Clinical Policy Bulletin:
Quantitative EEG (Brain Mapping)

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Policy

I. Aetna considers the use of quantitative EEG (brain mapping), also known as BEAM (Brain Electrical Activity Mapping), medically necessary only as an adjunct to traditional EEG for any of the following:

1. For ambulatory recording of EEG to facilitate subsequent expert visual EEG interpretation; or
2. For continuous EEG monitoring by frequency-trending to detect early, acute intracranial complications in the operating room or intensive care unit (ICU); or
3. For evaluation of certain members with symptoms of cerebrovascular disease whose neuroimaging and routine EEG studies are not conclusive; or
4. For evaluation of dementia and encephalopathy when the diagnosis remains unresolved after initial clinical evaluation; or
5. For screening for possible epileptic seizures in high-risk ICU members; or
6. For screening for possible epileptic spikes or seizures in long-term EEG monitoring; or
7. For topographic voltage and dipole analysis in pre-surgical evaluations for intractable epilepsy.

II. In accordance with the American Academy of Neurology/American Clinical Neurophysiology Society's assessment and available evidence, Aetna considers the use of quantitative EEG experimental and investigational for all other indications, including any of the following diagnoses because there is inadequate scientific evidence to prove its clinical usefulness for these indications:

Alcoholism
Asperger syndrome and other autism spectrum disorders
Attention disorders
Bipolar disorder
Depression
Drug abuse
Fibromyalgia
Hypoxic ischemic encephalopathy
Insomnia
Learning disability
Mild or moderate head injury
Panic disorder
Parkinson's disease
Post-concussion syndrome
Predicting response to psychotropic medication
Schizophrenia
Sepsis-associated encephalopathy prognosis
Sports concussion (diagnosis and assessment of recovery)
Tinnitus.

See also CPB 0480 - Tourette Syndrome.

Background

Quantitative EEG (qEEG) is a method of analyzing the electrical activity of the brain to derive quantitative patterns that may correspond to diagnostic information and/or cognitive deficits.

Quantitative EEG, a technique for topographic display and analysis of brain electrophysiological data, has been proposed for use in the diagnosis of various psychiatric disorders. Clinical studies have demonstrated distinctive forms of brain electrical activity in psychiatric conditions including attention deficit disorder, schizophrenia, major depression, and obsessive-compulsive disorder. However, the clinical significance of these distinctive patterns of brain wave activity is unknown. Thus the role of quantitative EEG in diagnosis, evaluation of disease progression, and treatment of these conditions has yet to be elucidated. A report from the American Academy of Neurology and the American Clinical Neurophysiology Society concluded that quantitative EEG remains investigational for clinical use in post-concussion syndrome, mild-to-moderate head injury, learning disability, attention disorders, schizophrenia, depression, alcoholism, and drug abuse.

Clinical studies have demonstrated distinctive forms of brain electrical activity in neurologic and psychiatric conditions including learning disabilities, autism, traumatic brain injury, coma, schizophrenia, major depression, and obsessive-compulsive disorder. However, the clinical significance of these distinctive patterns of brain wave activity is unknown. Thus the role of quantitative EEG in diagnosis, evaluation of disease progression, and treatment of these conditions has yet to be elucidated.

Quantitative EEG has been proposed for use in a broad array of potential applications. This evidence has focused on the diagnostic accuracy of qEEG. There is, however, a paucity of evidence regarding its clinical utility.
There are no current guidelines from leading medical professional organizations recommending the use of quantitative EEG as a screening test for neurological and psychiatric conditions. In addition, there are no peer-reviewed published prospective studies of the use of quantitative EEG screening for these conditions showing that management is altered such that clinical outcomes are improved.

There are no published clinical studies demonstrating that use of quantitative EEG reduces the number of imaging studies or other follow-up tests. In addition, there are no current guidelines from leading medical professional organizations recommending the use of quantitative EEG either as a prerequisite to, or as a replacement for, imaging studies.

While there is some evidence that electroencephalograph activity differs between normal control subjects and subjects suffering from tinnitus, additional evidence is needed to evaluate the value of including quantitative EEG in a battery of electrophysiological tests for the clinical identification of a predominantly central type of tinnitus. In addition, there is little evidence to support the use of quantitative EEG to determine the need for change of medications in the treatment of tinnitus.

Some investigators have proposed use of quantitative EEG in psychiatric cases to facilitate selection of medications. However, there is a lack of reliable evidence from prospective studies demonstrating that clinical outcomes are improved by basing selection of psychotropic medications on quantitative EEG results compared to empiric selection. The FDA approved prescribing information for psychotropic medications includes no recommendation for use of quantitative EEG in selection or dosing, and there are no current guidelines from leading medical professional organizations recommending such use of quantitative EEG.

