Clinical Policy Bulletin:
Evoked Potential Studies

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Policy

I. Aetna considers evoked potential studies medically necessary for the following indications:

   A. Somatosensory evoked potentials (SEPs, SSEPs) or dermatosensory evoked potentials (DSEPs) are considered medically necessary for any of the following indications:

       1. To assess any decline which may warrant emergent surgery in unconscious spinal cord injury persons who show specific structural damage to the somatosensory system, and who are candidates for emergency spinal cord surgery; or
       2. To evaluate acute anoxic encephalopathy (within 3 days of the anoxic event); or
       3. To evaluate persons with suspected brain death; or
       4. To identify clinically silent brain lesions in multiple sclerosis suspects in order to establish the diagnosis, where multiple sclerosis is suspected due to presence of suggestive neurologic symptoms plus one or more other objective findings (brain plaques on MRI, clinical lesions by history and physical examination, and/or positive CSF (determined by oligoclonal bands detected by established methods (isoelectric focusing) different from any such bands in serum, or by an increased IgG index); or
       5. To localize the cause of a central nervous system deficit seen on exam, but not explained by lesions seen on CT or MRI; or
       6. To manage persons with spinocerebellar degeneration (e.g., Friedreich's ataxia, olivopontocerebellar (OPC) degeneration); or
       7. Unexplained myelopathy, or
       8. Intraoperative SSEPs under certain conditions (see I. B., below).

SEPs and DSEPs are considered experimental and investigational for all other indications because their effectiveness for indications other than the ones listed above has not been established.
B. **Intraoperative somatosensory evoked potentials (SSEPs)** performed either alone, or in combination with motor evoked potentials (MEPs) are considered medically necessary for monitoring the integrity of the spinal cord to detect adverse changes before they become irreversible during spinal, intracranial, orthopedic, or vascular procedures, when the following criteria are met:

1. A specially trained physician or a certified professional practicing within the scope of their license, who is not a member of the surgical team contemporaneously interprets the intraoperative evoked potentials during the operation; and
2. The evoked potential monitoring is performed in the operating room by dedicated trained technician; and
3. The clinician who performs the interpretation is monitoring no more than 3 surgical procedures at the same time; and
4. The clinician who performs the interpretation may do so remotely, but must provide direct, immediate communication of intraoperative evoked potential results to the technician and surgeon during the operation.

Intra-operative SEP monitoring, with or without MEPs, may be appropriate for the following types of surgery (not an all-inclusive list):

1. Spinal Surgeries:
   a. Correction of scoliosis or deformity of the spinal cord involving traction on the cord
   b. Decompression of the spinal cord where function of the spinal cord is at risk
   c. Removal of spinal cord tumors
   d. Surgery as a result of traumatic injury to the spinal cord
   e. Surgery for arteriovenous (AV) malformation of the spinal cord

2. Intracranial Surgeries:
   a. Chiari malformation surgery
   b. Correction of cerebral vascular aneurysms
   c. Deep brain stimulation
   d. Endolymphatic shunt for Meniere's disease
   e. Microvascular decompression of cranial nerves (e.g., optic, trigeminal, facial, auditory nerves)
   f. Oval or round window graft
   g. Removal of cavernous sinus tumors
   h. Removal of tumors that affect cranial nerves
   i. Resection of brain tissue close to the primary motor cortex and requiring brain mapping
   j. Resection of epileptogenic brain tissue or tumor
   k. Surgery as a result of traumatic injury to the brain
   l. Surgery for intracranial AV malformations
3. Vascular Surgeries:

   a. Arteriography, during which there is a test occlusion of the carotid artery
   b. Circulatory arrest with hypothermia (does not include surgeries performed under circulatory bypass such as CABG, and ventricular aneurysms)
   c. Distal aortic procedures, where there is risk of ischemia to the spinal cord
   d. Surgery of the aortic arch, its branch vessels, or thoracic aorta, including carotid artery surgery, when there is risk of cerebral ischemia.

Intra-operative SSEPs with or without MEPs are considered experimental and investigational for all other indications (e.g., scapula-thoracic fusion surgery) because their effectiveness for indications other than the ones listed above has not been established.

**Note:** Depending on the clinical condition being investigated, it may be medically necessary to test several nerves in one extremity and compare them with the opposite limb.

**Note:** Intra-operative evoked potential studies have no proven value for lumbar surgery below (distal to) the end of the spinal cord; the spinal cord ends at L1-L2.

**Note:** Post-operative SEP or MEP monitoring is not considered medically necessary for individuals who have undergone intra-operative SEP or MEP monitoring.

**Note:** The NIM-Spine System received 510(k) clearance from the Food and Drug Administration (FDA) in June 2003. It offers 2 types of monitoring modalities: (i) electromyography, and (ii) MEP.

**Note on documentation requirements:** The physician’s SEP report should note which nerves were tested, latencies at various testing points, and an evaluation of whether the resulting values are normal or abnormal. See appendix for additional details on documentation requirements.

II. **Visual evoked potentials (VEPs)** are considered medically necessary for any of the following indications:

   A. To diagnose and monitor multiple sclerosis (acute or chronic phases); or
   B. To evaluate signs and symptoms of visual loss in persons who are unable to communicate (e.g., unresponsive persons, etc); or
   C. To identify persons at increased risk for developing clinically definite multiple sclerosis (CDMS); or
   D. To localize the cause of a visual field defect, not explained by lesions seen on CT or MRI, metabolic disorders, or infectious diseases.
Standard or automated VEPs are considered experimental and investigational for routine screening of infants and other persons; evidence-based guidelines from leading medical professional organizations and public health agencies have not recommended VEP screening of infants. VEPs are considered experimental and investigational for all other indications because their effectiveness for indications other than the ones listed above has not been established.

III. Brain stem auditory evoked response (BAER)** is considered medically necessary for any of the following:

A. For cerebral vascular surgery; or
B. For Chiari malformation surgery; or
C. For intra-operative monitoring during microvascular decompression of cranial nerve when decompression is performed via the intracranial posterior fossa approach; or
D. For intra-operative monitoring during resection of chordoma, odontoidectomy, decompression of tumor from anterior brainstem/high spinal cord; or
E. To assess brain death or profound metabolic coma in selected cases where diagnosis or outcome is unclear from standard tests (e.g., EEG); or
F. To assess recovery of brainstem function after a lesion compressing the brainstem has been surgically removed; or
G. To diagnose and monitor demyelinating and degenerative diseases affecting the brain stem (e.g., central pontine myelinolysis, olivopontocerebellar (OPC) degeneration, etc.); or
H. To diagnose post-meningitic deafness in children; or
I. To diagnose suspected acoustic neuroma; or
J. To evaluate infants and children who have suspected hearing loss that can not be effectively measured or monitored through audiometry; or
K. To localize the cause of a central nervous system deficit seen on examination, but not explained by CT or MRI; or
L. To screen infants and children under age 5 for hearing loss. Note: For purposes of neonatal screening, only limited auditory evoked potentials or limited evoked otoacoustic emissions are considered medically necessary. Neonates who fail this screening test are then referred for comprehensive auditory evoked response testing or comprehensive otoacoustic emissions. Comprehensive auditory evoked response testing and comprehensive otoacoustic emissions are considered experimental and investigational for neonatal screening because there is a lack of evidence of the value of comprehensive testing over the limited auditory evoked potentials or limited otoacoustic emissions for this indication.

BAERs are considered experimental and investigational for all other indications because their effectiveness for indications other than the ones listed above has not been established.

** Also known as auditory evoked potentials (AEPs), brainstem auditory evoked potentials (BAEP), BERA, BSER, and BSRA.
Aetna considers the following studies and indications to be experimental and investigational because they have not been proven necessary to aid in diagnosis or alter the management of the member:

Auditory evoked potentials to determine gestational age or conceptual age in pre-term neonates;
BAERs as a test to identify persons at increased risk for developing clinically definite multiple sclerosis (CDMS);
BAERs for syringomyelia and syringobulbia;
Cognitive evoked potentials (also known as auditory or visual P300 or P3 cognitive evoked potentials) to diagnose cognitive dysfunction in persons with dementia (e.g., Alzheimer's disease and Parkinson's disease) or to identify the etiology of depression in persons with chronic demyelinating disease;
Cortical auditory evoked response (CAER) for the diagnosis of depression, attention deficit/hyperactivity disorder, autism, or any other indication;
Event-related potentials for the diagnosis of attention deficit/hyperactivity disorder (see CPB 0426 - Attention Deficit/Hyperactivity Disorder) or post-traumatic stress disorder, or assessment of amyotrophic lateral sclerosis, brain injury, or evaluation of comatose persons;
Evoked potential studies for Kennedy's syndrome/disease;
Gustatory evoked potentials for diagnosing taste disorders (see CPB 0390 - Smell and Taste Disorders: Diagnosis);
Intraoperative BAER during stapedectomy, tympanoplasty and ossicle reconstruction
Intraoperative MEP during implantation of a spinal cord stimulator
Intraoperative saphenous nerve somatosensory evoked potential for monitoring the femoral nerve during transposas lumbar lateral interbody fusion
Intraoperative SSEP of the facial nerve for submandibular gland excision or parotid gland surgery, during hip replacement surgery, implantation of a spinal cord stimulator, off-pump coronary artery bypass surgery, and for thyroid surgery and parathyroid surgery (because they have not been proven necessary to aid in diagnosis or alter the management of individual undergoing surgical treatment)
Intraoperative visual evoked potentials (e.g., for pituitary surgery, during intracranial surgery for arterio-venous malformation);
Motor evoked potentials other than for intraoperative use with SSEPs (e.g., facial MEPs during cerebellopontine angle and skull base tumor surgery);
SEPs for radiculopathies and peripheral nerve lesions where standard nerve conduction velocity studies are diagnostic (see CPB 0502 - Nerve Conduction Velocity Studies);
SEPs for the diagnosis of carpal tunnel syndrome/ulnar nerve entrapment;
SEPs in conscious persons with severe spinal cord or head injuries (the standard neurologic examination is the most direct way to evaluate any deficits);
SEPs in diagnosis of cervical spondylotic myeloradiculopathy;
SEPs in the diagnosis of thoracic outlet syndrome;
SEPs in the diagnosis or management of acquired metabolic disorders (e.g., lead toxicity, B12 deficiency);
SEPs in the diagnosis or management of amyotrophic lateral sclerosis (ALS);
SEPs for pectus excavatum surgery;
SEPs for prostate surgery;
VEPs for detecting amnestic mild cognitive impairment
VEPs for syringomyelia, syringobulbia, and evaluation of vigabatrin (Sabril)-
associated retinal toxicity, screening Plaquenil (hydroxychloroquine) toxicity, as
prognostic tests in neonates with perinatal asphyxia and hypoxic-ischemic
encephalopathy;
Vestibular evoked myogenic potentials (VEMP) (e.g., for differentiation of
Meniere disease from vestibular migraine)

Background

Evoked potentials measure conduction velocities of sensory pathways in the central
nervous system using computerized averaging techniques. Three types of evoked
potentials are routinely performed: (i) somatosensory; (ii) visual; and (iii) brainstem
auditory. In each of these tests a peripheral sense organ is electrically stimulated and
conduction velocities are recorded for central somatosensory pathways located in the
posterior columns of the spinal cord, brain stem, and thalamus, and the primary
sensory cortex located in the parietal lobes.

