: Visions for mobile brain imaging and neurofeedback. Int J Psychophysiol. 2014;91(1):54-66. doi:10.1016/j.ijpsycho.2013.08.007
276. Peake JM, Kerr G, Sullivan JP. A critical review of consumer wearables, mobile applications, and equipment for providing biofeedback, monitoring stress, and sleep in physically active populations. Front Physiol. 2018;9:743. doi:10.3389/fphys.2018.00743
277. Polinder S, Cnossen MC, Real RGL, et al. A multidimensional approach to post-concussion symptoms in mild traumatic brain injury. Front Neurol. 2018;9:1113. doi:10.3389/fneur.2018.01113
278. Dinh MM, Bein K, Roncal S, et al. Redefining the golden hour for severe head injury in an urban setting: The effect of prehospital arrival times on patient outcomes. Injury. 2013;44(5):606-610. doi:10.1016/j.injury.2012.01.011
279. Hu W, Freudenberg V, Gong H, Huang B. The "Golden Hour" and field triage pattern for road trauma patients. J Safety Res. 2020;75:57-66. doi:10.1016/j.jsr.2020.08.001