Crumbley and associates (2005) examined the use of quantitative EEG in predicting response to psychotropic medication. The clinical outcomes of 2 groups of patients were compared: (i) those with prescribed medication regimens that were concordant with the quantitative EEG predictors, and (ii) those whose medication regimens were discordant with the quantitative EEG predictors. Participants included 70 adolescent inpatients who were administered quantitative EEG upon admission. The results indicated no significant difference in clinical outcome between the two groups. The failure of this study to find significant differences in patient outcomes questions this particular use of the quantitative EEG (Crumbley et al, 2005).

John and Prichep (2006) noted that as quantitative EEG and pharmaco-EEG have evolved, a vast body of facts has been accumulated, describing changes in the EEG or event-related potentials observed in a variety of brain disorders or after administration of a variety of medications. With some notable exceptions, these studies have tended to be phenomenological rather than analytical. There has not been a systematic attempt to integrate these phenomena to provide better understanding of how the abnormal behaviors of a particular psychiatric patient might be related to the specific pattern of the deviant electrical activity, nor just how pharmacological reduction of that deviant activity may have resulted in more normal behavior.
There is insufficient evidence to support the use of quantitative EEG in the diagnosis and/or classification of attention-deficit hyperactivity disorder (ADHD) (Krull, 2009). Several studies have demonstrated differences in qEEG between groups of children with ADHD and normal children. However, these studies are limited by non-random assignment, lack of blinding, failure to consider comorbidities, and/or failure to control for pharmacologic therapy. In addition, the specificity of the findings for ADHD has not been demonstrated.

Snyder and Hall (2006) performed a meta-analysis on the use of quantitative EEG in evaluating patients with ADHD. The 9 eligible studies (n = 1,498) observed quantitative EEG traits of a theta power increase and a beta power decrease, summarized in the theta/beta ratio with a pooled effect size of 3.08 (95% confidence interval: 2.90 to 3.26) for ADHD versus controls (normal children, adolescents, and adults). These investigators concluded that this meta-analysis supports that a theta/beta ratio increase is a commonly observed trait in patients with ADHD relative to normal controls. Moreover, they noted that since it is known that the theta/beta ratio trait may arise with other conditions, a prospective study covering differential diagnosis would be needed to determine generalizability to clinical applications. Furthermore, standardization of the quantitative EEG technique is also needed, specifically with control of mental state, drowsiness, and medication.

Although QEEG may prove to be helpful in the diagnosis and/or classification of ADHD in the future, at present, there is insufficient evidence to support its use in clinical populations.

Much of the literature submitted focuses on the use of QEEG in the early detection of dementia. Although several markers of early dementia have been reported in the literature, there is a lack of evidence that early detection of dementia alters clinical management such that outcomes are improved, especially given the lack of robust treatments available.

An assessment by the Swedish Office of Health Technology Assessment (SBU, 2008) found insufficient evidence to support the use of quantitative EEG in dementia. The SBU assessment stated: "[t]here is limited evidence that either visually rated EEG or qEEG helps the diagnostic workup differentiate AD (Alzheimer’s Disease) patients from controls or AD from other dementia disorders."

Klassen et al (2011) evaluated qEEG measures as predictive biomarkers for the development of dementia in Parkinson disease (PD). Preliminary work shows that qEEG measures correlate with current PD cognitive state. A reliable predictive qEEG biomarker for PD dementia (PD-D) incidence would be valuable for studying PD-D, including treatment trials aimed at preventing cognitive decline in PD. A cohort of subjects with PD in the authors’ brain donation program utilizes annual pre-mortem longitudinal movement and cognitive evaluation. These subjects also undergo biennial EEG recording. EEG from subjects with PD without dementia with follow-up cognitive evaluation was analyzed for qEEG measures of background rhythm frequency and relative power in δ, α, and β bands. The relationship between the time to onset of dementia and qEEG and other possible predictors was assessed by using Cox regression. The hazard of developing
dementia was 13 times higher for those with low background rhythm frequency (lower than the grand median of 8.5 Hz) than for those with high background rhythm frequency (p < 0.001). Hazard ratios (HRs) were also significant for greater than median bandpower (HR = 3.0; p = 0.004) compared to below, and for certain neuropsychological measures. The HRs for δ, α, and β bandpower as well as baseline demographic and clinical characteristics were not significant. The authors concluded that qEEG measures of background rhythm frequency and relative power in the band are potential predictive biomarkers for dementia incidence in PD. These QEEG biomarkers may be useful in complementing neuropsychological testing for studying PD-D incidence.

