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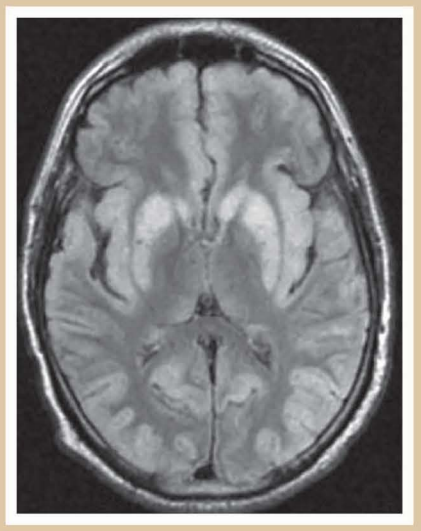

Series 1

Practice Pearls in NEUROLOGY

AV Srinivasan • D Vasudevan • K Bhanu

Foreword

Padma Shri Professor CU Velmurugendran

JAYPEE

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CHAPTER 4

Medically Refractory Epilepsy: A Clinical Approach

M Madhusudanan

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INTRODUCTION

Patients with epilepsy whose seizures sometimes not controlled with antiepileptic drug (AED) therapy are number of different terms may be used to describe these includes 'uncontrolled,' 'intractable,' 'refractory,' 'drug resistant.' or in simple terms drug-resistant epilepsy (DRE). That means seizures continue to occur in spite of treatment with a maximally tolerated dose of a first-line AED as monotherapy or in at least one combination with an adjuvant medication. The terms 'intractable' or 'medically refractory' are interchangeable.

MAGNITUDE OF THE PROBLEM OF INTRACTABLE EPILEPSY

It has been signified that 70–80% of persons developing seizures for the first time will eventually achieve remission, whereas the remaining 20–30% will continue to have recurrent seizures despite of medication.[1] If we extrapolate it to Indian population, about 1 million people will be suffering from refractory epilepsy.

IMPACT OF INTRACTABLE EPILEPSY

Medically refractory seizures produce many psychological and social issues. Recurrent seizures impair socialization and psychological development during formative years and may lead to an inability to obtain gainful employment, education or driving privileges. The intractable epilepsy in particular causes low self-esteem. Cognitive impairment can occur by refractory epilepsy as well as by the effects of AED therapy. Successful treatment of a seizure disorder can improve psychological, social, sexual, and behavioral functions.

DEFINITION OF INTRACTABLE EPILEPSY

Patients with uncontrolled seizure or with intolerable side effects that interfere with the quality of life, despite maximally tolerated dosage of one or more AEDs are referred to as those with intractable epilepsy. For how long the patient should have uncontrolled seizures, or what should be the frequency of seizures to designate intractability varies and individualized.

A task force of the International League against Epilepsy proposed that drug-resistant be defined as the failure of adequate trials of 2 tolerated, appropriately chosen and administered antiepileptic drugs (whether single or in combination) to achieve seizure freedom.[2] They also recommended replacing the term 'intractable' with 'drug-resistant' epilepsy (DRE).

Risk Factors for Intractability

Many prospective studies have been done to identify factors that predict the risk of DRE.

- *The response to the first AED:* It is the important predictive factor in both adult and pediatric groups.[3-7] While more than half of patients respond to the first AED < 20% are likely to respond to subsequent drug trials. With each number of failed AED trials, the risk of DRE increases.
- *A high number of seizures prior to diagnosis and treatment*[3,4,8-13] *is another consistently identified risk factor for DRE.*
- *The underlying etiology and seizure classification:* Idiopathic syndromes, for both generalized and focal epilepsy, have a better prognosis than symptomatic seizures in both pediatric and adult populations.[5,8,9,14-17] Nonidiopathic localization-related epilepsy underlies most of the cases of DRE in adults. Among the localization-related epilepsies, vascular cause associated epilepsies are more treatment responsive than those with mesial temporal sclerosis (MTS), cortical dysgenesis, or dual pathology.[4,16-18]
- *Certain pediatric epilepsy syndromes:* Early (neonatal) myoclonic encephalopathy, early infantile epileptic encephalopathy, Lennox Gastaut syndrome, and Rasmussen encephalitis are unfailingly refractory.
- *Other risk factors:* Other factors associated with the risk of DRE include status epilepticus,[5,16,19] a longer duration of epilepsy,[14,15] a family history of epilepsy,[8,9,20] a history of febrile seizures,[20] and abnormal EEG.[8] An abnormal neurologic examination or developmental delay are also identified as risk factors for DRE in some studies.[9,16,19]

CAUSES OF REFRACTORY EPILEPSY

Errors can occur in the diagnosis or in management.

