

# Imaging Strategies for Epilepsy

Blake A. Johnson

Epilepsy is a heterogeneous group of disorders with multiple causes. A common neurologic condition worldwide, up to 8% of the population will experience at least one seizure in their lifetime (So 1995). Characterization of seizures to facilitate treatment and localization of patients was previously based on clinical evaluation, which was imprecise at best. Based on clinical criteria alone, it is often difficult to distinguish different seizure types, such as temporal lobe seizures from those with an extratemporal lobe origin. For example, even though experiential and visual seizures are typically associated with temporal lobe abnormalities, a percentage of seizures of this type will be extratemporal in origin (Manford 1996). The introduction of electroencephalography (EEG) in the 1950's provided a method to localize and characterize seizure foci within the brain. This ushered in the surgical era for the treatment of epilepsy due to brain lesions (Bailey and Gibbs 1951). To this day, intensive electroencephalographic monitoring has an important role for evaluating seizure patients (Gumnit 1986). However, even video-monitored EEG is usually insufficient to make a definitive diagnosis. Anatomic imaging, as well as ictal and interictal brain perfusion studies, can be pivotal in making the correct diagnosis (Blend 1997). Advanced imaging studies are essential for the evaluation of patients who are surgical candidates.

## CLINICOPATHOLOGY

Epilepsy has a profound impact on the lives of patients, regardless of age group. Catastrophic seizures of infancy and early childhood may be life-threatening (Engel 1996). There is invariably developmental delay with catastrophic seizures of infancy and childhood. On the other end of the spectrum, it is now recognized that epilepsy is more common in the elderly than is generally recognized. With this age group expanding faster than others it is likely that clinicians and imagers will encounter an increasing number of epilepsy patients. From a clinical standpoint, management of the elderly seizure patient is difficult. This is because metabolism changes with age, making titration of drug levels more difficult. The causes of epilepsy in older patients differs from those seen in the younger population as well. Seizures in older patients are often a result of prior stroke or other central nervous system insult (Pourmand

1996).

Clinical management of epilepsy patients requires a working knowledge of seizure syndromes, causes, and imaging features. Therapeutic options vary greatly, and success is based upon recognition of seizure type as well as the underlying cause. It is vital that the clinician accurately diagnose the epilepsy syndrome and employ the appropriate anti-epileptic drugs (Cloyd). The wrong pharmacotherapy for a given epilepsy syndrome may actually exacerbate seizure activity. The mechanism of seizure generation and activity has not been completely elucidated, although there is morphological evidence regarding the sequelae of epilepsy. The cerebral consequences of seizures may include generalized cerebral damage, cerebral hemiatrophy and mesial temporal sclerosis (Chan). MR spectroscopy studies which show elevated glutamate and other metabolites in the medial temporal lobes after prolonged seizure activities. This supports the theory that excitatory amino acid neurotransmitters play a role in this pathologic pathway. Regardless of the actual mechanism of seizure generation and cerebral insults, pharmacological and surgical interventions are more successful when a selective approach is utilized. It is therefore essential for imagers to be familiar with seizure syndrome classification, causes and imaging manifestations (Table 1).

## Etiology

Differentiating partial (focal) from generalized seizures is a primary step in evaluating seizure patients. Those with partial seizures are more likely to have a focal structural abnormality amenable to an

imaging diagnosis and potentially surgical cure. Biochemical screening in young patients with seizures is important to exclude an underlying metabolic disorder (Gibbs 1997).

In general, patients with temporal lobe epilepsy fare better and respond to treatment more often than those with extra temporal epilepsy. Those with extra temporal epilepsy generally have better outcome if the seizure is lesional in nature. There are a number of disease categories which may be associated with lesional epilepsy (Table 2).