For patients with symptomatic nerve root compression, the accurate identification of the
particular nerve root(s) that are causing symptoms is an essential prerequisite to
surgical intervention. The history and physical examination may be helpful in identifying
the particular peripheral nerve root that is affected, but these are often inconclusive.
Patients often have difficulty defining the distribution of pain or sensory symptoms, and
the physical examination may be completely normal even in patients with severe pain.
Imaging studies may be helpful, but are frequently normal or reveal abnormalities of
uncertain clinical relevance. Moreover, because structural abnormalities are commonly
seen in imaging studies of normal asymptomatic middle-aged or elderly subjects, it is
difficult to determine whether any such abnormalities that are identified in pain patients
are related to their symptoms. In addition, imaging studies may show equivocal
changes or anatomic abnormalities at multiple levels, making it impossible to determine
which nerve root is responsible for the patient’s symptoms. In these circumstances,
evoked potentials may be used to measure nerve root function and thereby more
accurately identify the precise nerve roots responsible for the patient’s symptoms.

Somatosensory evoked potentials (SEPs or SSEPs) (also known as cerebral sensory
evoked potentials) augment the sensory examination and are most useful in assessing
the spinal nerve roots, spinal cord, or brain stem for evidence of delayed nerve
conduction. Dermatomal somatosensory evoked potentials (DSEPs) are elicited by
stimulating the skin “signature” areas of specific nerve roots. Both techniques involve
production and recording of small electrophysiological responses of the central nervous
system that follow sequential electrical stimulation of peripheral nerves. These small
electrophysiological responses are extracted from the background noise of
electroencephalography (EEG), usually by signal averaging techniques. Delays in
signal propagation suggest lesions of the central sensory pathways. Although
controversial, evoked potentials have been used to assess the prognosis of children
with spinal cord lesions, brain malformations, and neurodegenerative diseases, as well
as young children who are at risk for brain injury, such as preterm infants.
Somatosensory evoked potentials measurements have been used to predict outcome
in spinal cord injury; however, signal changes on MRI actually may be more useful in
determining the severity of injury. Hemorrhage within the spinal cord is readily
identified on MRI, and such hemorrhage is predictive of injury severity. Intra-operative SSEP measurements are useful in complex neurologic, orthopedic, and vascular surgical procedures as a means of gauging nerve injury during surgery (e.g., resection of cord tumors).

Somatosensory evoked potentials are altered by conditions that affect the somatosensory pathways, including both focal lesions (such as strokes, tumors, cervical spondylosis, syringomyelia) and diffuse diseases (such as hereditary systemic neurologic degeneration, subacute combined degeneration, and vitamin E deficiencies).

Somatosensory evoked potentials may detect clinically silent brain lesions in multiple sclerosis suspects. Although SEP abnormalities alone are insufficient to establish the diagnosis of multiple sclerosis, the diagnosis can be established when there is also other objective findings (brain plaques on MRI, clinical lesions by history and physical examination, and/or positive CSF (determined by oligoclonal bands detected by established methods (isoelectric focusing) different from any such bands in serum, or by an increased IgG index)).

Fifty to 60 % of multiple sclerosis patients have other concurrent demyelinating lesions that may not be clinically evident, and SSEP may be helpful in documenting these abnormalities. Somatosensory evoked potentials abnormalities are also produced by other diseases affecting myelin (adrenoleukodystrophy and adrenomyelo-neuropathy, metachromatic leukodystrophy, Pelizaeus-Merzbacher disease). In adrenoleukodystrophy and adrenomyeloneuropathy, SSEP abnormalities may be present in asymptomatic heterozygotes. Abnormally large amplitude SEPs, reflecting enhanced cortical excitability, are seen in progressive myoclonus epilepsy, in some patients with photosensitive epilepsy, and in late infantile ceroid lipofuscinosis.

Studies have demonstrated a statistically significant association between abnormal visual evoked potentials (VEPs) and an increased risk of developing clinically definite multiple sclerosis (CDMS). In these studies, patients with suspected MS were 2.5 to 9 times as likely to develop CDMS as patients with normal VEPs. Visual evoked potentials sensitivities ranged from 25 % to 83 %. Visual evoked potentials improved the ability to predict which MS suspects will develop CDMS by as much as 29 %.

Measurement of visual evoked responses (VERs) is the primary means of objectively testing vision in infants and young children suspected of having disorders of the visual system, where the child is too young to report differences in color vision or to undergo assessment of visual fields and visual acuity. A flashing stroboscope or an alternating checkerboard pattern is presented and the wave patterns are recorded. In an infant, vision may be reliably tested using a flashing light during quiet sleep. Lesions affecting the visual pathways can be localized by noting the presence of decreased amplitudes or increased latencies of VERs, and by determining whether VER abnormalities involve one or both eyes. Visual evoked responses are also useful for testing vision in other persons who are not able to communicate.

Brain stem auditory evoked responses (BAERs) are electrical potentials that are produced in response to an auditory stimulus and are recorded from disk electrodes attached to the scalp. Depending on the amount of time elapsed between the "click" stimulus and the auditory evoked response, potentials are classified as early (0 to 10 msec), middle (11 to 50 msec), or late (51 to 500 msec). The early potentials reflect electrical activity at the cochlea, 8th cranial nerve, and brain stem levels; the latter
potentials reflect cortical activity. In order to separate evoked potentials from background noise, a computer averages the auditory evoked responses to 1,000 to 2,000 clicks. Early evoked responses may be analyzed to estimate the magnitude of hearing loss and to differentiate among cochlea, 8th nerve, and brainstem lesions.

The clinical utility of BAER over standard auditory testing is due to several of BAER's characteristics: (i) BAER's resistance to alteration by systemic metabolic abnormalities, medications or pronounced changes in the state of consciousness of the patient; and (ii) the close association of BAER waveform abnormalities to underlying structural pathology. Brain stem auditory evoked responses have been proven effective for differentiating conductive from sensory hearing loss, for detecting tumors and other disease states affecting central auditory pathways (e.g., acoustic neuromas), and for noninvasively detecting hearing loss in patients who can not cooperate with subjective auditory testing (e.g., infants, comatose patients). BAER is the test of choice to assess hearing in infants and young children. It is most useful for following asphyxia, hyperbilirubinemia, intracranial hemorrhage, or meningitis or for assessing an infant who has trisomy. BAER also is useful in the assessment of multiple sclerosis or other demyelinating conditions, coma, or hysteria. Audiometric analysis using multiple sound frequencies is usually preferred over BAER for testing hearing in cooperative patients who are able to report when sounds are heard. Evidence is insufficient at this time to recommend BAER as a useful test to identify patients at increased risk for developing CDMS.

Studies of cognitive evoked potentials (also known as the P300 or P3 cognitive evoked potentials) have been used in research settings to correlate changes in cognitive evoked potentials with clinical changes in cognitive function in patients with dementia (e.g., Alzheimer's disease and Parkinson's disease) and identify the etiology of depression in patients with chronic demyelinating disease. However, there is insufficient evidence regarding the effectiveness of cognitive evoked potential studies in diagnosing or rendering treatment decisions that would affect health outcomes. Furthermore, there is a lack of studies comparing cognitive evoked potential studies with standard neuropsychiatric and psychometric tests used in diagnosing cognitive dysfunction.

The American Academy of Pediatrics (AAP) Task Force on Newborn and Infant Hearing and the Joint Committee on Infant Hearing (JCIH) endorse the implementation of universal newborn hearing screening. Screening should be conducted before discharge from the hospital whenever possible. Physicians should provide recommended hearing screening, not only during early infancy but also through early childhood for those children at risk for hearing loss (e.g., history of trauma, meningitis) and for those demonstrating clinical signs of possible hearing loss.

The U.S. Preventive Services Task Force (USPSTF) recommends screening for hearing loss in all newborn infants. All infants should be screened before 1 month of age. Those infants who do not pass the newborn screening should undergo audiologic and medical evaluation before 3 months of age for confirmatory testing. Because of the elevated risk of hearing loss in infants with risk indicators (e.g., neonatal intensive care unit admission for 2 or more days; syndromes associated with hearing loss, such as Usher syndrome and Waardenburg syndrome; family history of hereditary childhood hearing loss; craniofacial abnormalities; and congenital infections such as cytomegalovirus, toxoplasmosis, bacterial meningitis, syphilis, herpes, and rubella), an
expert panel recommends that these children undergo periodic monitoring for 3 years. The USPSTF found good evidence that newborn hearing screening leads to earlier identification and treatment of infants with hearing loss and improves language outcomes. However, additional studies detailing the correlation between childhood language scores and functional outcomes (e.g., school attainment and social functioning) are needed.

Two types of tests are commonly used to screen for congenital hearing loss: (i) otoacoustic emissions (OAEs) and (ii) auditory brainstem response (ABR) (Helfand et al, 2001). Otoacoustic emissions testing evaluates the integrity of the inner ear (cochlea). In response to noise, vibrations of the hair cells in a healthy inner ear generate electrical responses, known as otoacoustic emissions. The absence of OAEs indicates that the inner ear is not responding appropriately to sound. Transient evoked otoacoustic emissions (TEOAEs) are generated in response to wide-band clicks, while distortion product otoacoustic emissions (DPOAE) are a response to tones. Both stimuli are presented via a light-weight ear canal probe. A microphone picks up the signal, and multiple responses are averaged to get a specific repeatable waveform. Otoacoustic emissions are used in screening and diagnosis of hearing impairments in infants, and in young children and patients with cognitive impairments (e.g., mental retardation, dementia) who are unable to respond reliably to standard hearing tests. Otoacoustic emissions are also useful for evaluating patients with tinnitus, suspected malingering, and for monitoring cochlear damage from ototoxic drugs.

The ABR is an electrophysiological response generated in the brainstem in response to auditory signals and composed of either clicks or tones. The stimulus is delivered via earphones or an inserted ear probe, and scalp electrodes pick up the signal. Auditory brainstem response evaluates the integrity of the peripheral auditory system and the auditory nerve pathways up to the brainstem and is able to identify infants with normal cochlear function but abnormal 8th-nerve function (auditory neuropathy). For purposes of neonatal screening, a limited ABR is performed in the nursery using a significantly low intensity level (35 to 40 dB) to rule out marked hearing loss (Schwartz and Schwartz, 1990; Scott and Bhattacharyya, 2002). If testing at this level fails to elicit a response, the infant is referred to an audiolgic laboratory for a comprehensive ABR, involving testing at many different intensity levels.

Typically, screening programs use a 2-stage screening approach (either OAE repeated twice, OAE followed by ABR, or ABR repeated twice). Criteria for defining a "pass" or "fail" on the initial screening test vary widely. Comprehensive (diagnostic) OAEs or ABRs are used to diagnose hearing impairments identified by limited (screening) tests.

