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# Clinical electrophysiologic assessments and mild traumatic brain injury: State-of-the-science and implications for clinical practice

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ABSTRACT

Conventional and quantitative electroencephalography (EEG and qEEG, respectively) may enhance clinical diagnosis and treatment planning provided to persons with mild traumatic brain injury (mTBI) and postconcussive symptoms. Effective and appropriate use of EEG and qEEG in this context requires expert-level knowledge of these technologies, mTBI, and the differential diagnosis for postconcussive symptoms. A practical and brief review from the perspective of a clinician–scientist engaged principally in the care and study of persons with mTBI therefore may be of use and value to other clinicians and scientists interested in these matters. Toward that end, this article offers an overview of the current applications of conventional EEG and qEEG to the study and clinical evaluation of persons with mTBI. The clinical case definition of TBI, the differential diagnosis of post-injury neuropsychiatric disturbances, and the typical course of recovery following mTBI are reviewed. With this background and context, the strengths and limitations of the literature describing EEG and qEEG studies in this population are considered. The implications of this review on the applications of these electrophysiologic assessments to the clinical evaluation of persons with mTBI and postconcussive symptoms are then considered. Finally, suggestions are offered regarding the design of future studies using these technologies in this population. Although this review may be of interest and value to professionals engaged in clinical or research electrophysiology in their daily work, it is intended to serve more immediately the needs of clinicians less familiar with these types of clinical electrophysiologic assessments.



## 1. Introduction

Electroencephalography (EEG) was the first clinical neurodiagnostic assessment to demonstrate abnormal brain function following traumatic brain injury (TBI) (Glaser and Sjaardema, 1940; Jasper et al., 1940; Williams, 1941). In the 70 years since these initial observations, the field of clinical electrophysiology evolved to include quantitative EEG (qEEG), evoked and event-related potentials (EPs and ERPs, respectively), magnetoencephalography (MEG) and magnetic source imaging (MSI), among others electrophysiologic recording techniques. While both conventional and advanced clinical electrophysiology offer the potential to improve clinical diagnosis and treatment planning, their effective and appropriate use necessitates advanced knowledge of human electrophysiology, electrophysiologic recording methods and interpretation, as well as the knowledge and skills needed to use appropriately electrophysiologic recording equipment and software. When applied to the clinical or research evaluation of persons with mild TBI (mTBI), effective and appropriate use of these technologies and techniques also requires an in-depth understanding of the epidemiology, pathophysiology, and natural history of mTBI as well as an understanding of the broad differential diagnosis of conditions that may co-occur with or mimic (clinically and electrophysiologically) mTBI.

These knowledge and skill requirements present substantial obstacles to the routine application of clinical electrophysiology to the assessment and treatment of persons with mTBI. They also limit the accessibility and usefulness of information gleaned from clinical electrophysiologic studies to clinicians working outside this niche area of clinical neuroscience. Complicating these issues is a history of contentious, and at least occasionally acrimonious, debate about the role, usefulness, and legitimate applications of clinical electrophysiology to the clinical evaluation, study, and (especially) forensic assessment of persons with mTBI. A practical and brief review of these matters from the perspective of a clinician–scientist engaged principally in the care and study of persons with mTBI, and only secondarily involved in the use of electrophysiologic assessments for clinical and research purposes, therefore may be of use and value to other clinicians and scientists interested in these matters.

Toward that end, this article offers an overview of the current applications of conventional EEG and qEEG to the study and clinical

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evaluation of persons with mTBI. This review is complementary to that offered by Nuwer et al. (2005), to which readers are directed for additional consideration of this subject. As the diagnosis of mTBI rests first and foremost on clinical history and evaluations, the clinical case definition of TBI, the differential diagnosis of post-injury neuropsychiatric disturbances, and the typical course of recovery from mTBI are reviewed. It is in this context that the role and value of electrophysiologic assessments, as well as the relative strengths and limitations of such assessments, are considered. Although qEEG also is sometimes used to guide therapeutic interventions (e.g., neurofeedback) (Thornton and Carmody, 2009) and, in other contexts, to select and/or predict pharmacologic treatment response (Hunter et al., 2010; Leuchter et al., 2009a, 2009b), these issues will not be addressed in this review. Finally, possible future clinical and research applications of electrophysiologic assessments of mTBI are considered. Although this review may be of interest and value to professionals engaged in clinical or research electrophysiology in their daily work, it is intended to serve more immediately the needs of clinicians less familiar with these types of neurodiagnostic studies.