Marzano and colleagues (2008) stated that in the last 2 decades quantitative EEG analysis has been used to examine the neurophysiological characteristics of insomnia. These studies provided evidence in support of the hypothesis that primary insomnia is associated with hyper-arousal of central nervous system and altered sleep homeostasis. However, these researchers have here underlined that these results have intrinsic methodological problems, mainly related to constraints of standard assessment in clinical research. They have proposed that future studies should be performed on larger samples of drug-free patients, using within-subjects designs and longitudinally recording patients adapted to sleep laboratory. All these methodological improvements will allow to partial out the contribution of individual differences, pharmacological influences and first-night effects on EEG frequencies. Moreover, they have discussed the potential relevance of recent findings from basic research concerning local changes during physiological sleep, which could be extended to the study of insomnia.

Hargrove and colleagues (2010) stated that there is increasing acceptance that pain in fibromyalgia (FM) is a result of dysfunctional sensory processing in the spinal cord and brain, and a number of recent imaging studies have demonstrated abnormal central mechanisms. These researchers compared quantitative electroencephalogram (qEEG) measures in 85 FM patients with age- and gender-matched controls in a normative database. A statistically significant sample (minimum 60 seconds from each subject) of artifact-free EEG data exhibiting a minimum split-half reliability ratio of 0.95 and test-retest reliability ratio of 0.90 was used as the threshold for acceptable data inclusion. Electroencephalograms of FM subject were compared to EEGs of age- and gender-matched healthy subjects in the Lifespan Normative Database and analyzed using NeuroGuide 2.0 software. Analyses were based on spectral absolute power, relative power and coherence. Clinical evaluations included the Fibromyalgia Impact Questionnaire (FIQ), Beck Depression Inventory and Fischer dolorimetry for pain pressure thresholds. Based on Z-statistic findings, the EEGs from FM subjects differed from matched controls in the normative database in 3 features: (i) reduced EEG spectral absolute power in the frontal International 10-20 EEG measurement sites, particularly in the low- to mid-frequency EEG spectral segments; (ii) elevated spectral relative power of high frequency components in frontal/central EEG measurement sites; and (iii) widespread hypo-coherence, particularly in low- to mid-frequency EEG spectral segments, in the frontal EEG measurement sites. A consistent and significant negative correlation was found between pain severity and the magnitude of the EEG abnormalities. No relationship between EEG findings and medicine use was found. The authors concluded that qEEG analysis reveals significant differences between FM patients compared to age- and gender-
matched healthy controls in a normative database, and has the potential to be a clinically useful tool for assessing brain function in FM patients.

Hathi et al (2010) assessed an EEG-based index, the Cerebral Health Index in babies (CHI/b), for identification of neonates with high Sarnat scores and abnormal EEG as markers of hypoxic ischemic encephalopathy (HIE) after perinatal asphyxia. This was a retrospective study using 30-min EEG data collected from 20 term neonates with HIE and 20 neurologically normal neonates. The HIE diagnosis was made on clinical grounds based on history and examination findings. The maximum-modified clinical Sarnat score was used to grade HIE severity within 72 hrs of life. All neonates underwent 2-channel bedside EEG monitoring. A trained electroencephalographer blinded to clinical data visually classified each EEG as normal, mild or severely abnormal. The CHI/b was trained using data from Channel 1 and tested on Channel 2. The CHI/b distinguished among HIE and controls (p < 0.02) and among the 3 visually interpreted EEG categories (p < 0.0002). It showed a sensitivity of 82.4 % and specificity of 100 % in detecting high grades of neonatal encephalopathy (Sarnat 2 and 3), with an area under the receiver operator characteristic (ROC) curve of 0.912. CHI/b also identified differences between normal versus mildly abnormal (p < 0.005), mild versus severely abnormal (p < 0.01) and normal versus severe (p < 0.002) EEG groups. An ROC curve analysis showed that the optimal ability of CHI/b to discriminate poor outcome was 89.7 % (sensitivity: 87.5 %; specificity: 82.4 %). The authors concluded that the CHI/b identified neonates with high Sarnat scores and abnormal EEG. These results support its potential as an objective indicator of neurological injury in infants with HIE.