Auditory brainstem response and OAE have limitations that affect their accuracy in certain patients. Both require a sleeping or quiet child. Middle-ear effusion or debris in the external canal can compromise the accuracy of these tests. Otoacoustic emissions and ABR test the peripheral auditory system and 8th nerve pathway to the brainstem, respectively. They are not designed to identify infants with central hearing deficits. Therefore, infants with risk factors for central hearing deficits, particularly those who have congenital Cytomegalovirus infection or prolonged severe hypoxia at birth, may pass their newborn hearing screens with either OAE or ABR, but develop profound hearing loss in early infancy.

The newer generation of automated screeners are easy to use and do not require highly trained staff. However, equipping hospitals with equipment and sufficient staff
can be costly, the staff must be trained to understand the limitations of the techniques, and ongoing quality control is essential to achieve accurate, consistent test results. The importance of technique is illustrated by the results of multicenter studies of universal screening, in which the rates of false positive and technically inadequate examinations varied 10-fold among sites.

There are differences between the guidelines with respect to the screening technology that is endorsed. The Joint Committee on Infant Hearing recommends that all infants have access to screening using a physiologic measure (either otoacoustic emissions [TEOAE or DPOAE] and/or ABR). The AAP states that although additional research is necessary to determine which screening test is ideal, EOAE and/or ABR are presently the screening methods of choice. The AAP defers recommending a preferred screening test. The USPSTF recommends a 1- or 2-step validated protocol, stating that OAEs followed by ABR in those who failed the first test is a frequently used protocol. Well-maintained equipment, thoroughly trained staff, and quality control programs are also recommended to avoid false-positive tests.

Cortical auditory evoked responses (CAERs) measure the later-occurring auditory evoked potentials reflecting cortical activity in response to an auditory stimulus (UBC, 2005). Cortical auditory evoked responses have a long latency, compared to the short latency auditory evoked responses; they have been used in clinical research to evaluate the timing, sequence, strength, and anatomic location of brain processes involved with the perception of sounds. Current research underway concerns the use of CAERs to understand the brain processes underlying basic hearing percepts such as loudness, pitch, and localisation, as well as those processes involved with speech perception (UBC, 2005).

Vestibular evoked myogenic potentials (VEMP), also known as click evoked neurogenic vestibular potentials, are presumed to originate in the saccule. They are recorded from surface electrodes over the sternocleidomastoid muscles, and can be activated by means of brief, high-intensity acoustic stimuli. Papanastasiou et al (2003) stated that VEMP testing is a possible new diagnostic technique that may be specific for the vestibular pathway. It has potential use in patients with symptoms of dizziness, subclinical symptoms in multiple sclerosis, and in disorders specific for the vestibular nerve. There is a lack of reliable evidence from well controlled, prospective studies demonstrating that VEMP testing alters management such that clinical outcomes are improved. Current evidence-based guidelines on the management of neurological disorders from leading medical professional organizations have not incorporated VEMP testing in diagnostic and treatment algorithms. The American Academy of Neurology considered VEMP as an investigational technique (Fife et al, 2000). Guidelines prepared for the State of Colorado (DLE, 2006) state that VEMP "is currently a research tool and is not recommended for routine clinical use." In a review of the literature, Rauch (2006) states that VEMP holds great promise for diagnosing and monitoring Ménière's disease and some other neurotologic disorders. Rauch notes, however, that the methods, equipment, and applications for vestibular evoked myogenic potential testing are not yet standardized, and many aspects of vestibular evoked myogenic potential and its use have not yet been adequately studied or described.

Akkuzu et al (2006) examined the role of VEMP in benign paroxysmal positional vertigo (BPPV) and Meniere's disease, and ascertained if this type of testing is valuable for
assessing the vestibular system. The 62 participants included 17 healthy controls and 45 other subjects selected from patients who presented with the complaint of vertigo (25 diagnosed with BPPV and 20 diagnosed with Meniere's disease). Vestibular evoked myogenic potentials were recorded in all subjects and findings in each patient group were compared with control findings. Vestibular evoked myogenic potentials for the 30 affected ears in the 25 BPPV patients revealed prolonged latencies in 8 ears and decreased amplitude in 1 ear (9 abnormal ears; 30% of total). The recordings for the 20 affected ears in the Meniere's disease patients revealed 4 ears with no response, 6 ears with prolonged latencies (10 abnormal ears; 50% of total). Only 2 (5.9%) of the 34 control ears had abnormal VEMP. The rate of VEMP abnormalities in the control ears was significantly lower than the corresponding rates in the affected BPPV ears and the affected Meniere's ears that were studied (p = 0.012 and p < 0.001, respectively). The results suggested that testing of VEMP is a promising method for diagnosing and following patients with BPPV paroxysmal positional vertigo and Meniere's disease.

Brantberg et al (2007) studied VEMP in response to sound stimulation (500 Hz tone burst, 129 dB SPL) in 1,000 consecutive patients. Vestibular evoked myogenic potentials from the ear with the larger amplitude were evaluated based on the assumption that the majority of the tested patients probably had normal vestibular function in that ear. Patients with known bilateral conductive hearing loss, with known bilateral vestibular disease and those with Tullio phenomenon were not included in the evaluation. It was found that there was an age-related decrease in VEMP amplitude and an increase in VEMP latency that appeared to be rather constant throughout the whole age span. Vestibular evoked myogenic potentials data were also compared to an additional group of 10 patients with Tullio phenomenon. Although these 10 patients did have rather large VEMP, equally large VEMP amplitudes were observed in a proportion of unaffected subjects of a similar age group. Thus, the findings of a large VEMP amplitude in response to a high-intensity sound stimulation is not, per se, distinctive for a significant vestibular hypersensitivity to sounds.

Muyts et al (2007) provided an overview of vestibular function testing and highlights the new techniques that have emerged during the past 5 years. Since the introduction of video-oculography as an alternative to electro-oculography for the assessment of vestibular-induced eye movements, the investigation of the utricle has become a part of vestibular function testing, using unilateral centrifugation. Vestibular evoked myogenic potentials have become an important test for assessing saccular function, although further standardization and methodological issues remain to be clarified. Galvanic stimulation of the labyrinth also is an evolving test that may become useful diagnostically. The authors concluded that a basic vestibular function testing battery that includes ocular motor tests, caloric testing, positional testing, and earth-vertical axis rotational testing focuses on the horizontal semicircular canal. Newer methods to investigate the otolith organs are being developed. These new tests, when combined with standard testing, will provide a more comprehensive assessment of the complex vestibular organ.

Magnetic stimulation of the brain and spine elicits so-called motor evoked potentials (MEPs) (Goetz, 2005). The latency of the motor responses can be measured, and central conduction time can be estimated by comparing the latency of the responses elicited by cerebral and spinal stimulation. Abnormalities have been described in patients with a variety of central disorders including multiple sclerosis, amyotrophic lateral sclerosis, stroke, and certain degenerative disorders. An assessment by the
McGill University Health Centre on use of intraoperative neurophysiological monitoring during spinal surgery stated that there is sufficient evidence to support the conclusion that intraoperative spinal monitoring using SSEPs and MEPs during surgical procedures that involve risk of spinal cord injury is an effective procedure that is capable of substantially diminishing this risk (Erickson et al, 2005). The report explained that intra-operative spinal cord injury during spinal surgery generally compromises both motor and somatosensory pathways; therefore the use of both of these independent techniques in parallel has been proposed and is seen as a safeguard should one of the monitoring techniques fail. Combination of SSEP monitoring with MEP monitoring is also proposed to reduce false-positive results, and eliminate the need for the wake-up test. The assessment identified 11 studies, all case series, of the combined use of SSEPs and MEPs in neurophysiological monitoring during spinal surgery. The assessment found that, in several reports, combined SSEP and MEP monitoring was shown to have greater sensitivity than SSEP alone. The report also noted that the addition of MEP monitoring where SSEP monitoring is already being performed is considered to be relatively straightforward, adding little to the overall effort and resources employed in intraoperative neurophysiological monitoring.

A study by Schwarz, et al. (2007) illustrated the advantage of intraoperative monitoring of spinal cord motor tracts directly by recording motor evoked potentials in addition to somatosensory evoked potentials. Investigators reviewed the intraoperative neurophysiological monitoring records of 1121 consecutive patients (834 female and 287 male) with adolescent idiopathic scoliosis (mean age, 13.9 years) treated between 2000 and 2004 at four pediatric spine centers. The same group of experienced surgical neurophysiologists monitored spinal cord function in all patients with use of a standardized multimodality technique with the patient under total intravenous anesthesia. A relevant neurophysiological change (an alert) was defined as a reduction in amplitude (unilateral or bilateral) of at least 50% for somatosensory evoked potentials and at least 65% for transcranial electric motor evoked potentials compared with baseline. The investigators reported that 38 (3.4%) of the 1121 patients had recordings that met the criteria for a relevant signal change (i.e., an alert). Of those 38 patients, 17 showed suppression of the amplitude of motor evoked potentials in excess of 65% without any evidence of changes in somatosensory evoked potentials. In nine of the 38 patients, the signal change was related to hypotension and was corrected with augmentation of the blood pressure. The remaining 29 patients had an alert that was related directly to a surgical maneuver. Three alerts occurred following segmental vessel clamping, and the remaining 26 were related to posterior instrumentation and correction. Nine (35%) of these 26 with an instrumentation-related alert, or 0.8% of the cohort, awoke with a transient motor and/or sensory deficit. Seven of these nine patients presented solely with a motor deficit, which was detected by intraoperative monitoring of motor evoked potentials in all cases, and two patients had only sensory symptoms. The investigators reported that somatosensory evoked potential monitoring failed to identify a motor deficit in four of the seven patients with a confirmed motor deficit. Furthermore, when changes in somatosensory evoked potentials occurred, they lagged behind the changes in transcranial electric motor evoked potentials by an average of approximately five minutes. With an appropriate response to the alert, the motor or sensory deficit resolved in all nine patients within one to 90 days.
The clinical utility of MEPs outside of the operative setting, however, is unclear and at the present time the magnetic stimulation of central structures is regarded as investigational (Goetz, 2003; Miller, 2005).

In a prospective consecutive case series study, Lee et al (2009) evaluated the side effects of microvascular decompression (MVD) on hearing and described the main intra-operative ABR changes. The study included 22 patients who underwent MVD with monitoring of ABRs. The latency prolongation and wave loss were analyzed at each surgical step, which were decided arbitrarily. Patients were divided into 4 groups depending on degree of change of wave V. Group 1 consisted of minimal change, whereas group 4 was permanent loss of wave V. Hearing changes were evaluated in 20 patients in the 4 groups who were available for post-operative hearing results. Loss of wave I, III, and V occurred with 6 %, 13 %, and 9 % of surgical actions, respectively. Wave III disappearance was identified as the earliest and most sensitive sign and was usually preceded by the disappearance of wave V. The greatest prolongation of wave V at more than 1.0 ms developed statistically significant sensorineural hearing loss in the range of 10 dB. One patient in group 4 experienced deafness. The authors concluded that in addition to the significant delay of wave V, useful recognition of early changes of wave III is possible and enables a change of microsurgical maneuvers to favor ABR recovery.

