American College of Radiology ACR Appropriateness Criteria[®]

<u>Clinical Condition:</u>

Epilepsy

Variant 1:

Chronic epilepsy, poor therapeutic response. Surgery candidate.

Radiologic Procedure	Rating	Comments	RRL*
MRI head without contrast	8		None
MRI head without and with contrast	8		None
FDG-PET head	7	May be helpful in pre-op planning.	High
CT head without and with contrast	6		Low
fMRI head	5	May be helpful in pre-op planning.	None
NUC SPECT head	5	May be helpful in pre-op planning.	High
MEG/MSI	5	Data probably equivalent to BOLD and SPECT.	None
CT head without contrast	5		Low
MRA head	3		None
<u>Rating Scale:</u> 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Variant 2:

New onset seizure. ETOH, and/or drug related.

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with contrast	8	In the acute or emergency setting, CT may be the imaging study of choice.	None
MRI head without contrast	7	In the acute or emergency setting, CT may be the imaging study of choice.	None
CT head without and with contrast	6	In the acute or emergency setting, CT may be the imaging study of choice.	Low
CT head without contrast	5	In the acute or emergency setting, CT may be the imaging study of choice.	Low
MRA head	2		None
fMRI head	2		None
NUC SPECT head	2		High
FDG-PET head	2		High
MEG/MSI	2		None
<u>Rating Scale:</u> 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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Clinical Condition:

Epilepsy

Variant 3:

New onset seizure. Aged 18-40 years.

Radiologic Procedure	Rating	Comments	RRL*
MRI head without contrast	8	In the acute or emergency setting, CT may be the imaging study of choice.	None
MRI head without and with contrast	7	In the acute or emergency setting, CT may be the imaging study of choice.	None
CT head without and with contrast	6	In the acute or emergency setting, CT may be the imaging study of choice.	Low
CT head without contrast	5	In the acute or emergency setting, CT may be the imaging study of choice.	Low
NUC SPECT head	4		High
FDG-PET head	4		High
MRA head	2		None
fMRI head	2		None
MEG/MSI	2		None
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Variant 4:

New onset seizure. Older than age 40.

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with contrast	8	In the acute or emergency setting, CT may be the imaging study of choice.	None
MRI head without contrast	7	In the acute or emergency setting, CT may be the imaging study of choice.	None
CT head without contrast	5	In the acute or emergency setting, CT may be the imaging study of choice.	Low
NUC SPECT head	4		High
FDG-PET head	4		High
CT head without and with contrast	3	In the acute or emergency setting, CT may be the imaging study of choice.	Low
MRA head	2		None
fMRI head	2		None
MEG/MSI	2		None
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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Clinical Condition:

Epilepsy

New onset seizure. Focal neurological deficit.

Radiologic Procedure	Rating	Comments	RRL*
MRI head without contrast	8	In the acute or emergency setting, CT may be the imaging study of choice.	None
MRI head without and with contrast	8	In the acute or emergency setting, CT may be the imaging study of choice.	None
CT head without and with contrast	7	In the acute or emergency setting, CT may be the imaging study of choice.	Low
CT head without contrast	6	In the acute or emergency setting, CT may be the imaging study of choice.	Low
NUC SPECT head	3		High
FDG-PET head	3		High
MRA head	2		None
fMRI head	2		None
MEG/MSI	2		None
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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Expert Panel on Neurologic Imaging: John P. Karis, MD¹; David J. Seidenwurm, MD²; Patricia C. Davis, MD³; James A. Brunberg, MD⁴; Robert L. DeLaPaz, MD⁵; Pr. Didier Dormont⁶; David B. Hackney, MD⁷; John E. Jordan, MD⁸; Suresh Kumar Mukherji, MD⁹; Patrick A. Turski, MD¹⁰; Franz J. Wippold II, MD¹¹; Robert D. Zimmerman, MD¹²; Michael W. McDermott, MD¹³; Michael A. Sloan, MD, MS.¹⁴

Summary of Literature Review

Epilepsy is a common disorder, affecting approximately 0.5% to 1.0% of the United States population at any time with an incidence of 30.9 to 56.8 per 100,000 [1]. It has been estimated that about 7%-8% of the population experiences at least one epileptic seizure during their lifetimes [2]. The basic mechanism of epileptic seizures has not been fully elucidated.