Developmental abnormalities include neuronal migration disorders, neurocutaneous syndromes, hamartomas and vascular malformations. The most commonly encountered are hemimegalencephaly, Sturge-Weber Syndrome, large porencephalic cysts and other diffuse unilateral cortical malformations. Focal cortical dysplasia (FCD) differs from pachygyria, polymicrogyria and hemimegalencephaly because it is not associated with significant malformations of the cortical gyri. This histologically distinct lesion is found in up to 15% of seizure patient surgical specimens depending on the patient (Yagishita 1997). In patients with tuberous sclerosis, significantly more tubers are generally found in patients with seizure onset before one year of age and with mental disability (Shepard 1995). In fact, tuberous sclerosis patients without seizures generally do not have mental disability (Shepard 1995). Unlike tuberous sclerosis patients, seizures are relatively uncommon in patients with neurofibromatosis type I, and have a natural history similar to that of other epilepsy patients (Korf 1993). Rasmussen's encephalitis is another cause of seizures,

**Table 1. International Classification of Epileptic Seizures**

### I. PARTIAL SEIZURES

- Simple partial seizures
  - with motor signs
  - with somatosensory or special sensory symptoms
  - with autonomic symptoms
  - with psychic symptoms
- Complex partial seizures
  - starting as simple partial (+/- automatisms)
  - impaired consciousness at onset (+/- automatisms)
- Partial seizures evolving into secondarily generalized

### II. GENERALIZED SEIZURES

- Absence seizures
- Myoclonic seizures
- Clonic seizures
- Tonic seizures
- Tonic-clonic seizures
- Atonic seizures

### III. UNCLASSIFIED SEIZURES

which generally has an onset later in life (Engel 1996). This is postulated to be immune mediated in origin (Kim 1995). Strategically located neoplasms may also give rise to seizures as well. Infections, cerebrovascular disease and even trauma may cause epileptogenic lesions (Table 2).

The most common pathological substrate in patients with temporal lobe seizures is mesial temporal sclerosis (Mayanagi 1996, Kuzniecky 1987, Jack 1996, Jack 1996 commentary). Pathologically, there is loss of pyramidal neurons in the Sommer sector (H1) and the end folium (H3-5) of the hippocampal formation (Berkovic 1991). This entity was previously diagnosed postoperatively, with pathological demonstration of neuronal loss and gliosis. The prospective identification of mesial temporal sclerosis using high resolution MR has drastically increased the success rate of properly selected surgical patients.

Whether mesial temporal sclerosis is the result of, or the cause of (or both), seizures is debated. There is usually a history of febrile seizures, especially early and prolonged seizures (Mayanagi 1996, Berkovic 1991, Kodama 1995). There is evidence that a variety of insults to the brain within the first five years of life may precipitate this clinical condition (Engel 1996). It has been observed in the past that MTS is uncommon in young children with temporal lobe epilepsy, which was consistent with the hypothesis that hippocampal sclerosis occurs later in the course of recurrent complex partial seizures. Others contend that hippocampal sclerosis is more common in children than is currently recognized, and that it is a cause, rather than a consequence of, temporal lobe epilepsy (Harvey 1995). It is now recognized that MTS may ensue in a relatively short period following the inciting event, such as febrile seizure (Murakami 1996).

## IMAGING

The lack of sensitivity for the detection of subtle parenchymal abnormalities renders CT inadequate for evaluating seizure patients. MR is far superior for evaluating lesional and other forms of epilepsy (Kuzniecky, 1987, Bronen 1996). High resolution MR now allows for diagnosis of previously undetected pathology which is not revealed on CT or even conventional MR (Barkovich 1995). For example, patients with focal cortical dysplasia may have subtle indistinction of the gray white matter junction with signal abnormalities in the subjacent white matter (Yagishita 1997). Additional forms of migration abnormalities such as polymicrogyria, pachygyria and heterotopic

