

## 2011 Imaging Criteria

Positron Emission Tomography (PET), Brain<sup>(1)</sup>

ICD-9-CM: 92.11, 92.12

CPT: 78608, 78609

I/O Setting: Outpatient

## INDICATION(S)

- 100 Primary brain tumor
- 200 Seizure
- 300 Suspected Parkinson's disease
- 400 Movement disorder
- 500 Nonacute onset mental status changes/dementia

- 100 Primary brain tumor **[One]**<sup>(2, 3)</sup>
  - 110 MRI nondiagnostic for tumor extent and radiosurgery/surgical resection planned<sup>(4)</sup>
  - 120 MRI nondiagnostic for tumor recurrence/radiation necrosis and intervention planned<sup>(5)</sup>
- 200 Seizure **[All]**<sup>(6, 7)</sup>
  - 210 Refractory seizures with therapeutic blood levels of anticonvulsant
  - 220 MRI nondiagnostic for etiology/extent of seizure focus<sup>(8)</sup>
  - 230 Interventional procedure/surgery planned<sup>(9, 10)</sup>
- 300 Suspected Parkinson's disease **[Both]**<sup>(11, 12, 13)</sup>
  - 310 MRI/CT nondiagnostic for etiology of Sx/findings<sup>(14)</sup>
  - 320 Continued Sx/findings **after** Rx **[One]**<sup>(15)</sup>
    - 321 Carbidopa-levodopa ≥ 3 wks
    - 322 Dopamine receptor agonists ≥ 3 wks<sup>(16)</sup>
    - 323 Monamine oxidase B inhibitors ≥ 3 wks<sup>(17)</sup>
    - 324 Catechol-O-methyltransferase (COMT) inhibitors ≥ 3 wks<sup>(18)</sup>
    - 325 Anticholinergics ≥ 3 wks<sup>(19)</sup>
    - 326 Amantadine ≥ 3 wks<sup>(20)</sup>
- 400 Movement disorder **[One]**
  - 410 Suspected Huntington's chorea **[Both]**<sup>(21)</sup>
    - 411 MRI nondiagnostic for Huntington's chorea<sup>(22)</sup>
    - 412 Genetic testing not feasible/refused<sup>(23)</sup>
  - 420 Progressive ataxia of undetermined etiology and MRI nondiagnostic for etiology of ataxia<sup>(24, 25)</sup>

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- 500 Nonacute onset mental status changes/dementia **[Both]**<sup>(26, 27)</sup>
- 510 Metabolic evaluation normal/nondiagnostic
- 520 MRI/CT nondiagnostic for etiology of Sx/findings<sup>(28)</sup>

## Notes

**(1)**

While MRI and CT image anatomy, PET scans image physiology, including disease activity. PET requires the IV injection or inhalation of a tracer labeled with a positron-emitting radionuclide which accumulates in the studied tissue. The radionuclide emits positrons, which are imaged during radioactive decay. Recent improvements in both hardware and software used in PET devices have created a clinical tool which provides improved resolution, image quality, and shorter acquisition study time (Miller and DiCarli, *Am J Geriatr Cardiol* 2007; 16(6): 355-362).

**(2)**

The most common primary brain tumors in adults are gliomas and meningiomas, with gliomas accounting for more than 80% of primary brain tumors (Chandana et al., *Am Fam Physician* 2008; 77(10): 1423-1430; Grant, *J Neurol Neurosurg Psychiatry* 2004; 75 Suppl 2: ii18-23).

**(3)**

PET scanning identifies the rate of glucose uptake and is used to detect the metabolic differences between normal brain tissue, low-grade and high-grade gliomas, and radionecrosis (Jacobs et al., *NeuroRx* 2005; 2(2): 333-347). Information gained from PET may suggest the histologic grade, determine the extent of the primary brain tumor, and provide preoperative guidance of a surgical approach and subsequent management. Additionally, PET scanning may help distinguish between inflammatory, infectious, and neoplastic causes of a mass lesion that are indistinguishable by CT or MRI (Miletich, *Neurol Clin* 2009; 27(1): 61-88; Omuro et al., *Lancet Neurol* 2006; 5(11): 937-948). Newer neuroimaging techniques being used to identify hallmarks of neoplastic lesions include magnetic resonance spectroscopy (MRS) and single photon emission computed tomography (SPECT) of the brain. MRS assists in identification of areas of high metabolism or cell turnover, while SPECT identifies areas of increased blood perfusion (Jacobs et al., *NeuroRx* 2005; 2(2): 333-347).