The classification of epileptic seizures by the International League Against Epilepsy was last revised in 1989 (Appendix A) [3]. The classification is important because etiologic diagnosis, appropriate treatment, and accurate prognostication all depend on the correct identification of seizures and epilepsy. There are two main seizure types: partial seizures and primary generalized seizures. Partial (formerly referred to as focal) seizures show either clinical or EEG evidence of onset from a localized area within the cerebral hemisphere. The nature of the signs and symptoms in most cases indicate the region of the brain involved by the epileptic process. Partial seizures are designated as simple or complex. Complex partial seizures are associated with loss of consciousness. In simple seizures, the epileptic process is usually confined to neocortical structures, and the limbic system and brainstem are spared. Most simple seizures are less disabling than those associated with loss of consciousness. Partial seizures can spread and develop into secondarily generalized seizures. Primary generalized seizures originate simultaneously from both cerebral hemispheres, and clinical manifestations involve both sides of the body. Primary generalized seizures first occur at an earlier age, and are more likely to be associated with

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a family history of seizure disorders, but are less likely to be associated with focal cerebral lesions. Some seizures remain unclassified because the underlying mechanism of their origin or propagation is unknown [2].

Certain types of seizure disorders are likely to be associated with structural brain lesions, including tumors, infection, infarction, traumatic brain injury, vascular malformations, developmental abnormalities, and seizureassociated brain pathology (Appendix B) [4], whereas others are not. Hence, knowledge of seizure types helps to determine whether neuroimaging is clinically indicated and what type of study is appropriate.

While the imaging evaluation of epilepsy was greatly advanced by the clinical introduction of computed tomography (CT) in the early 1970s [5,6] because of its superior soft tissue contrast, multiplanar imaging capability, and lack of beam hardening artifacts, virtually all the substrates of epilepsy are visualized with greater sensitivity and accuracy by magnetic resonance imaging (MRI) [7-15]. As a result, MRI has become the modality of choice for high-resolution structural imaging in epilepsy. Although routine evaluation techniques of all clinically available scanner field strengths may be sufficient for determining mass lesions, optimized protocols for scans obtained on high-field (>1.5 T) scanners may be necessary for evaluating partial complex epilepsy, requiring scrutiny of the hippocampus and temporal lobe for atrophy and subtle signal alteration, as well as for detecting certain structural abnormalities such cortical dysplasias, hamartomas, and other as developmental abnormalities [8,9,16-21]. Anatomic imaging identifies focal abnormality in up to 51% of patients with partial epilepsy [22]. With the widespread clinical availability of high-performance MRI systems, a comprehensive MRI examination, with functional techniques providing additional information, adding corroborative information, and improving overall accuracy, may in the future be of even greater value in epilepsy.

Although the data provided by MRI are essential in the presurgical evaluation of patients with medically refractory epilepsy, structurally detectable abnormalities are absent in many patients. In these patients, functional studies provide useful information on the location of the seizure focus. Functional imaging techniques, including positron emission tomography (PET), single-photon emission computed tomography (SPECT), magnetic source imaging (MSI), and functional MRI (fMRI), have contributed to the presurgical evaluation of patients with epilepsy [18-20,23-41].