**Table 2. Lesions associated with epilepsy**

<p><b>DEVELOPMENTAL DISORDERS</b></p> <p>Neuronal migration disorders</p> <ul style="list-style-type: none"> <li>- Focal cortical dysplasia</li> <li>- Hemimegalencephaly</li> <li>- Neuronal heterotopia</li> <li>- Lissencephaly/pachygyria</li> <li>- Generalized cortical dysplasia</li> <li>- Polymicrogyria</li> </ul> <p>Neurocutaneous Syndromes</p> <ul style="list-style-type: none"> <li>- Tuberous sclerosis</li> <li>- Sturge-Weber Syndrome</li> </ul> <p>Hamartomas</p> <p>Vascular malformations</p> <ul style="list-style-type: none"> <li>- Arteriovenous malformation</li> <li>- Cavernous malformation</li> </ul> <p><b>POSTINFECTIOUS/AUTOIMMUNE</b></p> <ul style="list-style-type: none"> <li>- Rasmussen's syndrome</li> </ul>	<p><b>NEOPLASM</b></p> <p>Gliomas</p> <ul style="list-style-type: none"> <li>- Astrocytoma (diffuse, JPA)</li> <li>- Oligodendroglioma</li> <li>- Mixed oligoastrocytoma</li> <li>- Ganglioglioma</li> <li>- DNET</li> </ul> <p><b>INFECTION</b></p> <ul style="list-style-type: none"> <li>- Active lesions</li> <li>- Sclerosis</li> </ul> <p><b>VASCULAR</b></p> <ul style="list-style-type: none"> <li>- Infarction</li> <li>- Vascular malformation</li> </ul> <p><b>TRAUMA</b></p>
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gray matter are best imaged with MR due to its multiplanar capabilities and superior contrast resolution.

Other offending lesions that may be detected include tumor, vascular malformations, cerebrovascular disease, hamartomas, and infection (Engel 1996). Sensitivity for the detection of tumors, vascular malformations and migrational disorders approaches 100%. Tumors tend to be low grade, and are generally well circumscribed, with minimal edema, and minimal or no necrosis (Jack NI Clinics). The frontal and temporal cortex is the most common location for epileptogenic tumors.

Although it is an uncommon cause of seizures in developed countries, infection is a common etiology in developing countries. Tuberculosis and cysticercosis are the main sources of intracranial infectious lesions. In many endemic regions, these conditions are the most common causes of seizures. Tuberculosis may be associated with meningeal and/or parenchymal involvement. Granulomas, and less commonly abscesses, in the frontal and temporal lobe distribution may serve as a seizure focus. The imaging appearance of neurocysticercosis depends on the stage of infection. Live cysticerci (stage 1: vesicular) are immunologically transparent and do not evoke an inflammatory response in the adjacent brain parenchyma. The cystic lesions have a fine capsule and a central fluid-filled cavity. The CT and MR appearance parallels that of CSF. On MR, a characteristic small mural nodule containing the scolex increases the specificity of the findings. The mural nodule is usually slightly hyperintense on T1 weighted images. When the larva dies, an inflammatory response is elicited (stage 2: colloid vesicular). Perifocal edema and peripheral enhancement of the

thickened cyst wall are the imaging manifestations. This appearance is indistinguishable from other rim-enhancing lesions unless it is imaged while the scolex is still intact. The central fluid becomes turbid and displays higher density than CSF on CT, and may show T1 shortening on MR. With involution of the lesion (stage 3: granular nodular), perifocal edema decreases, but solid or rim enhancement may persist for weeks to months. The lesions are iso- to hyperdense on CT and iso- to hypointense on T1- and T2-weighted MR images. As the lesion continues to evolve, calcification occurs (stage 4: nodular calcific), resulting in the characteristic appearance of the final stage, which is best demonstrated on CT. Because the detection of calcium provides both improved sensitivity and specificity, CT still plays a critical role in the diagnosis of many CNS infections.

An acute infection which is often associated with seizures is herpes simplex encephalitis. This entity is seen in both developed and undeveloped countries, and represents a common cause of sporadic encephalitis. On MR, findings are usually present within 48 hours following the onset of symptoms. On T1-weighted images (T1WI), low signal intensity in the medial temporal lobes is characteristically bilateral and asymmetric. Both cortex and white matter are affected. Long-TR images reveal high signal intensity in the involved areas, which typically include the medial temporal lobes, insulae, inferior frontal lobes and occasionally the parietal lobes. Later, the cingulate gyrus may be affected. Involvement of the convexities, occipital cortex, brainstem and cranial nerves have also been reported. Petechial hemorrhage is characteristic, but only occasionally identified. Gross hemorrhage is

seen in a minority of cases. Following contrast administration, irregular or gyriform enhancement may occur after 5-6 days, initially involving the meningeal surface and subsequently seen within the parenchyma. Changes on MR develop rapidly, frequently concomitant with the clinical course. Because of the grave consequences of delayed treatment, a high clinical suspicion is sufficient cause to initiate therapy even if imaging findings are initially absent.

