American College of Radiology
ACR Appropriateness Criteria®

Clinical Condition: **Focal Neurologic Deficit**

Variant 1: **Single focal neurologic deficit, acute onset, stable or incompletely resolving.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with IV contrast	8	Both CT and MRI may be necessary. CT screens for suspected hemorrhage in the acute setting and MRI screens for infarction and masses.	O
CT head without IV contrast	8	Both CT and MRI may be necessary. CT screens for suspected hemorrhage in the acute setting and MRI screens for infarction and masses.	☢ ☢ ☢
MRI head without IV contrast	7		O
MRA head and neck without and with IV contrast	7		O
MRA head and neck without IV contrast	7		O
CTA head and neck with IV contrast	7		☢ ☢ ☢
CT head perfusion with IV contrast	7		☢ ☢ ☢
MRI head perfusion with IV contrast	7		O
CT head without and with IV contrast	5	If MRI is unavailable or contraindicated. Consider CT perfusion.	☢ ☢ ☢
CT head with IV contrast	4		☢ ☢ ☢
MR spectroscopy head without IV contrast	4		O
MRI functional (fMRI) head without IV contrast	3		O
Tc-99m HMPAO SPECT head	3	For problem solving in HIV/AIDS.	☢ ☢ ☢ ☢
Arteriography cervicocerebral	3	For problem solving.	☢ ☢ ☢
FDG-PET/CT head	2		☢ ☢ ☢ ☢
Thallium-201 SPECT head	2	For problem solving in HIV/AIDS.	☢ ☢ ☢ ☢
Rating Scale: **1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate**			***Relative Radiation Level**

Clinical Condition: **Focal Neurologic Deficit**

Variant 2: **Single focal neurologic deficit, acute onset, completely resolving.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with IV contrast	8	Both CT and MRI may be necessary. CT screens for suspected hemorrhage in the acute setting and MRI screens for infarction and masses.	O
CT head without IV contrast	8	Both CT and MRI may be necessary. CT screens for suspected hemorrhage in the acute setting and MRI screens for infarction and masses.	☢ ☢ ☢
MRI head without IV contrast	7		O
MRA head and neck without and with IV contrast	7		O
MRA head and neck without IV contrast	7		O
CTA head and neck with IV contrast	7		☢ ☢ ☢
CT head without and with IV contrast	6	If MRI is unavailable or contraindicated. Consider CT perfusion.	☢ ☢ ☢
CT head with IV contrast	4		☢ ☢ ☢
CT head perfusion with IV contrast	4		☢ ☢ ☢
MRI head perfusion with IV contrast	4		O
MRI functional (fMRI) head without IV contrast	3		O
MR spectroscopy head without IV contrast	3		O
Tc-99m HMPAO SPECT head	3	For problem solving in HIV/AIDS.	☢ ☢ ☢ ☢
Thallium-201 SPECT head	3	For problem solving in HIV/AIDS.	☢ ☢ ☢ ☢
Arteriography cervicocerebral	3	For problem solving.	☢ ☢ ☢
FDG-PET/CT head	1		☢ ☢ ☢ ☢
Rating Scale: **1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate**			***Relative Radiation Level**

Clinical Condition: **Focal Neurologic Deficit**

Variant 3: **Single focal neurologic deficit, acute onset, progressive.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with IV contrast	8	Both CT and MRI may be necessary. CT screens for suspected hemorrhage in the acute setting and MRI screens for infarction and masses.	O
MRI head without IV contrast	8	Both CT and MRI may be necessary. CT screens for suspected hemorrhage in the acute setting and MRI screens for infarction and masses.	O
CT head without IV contrast	8	Both CT and MRI may be necessary. CT screens for suspected hemorrhage in the acute setting and MRI screens for infarction and masses.	☢☢☢
MRA head and neck without and with IV contrast	7		O
MRA head and neck without IV contrast	7		O
CTA head and neck with IV contrast	7		☢☢☢
CT head perfusion with IV contrast	7		☢☢☢
MRI head perfusion with IV contrast	7		O
CT head without and with IV contrast	6	If MRI is unavailable or contraindicated. Consider CT perfusion.	☢☢☢
CT head with IV contrast	4		☢☢☢
MR spectroscopy head without IV contrast	4		O
MRI functional (fMRI) head without IV contrast	3		O
Tc-99m HMPAO SPECT head	3	For problem solving in HIV/AIDS.	☢☢☢☢
Thallium-201 SPECT head	3	For problem solving in HIV/AIDS.	☢☢☢☢
Arteriography cervicocerebral	3	For problem solving.	☢☢☢
FDG-PET/CT head	1		☢☢☢☢
Rating Scale: **1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate**			***Relative Radiation Level**