**(4)**

MRI is the preferred imaging modality for evaluation of a suspected or known brain tumor. In addition to providing information on the size and location of the tumor, MRI may demonstrate mass effect, edema, hemorrhage, necrosis, or signs of increased intracranial pressure (Jacobs et al., *NeuroRx* 2005; 2(2): 333-347). If sufficient information is obtained from MRI to determine an appropriate management plan (e.g., resection, radiation therapy, stereotactic radiosurgery), then PET or SPECT is not necessary. If, however, surgical resection is planned and there are questions as to the extent of tumor involvement, PET or SPECT may be useful in the preoperative evaluation (Miletich, *Neurol Clin* 2009; 27(1): 61-88; Chandana et al., *Am Fam Physician* 2008; 77(10): 1423-1430). The decision to obtain a PET or SPECT is a matter of clinical judgement.

**(5)**

In patients who have undergone radiation therapy for treatment of a brain tumor, follow-up MRI may fail to distinguish radiation necrosis from possible tumor recurrence. Either recurrence or necrosis can present with worsening of baseline neurologic signs and symptoms or the development of new deficits (Hustinx et al., *Radiol Clin North Am* 2005; 43(1): 35-47; Jacobs et al., *NeuroRx* 2005; 2(2): 333-347). PET or SPECT may help make this distinction, particularly if resection or additional radiation therapy is being considered. Additionally, with the increased use of stereotactic radiosurgery, the incidence of radiation necrosis is likely to increase (Chen, *J Nucl Med* 2007; 48(9): 1468-1481). The decision to obtain a PET or SPECT is a matter of clinical judgement.

**(6)**

A seizure focus on PET or SPECT performed between episodes of seizures (interictal examinations) typically appears as hypoperfusion on the scan; ictal examinations (performed during seizures) will be seen as a focus of increased activity or hyperperfusion (Miletich, *Neurol Clin* 2009; 27(1): 61-88; Karis, *AJNR Am J Neuroradiol* 2008; 29(6): 1222-1224). MRI is the preferred modality for evaluation of seizures; should MRI be nondiagnostic, PET scanning with <sup>18</sup>F-fluorodeoxyglucose (FDG) may be considered. FDG PET is particularly helpful in evaluating complex partial seizures of the temporal and frontal lobes (Newberg and Alavi, *Radiol Clin North Am* 2005; 43(1): 79-92). The decision to obtain a PET or SPECT is a matter of clinical judgement.

**(7)**

Sensitivity for detecting relative temporal lobe hypometabolism with FDG PET in temporal lobe epilepsy ranges between 80% to 90% (Karis, *AJNR Am J Neuroradiol* 2008; 29(6): 1222-1224; Knowlton, *Epilepsy Behav* 2006; 8(1): 91-101). The specificity of FDG PET for earmarking the exact location and extent of the seizure focus is considerably less, due in part to more diffuse hypometabolism in the temporal lobe (Knowlton, *Epilepsy Behav* 2006; 8(1): 91-101).

**(8)**

While MRI is the preferred initial imaging study, not all epileptogenic foci can be localized accurately with this modality (Warwick, *Metab Brain Dis* 2004; 19(1-2): 113-123). In approximately 20% of patients with chronic focal seizures, pathology is not seen on inspection of high resolution MRI (Koepp and Woermann, *Lancet Neurol* 2005; 4(1): 42-53).

**(9)**

The goal of resection of the seizure foci is to remove the foci without causing significant functional impairment. When the primary seizure focus remains obscure, stereotactic placement of electrodes may be performed to assist with neurosurgical planning. Surgery may include resection or stereotactic ablation of the seizure foci (Kuzniecky, *NeuroRx* 2005; 2(2): 384-393; Newberg and Alavi, *Radiol Clin North Am* 2005; 43(1): 79-92; Chapell et al., *Evid Rep Technol Assess (Summ)* 2003; (77): 1-8).

**(10)**

Stereotactic radiosurgery is an emerging treatment for patients with intractable seizures. The procedure is performed when the location or extent of a lesion precludes open surgery, or as an alternative to open surgery in selected cases. A highly focused beam of radiation is aimed at the lesion. This is accomplished by heavy ion beams, the gamma knife, or a linear accelerator. This technique differs from conventional radiotherapy, which exposes large areas of intracranial tissue to broad fields of radiation (Romanelli and Ansel, *Lancet Neurol* 2006; 5(7): 613-620).