Polo and Fischer (2009) stated that BAEP monitoring is a useful tool to decrease the danger of hearing loss during pontocerebellar angle surgery, particularly in MVD. Critical complications arising during MVD surgery are the stretching of the VIII nerve -- the main cause of hearing loss -- labyrinthine artery manipulation, direct trauma with instruments, or a nearby coagulation, and at end of the surgery necrocompression of the cochlear nerve by the prosthesis positioned between the conflicting vessel(s) and the VIIth-VIIIth nerve complex. All these dangers warrant the use of BAEP monitoring during the surgical team's training period. Based on delay in latency of peak V, these investigators established warning thresholds that can provide useful feedback to the surgeon to modify the surgical strategy: the initial signal at 0.4 ms is considered the safety limit. A second signal threshold at 0.6 ms (warning signal for risk) corresponds to the group of patients without resultant hearing loss. The third threshold characterized by the delay of peak V is at 1 ms (warning signal for a potentially critical situation). BAEP monitoring provides the surgeon with information on the functional state of the auditory pathways and should help avoid or correct maneuvers that can harm hearing function. BAEP monitoring during VIIth-VIIIth complex surgery, particularly in MVD of facial nerves for hemifacial spasm (HFS) is very useful during the learning period.

Huang and colleagues (2009) determined the reliability of (i) intra-operative monitoring by stimulated electromyography (EMG) of the facial nerve to predict the completeness of MVD for HFS, and (ii) BAEP to predict post-operative hearing disturbance. These investigators conducted a prospective study of 36 patients who received MVD for HFS. They confirmed the disappearance of an abnormal muscle response in the facial nerve EMG to predict the completeness of MVD, and performed BAEP monitoring to predict post-operative hearing disturbance. The sensitivity, specificity and accuracy of facial nerve EMG and BAEP monitoring were evaluated. The sensitivity, specificity and accuracy of facial nerve EMG were 0.97, 1.0 and 0.97, respectively, and that for BAEP monitoring were 1.0, 0.94 and 0.94, respectively. There was 1 false-positive result for
facial nerve EMG, and 2 false-positive results for BAEP monitoring. No false-negative result was encountered for either EMG or BAEP monitoring. Facial nerve EMG correctly predicted whether MVD was successful in 35 out of 36 patients, and BAEP correctly predicted whether there was post-operative hearing disturbance in 34 out of 36 patients. The authors concluded that intra-operative facial nerve EMG provides a real-time indicator of successful MVD during an operation while BAEP monitoring may provide an early warning of hearing disturbance after MVD.

In a systematic review, Fehlings et al (2010) examined if intra-operative monitoring (IOM) is able to sensitively and specifically detect intra-operative neurological injury during spine surgery and to assess whether IOM results in improved outcomes for patients during these procedures. Two independent reviewers assessed the level of evidence quality using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) criteria, and disagreements were resolved by consensus. A total of 103 articles were initially screened and 32 ultimately met the pre-determined inclusion criteria. These researchers determined that there is a high level of evidence that multi-modal (SSEP and MEP) IOM is sensitive and specific for detecting intra-operative neurological injury during spine surgery. On the other hand, there is very low evidence from the literature that uni-modal SSEPs or MEPs are valid diagnostic tests for measuring intra-operative neurological injury. There is a low level of evidence that IOM reduces the rate of new or worsened peri-operative neurological deficits (a grade of "low" means that further research is very likely to have an important impact on the confidence in the estimate of effect and is likely to change the estimate). There is very low evidence that an intra-operative response to a neuromonitoring alert reduces the rate of peri-operative neurological deterioration (a grade of "very low" means that any estimate of effect is very uncertain). The authors concluded that based on strong evidence that multi-modality intra-operative neuromonitoring is sensitive and specific for detecting intra-operative neurological injury during spine surgery, it is recommended that the use of multi-modality intra-operative neuromonitoring be considered in spine surgery where the spinal cord or nerve roots are deemed to be at risk, including procedures involving deformity correction and procedures that require the placement of instrumentation. Furthermore, they stated that there is a need to develop evidence-based protocols to deal with intra-operative changes in multi-modality intra-operative neuromonitoring and to validate these prospectively. Intra-operative EMG monitoring was not recommended as a means of neurophysiological monitoring during spinal surgery.

Balzer, et al. (1998) reported on the results of a descriptive case series of the use of somatosensory evoked potentials during lumbosacral spine surgery. SSEPs and EMG activity were simultaneously recorded for 44 patients who underwent surgical procedures to decompress and stabilize the lumbosacral spine, using pedicle screw instrumentation. Indications included degenerative spondylolisthesis (22), pars fracture with spondylolisthesis (9), failed back syndrome (7), burst/compression fracture (4), and instability from metastasis (2). The specific level of the lumbar spine for each procedure included in this series was not reported. All neurosurgical procedures were performed by a single surgeon. The authors reported that, in two cases, changes in SSEPs and spontaneous EMG activity were noted and were correlated with postoperative patient complaints.

Rothstein (2009) stated that the early recognition of comatose patients with a hopeless prognosis -- regardless of how aggressively they are managed -- is of utmost
importance. Median SSEP supplement and enhance neurological examination findings in anoxic-ischemic coma and are useful as an early guide in predicting outcome. The key finding is that bilateral absence of cortical evoked potentials reliably predicts unfavorable outcome in comatose patients after cardiac arrest. The author studied 50 comatose patients with preserved brainstem function after cardiac arrest. All 23 patients with bilateral absence of cortical evoked potentials died without awakening. Neuropathological study in 7 patients disclosed widespread ischemic changes or frank cortical laminar necrosis. The remaining 27 patients with normal or delayed central conduction times had an uncertain prognosis because some died without awakening or entered a persistent vegetative state. The majority of patients with normal central conduction times had a good outcome, whereas a delay in central conduction times increased the likelihood of neurological deficit or death. Greater use of SSEP in anoxic-ischemic coma would identify those patients unlikely to recover and would avoid costly medical care that is no avail.

An UpToDate review on “Hypoxic-ischemic brain injury: Evaluation and prognosis” (Weinhouse and Young, 2012) states that several ancillary tests have been studied in the period after anoxic injury; these are often helpful at arriving at an earlier prognostic determination than would be possible with clinical testing alone. Somatosensory evoked potentials are the averaged electrical responses in the central nervous system to somatosensory stimulation. Bilateral absence of the N20 component of the SSEP with median nerve stimulation at the wrist in the 1st week (usually between 24 and 72 hours) from the arrest has a pooled likelihood ratio of 12.0 (95% confidence interval [CI]: 5.3 to 26.6) and a false-positive rate of zero % for an outcome no better than persistent vegetative state. Repeated testing should be considered when the N20 responses are present in the first 2 to 3 days from the cardiac arrest, as they may later disappear. The clinical operating characteristics of other evoked potentials (brainstem, auditory, visual, middle latency, and event-related) have not been adequately evaluated. Somatosensory evoked potentials are the best validated and most reliable of the ancillary tests currently available for clinical use.

The Quality Standards Subcommittee of the American Academy of Neurology's Practice Parameter on “Prediction of outcome in comatose survivors after cardiopulmonary resuscitation (an evidence-based review)” (Wijdicks et al, 2006) recommended the assessment of poor prognosis can be guided by the bilateral absence of cortical SSEPs (N20 response) within 1 to 3 days (recommendation level B).

Raggi et al (2010) noted that amyotrophic lateral sclerosis (ALS) is increasingly recognized to be a multi-system disease, involving associative areas in addition to the motor cortex and therefore affecting cognition. Patients with ALS may present with subtle behavioral and executive dysfunctions or, less frequently, with a manifest fronto-temporal dementia. Event-related potentials (ERPs) are a high-temporal resolution technique, which can be used to explore the presence of cognitive dysfunction. All the primary studies reviewed here have shown ERP abnormalities in groups of non-demented patients affected by sporadic ALS compared to healthy controls. The ERP results support findings of neuropsychological and imaging studies. The authors concluded that prospective studies combining simultaneous neuropsychological and imaging investigations are needed to assess the possible role of ERPs in the early detection and follow-up of cognitive dysfunction in ALS patients.
The U.S. Preventive Services Task Force (USPSTF) has not recommended vision screening of infants and young children. The 2011 USPSTF recommendation does not support vision screening for children less than 3 years of age, as it concludes that the current evidence is insufficient to assess the balance of benefits and harms to this subpopulation. This position is consistent with the current recommendations of the American Academy of Ophthalmology and the American Association for Pediatric Ophthalmology and Strabismus and other professional organizations.

In a review on "Facial nerve monitoring during cerebellopontine angle and skull base tumor surgery", Acioly et al (2013) stated that intraoperative neuromonitoring has been established as one of the methods by which modern neurosurgery can improve surgical results while reducing morbidity. Despite routine use of intraoperative facial nerve (FN) monitoring, FN injury still is a complication of major concern due to severe negative impact on patient's quality of life. Through searches of PubMed, these investigators provided a systematic review of the current literature up to February, 2011, emphasizing all respects of FN monitoring for cerebellopontine angle and skull base tumor surgery from description to current success on function prediction of standard and emerging monitoring techniques. Currently, standard monitoring techniques comprise direct electrical stimulation (DES), free-running electromyography (EMG), and facial motor evoked potential (FMEP). These researchers included 62 studies on function prediction by investigating DES (43 studies), free-running EMG (13 studies), and FMEP (6 studies) criteria. DES mostly evaluated post-operative function by using absolute amplitude, stimulation threshold, and proximal-to-distal amplitude ratio, whereas free-running EMG used the train-time criterion. The prognostic significance of FMEP was assessed with the final-to-baseline amplitude ratio, as well as the event-to-baseline amplitude ratio and waveform complexity. The authors concluded that although there is a general agreement on the satisfactory functional prediction of different electrophysiological criteria, the lack of standardization in electrode montage and stimulation parameters precludes a definite conclusion regarding the best method. Moreover, studies emphasizing comparison between criteria or even multi-modal monitoring and its impact on FN anatomical and functional preservation are still lacking in the literature.

Mauguiere et al (1997) examined if abnormalities of central conduction could be detected prospectively in patients with epilepsy treated with vigabatrin (VGB) as long-term add-on medication. A total of 201 patients with refractory partial epilepsy were enrolled and monitored for as long as 2 years. Vigabatrin was added to the treatment at an average dose of 2 to 3g/day. Conduction in somatosensory and visual pathways was assessed by median nerve SEP and pattern VEP recordings performed at inclusion and once every 6 months. The upper limit and test-retest variability of EP latencies were evaluated at time of enrollment in the patient group. Prolonged N13-N20 or P14-N20 SEP intervals and P100 VEP latency greater than 2.5 SD above the baseline mean, observed on repeated runs in the same session and exceeding the test-retest variability at enrollment were considered to indicate central conduction slowing. A total of 109 patients completed the 2-year study period, and 92 discontinued VGB, of whom 37 were monitored with regard to EP until the end of the study. No consistent change in SEP or VEP was observed in the entire group during VGB treatment. The number of occasional EP values outside the baseline range in patients treated with VGB similar to that in patients whose VGB treatment had been discontinued. The
authors concluded that they detected no evidence of changes in SEP and VEP attributable to altered neuronal conduction in the CNS during long-term VGB treatment.