Clinical Condition: **Focal Neurologic Deficit**

Variant 4: **Single or multiple focal neurologic deficits, subacute onset, progressive or fluctuating.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with IV contrast	8		O
MRI head without IV contrast	8		O
CT head without IV contrast	7	Acute screening.	☢ ☢ ☢
MRA head and neck without and with IV contrast	6		O
MRA head and neck without IV contrast	6		O
CT head without and with IV contrast	6	If MRI is unavailable or contraindicated. Consider CT perfusion.	☢ ☢ ☢
CTA head and neck with IV contrast	6	For suspected vascular abnormality.	☢ ☢ ☢
CT head perfusion with IV contrast	5		☢ ☢ ☢
MRI head perfusion with IV contrast	5		O
CT head with IV contrast	4		☢ ☢ ☢
MR spectroscopy head without IV contrast	4	For selected cases.	O
MRI functional (fMRI) head without IV contrast	3		O
Tc-99m HMPAO SPECT head	3	For problem solving in HIV/AIDS.	☢ ☢ ☢ ☢
Thallium-201 SPECT head	3	For problem solving in HIV/AIDS.	☢ ☢ ☢ ☢
Arteriography cervicocerebral	3	For problem solving.	☢ ☢ ☢
FDG-PET/CT head	2		☢ ☢ ☢ ☢
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

Clinical Condition: **Focal Neurologic Deficit**

Variant 5: **Unexplained acute confusion or altered level of consciousness.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with IV contrast	8	Both CT and MRI may be necessary. CT screens for suspected hemorrhage in the acute setting and MRI screens for infarction and masses.	O
MRI head without IV contrast	8	Both CT and MRI may be necessary. CT screens for suspected hemorrhage in the acute setting and MRI screens for infarction and masses.	O
CT head without IV contrast	8	Both CT and MRI may be necessary. CT screens for suspected hemorrhage in the acute setting and MRI screens for infarction and masses.	☢☢☢
MRA head and neck without and with IV contrast	6		O
MRA head and neck without IV contrast	6		O
CTA head and neck with IV contrast	6	For suspected vascular abnormality.	☢☢☢
CT head without and with IV contrast	5	If MRI is unavailable or contraindicated. Consider CT perfusion.	☢☢☢
CT head with IV contrast	4		☢☢☢
MRI functional (fMRI) head without IV contrast	3		O
MR spectroscopy head without IV contrast	3		O
FDG-PET/CT head	3		☢☢☢☢
Tc-99m HMPAO SPECT head	3		☢☢☢☢
Thallium-201 SPECT head	3		☢☢☢☢
CT head perfusion with IV contrast	3		☢☢☢
MRI head perfusion with IV contrast	3		O
Arteriography cervicocerebral	2		☢☢☢
Rating Scale: **1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate**			***Relative Radiation Level**

FOCAL NEUROLOGIC DEFICIT

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Summary of Literature Review

Introduction/Background

A focal neurological deficit consists of a set of symptoms or signs in which causation can be localized to an anatomic site within the central nervous system (CNS). The presumed site of the pathologic abnormality within the brain or spinal cord is typically deduced by thoughtful consideration of the patient's history and physical examination prior to imaging. The clinical localization of a suspected lesion is extremely useful (and should be encouraged on the part of the examining physician) in that it assists the radiologist in directing the imaging portion of the evaluation. Focal neurological deficits may develop suddenly or may evolve chronically. Once a deficit occurs, it may remain stable, worsen in a continuous or step-like fashion, or resolve. Resolution may be partial or complete.

Additionally, deficits may be unifocal, implying a single lesion, or multifocal, suggesting multiple discrete lesions. A patient presenting with a focal neurological deficit should be considered for imaging of the appropriate portion of the neuraxis. The presentation may suggest causation. For example, an acute temporal course prompts evaluation for cerebral infarction, but a more chronically progressive course is often due to a mass lesion. Specific disease entities are fully reviewed in separate ACR Appropriateness Criteria® topics. The patient who presents with a focal disorder of motor or sensory function caused by intracranial pathology is addressed in this summary.

