Practice Guideline: Use of Quantitative EEG for the Diagnosis of Mild Traumatic Brain Injury: Report of the Guideline Committee of the American Clinical Neurophysiology Society

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Summary: Despite many decades of research, controversy regarding the utility of quantitative EEG (qEEG) for the accurate diagnosis of mild traumatic brain injury (mTBI) remains. This guideline is meant to assist clinicians by providing an expert review of the clinical usefulness of qEEG techniques for the diagnosis of mTBI. This guideline addresses the following primary aim: For patients with or without posttraumatic symptoms (abnormal cognition or behavior), does qEEG either at the time of injury or remote from the injury, as compared with current clinical diagnostic criteria, accurately identify those patients with mTBI (i.e., concussion)? Secondary aims included differentiating between mTBI and other diagnoses, detecting mTBI in the presence of central nervous system medications, and pertinence of statistical methods for measurements of qEEG components. It was found that for patients with or without symptoms of abnormal cognition or behavior, current evidence does not support the clinical use of qEEG either at the time of the injury or remote from the injury to diagnose mTBI (level U). In addition, the evidence does not support the use of qEEG to differentiate mTBI from other diagnoses or detect mTBI in the presence of central nervous system medications, and suitable statistical methods do not exist when using qEEG to identify patients with mTBI. Based upon the current literature review, qEEG remains an investigational tool for mTBI diagnosis (class III evidence).

Key Words: qEEG, mTBI, Diagnosis, Guideline.

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The traditional visual review of EEG waveforms focuses on the assessment of normal brain rhythms and detection of pathological, including epileptiform, activity. However, visual unassisted precise quantification of various frequencies is difficult. Likewise, visual analysis cannot resolve interrelated brain network oscillations or how a signal might relate to normal or abnormal cognition or consciousness.[1] Over the past several decades, investigators have tried to address these limitations using computer-driven algorithms, which have broadly been referred to as quantitative EEG (qEEG).[2]

One application of qEEG has been in the field of mild traumatic brain injury (mTBI). Traumatic brain injury is defined as a physical injury to the brain causing compression or tearing of the tissue. Initial clinical symptoms related to mTBI may be minimal, but cognitive and psychiatric symptoms may become chronic and last for weeks or months.[3] The rapid and accurate identification of mTBI is an important issue for populations, such as the military and sports athletes, and its correct diagnosis and prognosis have many medicolegal consequences.[4] Quantitative EEG technology has become a promising, yet controversial, tool within this field because it has been felt that it can serve as a rapid tool to detect pathological brain patterns after mTBI.

A review of qEEG was completed by the American Clinical Neurophysiology Society and the American Academy of Neurology (AAN) in 1996 as part of a joint guideline to assess the clinical usefulness of the technique.[5] At that time, it was reported that qEEG had class-I (one or more well-designed, prospective, blinded, controlled clinical studies) and class-II (one or more well-designed clinical studies such as case–control, cohort, etc.) evidence and type-A (strong positive recommendation, based in class-I or overwhelming class-II evidence) and type-B (positive recommendation, based on class II evidence) recommendation for use as an adjunctive tool in epilepsy and intraoperative/intensive care monitoring. Most other uses of qEEG had class II and class III (expert opinion, nonrandomized historical controls, or case reports of one or more) evidence and type D recommendation (negative recommendation, based on inconclusive or conflicting class-II evidence), including in patients with postconcussion syndrome and mild/moderate head injury.

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Because the original guidelines were published over 20 years ago, there has been a great deal of advancement in the field of qEEG, particularly in the areas of critical care monitoring and spike/seizure detection in epilepsy monitoring units. The prior guideline addressed a wide variety of clinical and investigational uses. Subsequently, a recent AAN Assessment updated the use of qEEG in the assessment of patients with possible attention-deficit hyperactivity syndrome.[6] This guideline is focused on qEEG as related to the diagnosis of mTBI because of its important medicolegal ramifications.

This guideline addresses the following primary aim: For patients with or without posttraumatic symptoms (abnormal cognition or behavior), does qEEG either at the time of injury or remote from the injury, as compared with current clinical diagnostic criteria, accurately identify those patients with mTBI (i.e., concussion)? Secondary aims included differentiating between mTBI and other diagnoses, detecting mTBI in the presence of central nervous system medications, and pertinence of statistical methods for measurements of qEEG components.