## 2. TBI clinical case definition

### 2.1. Biomechanically-induced TBI

The *Guidelines for Surveillance of Central Nervous System Injury* developed by the United States Centers for Disease Control and Prevention (Marr and Coronado, 2002) define traumatic brain injury as an event involving an injury to the head (brain) due to blunt or penetrating trauma (i.e., application of a biomechanical force) or from acceleration–deceleration forces that produces an immediately apparent disruption of brain function and/or structure. Evidence of disrupted brain function and/or structure may include one or more of the following: a) loss of consciousness (LOC), i.e., complete loss of arousal with unresponsiveness to internal and external stimuli; b) posttraumatic amnesia (PTA), i.e., dense impairment of new learning immediately following the event, usually producing an inability to recall the injury, events immediately following it, and, in some cases, events immediately preceding it; c) alteration of consciousness (AOC), i.e., feeling 'dazed,' 'disoriented,' or 'confused'; d) focal neurological signs, i.e., motor, sensory, or reflex abnormalities, aphasia or dysphasia, or seizures (focal or generalized); e) abnormalities on formal neuropsychological testing performed in the peri-injury period; f) intracranial lesion(s) consistent with neurotrauma on computed tomography (CT) or magnetic resonance imaging (MRI) studies of the brain, i.e., subdural, epidural, intraparenchymal, or subarachnoid hemorrhage(s), diffuse or multifocal axonal injury, cerebral contusion or laceration, or penetration of brain tissue by a foreign body.

This TBI clinical case definition excludes injuries that produce head injury without brain injury — in other words, injuries resulting in lacerations or contusions of the face, eye, or scalp, and fractures of facial bones, skull, or neck but that fail to disrupt brain function and/or structure in the ways described above. Additionally, brain injuries resulting from perinatal trauma, hypoxic–ischemic (anoxic), inflammatory, toxic, or metabolic encephalopathies, primary ischemic or hemorrhagic strokes, seizure disorders, intracranial surgery, and cerebral neoplasms also fall outside this definition of TBI.

### 2.2. Blast-related TBI

In military settings, blast-related biomechanical forces are also permitted to fulfill the first element of the TBI clinical case definition (Iverson et al., 2009; Terrio et al., 2009). The biomechanical forces produced by explosive devices include: primary blast effect, which refers the combination of effective overpressure and positive pulse duration (blast wave); secondary effects, which refers to objects displaced by the blast striking or penetrating the body/brain; tertiary effects, which refers to the body/brain displaced by a blast striking objects or ground. In addition to biomechanical forces, blast events also may complicate biomechanically-induced injury through a variety of other (quaternary or miscellaneous) factors, including burns, exposure to toxic gasses and dust, hypoxia due to airway compromise or toxic exposure, structural collapse, body rupture, and psychological trauma associated with the blast event (Cernak and Noble-Haeusslein, 2010; Leung et al., 2008; Warden, 2006). The relative contributions of primary vs. secondary, tertiary, and quaternary effects to blast-induced neurotrauma are matters of considerable debate (Cernak and Noble-Haeusslein, 2010; Iverson et al., 2009) as are the relationships between blast-related mTBI and persistent postconcussive symptoms (Hoge et al., 2008; Wilk et al., 2010). Although it is not unreasonable to suggest that primary blast effects may injure the brain through a combination of concurrent and interactive systemic, local, and cerebral responses (Cernak and Noble-Haeusslein, 2010), exposure to a blast wave of magnitude sufficient to induce neurotrauma through primary blast effect frequently entails exposure to potentially injurious secondary and tertiary blast effects as well — especially rapid acceleration/deceleration forces.

For the purpose of establishing the occurrence of mTBI in the setting of blast exposure, disentangling the relative contributions of primary vs. other blast effects is less important than simply noting that the blast exposure produced a physical force that resulted in an immediately apparent disruption of brain function and/or structure of the types described in the Guidelines for Surveillance of Central Nervous System Injury (Marr and Coronado, 2002). If clinical (or research) needs necessitate specifically mentioning that blast was involved in the injury event, it is both sufficient and accurate to refer to such injuries as 'blast-related mTBI' — a term that encompasses the combined contributions of primary, secondary, tertiary, and quaternary effects of blast exposure to mTBI.

### 2.3. Applying TBI clinical case definition in clinical practice

Whether evaluating patients in civilian, military/Veterans Health Administration, or forensic contexts, clinicians must remain mindful that both the CDC definition of TBI and that advanced by the American Congress of Rehabilitation Medicine (ACRM) (Kay et al., 1993) reject the notion that there is any single pathognomonic clinical symptom or sign of TBI. Instead, these definitions identify the types of clinical symptoms and signs, as well as some of the common exclusory conditions, that allow clinicians to assert with reasonable confidence that an event experienced by a patient is characterized fairly and accurately as mTBI. As a consequence of the flexibility inherent in these clinical case definitions, however, there is considerably heterogeneity within the diagnostic category of mTBI (discussed briefly in Section 3, below). This heterogeneity presents challenges not only to clinical diagnosis but also to the study and interpretation of findings derived from studies of persons with mTBI using either conventional EEG or qEEG (discussed in Sections 5 and 6, below). Applying these clinical case definitions to any individual clinical history and examination findings (including those derived from clinical electrophysiologic assessments) therefore remains a matter in which individual clinician judgment is required (Montgomery, 2006). Clinical judgments about mTBI diagnosis rely on the clinician's ability to undertake a thorough and thoughtful consideration of all explanations for event-associated disturbances of consciousness among persons experiencing physical and/or psychological trauma; as discussed in the next section of this article, exercising such judgment requires clinicians to consider the differential diagnosis for such disturbances.

## 3. Mild traumatic brain injury vs. postconcussive symptoms

It is with this in mind that a distinction must be made between mTBI and the symptoms that a patient may experience thereafter. Mild TBI