Zgorzalewicz and Galas-Zgorzalewicz (2000) estimated the effects of VGB as add-on therapy on VEP and BAEP. The investigation covered 100 epileptic patients from 8 to 18 years of age. The treatment included therapy with carbamazepine (CBZ) or valproate acid (VPA) using slow release formulations of these anti-epileptic drugs (AEDs). Combination therapy was administered using add-on VGB in the recommended dose 57.4 +/- 26.5 mg/kg body weight/day. VEP and BAEP were recorded by means of Multiliner (Toennies, Germany). The obtained values were compared with age-matched control group. Compared to control groups, significant differences in epileptic groups emerged in latencies of the peak III, V along with the inter-peak intervals I-III of BAEP. Also VEP studies showed the reduction of N75/P100 and P100/N145 amplitudes. The authors concluded that adding VGB did not significantly increase the percentage of pathological abnormalities observed from EPs.

In a prospective cohort study, Zuniga et al (2012) characterized both cervical and ocular vestibular-evoked myogenic potential (cVEMP, oVEMP) responses to air-conducted sound (ACS) and midline taps in Meniere disease (MD), vestibular migraine (VM), and controls, and determined if cVEMP or oVEMP responses can differentiate MD from VM. Unilateral definite MD patients (n = 20), VM patients (n = 21) by modified Neuhauser criteria, and age-matched controls (n = 28) were included in this study; cVEMP testing used ACS (clicks), and oVEMP testing used ACS (clicks and 500-Hz tone bursts) and midline tap stimuli (reflex hammer and Mini-Shaker). Outcome parameters were cVEMP peak-to-peak amplitudes and oVEMP n10 amplitudes. Relative to controls, MD and VM groups both showed reduced click-evoked cVEMP (p < 0.001) and oVEMP (p < 0.001) amplitudes. Only the MD group showed reduction in tone-evoked amplitudes for oVEMP. Tone-evoked oVEMPs differentiated MD from controls (p = 0.001) and from VM (p = 0.007). The oVEMPs in response to the reflex hammer and Mini-Shaker midline taps showed no differences between groups (p > 0.210). The authors concluded that using these techniques, VM and MD behaved similarly on most of the VEMP test battery. A link in their pathophysiology may be responsible for these responses. The data suggested a difference in 500-Hz tone burst-evoked oVEMP responses between MD and MV as a group. However, no VEMP test that was investigated in segregated individuals with MD from those with VM.

Heravian et al (2011) assessed the usefulness of color vision, photo stress recovery time (PSRT), and VEP in early detection of ocular toxicity of hydroxychloroquine (HCQ), in patients with rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE). A total of 86 patients were included in the study and divided into 3 groups: with history of HCQ use: interventional 1 (Int.1) without fundoscopic changes and Int.2 with fundoscopic changes; and without history of HCQ use, as control. Visual field, color vision, PSRT and VEP results were recorded for all patients and the effect of age, disease duration, treatment duration and cumulative dose of HCQ on each test was assessed in each group. There was a significant relationship among PSRT and age, treatment duration, cumulative dose of HCQ and disease duration (p < 0.001 for all). Color vision was normal in all the cases. P100 amplitude was not different between the 3 groups (p = 0.846), but P100 latency was significantly different (p = 0.025) and for Int.2 it was greater than the others. The percentage of abnormal visual fields for Int.2 was more than Int.1 and control groups (p = 0.002 and p = 0.005, respectively), but Int.1 and control groups were not significantly different (p > 0.50). In the early stages of
maculopathy. P100 latencies of VEP and PSRT are useful predictors of HCQ ocular toxicity. In patients without ocular symptoms and fundoscopic changes, the P100 latency of VEP predicts more precisely than the others.

Current guidelines from the American Academy of Ophthalmology do not recommend visual evoked potentials for screening or diagnosis of hydroxychloroquine toxicity (Marmor, et al., 2011; Karmel, 2011; Scechtman and Karpecki, 2011). Scechtman and Karpecki (2011) noted that the 2011 testing guidelines for patients on Plaquenil listed (i) dilated fundus examination, (ii) automated 10-2 VF, (iii) spectral domain optical coherence tomography (SD-OCT), fundus autofluorescence (FAF) or multi-focal electroretinography (mERG) (if available), and (iv) photography as screening tests. Visual evoked potentials were not mentioned as a screening tool. Furthermore, the screening guidelines on “Hydroxychloroquine toxicity” by Schwartz and Mieler (2011) did not mention the use of VEP. An UpToDate Drug Information on “Hydroxychloroquine” notes that “Ophthalmologic exam at baseline and every 3 months during prolonged therapy (including visual acuity, slit-lamp, fundoscopic, and visual field exam); muscle strength (especially proximal, as a symptom of neuromyopathy) during long-term therapy”. Visual evoked potentials were not mentioned as a screening tool. Also, an UpToDate review on “Antimalarial drugs in the treatment of rheumatic disease” (Wallace, 2013) does not mention the use of VEPs.

In a systematic review, van Laerhoven et al (2013) examined the prognostic value of currently used clinical tests in neonatal patients with perinatal asphyxia and hypoxic-ischemic encephalopathy (HIE). Searches were made on MedLine, Embase, Central, and CINAHL for studies occurring between January 1980 and November 2011. Studies were included if they (i) evaluated outcome in term infants with perinatal asphyxia and HIE, (ii) evaluated prognostic tests, and (iii) reported outcome at a minimal follow-up age of 18 months. Study selection, assessment of methodological quality, and data extraction were performed by 3 independent reviewers. Pooled sensitivities and specificities of investigated tests were calculated when possible. Of the 259 relevant studies, 29 were included describing 13 prognostic tests conducted 1,631 times in 1,306 term neonates. A considerable heterogeneity was noted in test performance, cut-off values, and outcome measures. The most promising tests were amplitude-integrated electroencephalography (sensitivity 0.93, [95 % CI: 0.78 to 0.98]; specificity 0.90 [0.60 to 0.98]), EEG (sensitivity 0.92 [0.66 to 0.99]; specificity 0.83 [0.64 to 0.93]), and VEPs (sensitivity 0.90 [0.74 to 0.97]; specificity 0.92 [0.68 to 0.98]). In imaging, diffusion weighted MRI performed best on specificity (0.89 [0.62 to 0.98]) and T1/T2-weighted MRI performed best on sensitivity (0.98 [0.80 to 1.00]). Magnetic resonance spectroscopy demonstrated a sensitivity of 0.75 (0.26 to 0.96) with poor specificity (0.58 [0.23 to 0.87]). The authors concluded that this evidence suggested an important role for amplitude-integrated electroencephalography, EEG, VEPs, and diffusion weighted and conventional MRI. Moreover, they stated that given the heterogeneity in the tests’ performance and outcomes studied, well-designed, large prospective studies are needed.

In a retrospective analysis of a case series, Silverstein et al (2014) described a novel technique to monitor femoral nerve function by analyzing the saphenous nerve SSEP during transpsoas surgical exposures of the lumbar spine. Institutional review board approval was granted for this study and the medical records along with the intraoperative monitoring reports from 41 consecutive transpsoas lateral interbody fusion procedures were analyzed. The presence or absence of intraoperative changes
to the saphenous nerve SSEP was noted and the post-operative symptoms and physical examination findings were noted. Changes in SSEP were noted in 5 of the 41 surgical procedures, with 3 of the patients waking up with a femoral nerve deficit. None of the patients with stable SSEP's developed sensory or motor deficits post-operatively. No patient in this series demonstrated intraoperative EMG changes indicative of an intraoperative nerve injury. The authors concluded that saphenous nerve SSEP monitoring may be a beneficial tool to detect femoral nerve injury related to transpsoas direct lateral approaches to the lumbar spine. These preliminary findings need to be validated by well-designed studies.

Fix and colleagues (2015) noted that amnesic mild cognitive impairment (MCIa) is often characterized as an early stage of Alzheimer's dementia (AD). The latency of the P2, an electroencephalographic component of the flash VEP (FVEP), is significantly longer in those with AD or MCIa when compared with controls. In a pilot study, these investigators examined the diagnostic accuracy of several FVEP-P2 procedures in distinguishing people with MCIa and controls. The latency of the FVEP-P2 was measured in participants exposed to a single flash condition and 5 double-flash conditions. The double-flash conditions had different inter-stimulus intervals between the pair of strobe flashes. Significant group differences were observed in the single-flash and 2 of the double-flash conditions. One of the double-flash conditions (100 ms) displayed a higher predictive accuracy than the single-flash condition, suggesting that this novel procedure may have more diagnostic potential. Participants with MCIa displayed similar P2 latencies across conditions, while controls exhibited a consistent pattern of P2 latency differences. These differences demonstrated that the double stimulation procedure resulted in a measurable refractory effect for controls but not for those with MCIa. The authors concluded that the pattern of P2 group differences suggested that those with MCIa have compromised cholinergic functioning that resulted in impaired visual processing. They stated that results from the present investigation lend support to the theory that holds MCIa as an intermediate stage between normal healthy aging and the neuropathology present in AD; and measuring the FVEP-P2 during several double stimulation conditions could provide diagnostically useful information about the health of the cholinergic system.

Appendix

Documentation Requirements:

I. All medical necessity criteria must be clearly documented in the member's medical record and made available upon request.

II. The member's medical record must contain documentation that fully supports the medical necessity for evoked potential studies. This documentation includes, but is not limited to, relevant medical history, physical examination, the anatomic location of the planned surgical procedure, the rationale for the location and modalities to be monitored, and results of pertinent diagnostic tests or procedures.

III. For the BAERs, the member's medical record should document the otologic exam describing both ear canals and tympanic membranes, as well as a gross hearing assessment. The medical record should also include the results of air and bone pure tone audiogram and speech audiometry.
IV. The physician's evoked potential report should note which nerves were tested, latencies at various testing points, and an evaluation of whether the resulting values are normal or abnormal.

V. Baseline testing prior to intraoperative neuromonitoring requires contemporaneous interpretation prior to the surgical procedure. To qualify for coverage of baseline testing, results of testing of multiple leads for signal strength, clarity, amplitude, etc., should be documented in the medical record. The time spent performing or interpreting the baseline electrophysiologic studies performed prior to surgery should not be counted as intraoperative monitoring, but represents separately reportable procedures. Testing performed during surgery does not qualify as baseline testing and is not a separately reportable procedure.

VI. For continuous intraoperative neurophysiology monitoring, from outside the operating room (remote or nearby) or for monitoring of more than one case while in the operating room, increments of less than 30 minutes should not be billed. For continuous intraoperative neurophysiology monitoring in the operating room with one on one monitoring requiring personal attendance, increments of less than 8 minutes should not be billed.