Despite extensive research, controversy regarding the utility of qEEG for the accurate diagnosis of mTBI remains. The guidelines presented below are meant to assist clinicians by providing an expert review of the clinical usefulness of qEEG techniques for the diagnosis of mTBI.

DESCRIPTION OF THE REVIEW PROCESS

The development of this guideline follows the 2011 AAN clinical process guideline development manual.[7] All author conflict of interests were reviewed and in compliance with AAN policy. An experienced methodologist (D.G.) supported the design of the project.

Multiple databases (Medline, EMBASE, Science Citation Index) were initially searched by a medical research librarian using the following terms: EEG OR qEEG OR quantitative EEG AND mild traumatic brain injury OR concussion. The literature search based on these criteria yielded 598 abstracts, which were then reviewed independently by two authors (J.T., R.A.); 68 abstracts were determined to meet the inclusion/exclusion criteria. The full text of these articles was then reviewed independently by the authorship group, and from these, 29 articles met criteria for data extraction and grading. Nine articles were graded class III and included in this review (Fig. 1).

Risk of bias was ascertained using the latest version of the AAN guideline development manual for risk of bias evaluation.[7] Because of the wide diversity of methods and outcome measures in this literature, results were accepted from the included articles without further group-wise statistical synthesis. This was not decided a priori in hopes of not inadvertently excluding evidence.

Literature Inclusion and Exclusion Criteria

The following inclusion criteria were used (1) all languages, (2) dates January 1, 1996 (time of the last published guideline) to December 31, 2017, (3) human subjects, (4) randomized controlled trials, case–control studies, or retrospective case series, (5) studies related to the use of qEEG as a diagnostic tool for mTBI with outcomes related to frequency analysis, monitoring/trend analysis, source localization, topographic analysis, statistical analysis, comparison to normative values, or other signal analysis (i.e., coherence). The exclusion criterion was (1) case series with the number of participants less than 10.

ANALYSIS OF EVIDENCE

Diagnosis of mTBI

There has been no agreement in the literature concerning the choice of qEEG analysis method (Table 1).[8–12] Spectral analysis involving signal power quantification from delta to gamma bands is most commonly used. Five class III studies used spectral power as the qEEG method to assess mTBI.[8–12] Increased beta power was reported in only the left occipital head region for mTBI patients during non-rapid eye movement sleep (sleep cycle 1: $F = 4.454$; $P = 0.039$; sleep cycle 2: $F = 3.761$; $P = 0.047$; sleep cycle 3: $F = 7.455$; $P = 0.008$) in one study,[8] whereas another demonstrated increased beta asymmetry in only the frontal regions (control: $\mu = 1.12 \pm 0.33$; concussion: $\mu = 2.38 \pm 0.26$; $P = 0.01$).[11] Decreased alpha power for mTBI patients was reported in three of the studies.[9,11,12] One of these studies reported that athletes with a history of concussion had decreased alpha power asymmetry compared with controls (control: $\mu = 4.28 \pm 0.46$; concussion: $\mu = 3.02 \pm 0.22$; $P = 0.01$),[11] whereas another showed that those with mTBI and moderate–severe neuropsychological impairment had decreased global alpha power compared with those with mTBI and mild impairment ($\chi^2 = 6.47$; $P < 0.05$).[9] Another study during rapid eye movement sleep showed that lower delta power at two electrodes for patients with mTBI (C3 electrode: mTBI = 0.07 μ V^2; controls = 0.77 μ V^2; $P = 0.03$; O2 electrode: mTBI = 0.56 μ V^2; controls = 0.69 μ V^2; $P = 0.02$) and higher beta and gamma power during stage 2 non-rapid eye movement sleep (beta: mTBI = 0.07 μ V^2; controls = 0.06 μ V^2; $P = 0.04$; gamma: mTBI = 0.03 μ V^2; controls = 0.02 μ V^2; $P = 0.03$).[10] Similarly, increased delta power (percent of total electrode power) was reported for patients with mTBI (mTBI = 3.7 ± 0.2%; controls = 2.8 ± 0.2%; $P = 0.002$), but a decrease in alpha power was reported only for those with mTBI who had developed posttraumatic epilepsy (mTBI = 2.1 ± 0.1%; controls = 2.9 ± 0.2%; $P = 0.005$).[12] Studies using spectral power measurements were not done using diagnostic study designs, two of these included sleep recordings only,[8,10] and another did not clearly correct for multiple comparisons.[11]