Imaging Modalities

Many imaging tools are available to the clinician and radiologist for evaluating the focal neurological deficit. Application of these modalities largely depends on the presumptive or working diagnosis, the urgency of the clinical problem, availability of the modality, and comorbidities of the patient. Modalities such as computed tomography (CT) and magnetic resonance imaging (MRI) may be used as a first-line screening or definitive examination and tend to provide useful and frequently diagnostic anatomic information, whereas modalities such as positron emission tomography (PET), single-photon-emission computed tomography (SPECT), and cervicocerebral catheter angiography are usually reserved for solving more challenging clinical problems. Newer perfusion applications of both CT and MRI have also been proving useful for patients with conditions such as tumors and cerebrovascular disease.

Acute Focal Neurological Deficit

The sudden development of a single focal neurological deficit (Variant 1) suggests a vascular ischemic event such as an infarction. Infarctions typically remain stable in the immediate period of presentation or worsen due to complicating hemorrhage, edema, or infarction extension after hemorrhage (Variant 3). A deficit from a transient ischemic attack resolves within 24 hours (Variant 2). Neurologic deficits from acute reversible ischemia may take up to 30 days to completely resolve.

CT scanning is often used to screen patients for suspected infarction, but it may miss early cytotoxic edema. An obscured insular ribbon and a dense middle artery are signs indicating infarction but may be absent in a given patient. Diffusion-weighted imaging (DWI) MRI detects cytotoxic edema in the first few hours of an infarction and may remain positive for a week to 10 days. Spin-echo sequences before and after intravenous enhancement may add significant information as the infarction evolves. During the acute presentation, CT and MR perfusion may add information regarding the ischemic penumbra of an infarction [1,2]. CT angiography (CTA) may also be

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useful in evaluating the intra- and extra-cranial blood vessels. Catheter angiography is generally reserved for problem-solving. A detailed summary of ischemic vascular disease is in the ACR Appropriateness Criteria® on "Cerebrovascular Disease."

An intracerebral hemorrhage may also cause sudden onset of focal findings. The clinical examination may help to define the cause of the hemorrhage. A third cranial nerve palsy with associated abnormal pupillary function and headache, for example, suggests subarachnoid hemorrhage due to aneurysm rupture. Sudden hemiparesis in the setting of hypertension suggests a hemorrhage in the basal ganglia. CT is generally the preferred modality for initial screening for intracranial hemorrhage because of its availability, rapid scanning time, and sensitivity in detecting blood [3,4]. The newly identified "spot sign" described in patients undergoing CTA in the acute setting may be a useful indicator for prognosis [5,6]. MRI has been found to be sensitive for both acute and chronic blood products and, when available, can exclude hemorrhage in patients with a suspected infarction before intravenous administration of tissue plasminogen activator (tPA) [7]. Moreover, MRI has been shown to be superior to CT in detecting acute petechial hemorrhagic transformation in acute ischemic stroke. Kidwell et al [8] showed that with appropriate sequence selection, acquisition time of an MRI can be significantly decreased to about 10 to 15 minutes.

Traumatically induced or spontaneous subdural and epidural hematomas may also produce acute focal deficits. CT is the modality of choice for screening patients with suspected extra-axial hemorrhage and for detecting fractures. Surface renderings from CT data may also aid in diagnosis of the latter [9,10].

Rarely, Tc-99m HMPAO SPECT may be used for problem-solving in challenging cases of focal neurological deficit, especially in HIV/AIDS patients.

Subacute Progressive Focal Neurologic Deficit

Progressively worsening focal neurological deficits of subacute onset may be caused by an expanding intracranial lesion such as a primary or metastatic neoplasm (Variant 4). Subacute but more rapidly developing symptoms may be caused by an infectious lesion or expanding vascular malformation. Primary and secondary neoplasms and abscesses may produce progressive weakness, impaired speech, personality change, or a sensory deficit, depending on the location within the brain. Hemiplegia is the most common form of paralysis. Monoplegia and, less commonly, bilateral extremity weakness may also be caused by an intracranial process, although spinal cord and brain stem lesions should also be considered depending on the neurological examination. The cardinal signs of a mass lesion include headache, vomiting, and papilledema. This triad is usually caused by obstructive hydrocephalus or marked peritumoral vasogenic edema. Cranial nerve deficits accompanying contralateral weakness localize pathology to the brainstem.

Imaging studies are performed primarily to detect an intracranial mass lesion, whether neoplastic, infectious, or vascular, and to characterize the offending pathology. Ideally, imaging evaluation should be performed after the patient has undergone a physical examination in order to best direct the imaging protocol and even to clinically exclude a possible extracranial cause for the symptomotology.

CT is invaluable for detecting intracranial tumors, infections, and vascular lesions. A retrospective review by Brown et al [11] found that 20% of elderly patients (>70 years of age) presenting with neurological deficits had treatable lesions discovered with CT. The cohort most affected by the CT imaging was the group with neurological signs that were atypical of stroke and with unexplained confusion or altered sensorium.

Contrast agents yield additional information on CT. An increase in the iodine dose or scan delay time may reveal new lesions and can further increase the conspicuity of some lesions, sometimes yielding supplementary diagnostic information. Current-generation scanners have significantly improved sensitivity; however, some pathology is difficult to visualize with CT under any circumstances. This is especially true for white-matter disease and other lesions that may not produce significant mass effect. Also, compared with its ability to detect intraparenchymal lesions, CT is not as reliable for delineating leptomeningeal or dural disease. Moreover, it is unlikely to be of any benefit in atraumatic patients with neurological deficits that have completely resolved at the time of imaging.

Contrast-enhanced MRI is more sensitive than CT for detecting primary and secondary brain lesions and for defining the extent of disease. Even before the availability of MRI contrast agents, this modality surpassed CT in sensitivity for detecting intraparenchymal pathology. In addition to superior contrast resolution, MRI spares patients exposure to potentially damaging ionizing radiation. It also provides information that is unavailable by other noninvasive means, and sometimes it approaches the accuracy of a neuropathologic diagnosis. Intravenous gadolinium contrast especially increases the detection of intracranial metastatic disease. Contrast agents aid the

characterization of primary brain tumors, but they may not be essential for screening examinations. Stratification of patients who should receive contrast based on age may be beneficial. Metastatic disease affects all age groups, but the incidence increases significantly after the fourth decade. More than 75% of patients harboring metastases in the CNS are between 40 and 70 years of age. Gadolinium is better tolerated than iodine, so some centers follow an unenhanced CT scan with an unenhanced and enhanced MR scan. Although high-dose enhanced MRI results in increased lesion contrast, apparent size, and border definition compared with single-dose examinations [12], concern about nephrogenic systemic fibrosis has dampened enthusiasm for routine use of double- or triple-dose contrast scans, especially in patients with renal disease. Use of agents with greater relaxivity, such as gadobenate dimeglumine, may improve conspicuity of lesions at acceptable dose levels [13].

MRI is especially useful for evaluating the posterior fossa, a region often less well visualized with CT because of artifact. A posterior fossa mass is suspected in patients presenting with increased intracranial pressure, cerebellar signs, and/or cranial nerve deficits. Brain stem pathology is a potential source of concomitant extremity and cranial nerve deficits. Neoplasms, vascular lesions, and occasionally infections may involve the pons, midbrain, or medulla. Up to 22% of cavernous malformations occur in the brainstem. MRI is superior not only for detecting brain stem lesions but also for characterizing hemorrhagic residua. Brain stem ischemia typically occurs in older adults, but it may rarely affect children. Suspected brain stem and other posterior fossa pathologies argue strongly for MRI over CT because of CT artifact caused by adjacent bony structures. Enhanced MRI is also the modality of choice for patients with cranial neuropathy. (See the ACR Appropriateness Criteria® on "Cranial Neuropathy.")

While CT may be preferable for evaluating bony trauma, acute subarachnoid blood, and some head and neck disorders, MRI has become the modality of choice for most CNS disorders. Of course, nonavailability of MRI, MR-incompatible life support apparatus, ferromagnetic aneurysm clips, and other contraindications to MRI will prompt CT, even for diseases best evaluated with MRI. Hemorrhagic lesions are characterized more accurately with MRI. Although it is often impossible to distinguish tumoral hemorrhage from other causes on CT, features are often detected on MRI that suggest an underlying malignancy. Although CT is more sensitive for detecting small calcifications associated with vascular malformations, MRI is more sensitive for detecting the small hemorrhagic foci commonly associated with vascular malformations, and it provides a more specific imaging appearance.

Despite the exceptionally good tissue contrast resolution of MRI, the anatomic images may be insufficient for neurosurgeons who are contemplating resection of a lesion that borders eloquent cortex. Distortion of the motor strip and other vital parenchyma may occur secondary to an expanding adjacent mass. The functional plasticity of the brain may not be reflected on conventional anatomic imaging studies. Preoperative (or preradiation) functional MRI (fMRI) for mapping of eloquent cortex more precisely delineates motor and speech areas and may contribute to surgical and treatment planning [14]. Such studies may supplant or accompany intraoperative neurophysiological testing for mapping the motor strip prior to resection of brain tumors. Additional functional information can be provided by diffusion tensor tractography. This method is being used in some centers for mapping the deflection of fibers carrying eloquent signals in the vicinity of the contemplated surgical bed [15,16]. Such functional studies may also obviate amytal testing.

In previously treated patients with brain neoplasms presenting with new neurological complaints, distinguishing radiation necrosis from tumor recurrence is a diagnostic challenge. These lesions, which may have a similar appearance on enhanced MRI, call for significantly different clinical management. Nuclear medicine SPECT or PET studies may provide improved specificity. However, these modalities are not universally reliable for making this distinction [17]. MR spectroscopy with perfusion may also prove useful for distinguishing radiation necrosis from tumor recurrence. Catheter angiography has traditionally been used to assess tumor vascularity. More recently, evaluation of tumor vascularity using dynamic MRI has been validated [18].

Localized infection may also produce focal neurological signs and symptoms. Neurological deficits due to infection tend to evolve more quickly than those due to tumor. Patients with parenchymal infectious lesions often have no fever or other systemic signs of infection, and may have a normal cerebrospinal fluid (CSF) profile; if fever is present, it is nonspecific. Brain abscesses may result from a wide variety of organisms, including gram-positive and gram-negative bacteria and various fungi. Blood-borne abscesses may develop in the brain as a result of cyanotic heart disease, pulmonary anterior-venous fistula, or bacterial endocarditis. Direct spread of organisms may also result in brain abscesses as a complication of sinusitis, chronic otitis or mastoiditis, and post-traumatic or congenital transgression of the dura. Intracerebral abscesses may also develop by direct venous spread from extradural infections. An early diagnosis of a brain abscess or its earlier stage of "cerebritis" guides appropriate

treatment, including the careful selection of antibiotics, drainage of the abscess cavity, and correction of the original source of the infection, particularly if the abscess is secondary to sinus or middle ear infection.

Since the introduction of CT, the overall mortality rate due to abscesses has decreased from more than 40% to less than 5%. The CT appearance of infectious masses has been well described. Earlier detection in combination with improved therapeutic measures for intracranial infections has produced a significant decrease in complications such as extension to extra-axial spaces, hemorrhage, infarction, compartmental herniation, and death. Although it is less sensitive for detecting small calcifications, MRI provides greater sensitivity for assessing intracranial abscess and granulomas, and may be more specific. However, even in endemic areas, the imaging appearance of such lesions is not specific enough to obviate histological confirmation before treatment.

Contrast-enhanced images augment the sensitivity of CT and noncontrast MR brain imaging. The efficacy of enhanced MRI scans has been demonstrated in children and adults. MRI is superior to CT for evaluating parenchymal abscesses and their complications. It is also more sensitive for evaluating extra-axial infection. MRI demonstrates almost pathognomonic findings in a mature abscess due to the shortening of the T1 and T2 relaxation times in the abscess wall, resulting in hyperintensity on T1-weighted and hypointensity on T2-weighted images. DWI MRI may allow differentiation of brain abscess from necrotic or cystic brain tumors [19]. The ring configuration seen in tumor on spin-echo sequences aids in differentiating the finding from the solid, central restricted diffusion seen in an abscess. The restricted diffusion found in extradural epidermoids may be confused with empyema, but correlation with spin-echo images and clinical findings is useful [20].

MRI, and particularly MR venography (MRV), may also be useful for demonstrating secondary venous occlusive disease, a frequent complication of chronic mastoiditis with superimposed acute infection. Despite advances in MRV, catheter cerebral angiography remains the gold standard.

CT is considered superior for demonstrating bone abnormalities in inflammatory ear disease and may also provide useful additional information in cases of sinusitis. CT remains the standard modality for diagnosing sinusitis, but MRI is often necessary to exclude intracranial complications of sinusitis such as meningitis or abscess [21]. CT or MRI is also necessary for stereotactic aspiration of abscess cavities [22,23]. MR spectroscopy may be useful for demonstrating abscesses, because specific resonance peaks have been shown in the contents in the abscess [24]. Conspicuity may be further enhanced by magnetization transfer imaging techniques, although the latter have not been widely adopted in everyday practice.

Patients infected with human immunodeficiency virus (HIV) and those with acquired immunodeficiency syndrome (AIDS) exhibiting focal neurological symptoms should undergo cranial imaging in order to guide clinical management. In addition to contributing to clinical management, imaging findings also have prognostic implications in AIDS patients. The presence of focal lesions or atrophy significantly increases the risk of death in patients with AIDS when compared to AIDS patients with normal neuroimaging examinations. The risk is even greater if both focal lesion and atrophy are present. The treatment for the most common intracranial lesions in these patients must be instituted promptly. MRI is superior to CT for detecting white-matter lesions and vasogenic edema. Despite the excellent ability of MRI to delineate lesions, distinguishing between lesions caused by toxoplasmosis and primary CNS lymphoma is often difficult on the basis of anatomic imaging alone. Some MRI features may favor one diagnosis over the other, but the distinction is often difficult. Although enhanced images have been shown to provide additional information in AIDS patients who present for cranial MRI, the value of routine use of gadolinium contrast agents in AIDS patients has been challenged.

Thallium-201 uptake of lymphoma may be exploited by performing SPECT on AIDS patients presenting with intracranial lesions. Characterizing biochemical profiles of lesions using H-1 spectroscopy may provide another noninvasive and more specific method for differentiating these lesions [25]. Additional information may be obtained from perfusion MRI. Reduced regional cerebral blood volume (rCBV) in toxoplasmosis lesions has been described and compared with increased rCBV in lymphomas, thus allowing differentiation of mass lesions in AIDS patients caused by these diseases [26].

Chronic subdural hematomas may also produce a stepwise progressive neurological deficit if repetitive rebleeding has occurred. CT is the modality of choice for screening in this circumstance.

Fluctuating Focal Neurological Deficit

Focal neurological deficits that have a stuttering course or localize to multiple locations may be clinically challenging. One etiology is demyelination, most commonly caused by multiple sclerosis (MS) (Variant 4). MS is an inflammatory disease that primarily affects central myelin, secondarily injuring axons and their neurons of origin [27,28]. Although the mechanisms of injury are still being clarified, MS is considered an organ-specific

autoimmune disease [29,30]. Through a variety of possible mechanisms, including viral infection, a clone of T-cell lymphocytes becomes sensitized to specific myelin peptides. Relapses occur when the activated T-cell lymphocytes increase endothelial cell permeability and recruit macrophages, astrocytes, and other cells to cause focal inflammation and myelin destruction [31]. The management of this disorder has been radically changed recently by the availability of drugs that are effective in improving the natural course of the relapsing-remitting form [29,31,32].

When considering the appropriateness of imaging procedures for diagnosing MS, important factors include: 1) the likelihood that a given clinical presentation represents demyelinating disease or other disorder that can be imaged, and 2) the likelihood that the use of an imaging modality will change the management of the disorder. Up to 40% of patients with proven MS first present with paresthesias or other vague sensory symptoms. Pain can also be the first symptom. These patients often have negative MRI of the brain and spinal cord. Pursuing imaging beyond the standard screening MRI may not be indicated.

The sensitivity of CT of the brain for MS is low. Indirect findings, such as areas of hypodensity or brain atrophy, appear late in the disease and are nonspecific. MRI revolutionized the diagnosis and management of MS, which previously was diagnosed solely by clinical criteria and CSF analysis. In a study comparing high-field MRI (1.5T) to low-field MRI (0.23T), Ertl-Wagner et al [33] showed that high-field studies are far superior for diagnosing MS. As promising new therapies have become available, surveillance with MRI exceeds the neurological examination in sensitivity to disease activity [34,35]. Sequences such as fluid-attenuated inversion recovery (FLAIR) with fast-spin-echo acquisition and short T1 inversion recovery (STIR) have greatly improved lesion detection.

Brain MRI has been used in large therapeutic trials to monitor MS disease activity [36,37]. In relapsing-remitting and secondary progressive MS, serial T2-weighted MRI reveals 3-10 times as many new lesions as there are clinical relapses [38]. Gadolinium enhancement further increases the reliability and sensitivity of detecting active lesions [38]. In relapsing-remitting and secondary progressive MS, the presence of enhancement is more frequent during relapse and correlates well with clinical activity [39]. Enhancement is rare in primary progressive MS [40]. In benign MS, with a slow progression and little disability, enhancing lesions are also rare [41]. Delayed scanning and magnetization transfer may improve sensitivity [40,42].

MR spectroscopy may help clarify the pathophysiology underlying the diverse varieties of MS. Metabolic changes have been observed on MR spectroscopy before the appearance of lesions on MRI, but these applications have little utility in clinical practice at this time [43].

Unexplained Acute Confusion or Altered Level of Consciousness

Although confusion and altered level of consciousness are not considered focal neurological phenomena, these presentations are all too common in the field of emergency medicine and will be briefly discussed here (Variant 5). Impaired consciousness may range from mild inattention and clouding of the sensorium to stupor and coma. Substance intoxication, trauma, and cerebrovascular disease are frequent causes of coma in the typical large urban hospital. Unenhanced CT remains a reliable screening modality in this setting and is especially good when hemorrhage is suspected. Certainly, further evaluation with MRI including DWI may be useful. MRI better characterizes infarction and masses as previously noted. In cases of metabolic derangement such as uncontrolled diabetes mellitus or alcoholism, CT may evaluate for suspected trauma that may be associated with the metabolic condition. Advanced imaging studies such as fMRI, MR spectroscopy, FDG-PET, Tc-99m HMPAO SPECT, and thallium-201 SPECT have little use in the acute setting, but may have limited application as problem-solving techniques.

Summary

- Focal neurologic deficits typically localize to an anatomic site within the CNS and often point to a specific etiology such as ischemic cerebrovascular disease, hemorrhage, tumor, or abscess.
- Imaging evaluation of focal neurologic deficits is best performed in concert with a detailed clinical assessment.
- CT and MRI may be used as a first-line screening or definitive examination and tend to provide useful and frequently diagnostic anatomic information.
- Modalities such as PET, SPECT, and cervicocerebral catheter angiography are usually reserved for solving more challenging clinical problems.

Relative Radiation Level Information

Potential adverse health effects associated with radiation exposure are an important factor to consider when selecting the appropriate imaging procedure. Because there is a wide range of radiation exposures associated with different diagnostic procedures, a relative radiation level (RRL) indication has been included for each imaging examination. The RRLs are based on effective dose, which is a radiation dose quantity that is used to estimate population total radiation risk associated with an imaging procedure. Patients in the pediatric age group are at inherently higher risk from exposure, both because of organ sensitivity and longer life expectancy (relevant to the long latency that appears to accompany radiation exposure). For these reasons, the RRL dose estimate ranges for pediatric examinations are lower as compared to those specified for adults (see Table below). Additional information regarding radiation dose assessment for imaging examinations can be found in the ACR Appropriateness Criteria® Radiation Dose Assessment Introduction document.

Relative Radiation Level Designations		
Relative Radiation Level*	**Adult Effective Dose Estimate Range**	**Pediatric Effective Dose Estimate Range**
O	0 mSv	0 mSv
☢	<0.1 mSv	<0.03 mSv
☢ ☢	0.1-1 mSv	0.03-0.3 mSv
☢ ☢ ☢	1-10 mSv	0.3-3 mSv
☢ ☢ ☢ ☢	10-30 mSv	3-10 mSv
☢ ☢ ☢ ☢ ☢	30-100 mSv	10-30 mSv
*RRL assignments for some of the examinations cannot be made, because the actual patient doses in these procedures vary as a function of a number of factors (eg, region of the body exposed to ionizing radiation, the imaging guidance that is used). The RRLs for these examinations are designated as "Varies".		

Supporting Documents

For additional information on the Appropriateness Criteria methodology and other supporting documents go to www.acr.org/ac.

References

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