**(11)-DEF:**

Parkinson's disease is a progressive neurodegenerative disorder characterized by motor (e.g., resting tremor, rigidity, bradykinesia, gait disturbance, imbalance) and non-motor (e.g., cognitive impairment, disturbances in mood or impulse control) symptoms that can be present at all stages of the disease.

**(12)**

In the early stages of Parkinson's disease, there is loss of the dopaminergic neurons that project from the substantia nigra in the midbrain to the striatal complex (putamen and caudate nucleus) in the forebrain. This appears as striatal hypermetabolism on PET and SPECT scans. Early identification is important, as this state may be amenable to dopaminergic therapy (Miletich, *Neurol Clin* 2009; 27(1): 61-88; Piccini and Brooks, *Mov Disord* 2006; 21(12): 2035-2041; Piccini and Whone, *Lancet Neurol* 2004; 3(5): 284-290).

**(13)**

FDG PET and SPECT scans may assist in the early diagnosis of Parkinson's disease, as the cardinal features of the disease (rest tremor, bradykinesia and rigidity) are shared with other disorders. Additionally, PET may be used in guiding medical and interventional treatment (Miletich, *Neurol Clin* 2009; 27(1): 61-88; Eckert et al., *Neuroimage* 2005; 26(3): 912-921). While PET scan provides the highest resolution and sensitivity and has the ability to differentiate between normal and abnormal nigrostriatal innervation, it is less widely available and more costly than SPECT (Piccini and Brooks, *Mov Disord* 2006; 21(12): 2035-2041; Piccini and Whone, *Lancet Neurol* 2004; 3(5): 284-290). There currently is no consensus in the value of SPECT in the differential diagnosis of Parkinson's disease, despite widespread use (Vlaar et al., *BMC Neurol* 2007; 7: 27). The decision to obtain a PET or SPECT scan is a matter of clinical judgment.

**(14)**

CT and MRI imaging findings for suspected clinical Parkinsonism are usually nonspecific. The role of CT and MRI is often to confirm or eliminate differential diagnoses (Nandhagopal et al., *Neurology* 2008; 70(16 Pt 2): 1478-1488; Gallucci et al., *Radiol Clin North Am* 2008; 46(4): 799-817, vii).

**(15)**

The treatment of Parkinson's disease focuses on medication management with most drugs influencing the imbalance between dopamine and acetylcholine in the brain (Guttman et al., *CMAJ* 2003; 168(3): 293-301). The choice of medication should take into account patient age, clinical presentation, comorbidities, and lifestyle preferences (Schapira, *Arch Neurol* 2007; 64(8): 1083-1088; Horstink et al., *Eur J Neurol* 2006; 13(11): 1170-1185; National Institute for Clinical Excellence (NICE), Parkinson's disease. Clinical Guideline 35. 2006, 40).

**(16)**

Dopamine receptor agonists include pramipexole, ropinirole, and bromocriptine. Cabergoline is also used for the treatment of Parkinson's disease; studies found that it is well-tolerated and improved Parkinsonian symptomatology (Odin et al., *Acta Neurol Scand* 2006; 113(1): 18-24). Cabergoline is not, however, currently approved for such use in the United States.

**(17)**

MAOIs such as selegiline and rasagiline are used as adjunctive therapy in combination with levodopa for the treatment of Parkinson's disease and have been shown primarily to have a beneficial effect on motor fluctuations (Parkinson Study Group, *Arch Neurol* 2005; 62(2): 241-248; Miyasaki et al., *Neurology* 2002; 58(1): 11-17).

**(18)**

Catechol-O-methyltransferase (COMT) inhibitors (e.g., entacapone, tolcapone) block the metabolism of levodopa, increasing the amount of levodopa available to the brain. They are beneficial in patients with motor fluctuations and in patients with stable responses to levodopa. Entacapone is usually well-tolerated and dyskinesia, the most common side effect, is easily controlled with a reduction in the dose of levodopa (Brooks, *Neurology* 2004; 62(1 Suppl 1): S39-46). Entacapone is currently the COMT inhibitor of choice due to reports of liver toxicity associated with tolcapone (Dewey, *Neurology* 2004; 62(6 Suppl 4): S3-7).

**(19)**

Anticholinergic drugs include benztropine and trihexyphenidyl.