For the detection of focal sclerosis (mesial temporal sclerosis, post-trauma, post-infection), the sensitivity of MR is at least 80-95% (Jack 1995 NI clinics). Sclerosis manifests as increased signal on long-TR images. In mesial temporal sclerosis, hippocampal volume loss is another characteristic feature. In addition to the detection of sclerosis, intracranial masses and other lesions which may be responsible for seizure formation, MR may detect transient signal abnormalities in the brain parenchyma (Jaffe 1995, Aykut-Bingol 1997). Enhancement of these evanescent lesions suggests a transient defect in autoregulation and blood brain permeability.

The loss of neurons and gliosis provide the basis for the imaging manifestations of MTS. In some cases, edema may accompany gliosis as a cause for high signal on long-TR images (Kuzniecky 1987). Mapping of T2 relaxation times has been advocated for the detection of hippocampal sclerosis (Duncan 1996), but a qualitative analysis of the signal characteristics on high quality images is effective. Although the body of the hippocampus is most commonly involved, atrophy of the hippocampal tail and the amygdala occur in a minority of patients (Kuzniecky 1996). Atrophy of the involved hippocampus typically occurs at the level of the red nucleus on coronal MR images (Bronen 1997 AJR 875). Coronal images are essential for assessing hippocampal anatomy, due to the orientation of these structures. Both signal and morphological abnormalities may be obscure on axial images, and sagittal images do not allow direct side-to-side comparison. Associated limbic system structure abnormalities outside the hippocampal formations are also displayed on MR (Chan 1997). These changes may include atrophy of the fornix, mamillary bodies and ipsilateral cerebral hemispheres. Increased signal may be seen within the anterior temporal lobe on long-TR images (Chan 1997). For evaluating the degree of atrophy, volumetry or three-dimensional morphometry have been advocated for the assessment of hippocampal volumes (Free 1996, Cook 1992, Haller 1997). Because bilateral involvement may occur, advocates of

this technique maintain that a more sensitive means that visual inspection is necessary to assess hippocampal volume. However, surgical outcome studies indicate that patients with unilateral MTS and those with bilateral asymmetric MTS have essentially the same (favorable) postoperative prognosis for seizure control. Conversely, those with bilateral symmetric MTS or no MTS (ergo symmetric hippocampal formation volumes) have an unfavorable prognosis for seizure control following temporal lobectomy (Jack 1996 commentary). Patients with clearly defined unilateral mesial temporal sclerosis on MR have a 70 - 90% success rate following temporal lobectomy (Jack 1996 commentary, Kaskino 1995, Jack 1992, Zentner 1995).

Functional imaging allows assessment of the epileptic brain beyond morphologic mapping. Single proton emission computed tomography (SPECT) allows for the regional determination of epileptic foci (Jibika 1995, Blend 1997). With this modality, Tc-99m HMPAO is employed for determining brain perfusion. A seizure focus typically displays hypoperfusion between seizure episodes (interictal) and hyperperfusion during the event. Positron emission tomography (PET) can identify metabolic abnormalities associated with epileptic foci (Gaillard 1995). Metabolic aberrations detected on FDG PET can last up to 48 hours after a seizure (Leiderman 1994). The seizure focus will typically manifest as a focus of hypometabolism on interictal studies, and hypermetabolism on ictal interrogations. Asymmetry of mesial temporal lobe glucose metabolism is another positive predictor of good surgical outcome following temporal lobectomy (Manno 1994).

Magnetic resonance spectroscopy (MRS) provides a noninvasive technique for chemical analysis of the brain. A decrease in N-acetylaspartate (NAA) concentration or a decrease in the ratio of NAA relative to creatine and choline is demonstrated locally in patients with temporal lobe epilepsy (Ende 1997). The use of MRS as an adjunctive presurgical study will likely increase as this modality matures. Diffusion imaging is another potential adjunct for evaluating seizure patients. Even in the absence of MR signal abnormalities, diffusion abnormalities can be demonstrated after sustained seizures (Nakasu 1995). Further investigations are necessary to determine whether this can be employed to improve patient management.

