

# 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



**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.

activity sources for targeted treatment planning.

- Functional Assessment: Evaluates cognitive function, memory storage, and learning.

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 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 cingulate 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.

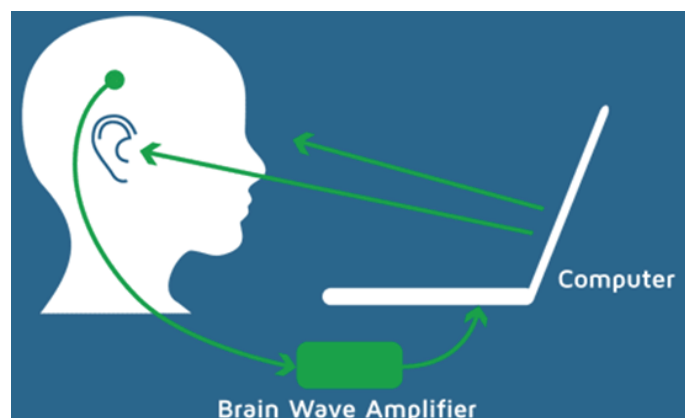


Figure 2B: BrainView utilizes a brain wave amplifier and advanced algorithms to analyze and extract detailed brain wave patterns.

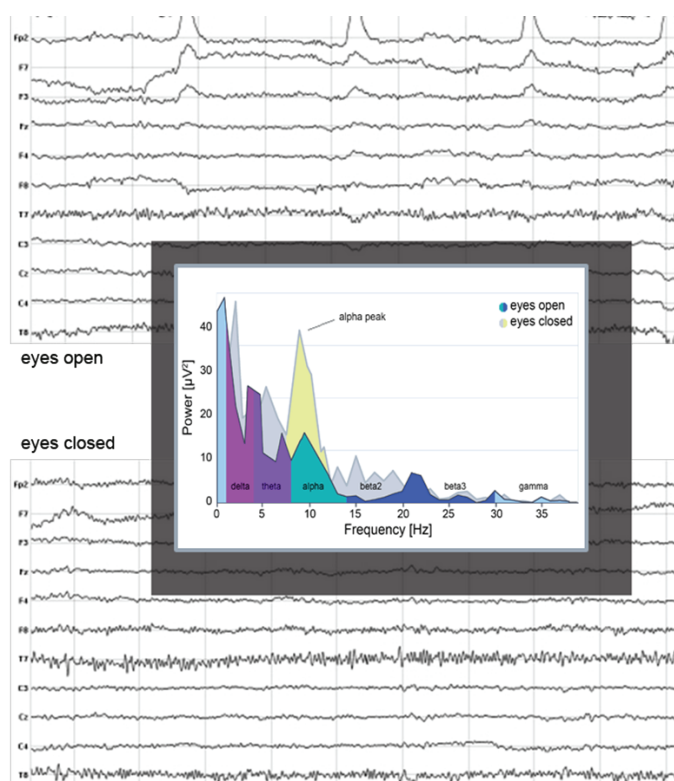


Figure 3: Example of BrainView's qEEG module identifying frequency and power domains during both eyes-open and eyes-closed sessions.

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, Michel, 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 detecting mTBI [247].

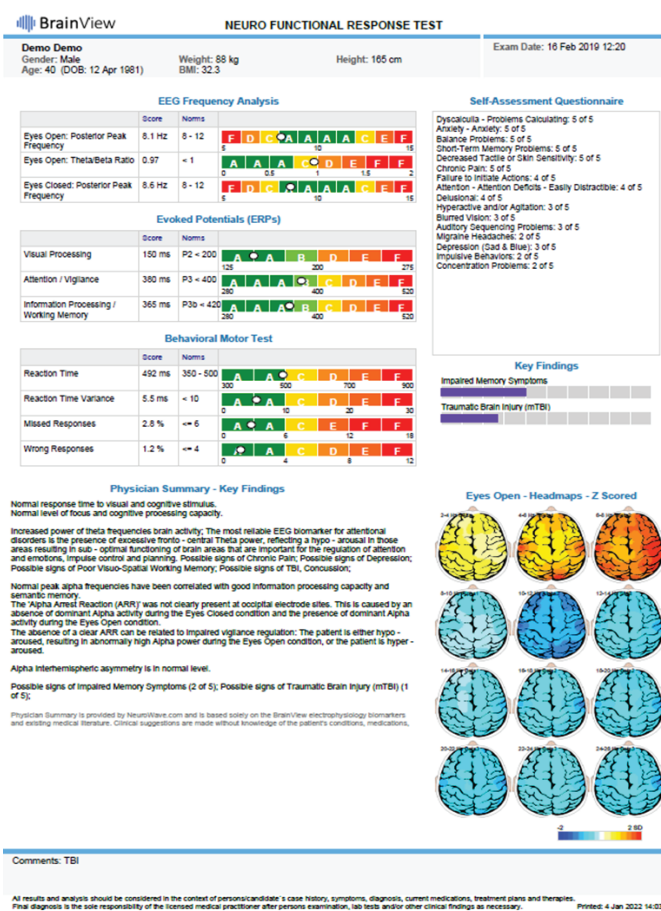
Conventional TBI diagnostics such as CT and fMRI have limitations [248]:

- 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.

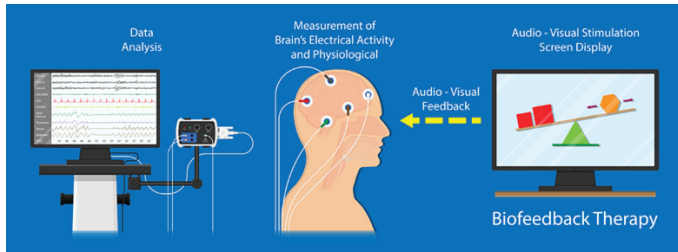
This system generates composite reports including theta/beta ratios and functional brain maps, aiding in diagnosis, treatment planning, and longitudinal tracking — especially when paired with neurofeedback.

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.

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