Two class III studies used a proprietary handheld frontal recording device (Brainscope, Brainscope Company Inc, Bethesda, MD).[13,14] One of the studies processed the collected EEG data offline to create a "TBI-index".[14] This measurement was compared with the New Orleans Criteria to predict which patients would have a positive head computed tomography finding. The TBI index had improved specificity over New Orleans Criteria (49.4 vs. 23.5%, respectively) to predict head computed tomography findings. Although the TBI index was compared with "age expected normal values", no controls were used in this study, and the lowest risk mTBI group, without head imaging ordered, was excluded. The other class-III study using the same recording technique used an offline multivariate analysis of seven qEEG

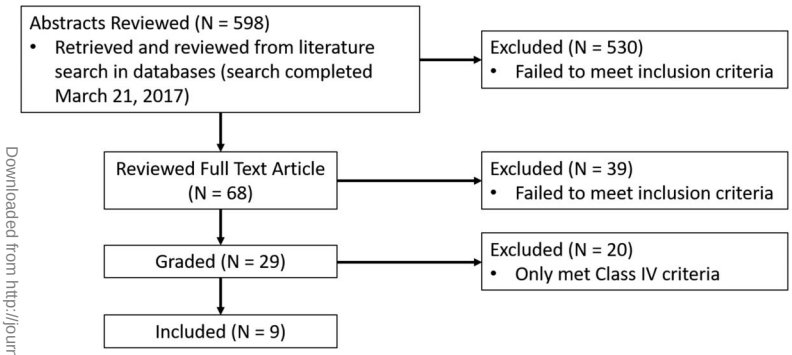

FIG. 1. Flow diagram showing number of abstract and full-text articles reviewed and excluded.

features.[13] These were found to be abnormal for patients with mTBI at day 0 (controls: $F = 0.52$; mTBI: $F = 2.5$; $P < 0.05$) and day 8 (controls: $F = 0.56$; mTBI: $F = 3.3$; $P < 0.05$), relative to the injury, but not at day 45 (controls: $F = 0.86$; mTBI: $F = 1.5$).

Three of the class-III studies used a variety of other analysis techniques, including EEG microstates, standardized low-resolution brain electromagnetic tomography (sLORETA) activation, wavelet entropy, and graph theory measures.[9,15,16] It was reported that patients with mTBI had durations of microstates, which decreased linearly with the degree of neuropsychological impairment (rho $= -0.541$; $P < 0.01$; $r = -0.573$; $P < 0.01$) and had reduced sLORETA activation in mTBI with moderate–severe neuropsychological impairment compared with patients with mTBI and no impairment ($F = 3.901$; $P = 0.036$).[9] Wavelet entropy was reduced at day 7 post-mTBI, and a recovery to baseline was slower after a second mTBI (occipital: $F = 179.18$; $P < 0.001$, parietal: $F = 181.98$; $P < 0.001$; temporal: $F = 98.17$; $P < 0.001$).[15] In this study, there was a comparison to an individual baseline but not against healthy controls. Graph theory measures have been reported with no small world topology differences between mTBI and control groups but regional increases in betweenness centrality (F4 electrode: $P = 0.05$; F10 electrode: $P = 0.02$) and mixed regional increases (F10 electrode: $P = 0.01$) and decreases (Fpz electrode: $P = 0.01$) in degree for patients with mTBI.[16] These were group level but not individual patient differences.

Evidence Synthesis

The evidence does not support the use of qEEG to accurately identify patients with mTBI either at the time of injury or remote from the injury.

Differentiation of mTBI from Related Diagnoses

Some of the studies attempted to differentiate mTBI from healthy controls and subgroups, such as those with/without pain or with/without posttraumatic epilepsy.[10,12] However, none of the class-III studies compared patients with mTBI with other neurological and psychiatric disorders (such as depression, autism, attention-deficit hyperactivity disorder, and migraine) that are also likely to have altered qEEG measures. Therefore, specificity of qEEG findings for the mTBI diagnosis could not be determined. Low-amplitude alpha also is considered to be a normal variation.[17]

Evidence Synthesis

The evidence does not support the use of qEEG to correctly differentiate mTBI from other disorders.