### MR Imaging Technique

A dedicated seizure protocol to include high resolution and T2-weighted images is a

requisite for maximizing the excellent sensitivity of MR for the detection of seizure foci. High resolution volumetric studies are best performed utilizing 1-1.5 mm thick partitions (Barkovich 1995). A coronal 3D SPGR sequence provides high resolution T1-weighted images which are ideal for evaluating hippocampal symmetry and volumes. In addition, subtle extra-temporal lobe abnormalities such as focal cortical dysplasia may be revealed on this sequence, even though conventional images fail to detect them due to partial volume averaging. Another advantage of the volume acquisition is that it may be manipulated after acquisition to compensate for patient rotation or to provide images in an alternate plane. An oblique turbo FSE sequence perpendicular to the long axis of the hippocampal formation provides an excellent means for assessing signal abnormalities. By suppressing the high signal of adjacent hyperintense cerebrospinal fluid, FLAIR improves the T2-weighted contrast of lesions in patients with mesial temporal sclerosis and other high signal pathology. This sequence can be used in lieu of, or in addition to, the FSE sequence. Hemosiderin-sensitive gradient echo sequences are another option, which maximizes sensitivity for small vascular lesions such as cavernous malformations, and other hemorrhagic residua such as that left in the wake of diffuse axonal injury. If conventional spin echo long TR images are acquired for the axial images, there is usually sufficient sensitivity for the detection of hemosiderin for screening purposes. We reserve the use of gradient echo images for those with an appropriate history or an abnormality on the basic seizure protocol (table 3). Enhanced images may provide additional information when a lesion is encountered on the unenhanced series, but they do not usually unveil pathology which was not seen on the precontrast exam.

### CONCLUSION

Familiarity with the various epilepsy syndromes allows the clinician to properly evaluate and treat seizure patients. Surgically remediable syndromes have the best outcome when intervention occurs early in the course of the disease. Early identification and therapy also enhances the likelihood that the patient will return to a normal and productive lifestyle (Engel 1996). Neuroimaging plays an important role in the evaluation of these patients. Congruence of imaging, metabolic, electrophysiologic and clinical determinations of the seizure focus portends the best outcome following surgery. Recognition of the imaging manifestations

described above provides an important contribution to the management of seizure patients.

**SEIZURE-Bibliography:**

Adamsbaum C, Pinton F, Rolland Y, Chiron C, Dulac O, Kalifa G. Accelerated myelination in early Sturge-Weber syndrome: MRI-SPECT correlations. *Pediatric Radiology* 1996; 26:759-62.

Aykut-Bingol C, Tekin S, Ince D, Aktan S. Reversible MRI lesions after seizures. *Seizure* 1997; 6:237-9.

Bailey P, Gibbs FA. The surgical treatment of psychomotor epilepsy. *JAMA* 1951; 145:365-70.

Barkovich AJ, Rowley HA, Andermann F. MR in partial epilepsy: Value of high-resolution volumetric techniques. *AJNR* 1995; 16:339-43.

Berkovic SF, Andermann F, Olivier A, et al. Hippocampal sclerosis in temporal lobe epilepsy demonstrated by magnetic resonance imaging. *Annals of Neurology* 1991; 29:175-82.

Bernard D, Walker PM, Baudouin-Poisson N, et al. Asymmetric metabolic profile in mesial temporal lobes: Localized H-1 MR spectroscopy in healthy right-handed and non-right-handed subjects. *Radiology* 1996; 199:381-9.

Blend MJ, de Leon OA, Jobe TH, Lin Q, Sychra JJ, Gaviria M. Cerebral perfusion SPECT imaging in epileptic and nonepileptic seizures. *Clinical Nuclear Medicine* 1997; 22:363-8.

Blume WT, Grabow JD, Darley FL, Aronson AE. Intracarotid amobarbital test of language and memory before temporal lobectomy for seizure control. *Neurology* 1973; 23:812-9.

Bronen RA, Fulbright RK, King D, et al. Qualitative MR imaging of refractory temporal lobe epilepsy requiring surgery: Correlation with pathology and seizure outcome after surgery. *AJR* 1997; 169:875-82.