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Clinical PET with fluorodeoxyglucose (FDG) provides a measure of glucose uptake and thus metabolism. A seizure focus will typically manifest as a focus of hypometabolism on interictal (between episodes of seizure activity) examinations and will be seen as a focus of increased metabolism on ictal (during seizure) examinations. Interictal FDG-PET is sensitive (84%) and specific (86%) by electroencephalogram (EEG) criteria to temporal lobe epilepsy (TLE) and 33% sensitive and 95% specific to extratemporal epilepsy. By comparison, structural imaging using a variety of MR field strengths and techniques yielded a sensitivity and specificity of 55% and 78%. SPECT utilizing perfusion agents such as 99mTc-HMPAO or 99mTc-Neurolite, as well as bolus MRI perfusion provide an assessment of regional cerebral blood flow rather than brain metabolism. A seizure focus will typically manifest as a focus of hypoperfusion on interictal examinations and will be seen as a focus of increased activity on ictal examinations. The utility of isolated interictal cerebral perfusion assessment in patients without anatomic imaging abnormality is limited [42,43]. The use of ictal/interictal subtraction imaging with coregistration on MRI and image-guided surgery datasets is proving to be more useful than interictal perfusion imaging alone [43]. Injection of the blood flow agent within 90 seconds of seizure onset does, however, appear to be required to demonstrate the expected localized increase in cerebral perfusion [44]. The use of perfusion techniques in epilepsy is therefore limited because of the technological challenge of injecting EEGmonitored patients within 90 seconds of seizure onset.

fMRI techniques include phosphorus and proton spectroscopy (MRS), perfusion, and blood oxygen level dependent (BOLD) activation. The widespread application of most of these techniques in clinical practice depends on the widespread availability of highperformance MR imagers capable of performing fast echo-planar pulse sequences (EPIs), as well as substantial data postprocessing capabilities.

MRS is a set of noninvasive techniques for in vivo chemical analysis of the brain, some of which can be performed on standard-performance clinical MR units. Although MRS has been used extensively for the past 30 years in molecular physics and chemistry, its application to the study of epilepsy is relatively recent. Widely available proton and phosphorus single-voxel techniques have consistently demonstrated metabolite changes in the epileptogenic region of the brain. MRS or chemical shift imaging (CSI) allows simultaneous acquisition of spectra from all brain regions. The pictorial display of MRS information facilitates comparison of the epileptogenic zone with the remainder of the brain and provides localizing information. CSI is not yet widely available in clinical practice. Initial studies suggest that both proton and phosphorus MRS may be useful adjunctive presurgical tests for localizing seizure foci in patients with partial epilepsy, particularly in difficult cases, potentially reducing the need for intracranial-depth electrode EEG recordings and those with extratemporal seizure foci [19,25,26,32,33,35].

Only magnetoencephalography (MEG) and EEG are capable of measuring epileptic brain activity directly and with high temporal resolution. The temporal resolution of PET, SPECT, and fMRI is poor by comparison (sec-min). Recent improvements in MEG technology now allow whole brain coverage and overlay of source information on MR or CT images (MSI). Available data indicate that interictal MEG can be an effective tool for localization of seizure foci, in patients with medical refractory partial epilepsy. Significant shortcomings include limited availability, high cost, and assessment limited to relatively superficial and tangential sources. Nonetheless, MSI does provide unique, accurate, and useful information about epileptogenic regions in the brain, and where available, has a potential role in the diagnostic workup of most patients with epilepsy [27,29,37,40].

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Table 1. Definitions

Seizure	A finite event of altered cerebral function because of excessive and abnormal electrical discharges in the brain cells. A clinical seizure is accompanied by signs and symptoms. When no overt signs or symptoms are present, the event is referred to as a subclinical or an electrographic seizure and can only be detected by electroencephalographic recording (EEG).
Provoked seizure	A seizure is said to be provoked when one or more acutely precipitating factors are identified. Also referred to as acute symptomatic seizures. Examples of acute brain disturbances that can provoke seizures include intracranial infections, strokes, head injuries, and medication withdrawal.
Remote unprovoked seizure	Seizure that occurs more than 1-2 weeks after the inciting factor (remote symptomatic or predisposing factor). This type of seizures carries a higher risk of recurrence than idiopathic unprovoked seizures.
Idiopathic unprovoked seizures	Seizure that occurs in the absence of acutely provoking or remotely predisposing factors.
Partial seizures	Seizure that originates from a localized area within the brain. Designated as complex partial if associated with loss of consciousness, and simple partial if not.
Generalized seizures	Seizure that originates simultaneously from both cerebral hemispheres.
Epilepsy	A chronic condition predisposing a person to recurrent epileptic seizures. The predisposition may be genetic or acquired.
Epilepsy syndrome	An epileptic disorder characterized by a cluster of signs and symptoms customarily occurring together.