CPT Codes / HCPCS Codes / ICD-9 Codes

Somatosensory evoked potentials (SEPs, SSEPs):

**CPT codes covered if selection criteria are met:**

- **95925** Short-latency somatosensory evoked potential study, stimulation of any/all peripheral nerves or skin sites, recording from the central nervous system; in upper limbs
- **95926** in lower limbs
- **95927** in the trunk or head
- **95938** Short-latency somatosensory evoked potential study, stimulation of any/all peripheral nerves or skin sites, recording from the central nervous system; in upper and lower limbs

**ICD-9 codes covered if selection criteria are met:**

- **333.0** Other degenerative diseases of the basal ganglia
  [olivopontocerebellar (OPC) degeneration]
- **334.0** Friedreich's ataxia
- **336.0 - 336.9** Other diseases of spinal cord [unexplained myelopathy]
- **340** Multiple sclerosis [with clinically silent lesions]
- **341.0 - 341.9** Other demyelinating diseases of central nervous system
- **348.1** Anoxic brain damage
348.82  Brain death

952.00 - 953.9  Spinal cord injury without evidence of spinal bone injury and injury to nerve roots and spinal plexus [unconscious]

ICD-9 codes not covered for indications listed in the CPB (not all-inclusive):

266.2  Other B-complex deficiencies [diagnosis and management of acquired metabolic disorders]

270.0 - 277.9  Other metabolic disorders [diagnosis and management of acquired metabolic disorders]

309.81  Posttraumatic stress disorder

314.00 - 314.01  Attention deficit disorder with or without mention of hyperactivity [ADD or ADHD]

315.10, 315.19  Other and unspecified spinal muscular atrophy [Kennedy's syndrome]

335.20  Amyotrophic lateral sclerosis [ALS]

353.0  Brachial plexus lesions [thoracic outlet syndrome]

354.0 - 355.9  Mononeuritis [radiculopathies, peripheral nerve lesions, carpal tunnel syndrome/nerve entrapment]

721.1  Cervical spondylosis with myelopathy

722.0 - 722.2  Displacement of intervertebral disc without myelopathy

722.70 - 722.73  Intervertebral disc disorder with myelopathy [radiculopathies]

723.4  Brachial neuritis or radiculitis [where standard nerve conduction velocity studies are diagnostic]

724.3  Sciatica [radiculopathies]

724.4  Thoracic or lumbosacral neuritis or radiculitis, unspecified [radiculopathies]

729.2  Neuralgia, neuritis, and radiculitis, unspecified

800.00 - 804.99  Fracture of skull [conscious]

805.00 - 805.9  Fracture of vertebral column [conscious]

806.00 - 806.9  Fracture of vertebral column with spinal cord injury [conscious]

850.00 - 854.19  Intracranial injury, excluding those with skull fracture [conscious]
952.00 - 953.9 Spinal cord injury without evidence of spinal bone injury and injury to nerve roots and spinal plexus [conscious]

961.2 Poisoning by heavy metal anti-infectives

984.0 - 984.9 Toxic effect of lead and its compounds (including fumes)

V80.0 Special screening for neurological conditions [indicates routine exam without signs or symptoms when reported alone]

V82.5 Special screening for chemical poisoning and other contamination [indicates routine exam without signs or symptoms when reported alone]

*Intra-operative somatosensory evoked potentials (SSEPs) performed either alone, or in combination with motor evoked potentials (MEPs):*

**CPT codes covered if selection criteria are met:**

95940 Continuous intraoperative neurophysiology monitoring in the operating room, one on one monitoring requiring personal attendance, each 15 minutes (List separately in addition to code for primary procedure)

95941 Continuous intraoperative neurophysiology monitoring, from outside the operating room (remote or nearby) or for monitoring of more than one case while in the operating room, per hour (List separately in addition to code for primary procedure)

**HCPCS codes covered if selection criteria are met:**

G0453 Continuous intraoperative neurophysiology monitoring, from outside the operating room (remote or nearby), per patient, (attention directed exclusively to one patient) each 15 minutes (list in addition to primary procedure)

*Intra-operative SEP monitoring, with or without MEPs, may be appropriate for the following types of surgery (not an all-inclusive):*

**CPT codes covered if selection criteria are met for intraoperative SEPs:**

22210 - 22226 Osteotomy of spine

22310 - 22328 Treatment of fracture and/or dislocation of vertebrae

22532 - 22819 Arthrodesis

22840 - 22855 Spinal instrumentation

31200 - 31230 Ethmoidectomy and maxillectomy

33320 - 33335 Repair of aorta or great vessels

33400 - 33417 Aortic valve procedures
33800 - 33853  Aortic anomalies procedures
33860 - 33877  Thoracic aortic aneurysm repair
61000 - 61070  Injection, drainage, or aspiration of skull meninges, and brain
61105 - 61253  Twist drill, burr hole(s), or trephine
61304 - 61576  Craniectomy or craniotomy
61600 - 61616  Definitive procedures of skull base
61618 - 61619  Repair and/or reconstruction of surgical defects of skull base
61623 - 61626  Endovascular therapy
61680 - 61711  Surgery for aneurysm, arteriovenous malformation or vascular
disease
61720 - 61791  Stereotaxis, intracranial
61850 - 61888  Neurostimulators (intracranial)
62000 - 62148  Repair of skull
62160 - 62165  Neuroendoscopy
62263 - 62319  Injection, drainage, or aspiration of spine and spinal cord
63001 - 63103  Exploration/decompression of spinal cord
63170 - 63308  Incision/excision intraspinal
63600 - 63615  Stereotaxis, spinal
63700 - 63710  Repair (spinal)
67570  Optic nerve decompression (e.g., incision or fenestration of optic
nerve sheath)
69666  Repair of oval window fistula
69667  Repair of round window fistula
69720  Decompression facial nerve, intratemporal; lateral to geniculate
ganglion
69725  including medial to geniculate ganglion
69740  Suture facial nerve, intratemporal, with or without graft or
decompression; lateral to geniculate ganglion
69745  including medial to geniculate ganglion
69805  Endolymphatic sac operation; without shunt
69806  with shunt
69915  Vestibular nerve section, translabyrinthine approach
69950  Vestibular nerve section, transcranial approach
69955  Total facial nerve decompression and/or repair (may include graft)
99173  Screening test of visual acuity, quantitative, bilateral

**CPT codes not covered for indications listed in the CPB for intraoperative SEPs:**

27130 - 27138  Total hip arthroplasty (includes conversion and revision to previous surgery)
33510 - 33548  Coronary artery bypass surgery
42410 - 42426  Excision of parotid tumor or parotid gland
42440  Excision of submandibular (submaxillary) gland
60000 - 60512  Thyroid and parathyroid surgery
61546  Craniotomy for hypophysectomy or excision of pituitary tumor, intracranial approach
61548  Hypophysectomy or excision of pituitary tumor, transnasal or transseptal approach, nonsterotatic
62165  Neuroendoscopy, intracranial; with excision of pituitary tumor, transnasal or trans-sphenoidal approach
63650  Percutaneous implantation of neurostimulator electrode array, epidural

**Other HCPCS codes related to the CPB:**

S8040  Topographic brain mapping

**ICD-9 codes covered if selection criteria are met for intraoperative SEPs:**

170.0  Malignant neoplasm of bones of skull and face, except mandible
170.2  Malignant neoplasm of vertebral column, excluding sacrum and coccyx
170.6  Malignant neoplasm of pelvic bones, sacrum, and coccyx
191.0 - 191.9  Malignant neoplasm of brain
192.0 - 192.3  Malignant neoplasm of cranial nerves, cerebral meninges, spinal cord, and spinal meninges
198.3  Secondary malignant neoplasm of brain and spinal cord
198.4  Secondary malignant neoplasm of other parts of nervous system
213.2  Benign neoplasm of vertebral column, excluding sacrum and coccyx
213.6  Benign neoplasm of pelvic bones, sacrum, and coccyx
225.0 - 225.4  Benign neoplasm of brain, cranial nerves, cerebral meninges, spinal cord, and spinal meninges
237.5 - 237.6  Neoplasm of uncertain behavior of brain and spinal cord, or meninges
239.6  Neoplasm of unspecified nature of brain
333.0 - 333.99  Other extrapyramidal disease and abnormal movement disorders [intractable]
345.00 - 345.91  Epilepsy [resection of brain tissue or tumor]
348.1  Anoxic brain damage
348.4  Compression of brain
348.5  Cerebral edema
350.8  Other specified trigeminal nerve disorders [compression]
351.8  Other facial nerve disorders [compression]
377.49  Other disorders of optic nerve [compression]
386.00 - 386.04  Meniere's disease [endolymphatic shunt placement]
386.10 - 386.19  Other and unspecified peripheral vertigo [vestibular resection]
386.2  Vertigo of central origin [vestibular resection]
388.5  Disorders of acoustic nerve [compression]
395.0 - 395.9  Diseases of aortic valve
424.1  Aortic valve disorders
440.0  Atherosclerosis of aorta
441.00 - 441.9  Dissection of aorta
442.81  Other aneurysm of artery of neck
443.21  Dissection of carotid artery
444.0  Arterial embolism and thrombosis of abdominal aorta
444.1  Arterial embolism and thrombosis of thoracic aorta
721.41 - 721.42  Thoracic or lumbar spondylosis with myelopathy
722.70 - 722.73  Intervertebral disc disorder, with myelopathy
722.80 - 722.83  Postlaminectomy syndrome
737.30 - 737.39  Kyphoscoliosis and scoliosis [correction involving traction]
737.43  Scoliosis associated with other conditions [correction involving traction]
741.00-741.03  Spina bifida with hydrocephalus
742.0  Encephalocele
742.2  Congenital reduction deformities of brain
747.81  Anomalies of cerebrovascular system [arteriovenous malformation brain]
747.82  Spinal vessel anomaly [arteriovenous malformation spine]
754.2  Congenital musculoskeletal deformities of spine [correction involving traction]
779.2  Cerebral depression, coma, and other abnormal cerebral signs
780.01  Coma [unconscious]
780.39  Other convulsions [resection of brain tissue or tumor]
780.4  Dizziness and giddiness [vertigo NOS]
781.0  Abnormal involuntary movements [intractable movement disorder]
800.00 - 804.99  Fracture of skull
805.0 - 806.9  Fracture of vertebral column
850.00 - 854.19  Intracranial injury, excluding those with skull fracture
952.00 - 953.9  Spinal cord injury without evidence of spinal bone injury and injury to nerve roots and spinal plexus
996.40 - 996.49  Mechanical complication of internal orthopedic device, implant, and graft
996.67  Infection and inflammatory reaction due to other internal orthopedic device, implant, and graft
Other complications due to other internal orthopedic device, implant, and graft