Presence of Medications

It is possible that a variety of medications as a covariate could have altered qEEG measures and acted as a confounder. None of the included class-III studies used a medication washout period or included their use in the analysis. Only one of the studies specifically excluded patients for "use of psychotropic medication or other drugs known to influence sleep or motor behavior."[10]

Evidence Synthesis

The evidence does not support the use of qEEG to reliably identify patients with mTBI in the presence of central nervous system medication.

Differentiation Between mTBI and State (Drowsiness)

The patient's state of alertness (e.g., awake, drowsy, asleep) is another potential confounder to consider for qEEG measurements because it can change spectral power in specific frequency bandwidths. Six of the nine class-III studies included in the analysis controlled qEEG findings for wakefulness. Four studies were completed while awake in an eyes closed state,[11,13,14,16] and two were performed during sleep because the primary outcome was a sleep-related measure.[8,10] None of the studies compared qEEG measurements with the patient in both waking and drowsy or sleeping states.

Evidence Synthesis

The evidence does not support the use of qEEG to differentiate between drowsiness and mTBI.

Statistical Considerations

There is no agreement in the literature related to the statistical measures for qEEG analyses. Because of the limited number of participants, testing at various time points, variability in number of electrodes, and frequency bins, a correction for multiple comparisons is necessary. The included class-III studies used a variety of statistical analysis tools, including analysis of variance or multivariate analysis of variance with Tukey post hoc

TABLE 1. Analysis of Evidence

Author Year	Class	Blind	Cohort Size	Controls Type	qEEG Method	Results	Notes
Arbour et al. 2015[8]	III	No	34	Yes Previous database	Spectral power	1 Higher beta power for mTBI in O1 in NREM 2) No other qEEG differences	1) No diagnostic study design 2) No awake qEEG
Corradini and Persinger 2014[9]	III	No	26	Yes Newly acquired	Spectral power Microstates sLORETA activation	1) Lower alpha power in mTBI with moderate–severe impairment 2) Decreased duration of microstates for mTBI with moderate–severe impairment 3) Reduced sLORETA activation for mTBI with moderate–severe impairment	1) Not controlled 2) No diagnostic study design
O'Neil et al. 2012[14]	III	No	119	No	Brainscope (handheld frontal recording)	1) TBI-index with improved specificity than NOC to predict positive head CT	1) Not controlled 2) Excludes lowest risk mTBI group (without head CT ordered)
Khoury et al. 2013[10]	III	No	24	Yes Newly acquired	Spectral power	1) Lower delta power for mTBI 2) Higher beta and gamma power for mTBI 3) No differences for mTBI without pain	1) No diagnostic study design 2) No awake qEEG
McCrea et al. 2010[13]	III	No	28	Yes Newly acquired	Brainscope (handheld frontal recording)	1) Multivariate analysis of 7 qEEG features were abnormal for mTBI at days 0 and 8 but not at day 45	1) Narrow spectrum of persons with and without disease
Moore et al. 2016[11]	III	No	52	Yes Newly acquired	Spectral power (high density EEG)	1) Decreased alpha and increased beta frontal asymmetry for mTBI	1) No correction for multiple comparisons
Slobounov et al. 2009[15]	III	No	21	No	Wavelet entropy	1) Entropy was reduced at 7 days post-mTBI 2) Recovery to baseline was slower after 2nd mTBI	1) No diagnostic study design 2) Comparison to baseline but not controls
Tomkins et al. 2011[12]	III	Yes	22	Yes Newly acquired	Spectral power	1) Increase in delta power for mTBI 2) Decrease in alpha power for mTBI + PTE only	1) Diagnostic study for PTE
Virji-Babul et al. 2014[16]	III	No	9	Yes Newly acquired	Graph theory	1) No small world topology differences 2) Regional increase in betweenness centrality 3) Regional mixed increases and decreases in degree	1) No diagnostic study design 2) Group level but not individual analyses

mTBI, mild traumatic brain injury; NOC, New Orleans Criteria; NREM, non-rapid eye movement; qEEG, quantitative electroencephalography.