Appendix A. Outline of the International Classification of Epileptic Seizures

I. Partial Seizures (seizures with focal onset)

- i) Simple partial seizures (consciousness not impaired)
 - (1) With motor signs
 - (2) With somatosensory or special-sensory symptoms
 - (3) With autonomic symptoms or signs
 - (4) With psychic symptoms (disturbance of higher cerebral functions)
- ii) Complex partial seizures (consciousness impaired)
 - (1) Starting as simple partial seizures
 - (a) Without automatisms
 - (b) With automatisms
 - (c) With impairment of consciousness at onset without automatisms (impairment of consciousness only)
- iii) Partial seizures evolving into secondarily generalized seizures.

II. Generalized Seizures

- i) Absence seizures and atypical absence seizures (may have the following components): Mild clonic, atonic, tonic, or autonomic activities, or automatic behavior
- ii) Myoclonic seizures
- iii) Clonic seizures
- iv) Tonic seizures
- v) Tonic-clonic seizures
- vi) Atonic seizures

III. Unclassified Epileptic Seizures

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Appendix B. Outline of the International Classification of Epilepsies and Epileptic Syndromes

I. Localization-Related (focal, local, partial) Epilepsies and Syndromes

- i) Idiopathic (with age-related onset)
 - (1) Benign childhood epilepsy with centrotemporal spike
 - (2) Childhood epilepsy with occipital paroxysms
 - (3) Primary reading epilepsy
- ii) Symptomatic
 - (1) Chronic progressive epilepsia partialis continua of childhood (Kojewnikow's syndrome)
 - (2) Temporal lobe epilepsies
 - (3) Frontal lobe epilepsies
 - (4) Parietal lobe epilepsies
 - (5) Occipital lobe epilepsies
- iii) Cryptogenic

II. Generalized Epilepsies and Syndromes

- i) Idiopathic (with age-related onset)
 - (1) Benign neonatal familial convulsions
 - (2) Benign neonatal convulsions
 - (3) Benign myoclonic epilepsy in infancy
 - (4) Childhood absence epilepsy (pyknolepsy)
 - (5) Juvenile absence epilepsy
 - (6) Juvenile myoclonic epilepsy (impulsive pete mal)
 - (7) Epilepsy with grand mal seizures on awakening
 - (8) Other generalized idiopathic epilepsies not defined above
 - (9) Epilepsies with seizures precipitated by specific modes of activation
- ii) Cryptogenic or symptomatic
 - (1) West syndrome (infantile spasms, Blitz-Nick-Salaam Krämpfe)
 - (2) Lennox-Gastaut syndrome
 - (3) Epilepsy with myoclonic-astatic seizures
 - (4) Epilepsy with myoclonic absences
- iii) Symptomatic
 - (1) Nonspecific etiology
 - (a) Early myoclonic encephalopathy
 - (b) Early infantile epileptic encephalopathy with suppression-burst
 - (c) Other symptomatic generalized epilepsies not defined above
 - (2) Specific syndromes
 - (a) Epileptic seizures complicating disease states

III. Epilepsies and Syndromes Undetermined Whether Focal or Generalized

- With both generalized and local seizures
- (1) Neonatal seizures
- (2) Severe myoclonic epilepsy in infancy
- (3) Epilepsy with continuous spike-waves during slow wave sleep
- (4) Acquired epileptic aphasia (Landau-Kleffner syndrome)
- (5) Other undetermined epilepsies not defined above

IV. Special Syndromes

i)

- i) Situation-related seizures
 - (1) Febrile convulsions
 - (2) Isolated seizures or isolated status epilepticus
- ii) Seizures occurring only with acute metabolic or toxic event

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