Bronen RA, Fulbright RK, Spencer DD, et al. Refractory epilepsy: Comparison of MR imaging, CT, and histopathologic findings in 117 patients. *Radiology* 1996; 201:97-105.

Cascino GD, Trenerry MR, Jack CR Jr, et al. Electrocorticography and temporal lobe epilepsy: Relationship to quantitative MRI and operative outcome. *Epilepsia* 1995; 36:692-6.

Chan S, Erickson JK, Yoon SS. Limbic system abnormalities associated with mesial temporal sclerosis: A model of chronic cerebral changes due to seizures. *RadioGraphics*; 1997; 17:1095-1110.

Chugani HT, Da Silva E, Chugani DC. Infantile spasms: III. Prognostic implications of bitemporal hypometabolism on positron emission tomography. *Annals of Neurology* 1996; 39:643-9.

Cloyd J, Leppik IE. Systematic approach to medical treatment of epilepsy. (? Citation)

Cook MJ, Fish DR, Shorvon SD, Straughan K, Stevens JM. Hippocampal volumetric and morphometric studies in frontal and temporal lobe epilepsy. *Brain* 1992; 115:1001-15.

Duncan JS, Bartlett P, Barker GJ. Technique for measuring hippocampal T2 relaxation time. *AJNR*

1996; 17:1805-10.

Ende GR, Laxter KD, Knowlton RC, et al. Temporal lobe epilepsy: Bilateral hippocampal metabolite changes revealed at proton MR spectroscopic imaging. *Radiology* 1997; 202:809-17.

Engel J Jr. Stratification of patients for epilepsy surgery. (? Citation)

Free SL, Li LM, Fish DR, Shorvon SD, Stevens JM. Bilateral hippocampal volume loss in patients with a history of encephalitis or meningitis. *Epilepsia* 1996; 37:400-5.

Gaillard WD, White S, Malow B, et al. FDG-PET in children and adolescents with partial seizures: Role in epilepsy surgery evaluation. *Epilepsy Research* 1995; 20:77-84.

Gibbs J, Appleton RE. The biochemical investigation of epilepsy in childhood. *Seizure* 1997; 6:193-200.

Gummit RJ. Intensive neurodiagnostic monitoring: Role in the treatment of seizures. *Neurology* 1986; 36:1340-6.

Haller JW, Banerjee A, Christensen GE, et al. Three-dimensional hippocampal MR morphometry with high-dimensional transformation of a neuroanatomic atlas. *Radiology* 1997; 202:504-10.

Harvey AS, Grattan-Smith JD, Desmond PM, Chow CW, Berkovic SF. Febrile seizures and hippocampal sclerosis: Frequent and related findings in intractable temporal lobe epilepsy of childhood. *Pediatric Neurology* 1995; 12:201-6.

Hermann BP, Wyler AR, Somes G, et al. Declarative memory following anterior temporal lobectomy in humans. *Behav Neurosci* 1994; 108:3-10.

Holmes MD, Dodrill CB, Ojemann GA, Wilensky AJ, Ojemann LM. Outcome following surgery in patients with bitemporal interictal epileptiform patterns. *Neurology* 1997; 48:1037-40.

Hufnagel A, Elger CE, Pels H, et al. Prognostic significance of ictal and interictal epileptiform activity in temporal lobe epilepsy. *Epilepsia* 1994; 35:1146-53.

Jack CR Jr. Hippocampal T2 relaxometry in epilepsy: Past, present, and future. *AJNR* 1996; 17:1811-4.

Jack CR Jr, Rydberg CH, Krecke KN, et al. Mesial temporal sclerosis: Diagnosis with fluid-attenuated inversion-recovery versus spin-echo MR imaging. *Radiology* 1996; 199:367-73.

Jack CR Jr, Sharbrough FW, Cascino GD, et al. MRI-based hippocampal volumetry: Correlation with outcome after temporal lobectomy. *Ann Neurol* 1992; 31:138-46.

Jaffe DJ, Latimer ME. Transient thalamic abnormality in association with seizure. *Journal of Child Neurology* 1995; 10:243-5.

