Clinical Policy Bulletin:
Quantitative EEG (Brain Mapping)

Number: 0221

Policy

I. Aetna considers the use of quantitative EEG (brain mapping), also known by the acronym 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
- Depression
Drug abuse
Fibromyalgia
Hypoxic ischemic encephalopathy
Insomnia
Learning disability
Mild or moderate head injury
Panic disorder
Post-concussion syndrome
Predicting response to psychotropic medication
Schizophrenia
Tinnitus.

See also CPB 480 - 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.

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.

Crumbley and associates (2005) examined the use of quantitative EEG in predicting response to psychotropic medication. The clinical outcomes of two 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 = 1498) 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, 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.

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: "There 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."

Marzano and colleagues (2008) stated that in the last two 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 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 (PD) 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 PD 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 PD.

CPT Codes / HCPCS Codes / ICD-9 Codes

CPT codes covered if selection criteria are met:

95961
+ 95962

Other CPT codes related to the CPB:

95812 - 95830

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
997.00 - 997.09  Nervous system complications

**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
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
The above policy is based on the following references:


