

**American College of Radiology
ACR Appropriateness Criteria®**

Clinical Condition: Focal Neurologic Deficit

Variant 1: Multiple focal neurologic deficits.

Radiologic Procedure	Rating	Comments	RRL*
MRI head without or without and with contrast	8	Consider increased contrast dose for problem solving in selected cases. Include diffusion weighted imaging. See comments regarding contrast in text under "Anticipated Exceptions."	None
CT head without contrast	7	Acute screening.	Med
MRA head and neck without or without and with contrast	6	For suspected vascular abnormality. See comments regarding contrast in text under "Anticipated Exceptions."	None
CT head without and with contrast	6	If MRI is unavailable or contraindicated. Consider CT perfusion.	Med
CTA head and neck	6	For suspected vascular abnormality.	Med
MRI spectroscopy head	4	For selected cases.	None
MRI functional (fMRI) head	3		None
NUC Tc-99m HMPAO SPECT head	3	For problem solving in HIV/AIDS.	High
NUC thallium-201 SPECT head	3	For problem solving in HIV/AIDS.	High
INV arteriography cervicocerebral	3	For problem solving.	Med
FDG-PET head	2		High
X-ray head	1		Min
<u>Rating Scale:</u> 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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Clinical Condition:**Focal Neurologic Deficit****Variant 2:****Single focal neurologic deficit, sudden onset, stable, or incompletely resolving.**

Radiologic Procedure	Rating	Comments	<u>RRL*</u>
MRI head without or without and with contrast	8	Include diffusion-weighted imaging. Both CT and MR may be necessary. CT screens for suspected hemorrhage in the acute setting and MR screens for infarction and masses. See comments regarding contrast in text under "Anticipated Exceptions."	None
CT head without contrast	8	Both CT and MR may be necessary. CT screens for suspected hemorrhage in the acute setting and MR screens for infarction and masses.	Med
MRA head and neck without or without and with contrast	7	See comments regarding contrast in text under "Anticipated Exceptions."	None
CTA head and neck	7		Med
CT head without and with contrast	5	If MRI is unavailable or contraindicated. Consider CT perfusion.	Med
MRI spectroscopy head	4		None
MRI functional (fMRI) head	3		None
NUC Tc-99m HMPAO SPECT head	3	For problem solving in HIV/AIDS.	High
INV arteriography cervicocerebral	3	For problem solving.	Med
FDG-PET head	2		High
NUC thallium-201 SPECT head	2	For problem solving in HIV/AIDS.	High
X-ray head	1		Min
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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Clinical Condition:**Focal Neurologic Deficit****Variant 3:****Single focal neurologic deficit, sudden onset, progressive.**

Radiologic Procedure	Rating	Comments	<u>RRL*</u>
MRI head without or without and with contrast	8	Include diffusion-weighted imaging. Both CT and MR may be necessary. CT screens for suspected hemorrhage in the acute setting and MR screens for infarction and masses. See comments regarding contrast in text under "Anticipated Exceptions."	None
CT head without contrast	8	Both CT and MR may be necessary. CT screens for suspected hemorrhage in the acute setting and MR screens for infarction and masses.	Med
MRA head and neck without or without and with contrast	7	See comments regarding contrast in text under "Anticipated Exceptions."	None
CTA head and neck	7		Med
CT head without and with contrast	6	If MRI is unavailable or contraindicated. Consider CT perfusion.	Med
MRI spectroscopy head	4		None
MRI functional (fMRI) head	3		None
NUC Tc-99m HMPAO SPECT head	3	For problem solving in HIV/AIDS.	High
NUC thallium-201 SPECT head	3	For problem solving in HIV/AIDS.	High
INV arteriography cervicocerebral	3	For problem solving.	Med
X-ray head	1		Min
FDG-PET head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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Clinical Condition:**Focal Neurologic Deficit****Variant 4:****Single focal neurologic deficit, completely resolving.**

Radiologic Procedure	Rating	Comments	<u>RRL*</u>
MRI head without or without and with contrast	8	Include diffusion-weighted imaging. Both CT and MR may be necessary. CT screens for suspected hemorrhage in the acute setting and MR screens for infarction and masses. See comments regarding contrast in text under "Anticipated Exceptions."	None
CT head without contrast	8	Both CT and MR may be necessary. CT screens for suspected hemorrhage in the acute setting and MR screens for infarction and masses.	Med
MRA head and neck without or without and with contrast	7	See comments regarding contrast in text under "Anticipated Exceptions."	None
CTA head and neck	7		Med
CT head without and with contrast	6	If MRI is unavailable or contraindicated. Consider CT perfusion.	Med
MRI functional (fMRI) head	3		None
MRI spectroscopy head	3		None
NUC Tc-99m HMPAO SPECT head	3	For problem solving in HIV/AIDS.	High
NUC thallium-201 SPECT head	3	For problem solving in HIV/AIDS.	High
INV arteriography cervicocerebral	3	For problem solving.	Med
X-ray head	1		Min
FDG-PET head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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Clinical Condition:**Focal Neurologic Deficit****Variant 5:****Unexplained acute confusion or altered level of consciousness.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without or without and with contrast	8	Include diffusion-weighted imaging. Both CT and MR may be necessary. CT screens for suspected hemorrhage in the acute setting and MR screens for infarction and masses. See comments regarding contrast in text under "Anticipated Exceptions."	None
CT head without contrast	8	Both CT and MR may be necessary. CT screens for suspected hemorrhage in the acute setting and MR screens for infarction and masses.	Med
MRA head and neck without or without and with contrast	6	For suspected vascular abnormality. See comments regarding contrast in text under "Anticipated Exceptions."	None
CTA head and neck	6	For suspected vascular abnormality.	Med
CT head without and with contrast	5	If MRI is unavailable or contraindicated. Consider CT perfusion.	Med
MRI functional (fMRI) head	3		None
MRI spectroscopy head	3		None
FDG-PET head	3		High
NUC Tc-99m HMPAO SPECT head	3		High
NUC thallium-201 SPECT head	3		High
INV arteriography cervicocerebral	2		Med
X-ray head	1		Min
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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FOCAL NEUROLOGIC DEFICIT

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Summary of Literature Review

A focal neurological deficit consists of a set of symptoms or signs in which causation can be localized to an anatomic site in the central nervous system. The site of the pathologic abnormality is typically deduced through the 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, continue to 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 entire 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.

Acute Focal Neurological Deficit

The sudden development of a focal neurological deficit 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 or edema. A deficit from a transient ischemic attack resolves within 24 hours. Neurologic deficits from acute reversible ischemia may take up to 30-days to

completely resolve. Computed tomography (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 (DW) magnetic resonance imaging (MRI) detects cytotoxic edema in the first few hours of an infarction and may remain positive for a week to ten days. Spin echo sequences before and after intravenous enhancement may add significant information as the infarction evolves. A detailed summary of ischemic vascular disease is the subject of another Appropriateness Criteria[®] topic.

An intracerebral hemorrhage may also cause sudden onset of focal findings. The clinical examination may help to define the cause of the hemorrhage. A pupil involving third cranial nerve palsy associated with 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 [1,2]. Recently, 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) [3]. Moreover, MRI has been shown to be superior to CT in detecting acute petechial hemorrhagic transformation in acute ischemic stroke. Kidwell et al [4] 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 for suspected extra-axial hemorrhage.

Chronic Progressive Focal Neurologic Deficit

Chronically worsening focal neurological deficits may be caused by an expanding intracranial lesion such as a primary or metastatic neoplasm. Subacute or more rapidly developing symptoms may be caused by an infectious lesion. 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 weakness may also be caused by an intracranial mass lesion. The latter is usually caused by cord compromise, but occasionally brain stem or cerebral pathology produces bilateral symptomatology. The cardinal signs of a mass lesion include headache,

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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 exclude an intracranial mass lesion, whether neoplastic, infectious, or vascular, and to characterize the offending pathology. These patients should undergo imaging evaluation after physical examination.

CT is invaluable for detecting intracranial tumors, infections, and vascular lesions. A retrospective review by Brown et al [5] 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.

It is well established that contrast agents yield additional information on CT. An increase in the iodine dose can 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.

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 allows multiplanar acquisition and 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. Whereas contrast agents allow the detection of metastases that are occult on unenhanced studies, virtually all primary brain neoplasms seen on enhanced images will also be identified on unenhanced sequences. 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 central nervous system (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.

High-dose enhanced MRI results in increased lesion contrast, apparent size, and border definition compared with single-dose examinations [6]. The administration of triple-dose MR contrast agents often reveals more lesions than does a single dose [6]. High-dose MRI is more sensitive for detecting intracerebral metastases than delayed standard-dose MRI [6]. Because there is evidence that resection of a solitary metastatic lesion (or a small number of lesions) improves patient survival, detection of a solitary lesion versus multiple lesions is likely to impact patient management. There is little argument that patients considered for surgical resection of a solitary metastatic nodule detected on noncontrast MRI studies or enhanced CT should undergo an enhanced MRI examination to exclude the presence of additional lesions. Certain patients may benefit from triple-dose contrast.

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 of brain stem lesions, but also for characterizing hemorrhagic residua. Brain stem ischemia is not uncommon in older adults, and it may rarely occur in 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.

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 central nervous system 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 MR. 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.

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Despite the high 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 for mapping of eloquent cortex more precisely delineates motor and speech areas and may contribute to surgical and treatment planning [7]. 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 [8,9]. Such functional studies may also obviate amygdala 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 single-photon emission computed tomography (SPECT) or positron emission tomography (PET) studies may provide improved specificity. However, these modalities are not universally reliable for making this distinction [10]. MR spectroscopy 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 [11].

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 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 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. Diffusion-weighted MRI may allow differentiation of brain abscess from necrotic or cystic brain tumors [12]. 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 [13].

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 [14]. CT or MRI is also necessary for stereotactic aspiration of abscess cavities [15,16]. MR spectroscopy may be useful for demonstrating abscesses, because specific resonance lines have been shown in the contents in the abscess [17]. Several studies have suggested the value of triple-dose contrast with MRI for increasing the conspicuity of abscesses. Conspicuity may be further enhanced by magnetization transfer imaging techniques, although the latter have not been widely

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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 [18]. 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 [19].

Chronic subdural hematomas may also produce a step-wise 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 cause is demyelination, most commonly caused by multiple sclerosis (MS). MS is an inflammatory disease that primarily affects central myelin, secondarily injuring axons and their neurons of origin [20,21]. Although the mechanisms of injury are still being clarified, MS is considered an organ-specific autoimmune disease [22,23]. 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 [24]. Recently, the management of this disorder has been radically changed by the availability of drugs that are effective in improving the natural course of the relapsing-remitting form [22,24,25].

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. Poorly detected by CT, MS is clearly depicted by MRI. In a study comparing high-field MRI (1.5T) to low-field MRI (.23T), Ertl-Wagner et al [26] showed that high-field studies are far superior for diagnosing MS. As promising new therapies for MS were evaluated in the early 1990s, it became clear that MRI was more sensitive to disease activity than the neurological evaluation, thus allowing for smaller sample sizes and, thereby, for more economical and faster therapeutic trials [27,28].

Because of its greater sensitivity for detecting edematous lesions next to CSF-filled spaces, fluid-attenuated inversion recovery (FLAIR) with fast spin-echo acquisition is quickly becoming a standard sequence in clinical MRI. In earlier studies, FLAIR images were found to be more sensitive for cord MS lesions than conventional T2-weighted images.

Large studies on the sensitivity and specificity of MRI for MS have used conventional MRI sequences. In a study of 303 patients referred because of the suspicion of MS, a "definite MS" reading on an MRI of the head was specific for MS (likelihood ratio: 24.9) and established the diagnosis, especially in patients clinically designated as probable MS before testing. However, MRI of the head was negative for MS in 25% and equivocal in 40% of the patients considered to have MS by the diagnostic review committee reviewing each patient's course after a 6-month follow-up. Studies of clinically definite MS yielded sensitivity for MRI of 70%-83%. Many of the patients with negative brain studies may have had spinal cord lesions that were undetected because the spinal cord was not systematically surveyed. In a group of 170 MS patients with symptoms and signs referable to the spinal

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cord or optic nerves, 20 (12%) had normal brain MRI [29]. Patients with a myelopathy often have brain lesions on MRI. Even in early studies, MRI was found to be more sensitive than CSF oligoclonal banding for diagnosing MS. MRI was also more sensitive than neurophysiological-evoked response studies [30].

Brain MRI has been used in large therapeutic trials to monitor MS disease activity [31,32]. In relapsing-remitting and secondary progressive MS, serial T2-weighted MRI reveals 3-10 times as many new lesions as there are clinical relapses [33]. Gadolinium enhancement further increases the reliability and sensitivity of detecting active lesions [33]. The sensitivity of MRI for detecting active brain lesions can be increased by injecting larger doses of contrast material, up to 0.3 mmol/kg (triple dose) [34-36].

In relapsing-remitting and secondary progressive MS, the presence of enhancement is more frequent during relapse and correlates well with clinical activity [29]. Enhancement is rare in primary progressive MS [35]. In benign MS, with a slow progression and little disability, enhancing lesions are also rare [37]. Delayed scanning and magnetization transfer may improve sensitivity [35,36].

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 [38].

Anticipated Exceptions

Nephrogenic systemic fibrosis (NSF), also known as nephrogenic fibrosing dermopathy) was first identified in 1997 and has recently generated substantial concern among radiologists, referring doctors and lay people. Until the last few years, gadolinium-based MR contrast agents were widely believed to be almost universally well tolerated, extremely safe and non-nephrotoxic, even when used in patients with impaired renal function. All available experience suggests that these agents remain generally very safe, but recently some patients with renal failure who have been exposed to gadolinium contrast agents (the percentage is unclear) have developed NSF [39-41], a syndrome that can be fatal. Further studies are necessary to determine what the exact relationships are between gadolinium-containing contrast agents, their specific components and stoichiometry, patient renal function and NSF. Current theory links the development of NSF to the administration of relatively high doses (eg, >0.2mM/kg) and to agents in which the gadolinium is least strongly chelated. The FDA has recently issued a “black box” warning concerning these contrast agents (http://www.fda.gov/cder/drug/InfoSheets/HCP/gcca_200705HCP.pdf).

This warning recommends that, until further information is available, gadolinium contrast agents should not be administered to patients with either acute or significant chronic kidney disease (estimated GFR <30 mL/min/1.73m²), recent liver or kidney transplant or hepato-renal syndrome, unless a risk-benefit assessment suggests that the benefit of administration in the particular patient clearly outweighs the potential risk(s) [39].

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. 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	Effective Dose Estimate Range
None	0
Minimal	< 0.1 mSv
Low	0.1-1 mSv
Medium	1-10 mSv
High	10-100 mSv

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