Jibiki I. Single photon emission computed tomography studies of partial epilepsies. *Psychiatry & Clinical Neurosciences* 1995; 49:S191-3.

Korf BR, Carrazana E, Holmes GL. Patterns of seizures observed in association with neurofibromatosis 1. *Epilepsia* 1993; 34:616-20.

Kuzniecky RI, Burgard S, Bilir E, et al. Qualitative MRI segmentation in mesial temporal sclerosis: Clinical correlations. *Epilepsia* 1996; 37:433-9.

Kuzniecky R, de la Sayette V, Ethier R, et al. Magnetic

resonance imaging in temporal lobe epilepsy: Pathological correlations. *Annals of Neurology* 1987; 22:341-7.

Leiderman DB, Albert P, Balish M, Bromfield E, Theodore WH. The dynamics of metabolic change following seizures as measured by positron emission tomography with fludeoxyglucose F 18. *Archives of Neurology* 1994; 51:932-6.

Manford M, Fish DR, Shorvon SD. An analysis of clinical seizure patterns and their localizing value in frontal and temporal lobe epilepsies. *Brain* 1996; 119:17-40.

Manno EM, Sperling MR, Ding X, et al. Predictors of outcome after anterior temporal lobectomy: Positron emission tomography. *Neurology* 1994; 44:2331-6.

Margerison JH, Corsellis JAN. Epilepsy and the temporal lobes: A clinical, electroencephalographic, and neuropathological study of the brain in epilepsy, with particular reference to the temporal lobes. *Brain* 1966; 89:499-530.

Mayanagi Y, Watanabe E, Kaneko Y. Mesial temporal lobe epilepsy: Clinical features and seizure mechanism. *Epilepsia* 1996; 37(Suppl 3):57-60.

Murakami N, Ohno S, Oka E, Tanaka A. Mesial temporal lobe epilepsy in childhood. *Epilepsia* 1996; 37(Suppl 3):52-6.

Nakasu Y, Nakasu S, Morikawa S, Uemura S, Inubushi T, Handa J. Diffusion-weighted MR in experimental sustained seizures elicited with kainic acid. *AJNR* 1995; 16:1185-92.

Pacia SV, Devinsky O, Perrine K, et al. Clinical features of neocortical temporal lobe epilepsy. *Annals of Neurology* 1996; 40:724-30.

Roberts DW, Darcey TM. The evaluation and image-guided surgical treatment of the patient with a medically intractable seizure disorder. *Neurosurgery Clinics of North America* 1996; 7:215-27.

Shepherd CW, Houser OW, Gomez MR. MR findings in tuberous sclerosis complex and correlation with seizure development and mental impairment. *AJNR* 1995; 16:149-55.

Trenerry MR, Jack CR Jr, Sharbrough FW, et al. Quantitative MRI hippocampal volumes: Association with onset and duration of epilepsy, and febrile convulsions in temporal lobectomy patients. *Epilepsy Research* 1993; 15:247-52.

Wada J, Rasmussen T. Intracarotid injection of Sodium Amytal for lateralization of cerebral speech dominance: Experimental and clinical observations. *J Neurosurg* 1960; 17:266-82.

Wieser HG, Engel J Jr, Williamson PD, Babb TL, Gloor P. Surgically remediable temporal lobe syndromes. In: Engel J Jr, ed. *Surgical Treatment of the Epilepsies*, 2nd ed. New York: Raven Press, 1993; 49-63.

Yagishita A, Arai N, Maehara T, Shimizu H, Tokumaru AM, Oda M. Focal cortical dysplasia: Appearance of MR images. *Radiology* 1997; 203:553-9.

Zentner J, Hufnagel A, Wolf HK, et al. Surgical treatment of temporal lobe epilepsy: Clinical, radiological, and histopathological findings in 178 patients. *J Neurol, Neurosurg, Psychiatry* 1995; 58:666-73.

<b>Table 3:</b>	<b>SEIZURE PROTOCOL</b>	<b>OPTIONAL</b>
	Sagittal T1WI	Coronal MPGR T2*WI
	Axial dual echo T2WI	Enhanced coronal T1WI
	Coronal 3D FSPGR	Turbo IR
	Oblique coronal turbo FSE	