Lopes et al (2010) examined and compared the brain cortical activity, as indexed by qEEG power, coherence and asymmetry measures, in panic disorder patients during an induced panic attack with a 35 % CO(2) challenge test and also in a resting condition. A total of 15 subjects with panic disorder were randomly assigned to both 35 % CO(2) mixture and atmospheric compressed air, in a double-blind study design, with EEG being recorded for a 20-min period. During induced panic attacks, a reduced right-sided frontal orbital asymmetry in the beta band, a decreased occipital frontal intra-hemispheric coherence in the delta band at both right and left sides, a left-sided occipital delta inter-hemispheric asymmetry and an increased relative power in the beta wave at T4 were observed. These data showed a disturbed frontal cortical processing, pointing to an imbalance of the frontal and occipital sites, common to both hemispheres, and an increased right posterior activity related to the high arousing panic attack condition. These findings corroborated the neuroanatomical hypothesis of panic disorder.

Velasques et al (2013) examined the relationship between cortical gamma coherence within patients with bipolar disorder and a control group during a pro-saccadic attention task. These investigators hypothesized that gamma coherence oscillations act as a main neural mechanism underlying information processing which changes in bipolar patients. A total of 32 subjects (12 healthy controls and 20 bipolar patients) were enrolled in this study. Participants performed a pro-saccadic attention task while their brain activity pattern was recorded using qEEG (20 channels). These researchers observed that the maniac group presented lower saccade latency when compared to depression and control groups. The main finding was a greater gamma coherence for control group in the right
hemisphere of both frontal and motor cortices caused by the execution of a pro-
saccadic attention task. The authors concluded that these findings suggested a
disrupted connection of the brain's entire functioning of maniac patients and
represented a deregulation in cortical inhibitory mechanism. Thus, these results
reinforce the hypothesis that greater gamma coherence in the right and left frontal
cortices for the maniac group produces a "noise" during information processing
and highlights that gamma coherence might be a biomarker for cognitive
dysfunction during the manic state. The authors stated that these findings need to
be confirmed in larger samples and in bipolar patients before start the
pharmacological treatment.

An UpToDate review on “Attention deficit hyperactivity disorder in children and
adolescents: Clinical features and evaluation” (Krull, 2013) states that “We do not
suggest qEEG for the evaluation of children with ADHD. Although the US Food
and Drug Administration has licensed the first EEG test for assessment of children
(6 to 17 years) for ADHD, and several studies have demonstrated differences in
qEEG between children with ADHD and normal children, the studies were limited
by non-random assignment, lack of blinding, failure to consider comorbidities,
and/or failure to control for pharmacologic therapy. In addition, the EEG patterns
differ in boys and girls. A 2013 meta-analysis of nine studies (including 1253
children with ADHD and 517 without ADHD) found significant heterogeneity and
concluded that EEG profiles (specifically an increased theta to beta ratio) cannot
be used to reliably diagnose ADHD (although they may be helpful for prognosis).
Current evidence is insufficient to support the use of qEEG over clinical evaluation
of symptoms and functional impairment for the diagnosis of ADHD”.

Kutcher et al (2013) summarized the evidence for the following
technologies/strategies related to diagnosing or managing sports-related
concussion: quantitative EEG, functional neuroimaging, head impact sensors,
telemedicine and mobile devices. Databases used were MEDLINE, PubMed,
Cochrane Controlled Trials Registers, SportDiscus, EMBASE, Web of Science and
ProQuest databases. Primary search keywords were concussion, sports
concussion and mild traumatic brain injury. The keywords used for secondary,
topic specific searches were quantitative electroencephalography, qEEG,
functional MRI, magnetoencephalography, near-infrared spectroscopy, positron
emission tomography, single photon emission CT, accelerometer, impact sensor,
telemetry, remote monitoring, robotic medicine, telemedicine, mobile device,
mobile phone, smart phone and tablet computer. The primary search produced
8,567 publications. The secondary searches produced 9 publications that
presented original data, included a comparison group in the study design and
involved sports-related concussion: 4 studies spoke to the potential of qEEG as a
diagnostic or management tool, while 5 studies addressed the potential of fMRI to
be used in the same capacity. The authors concluded that emerging technologies
and novel approaches that aid in sports concussion diagnosis and management
are being introduced at a rapid rate. Moreover, they stated that while some
technologies show promise, their clinical utility remains to be established.