V54.01 - V54.09

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

- 142.0 Malignant neoplasm of parotid gland
- 142.1 Malignant neoplasm of submandibular gland
- 195.0 Malignant neoplasm of head, face, and neck
- 210.2 Benign neoplasm of major salivary glands
- 230.0 Carcinoma in situ of lip, oral cavity, and pharynx
- 235.0 Neoplasm of uncertain behavior of major salivary glands
- 239.0 Neoplasm of unspecified nature of digestive system
- 240.0 - 246.9 Disorders of the thyroid gland
- 527.0 - 527.9 Diseases of the salivary glands
- 598.0 - 598.9 Urethral stricture
- 600.00 - 600.01 Hypertrophy (benign) of prostate
- 600.10 - 600.11 Nodular prostate
- 600.20 - 600.21 Benign localized hyperplasia of prostate
- 600.90 - 600.91 Hyperplasia of prostate, unspecified
- 754.81 Pectus excavatum

Intra-operative visual evoked potentials monitoring:

CPT codes covered if selection criteria are met:

- 0333T Visual evoked potential, screening of visual acuity, automated
- 95930 Visual evoked potential (VEP) testing central nervous system, checkerboard or flash
- +95940 Continuous intraoperative neurophysiology monitoring in the operating room, one on one monitoring requiring personal
attendance, each 15 minutes (List separately in addition to code for primary procedure)

+95941 Continuous intraoperative neurophysiology monitoring, from outside the operating room (remote or nearby) or for monitoring of more than one case while in the operating room, per hour (List separately in addition to code for primary procedure)

CPT codes not covered for indications listed in the CPB for intraoperative VEPs:

61680 - 61692 Surgery of intracranial arteriovenous malformation

HCPCS codes covered if selection criteria are met:

G0453 Continuous intraoperative neurophysiology monitoring, from outside the operating room (remote or nearby), per patient, (attention directed exclusively to one patient) each 15 minutes (list in addition to primary procedure)

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

747.81 Congenital anomalies of cerebrovascular system

Visual evoked potentials (VEPs):

CPT codes covered if selection criteria are met:

0333T Visual evoked potential, screening of visual acuity, automated [not covered for screening]

95930 Visual evoked potential (VEP) testing central nervous system, checkerboard or flash

ICD-9 codes covered if selection criteria are met (for members > 3 mos of age):

036.81 Meningococcal optic neuritis

054.3 Herpetic meningoencephalitis

055.0 Postmeasles encephalitis

056.01 Encephalomyelitis due to rubella

058.21 Human herpesvirus 6 encephalitis

058.29 Other human herpesvirus encephalitis

062.0 - 064 Mosquito-borne viral encephalitis, tick-borne viral encephalitis, and viral encephalitis transmitted by other and unspecified arthropods

088.81 Lyme disease

094.0 - 094.9 Neurosyphilis
<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>192.0 - 192.9</td>
<td>Malignant neoplasm of other and unspecified parts of nervous system</td>
</tr>
<tr>
<td>198.3 - 198.4</td>
<td>Secondary malignant neoplasm of brain and spinal cord</td>
</tr>
<tr>
<td>225.0 - 225.9</td>
<td>Benign neoplasm of brain and other parts of nervous system</td>
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<td>237.0 - 237.1</td>
<td>Neoplasm of uncertain behavior of endocrine glands and nervous system</td>
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<td>237.5 - 237.9</td>
<td>Neoplasm of uncertain behavior of brain and spinal cord, meninges, neurofibromatosis, and other and unspecified parts of nervous system</td>
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<td>239.6</td>
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<td>300.11</td>
<td>Conversion disorder</td>
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<td>333.0</td>
<td>Other degenerative diseases of the basal ganglia</td>
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<td>334.0 - 334.9</td>
<td>Spinocerebellar disease</td>
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<tr>
<td>340</td>
<td>Multiple sclerosis</td>
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<tr>
<td>341.0 - 341.9</td>
<td>Other demyelinating diseases of central nervous system</td>
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437.3  Cerebral aneurysm, nonruptured
780.01  Coma [unresponsive]
780.03  Persistent vegetative state [unresponsive, unable to communicate]
780.4  Dizziness and giddiness
781.2 - 781.4  Abnormality of gait, lack of coordination, and transient paralysis of limb
784.3  Aphasia [unable to communicate]
794.10 - 794.19  Nonspecific abnormal results of function studies of peripheral nervous system and special senses
850.4 - 853.19  Concussion with prolonged loss of consciousness without return to pre-existing conscious level
907.1 - 907.5  Late effect of injury to cranial nerve, spinal cord, nerve root(s), spinal plexus(es), and other nerves of trunk, peripheral nerve of shoulder girdle and upper limb, or peripheral nerve of pelvic girdle and lower limb
950.0 - 950.9  Injury to optic nerve and pathways

**ICD-9 codes not covered for indications listed in the CPB (for members > 3 mos of age) (not all-inclusive):**

084.0 - 084.9  Malaria
253.0 - 253.9  Disorders of the pituitary gland and its hypothalamic control
290.0 - 290.9  Senile and presenile organic psychotic conditions
291.2  Alcohol induced persisting dementia
292.82  Drug induced persisting dementia
309.81  Posttraumatic stress disorder
314.00 - 314.01  Attention deficit disorder [ADD or ADHD]
321.0  Alzheimer’s disease
332.0 - 332.1  Parkinson's disease
336.0  Syringomyelia and syringobulbia
647.4  Malaria complicating pregnancy, childbirth, or the puerperium
695.4  Lupus erythematosus
710.0  Systemic lupus erythematosus
771.2 Other congenital infections specific to the perinatal period

961.4 Poisoning by antimalarials and drugs acting on other blood protozoa

V20.2 Routine infant or child health check

V70.0 Routine general medical examination at a health care facility

V80.09 Special screening for other neurological conditions

E931.4 Antimalarials and drugs acting on other blood protozoa causing adverse effects in therapeutic use

ICD-9 codes not covered for indications listed in the CPB (for members < 3 mos of age/ neonatal screen):

084.0 - 084.9 Malaria

253.0 - 253.9 Disorders of the pituitary gland and its hypothalamic control

336.0 Syringomyelia and syringobulbia

345.40 - 345.61 Localization-related (focal)(partial) epilepsy and epileptic syndromes with complex partial seizures, and infantile spasms [not covered for vigabatrin (Sabril)-associated retinal toxicity]

695.4 Lupus erythematosus

710.0 Systemic lupus erythematosus

760.0 - 779.9 Certain conditions originating in the perinatal period

768.2 - 768.4 Fetal distress [in neonates]

768.5 - 768.6 Birth asphyxia [in neonates]

768.70 - 768.73 Hypoxic-ischemic encephalopathy [in neonates]

961.4 Poisoning by antimalarials and drugs acting on other blood protozoa

V20.2 Routine infant or child health check

V20.31 - V20.32 Health supervision for newborn under 8 days old to 28 days old

V27.0 - V27.9 Outcome of delivery

V29.0 - V39.2 Observation and evaluation of newborns and infants for suspected condition not found or liveborn infants according to type of birth

V72.0 Examination of eyes and vision [indicates routine screen without signs or symptoms when reported alone]
V80.09  Special screening for other neurological conditions

V80.2  Special screening for other eye conditions [indicates routine screen without signs or symptoms when reported alone]

E931.4  Antimalarials and drugs acting on other blood protozoa causing adverse effects in therapeutic use

**Intra-operative brain stem auditory evoked response (BAER) monitoring:**

CPT codes covered if selection criteria are met:

- **92585 - 92586**  
  Auditory evoked potentials for evoked response audiology and/or testing of the central nervous system; comprehensive and limited

- **+95940**  
  Continuous intraoperative neurophysiology monitoring in the operating room, one on one monitoring requiring personal attendance, each 15 minutes (List separately in addition to code for primary procedure)

- **+95941**  
  Continuous intraoperative neurophysiology monitoring, from outside the operating room (remote or nearby) or for monitoring of more than one case while in the operating room, per hour (List separately in addition to code for primary procedure)

**Intra-operative brain stem auditory evoked response (BAER) monitoring may be appropriate for the following types of surgery:**

- **22100**  
  Partial excision of posterior vertebral component (eg, spinous process, lamina or facet) for intrinsic bony lesion, single vertebral segment; cervical

- **+22101**  
  each additional segment (List separately in addition to code for primary procedure)

- **22110**  
  Partial excision of vertebral body, for intrinsic bony lesion, without decompression of spinal cord or nerve root(s), single vertebral segment; cervical

- **+22116**  
  each additional vertebral segment (List separately in addition to code for primary procedure)

- **22220**  
  Osteotomy of spine, including discectomy, anterior approach, single vertebral segment; cervical

- **+22226**  
  each additional vertebral segment (List separately in addition to code for primary procedure)

- **22548**  
  Arthrodesis, anterior transoral or extraoral technique, clivus-C1-C2 (atlas-axis), with or without excision of odontoid process

- **61343**  
  Craniectomy, suboccipital with cervical laminectomy for decompression of medulla and spinal cord, with or without dural graft (eg, Arnold-Chiari malformation)
61575 Transoral approach to skull base, brain stem or upper spinal cord for biopsy, decompression or excision of lesion requiring splitting of tongue and/or mandible (including tracheostomy)

62164 Neuroendoscopy, intracranial; with excision of brain tumor, including placement of external ventricular catheter for drainage

62165 with excision of pituitary tumor, transnasal or trans-sphenoidal approach

63001 Laminectomy with exploration and/or decompression of spinal cord and/or cauda equina, without facetectomy, foraminotomy or discectomy, (eg, spinal stenosis), one or two vertebral segments; cervical

**HCPCS codes covered if selection criteria are met:**

G0453 Continuous intraoperative neurophysiology monitoring, from outside the operating room (remote or nearby), per patient, (attention directed exclusively to one patient) each 15 minutes (list in addition to primary procedure

**ICD-9 codes covered if selection criteria are met:**

170.0 Malignant neoplasm of bones of skull and face, except mandible

170.2 Malignant neoplasm of vertebral column, excluding sacrum and coccyx

191.7 Malignant neoplasm of brain stem

192.2 Malignant neoplasm of spinal cord

756.0 Congenital anomalies of skull and face bones

**Brain stem auditory evoked response (BAER), comprehensive:**

**CPT codes covered if selection criteria are met:**

92585 Auditory evoked potentials for evoked response audiometry and/or testing of the central nervous system; comprehensive