analysis, nonparametric Kruskal–Wallis test, receiver operating curve, Mann–Whitney *U* test, or software packages with built-in statistical function (Brain Connectivity Toolbox).[18] Two of the studies specifically discussed the use of multiple comparison corrections.[11,16] It is noted that testing in 4 to 6 frequency bands, at 21 scalp electrodes, with 2 to 6 measurements each, and coherence across 21 × 20 electrode sites can generate many thousands of individual statistical comparisons. When judged at a likelihood of $P < 0.05$, hundreds of false-positive results can occur. When performing group-level analyses, these electrodes cannot be treated as independent observations but require the use of mixed models.

Evidence Synthesis

The evidence does not demonstrate that suitable statistical methods exist when using qEEG to identify patients with mTBI.

Neurophysiological Considerations

Because the neurophysiological studies reviewed here used heterogeneous methods for the measurement of qEEG and neuropsychological evaluations, there are multiple sources of possible bias. This can include differences in acquisition hardware and dissimilar practices, such as use of conventional electrodes versus whole head caps and whether the technician ensures adequate impedance before recording. Other potential sources of bias could include recording techniques, amplifier properties, and whether data are down sampled before analysis.

Clinical Context

Identification of individuals with acute or remote mTBI is an important goal, as it is a widespread health problem in both civilian and military populations with important medicolegal ramifications. Altered brain rhythms following mTBI have been purported to occur and are a possible neurophysiologic biomarker requiring further exploration. Rapidly evolving, numerous, nonstandardized, and unguided qEEG methods have led to significant controversy in the field.

This review reveals a diverse dearth of evidence-based qEEG diagnostic techniques for the identification of individuals with mTBI. The best studies used multiple different qEEG techniques, but even the most commonly used technique of spectral analysis had variations among studies with regards to recording parameters and analysis methods. Lack of standardization in qEEG acquisition leads to potentially inadequately performed recordings. In such a setting, an artifact could inadvertently be interpreted as an abnormality. It is recommended that qEEG studies be performed by a qualified EEG technician to ensure that high-quality data are acquired and that a qualified electroencephalographer should review the raw data for the presence of artifacts, drowsiness, or normal variants.

The identified literature does not inform about considerations regarding qEEG as a clinical diagnostic tool such as specificity for mTBI, effects of potential confounders such as patient state and medications, and appropriate statistics. The evaluation of any new diagnostic EEG test must evaluate several specific issues, which have previously been outlined if it is to be deemed clinically valuable to clinicians and is clinically relevant for patients.[19]

CONCLUSIONS AND RECOMMENDATIONS

Practice Recommendations

For patients with or without symptoms of abnormal cognition or behavior, current evidence does not support the clinical use of qEEG either at the time of the injury or remote from the injury to diagnose mTBI (level U). In addition, the evidence does not support the use of qEEG to differentiate mTBI from other diagnoses or detect mTBI in the presence of central nervous system medications, and suitable statistical methods do not exist when using qEEG to identify patients with mTBI. Based upon the current literature review, qEEG remains an investigational tool for mTBI diagnosis (class-III evidence).

Suggestions for Further Research

There have been no well-designed studies of qEEG-related methods for the diagnosis of mTBI. Optimal trials to validate qEEG as a tool for mTBI diagnosis would use well-accepted standards to initially define the disease population. The criteria to define a possible "abnormality" for a qEEG method should also be specified in advance of data collection. Although many qEEG measures may classify a measure as abnormal based on comparison to normative values, the test should be validated on participants different from the original cohort or normative database. Also, although qEEG methods may be able to discriminate between patients with mTBI and healthy controls, to be clinically useful, it should also be able to differentiate between mTBI patients and other conditions in the differential diagnosis.

To summarize, there are four important issues that would need to be addressed if a clinical practice change is to occur in patients with mTBI using qEEG: (1) definition of a gold-standard against which diagnostic performance of any qEEG modality could be measured, (2) consensus on methods for data acquisition, (3) analysis of multiple qEEG measures representing different neurophysiological aspects, and (4) inclusion of these metrics and use of appropriate statistical methods to develop a predictive, as opposed to merely an explanatory, model.