**(20)**

Amantadine has been used to improve the symptoms of early Parkinson's disease. It is not recommended as a drug of first choice as there is a lack of evidence of its efficacy and safety (National Institute for Clinical Excellence (NICE), Parkinson's disease. Clinical Guideline 35. 2006, 40; Crosby et al., *Cochrane Database Syst Rev* 2003; (1): CD003468).

**(21)-DEF:**

Huntington's chorea is a dominantly inherited neurodegenerative disorder that characteristically becomes manifest in midlife. Typically patients develop personality and behavioral changes, choreic movements, and slowly progressive dementia.

**(22)**

MRI abnormalities associated with Huntington's chorea in patients with moderate disability include atrophy of the putamen and caudate nucleus; the MRI may be normal in patients in the early stages of the disease. If MRI is nondiagnostic or normal, PET or SPECT may show metabolic abnormalities or changes in the striatal and extra-striatal regions, often before the onset of disabling symptoms (Harris et al., *Med Clin North Am* 2009; 93(2): 371-388; Montoya et al., *J Psychiatry Neurosci* 2006; 31(1): 21-29).

**(23)**

The diagnosis of Huntington's chorea can be made by genetic testing. If genetic testing cannot be performed because it is unavailable or the patient refuses testing, imaging is appropriate.

**(24)-DEF:**

Ataxia is incoordination or awkwardness in performance of a motor task. The term ataxia is often used to describe gait unsteadiness.

**(25)**

Progressive ataxia may be caused by a variety of neurodegenerative disorders including MS, Friedreich's ataxia, idiopathic sporadic cerebellar ataxia, tumors of the posterior fossa, and paraneoplastic cerebellar degeneration. While MRI provides excellent visualization of plaques typical of multiple sclerosis (MS) and may diagnose posterior fossa tumors, the differentiation of the remaining etiologies may be more difficult. If the MRI is nondiagnostic, PET may be helpful, as it will show hypometabolism in the cerebellum and brainstem for idiopathic sporadic cerebellar ataxia and will show hypermetabolism in the same areas for Friedreich's ataxia (Gilman et al., *Neurology* 2008; 71(9): 670-676).

**(26)**

Chronic mental status changes can have a number of etiologies, including metabolic disturbance, substance dependence, vitamin deficiency, and hereditary disease. Dementia is a specific type of chronic mental status change, without diminished awareness or altered consciousness. Prior to imaging, a thorough metabolic evaluation is indicated to assess for reversible or treatable etiologies. Although treatment of these disorders may not completely reverse cognitive dysfunction, they should be routinely screened for, and if present, treated. Depression and medication side effects also need to be excluded during the evaluation (Holsinger et al., *JAMA* 2007; 297(21): 2391-2404; National Institute for Health and Clinical Excellence (NICE), Dementia: supporting people with dementia and their carers in health and social care. Clinical guideline 42. 2006, 56 p.; Connelly and James, *Int J Geriatr Psychiatry* 2006; 21(1): 14-16). Imaging can exclude potentially treatable causes of dementia (e.g., subdural hematoma, brain tumor, normal pressure hydrocephalus) (Gallucci et al., *Radiol Clin North Am* 2008; 46(4): 799-817, vii; Whitwell and Jack, *Neurol Clin* 2007; 25(3): 843-857, viii).

**(27)**

PET scan may be useful in differentiating the cause of many common dementias which present with the nonacute onset of mental status changes. PET studies demonstrate characteristic patterns of hypometabolism, which may permit more precise diagnosis of Alzheimer's disease, Parkinson's disease with dementia, multi-infarct dementia, frontotemporal lobe dementia, and progressive supranuclear palsy (Whitwell and Jack, *Neurol Clin* 2007; 25(3): 843-857, viii; Newberg and Alavi, *Radiol Clin North Am* 2005; 43(1): 49-65; Coleman, *Neuroimaging Clin N Am* 2005; 15(4): 837-846, x).

**(28)**

In the patient with altered mental status, CT is mainly used to exclude other illnesses that may be amenable to treatment (e.g., brain tumors, subdural hematomas, hydrocephalus). MRI has increased specificity, improved tissue contrast, and the ability to detect focal temporal lobe abnormalities and subcortical vascular changes, as well as the ability to identify different patterns of atrophy (Waldemar et al., Eur J Neurol 2007; 14(1): e1-26; National Institute for Health and Clinical Excellence (NICE), Dementia: supporting people with dementia and their carers in health and social care. Clinical guideline 42. 2006, 56 p.).