**CPT codes not covered for indications listed in the CPB:**

69631 - 69633 Tympanoplasty & ossicle chain reconstruction

69660 - 69662 Stapedectomy

**ICD-9 codes covered if selection criteria are met (members > 3 mos of age):**

036.81 Meningococcal optic neuritis

046.3 Progressive multifocal leukoencephalopathy
054.3  Herpetic meningoencephalitis
055.0  Postmeasles encephalitis
056.01  Encephalomyelitis due to rubella
058.21  Human herpesvirus 6 encephalitis
058.29  Other human herpesvirus encephalitis
062.0 - 064  Mosquito-borne viral encephalitis, tick-borne viral encephalitis, and viral encephalitis transmitted by other and unspecified arthropods
088.81  Lyme disease
094.0 - 094.9  Neurosyphilis
191.0 - 191.9  Malignant neoplasm of brain
192.0 - 192.9  Malignant neoplasm of other and unspecified parts of the nervous system
198.3 - 198.4  Secondary malignant neoplasm of brain and spinal cord
225.0 - 225.9  Benign neoplasm of brain and other parts of nervous system
237.0 - 237.1  Neoplasm of uncertain behavior of endocrine glands and nervous system
237.5 - 237.9  Neoplasm of uncertain behavior of brain and spinal cord, meninges, neurofibromatosis, and other and unspecified parts of nervous system
239.6  Neoplasms of unspecified nature of brain
300.11  Conversion disorder
326  Late effects of intracranial abscess or pyogenic infection
333.0  Other degenerative diseases of the basal ganglia
334.0 - 334.9  Spinocebellar disease
340  Multiple Sclerosis
341.0 - 341.9  Other demyelinating diseases of the central nervous system
342.0 - 342.92  Hemiplegia and hemiparesis
343.0 - 343.9  Infantile cerebral palsy
348.0  Cerebral cysts
348.1  Anoxic brain damage
348.2  Benign intracranial hypertension
348.4  Compression of brain
348.5  Cerebral edema
348.82  Brain death [for members >3 months of age]
350.1 - 358.9  Trigeminal, facial, and other cranial nerve disorders, nerve root and plexus disorders, mononeuritis, neuropathy, and myoneural disorders
368.00 - 368.9  Visual disturbances
377.00 - 377.9  Disorders of the optic nerve and visual pathways
386.00 - 386.9  Vertiginous syndromes and other disorders of vestibular system
388.00 - 389.9  Other disorders of ear and hearing loss
430 - 435.9  Subarachnoid hemorrhage, intracerebral hemorrhage, other and unspecified intracranial hemorrhage, occlusion and stenosis of precerebral arteries, occlusion of cerebral arteries, and transient cerebral ischemia
437.1 - 437.2  Other generalized ischemic cerebrovascular disease or hypertensive encephalopathy
437.3  Cerebral aneurysm, nonruptured
741.00 - 741.03  Spina bifida with hydrocephalus
742.0  Encephalocele
742.2  Congenital reduction deformities of brain
763.0 - 763.9  Fetus or newborn affected by other complications of labor and delivery
779.2  Cerebral depression, coma, and other abnormal cerebral signs
780.01  Coma
780.03  Persistent vegetative state
780.4  Dizziness and giddiness
781.2 - 781.4  Abnormality of gait, lack of coordination, and transient paralysis of limb
794.10 - 794.19  Nonspecific abnormal results of function studies of peripheral nervous system and special senses
850.4 - 853.19  Concussion with prolonged loss of consciousness without return to pre-existing conscious level
907.1 - 907.5  Late effect of injury to cranial nerve, spinal cord, nerve root(s), spinal plexus(es), and other nerves of trunk, peripheral nerve of
shoulder girdle and upper limb, or peripheral nerve of pelvic girdle and lower limb

950.0 - 950.9 Injury to optic nerve and pathways

V20.1 - V20.2 Health supervision of other healthy infant or child receiving care or routine infant or child health check

V58.62 Long-term (current) use of antibiotics [damage due to ototoxic drugs]

V58.69 Long-term (current) use of other medications [damage due to ototoxic drugs]

V72.11 Encounter for hearing examination following failed hearing screening

ICD-9 codes not covered for indications listed in the CPB (members > 3 mos of age) (not all-inclusive):

290.0 - 290.9 Senile and presenile organic psychotic conditions

291.2 Alcohol induced persisting dementia

292.82 Drug induced persisting dementia

293.9 Unspecified transient mental disorder in conditions classified elsewhere

294.10 - 294.9 Other organic psychotic conditions (chronic)

295.20 - 295.25 Schizophrenic disorders, catatonic

295.90 - 295.95 Unspecified schizophrenia

296.00 - 296.99 Episodic mood disorders

298.0 Depressive type psychosis

299.00 - 299.01 Autistic disorder

300.4 Dysthymic disorder

309.81 Posttraumatic stress disorder

311 Depressive disorder, not elsewhere classified

314.00 - 314.01 Attention deficit disorder [ADD or ADHD]

331.0 Alzheimer’s disease
331.11  Pick's disease
331.19  Other frontotemporal dementia
331.82  Dementia with Lewy bodies
332.0 - 332.1  Parkinson's disease
335.10, 335.19  Other and unspecified spinal muscular atrophy [Kennedy's syndrome]
336.0  Syringomyelia and syringobulbia
781.1  Disturbances of sensation of smell and taste
V21.0 - V21.9  Constitutional states in development
V27.0 - V27.9  Outcome of delivery
V29.0 - V39.2  Observation and evaluation of newborns and infants for suspected condition not found or liveborn infants according to type of birth
V72.19  Other examination of ears and hearing [indicates routine exam without signs or symptoms when reported alone]
V80.3  Special screening for ear diseases [indicates routine exam without signs or symptoms when reported alone]

**ICD-9 codes not covered for indications listed in the CPB (for members < 3 mos of age/ neonatal screen):**

336.0  Syringomyelia and syringobulbia
760.0 - 779.9  Certain conditions originating in the perinatal period
V20.0 - V21.9  Health supervision of infant or child or constitutional states of development [neonatal screen]
V27.0 - V27.9  Outcome of delivery
V29.0 - V39.2  Observation and evaluation of newborns and infants for suspected condition not found or liveborn infants according to type of birth
V72.19  Other examination of ears and hearing [indicates routine exam without signs or symptoms when reported alone]
V80.3  Special screening for ear diseases [indicates routine exam without signs or symptoms when reported alone]

**Brain stem auditory evoked response (BAER), limited:**

**CPT codes covered if selection criteria are met:**

92586  Auditory evoked potentials for evoked response audiometry and/or testing of the central nervous system; limited
CPT codes not covered for indications listed in the CPB:

69631 - 69633  Tympanoplasty & ossicle chain reconstruction
69660 - 69662  Stapedectomy

ICD-9 codes covered if selection criteria are met:

036.81  Meningococcal optic neuritis
054.3  Herpetic meningoencephalitis
055.0  Postmeasles encephalitis
056.01  Encephalomyelitis due to rubella
058.21  Human herpesvirus 6 encephalitis
058.29  Other human herpesvirus encephalitis
062.0 - 064  Mosquito-borne viral encephalitis, tick-borne viral encephalitis, and viral encephalitis transmitted by other and unspecified arthropods
088.81  Lyme disease
094.0 - 094.9  Neurosyphilis
191.0 - 191.9  Malignant neoplasm of brain
192.0 - 192.9  Malignant neoplasm of other and unspecified parts of the nervous system
225.0 - 225.9  Benign neoplasm of brain and other parts of the nervous system
237.0 - 237.1  Neoplasm of uncertain behavior of endocrine glands and nervous system
237.5 - 237.9  Neoplasm of uncertain behavior of brain and spinal cord, meninges, neurofibromatosis, and other and unspecified parts of nervous system
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300.11  Conversion disorder
326  Late effects of intracranial abscess or pyogenic infection
333.0  Other degenerative diseases of the basal ganglia
334.0 - 334.9  Spinocerebellar disease
341.0 - 341.9  Other demyelinating diseases of the central nervous system
342.0 - 342.92  Hemiplegia and hemiparesis
343.0 - 343.9  Infantile cerebral palsy
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<tr>
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<td>Cerebral cysts</td>
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<td>Anoxic brain damage</td>
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<td>348.2</td>
<td>Benign intracranial hypertension</td>
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<td>Nonspecific abnormal results of function studies of peripheral nervous system and special senses</td>
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<td>850.4 - 853.19</td>
<td>Concussion with prolonged loss of consciousness without return to pre-existing conscious level</td>
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<td>Code Range</td>
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<td>907.1 - 907.5</td>
<td>Late effect of injury to cranial nerve, spinal cord, nerve root(s), spinal plexus(es), and other nerves of trunk, peripheral nerve of shoulder girdle and upper limb, or peripheral nerve of pelvic girdle and lower limb</td>
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<td>950.0 - 950.9</td>
<td>Injury to optic nerve and pathways</td>
</tr>
</tbody>
</table>

**V20.1 - V20.2**
Routine infant or child health check [*note - per ICD-9 guidelines - this is the correct code for hearing screen of infant or child over 28 days old as category V72 excludes routine hearing exam of infant or child*]

**V20.31 - V20.32**
Health supervision for newborn under 8 days old to 28 days old [*note - per ICD-9 guidelines - this is the correct code for neonatal hearing screen as category V72 excludes routine hearing exam of newborn*]

**V27.0 - V27.9**
Outcome of delivery

**V29.0 - V39.2**
Observation and evaluation of newborns and infants for suspected condition not found or liveborn infants according to type of birth

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<td>V58.62</td>
<td>Long-term (current) use of antibiotics [damage due to ototoxic drugs]</td>
</tr>
<tr>
<td>V58.69</td>
<td>Long-term (current) use of other medications [damage due to ototoxic drugs]</td>
</tr>
</tbody>
</table>

**V72.11**
Encounter for hearing examination following failed hearing screening

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

- 335.10 - 335.19 Other and unspecified spinal muscular atrophy [Kennedyâ€™s syndrome]
- 336.0 Syringomyelia and syringobulbia

**Evoked otoacoustic emissions:**

**CPT codes covered if selection criteria are met:**

- 92558 Evoked otoacoustic emissions, screening (qualitative measurement of distortion product or transient evoked otoacoustic emissions), automated analysis
- 92587 Distortion product evoked otoacoustic emissions; limited evaluation (to confirm the presence or absence of hearing disorder, 3-6 frequencies) or transient evoked otoacoustic emissions, with interpretation and report
- 92588 Comprehensive diagnostic evaluation (quantitative analysis of outer hair cell function by cochlear mapping, minimum of 12 frequencies), with interpretation and report
ICD-9 codes not covered for indications listed in the CPB (for comprehensive exam only for members < 3 mos. of age/ neonatal screen):

760.0 - 779.9  Certain conditions originating in the perinatal period

V20.0 - V21.9  Health supervision of infant or child or constitutional states of development

V27.0 - V27.9  Outcome of delivery

V29.0 - V39.2  Observation and evaluation of newborns and infants for suspected condition not found or liveborn infants according to type of birth

V72.19  Other examination of ears and hearing [indicates routine exam without signs or symptoms when reported alone]

V80.3  Special screening for ear diseases [indicates routine exam without signs or symptoms when reported alone]

Motor evoked potentials (other than intraoperative with SSEPs):

CPT codes not covered for indications listed in the CPB:

95928  Central motor evoked potential study (transcranial motor stimulation); upper limbs

95929  lower limbs

95939  Central motor evoked potential study (transcranial motor stimulation); in upper and lower limbs

Motor evoked potentials not covered intraoperatively:

CPT codes not covered for indications listed in the CPB:

63650  Percutaneous implantation of neurostimulator electrode array, epidural

The above policy is based on the following references:


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