Like standard EEG, it is important that the qEEG interpreters be blinded to the clinical status of the participants. The effect of potential qEEG signal confounders also needs to be understood and controlled for, including patient state and medications. In general, qEEG should only be interpreted for clinical purposes by a neurologist with neurophysiology training or another physician with Accreditation Council for Graduate Medical Education-certified neurophysiology training (or country equivalent) plus subspecialty board certification in neurophysiology. Finally, there is a need for careful statistical considerations given the number of measures necessary for qEEG acquisition, analysis, and interpretation. Any group-level differences would also need to be validated on an individual patient basis for it to be a clinically useful tool.

REFERENCES

1. Tononi G, Sporns O, Edelman GM. A measure for brain complexity: relating functional segregation and integration in the nervous system. Proc Natl Acad Sci U S A 1994;91:5033–5037.
2. Rapp PE, Keyser DO, Albano A, et al. Traumatic brain injury detection using electrophysiological methods. Front Hum Neurosci 2015;9:11.
3. DeKosky ST, Ikonomovic MD, Gandy S. Traumatic brain injury–football, warfare, and long-term effects. N Engl J Med 2010;363:1293–1296.
4. Hoge CW, McGurk D, Thomas JL, Cox AL, Engel CC, Castro CA. Mild traumatic brain injury in U.S. Soldiers returning from Iraq. N Engl J Med 2008;358:453–463.
5. Nuwer M. Assessment of digital EEG, quantitative EEG, and EEG brain mapping: report of the American Academy of Neurology and the American clinical neurophysiology society. Neurology 1997;49:277–292.
6. Nuwer MR, Buchhalter J, Shepard KM. Quantitative EEG in attention-deficit/hyperactivity disorder: a companion payment policy review for clinicians and payers. Neurol Clin Pract 2016;6:543–548.
7. Neurology) AAAo. Clinical practice guideline process manual. St. Paul, MN: The American Academy of Neurology, 2011.
8. Arbour C, Khoury S, Lavigne GJ, et al. Are NREM sleep characteristics associated to subjective sleep complaints after mild traumatic brain injury? Sleep Med 2015;16:534–539.
9. Corradini PL, Persinger MA. Spectral power, source localization and microstates to quantify chronic deficits from 'mild' closed head injury: correlation with classic neuropsychological tests. Brain Inj 2014;28:1317–1327.
10. Khoury S, Chouchou F, Amzica F, et al. Rapid EEG activity during sleep dominates in mild traumatic brain injury patients with acute pain. J Neurotrauma 2013;30:633–641.
11. Moore RD, Sauve W, Ellemberg D. Neurophysiological correlates of persistent psycho-affective alterations in athletes with a history of concussion. Brain Imaging Behav 2016;10:1108–1116.
12. Tomkins O, Feintuch A, Benifla M, Cohen A, Friedman A, Shelef I. Blood-brain barrier breakdown following traumatic brain injury: a possible role in posttraumatic epilepsy. Cardiovasc Psychiatry Neurol 2011;2011:765923.
13. McCrea M, Prichep L, Powell MR, Chabot R, Barr WB. Acute effects and recovery after sport-related concussion: a neurocognitive and quantitative brain electrical activity study. J head Trauma Rehabil 2010;25:283–292.
14. O'Neil B, Prichep LS, Naunheim R, Chabot R. Quantitative brain electrical activity in the initial screening of mild traumatic brain injuries. West J Emerg Med 2012;13:394–400.
15. Slobounov S, Cao C, Sebastianelli W. Differential effect of first versus second concussive episodes on wavelet information quality of EEG. Clin Neurophysiol : official J Int Fed Clin Neurophysiol 2009;120:862–867.
16. Virji-Babul N, Hilderman CG, Makan N, et al. Changes in functional brain networks following sports-related concussion in adolescents. J Neurotrauma 2014;31:1914–1919.
17. Niedermeyer's Electroencephalography. Basic principles, clinical applications, and related fields. 7 ed. New York, NY: Oxford University Press, 2017.
18. Rubinov M, Sporns O. Complex network measures of brain connectivity: uses and interpretations. Neuroimage 2010;52:1059–1069.
19. Nuwer MR. On the controversies about clinical use of EEG brain mapping. Brain Topogr 1990;3:103–111.