Furthermore, the American Medical Society for Sports Medicine’s position
statement on “Concussion in sport” (Harmon et al, 2013) did not mention the use
of quantitative EEG/brain mapping as a management tool.
Hosokawa et al (2014) noted that several studies have reported the presence of EEG abnormalities or altered evoked potentials (EPs) during sepsis. However, the role of these tests in the diagnosis and prognostic assessment of sepsis-associated encephalopathy remains unclear. These researchers performed a systematic search for studies evaluating EEG and/or EPs in adult patients with sepsis-associated encephalopathy. The following outcomes were extracted: (i) incidence of EEG/EP abnormalities; (ii) diagnosis of sepsis-associated delirium or encephalopathy with EEG/EP; (iii) outcome. Among 1,976 citations, 17 articles met the inclusion criteria. The incidence of EEG abnormalities during sepsis ranged from 12 % to 100 % for background abnormality and 6 % to 12 % for presence of tri-phasic waves. Two studies found that epileptiform discharges and electrographic seizures were more common in critically ill patients with than without sepsis. In 1 study, EEG background abnormalities were related to the presence and the severity of encephalopathy. Background slowing or suppression and the presence of tri-phasic waves were also associated with higher mortality. A few studies demonstrated that quantitative EEG analysis and EP could show significant differences in patients with sepsis compared to controls; but their association with encephalopathy and outcome was not evaluated. The authors concluded that abnormalities in EEG and EPs are present in the majority of septic patients. They stated that there is some evidence to support EEG use in the detection and prognostication of sepsis-associated encephalopathy, but further clinical investigation is needed to confirm this suggestion.

CPT Codes / HCPCS Codes / ICD-9 Codes

CPT codes covered if selection criteria are met:

95961  Functional cortical and subcortical mapping by stimulation and/or recording of electrodes on brain surface, or of depth electrodes, to provoke seizures or identify vital brain structures; initial hour of attendance by a physician or other qualified health care professional

+ 95962  each additional hour of attendance by a physician or other qualified health care professional (List separately in addition to code for primary procedure)

Other CPT codes related to the CPB:

95812 - 95830  Electroencephalography

HCPCS code covered if selection criteria are met:

S8040  Topographic brain mapping

ICD-9 codes covered if selection criteria are met (not all-inclusive):

046.0 - 046.9  Slow virus infection of central nervous system
290.0 - 290.9  Senile and presenile organic psychotic conditions

294.10  Dementia in conditions classified elsewhere without behavioral disturbance

294.11  Dementia in conditions classified elsewhere with behavioral disturbance

294.8  Other persistent mental disorders due to conditions classified elsewhere

323.71 - 323.72  Toxic encephalitis, myelitis, and encephalomyelitis

345.00 - 345.91  Epilepsy and recurrent seizures

348.1  Anoxic brain damage

348.30 - 348.39  Encephalopathy, not elsewhere classified

349.82  Toxic encephalopathy

433.00 - 438.9  Occlusion and stenosis of precerebral arteries, occlusion of cerebral arteries, transient cerebral ischemia, acute, but ill-defined cerebrovascular disease, other and ill-defined cerebrovascular disease, and late effects of cerebrovascular disease

780.33  Post traumatic seizures

780.39  Other convulsions

984.0  Toxic effect of inorganic lead compounds

**ICD-9 codes not covered for indications listed in the CPB:**

291.0 - 291.9  Alcoholic induced mental disorders

292.0 - 292.9  Drug induced mental disorders

295.00 - 295.95  Schizophrenic disorders

296.00 - 296.99  Episodic mood disorders [bipolar disorder]

298.0  Depressive type psychosis

299.00 - 299.91  Pervasive developmental disorders

300.01  Panic disorder without agoraphobia
300.4  Dysthymic disorder
303.00 - 303.93  Alcohol dependence syndrome
304.00 - 305.93  Drug dependence and nondependent abuse of drugs
307.41  Transient disorder of initiating or maintaining sleep
307.42  Persistent disorder of initiating or maintaining sleep
307.49  Other specific disorders of sleep of nonorganic origin
310.2  Post-concussion syndrome
311  Depressive disorder, not elsewhere classified
314.00 - 314.9  Hyperkinetic syndrome of childhood
315.00 - 315.9  Specific delays in development
327.00 - 327.09  Organic disorder of initiating or maintaining sleep [organic insomnia]
332.0 - 332.1  Parkinson's disease
388.30 - 388.32  Tinnitus
729.1  Myalgia and myositis, unspecified [fibromyalgia]
768.70 - 768.73  Hypoxic-ischemic encephalopathy (HIE)
780.51  Insomnia with sleep apnea, unspecified
780.52  Insomnia, unspecified
784.61  Alexia and dyslexia
850.00 - 854.19  Intracranial injury, excluding those with skull fracture
959.01  Head injury, unspecified
E917.0  Striking against or struck accidentally by objects or persons in sports without subsequent fall
V40.0  Problems with learning
V69.5  Behavioral insomnia of childhood
The above policy is based on the following references:


37. Krull KR. Attention deficit hyperactivity disorder in children and adolescents: Clinical features and evaluation. Last reviewed December 2013. UpToDate Inc., Waltham, MA.