Errors in Diagnosis

A. Nonepileptic paroxysmal events (syncope, pseudoseizures, etc.).

It is essential to consider errors in diagnosis in every case of intractable epilepsy. Before complex therapies are started, should be reviewed and executed for the diagnosis of epilepsy to be done. Up to 30% of all cases referred to epilepsy centers due to presumed intractable epilepsy, in fact, have nonepileptic seizures.[21]

Many of the nonepileptic seizures can closely mimic true epilepsy. So in any patient with refractory epilepsy, a reassessment of the condition is a must.

An epileptic seizure is usually defined as a brief and reversible alteration of ongoing behavior characterized by an excessive and disorderly neuronal discharge, usually at cortical level. Hence for the diagnosis of epilepsy, not only the paroxysmal nature of the neurological dysfunction is important, but we have to correlate this paroxysmal dysfunction with an abnormal or disorderly neuronal discharge. This is important because

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there are many other non-epileptic paroxysmal events, which are associated with transient neurological dysfunction, mimicking seizures. Hence an unequivocal diagnosis of epilepsy would require: (1) witnessing or videotaping of an episode of altered behavior and (2) demonstrating the characteristic EEG abnormality accompanying the clinical event.

The clinicians are handicapped in both respects. A physician is invariably at a disadvantage because he rarely witnesses a patient's seizure except in a case of frequent petit mal absence seizures where one can induce a seizure by asking the patient to hyperventilate. The second criteria to diagnose epilepsy, i.e. demonstrating an ictal EEG abnormality is almost impossible in our set up, since it requires a simultaneous video and EEG monitoring over an extended period of time, which is not practical most of the time. We have to depend up on the interictal EEG abnormality suggestive of seizure. This is also met with various pit falls. Even in genuine seizure, EEG can be negative in large proportion of cases. A single half-hour waking EEG with hyperventilation and photic stimulation shows epileptiform abnormality only in 30–55% of patients with a reasonably certain diagnosis of epilepsy. More over, detecting EEG abnormality does not always suggest a seizure disorder. The incidence of epileptiform abnormalities in nonepileptics is 2%.[22]

But we can increase the pick up rate of EEG abnormality by doing serial or multiple EEGs, doing a prolonged EEG, sleep and sleep deprivation EEG, recording immediately after a seizure, EEG recording after withdrawing anticonvulsant and recording using certain special electrodes. These are theoretical and not feasible for a practicing physician.

Hence, a detailed history obtained from both the patient and his relatives and /or friends who have witnessed the seizure is the key to correct diagnosis of epilepsy and separating it from nonepileptic paroxysmal events. We may have to ask the eye-witness to demonstrate or act out what he has actually seen. A home video recording by the eye witness using a mobile phone is very valuable for the clinician to arrive at the diagnosis. Prolonged EEG recording , and particularly a video-EEG recording is extremely useful in this setting.

When a patient presents with a history suggestive of seizure, we have to consider many conditions which can mimic seizure. The seizure generally presents with following symptomatology which can also be produced by other conditions.

The major categories of episodic symptoms caused by epilepsy include:

1. Loss of awareness
2. Generalized convulsive movements
3. Drop attacks
4. Transient focal motor attacks
5. Transient focal sensory attacks
6. Facial and eye movements
7. Psychic experiences
8. Aggressive or vocal out burst
9. Episodic phenomena in sleep
10. Prolonged confusional and fugue states.

Various disorders which may have to be considered under these headings are given below:

- **Loss of awareness with or without convulsive movements:**
  - Syncope, esp. convulsive syncope, micturition syncope, Pallid infantile syncope, cough syncope

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- Stokes-Adams attack
- Breath holding spells
- Confusional migraine
- Narcolepsy
- Panic attacks
- Hypoglycemic episodes
- Psychogenic seizures.

- **Drop attacks:**
  - Hyperekplexia
  - Cataplexy
  - VBI
  - Craniovertebral abnormalities (e.g. Arnold-Chiari malformation)
  - Third ventricular tumors
  - Vestibular disorders, e.g: Tumarkin's otolithic crisis
  - Idiopathic drop attacks.
- **Transient focal motor attacks:**
  - Paroxysmal kinesigenic choreo-athetosis
  - Tonic spasms of MS
  - Benign myoclonus of infancy.
- **Transient focal sensory attacks:**
  - Migraine
  - TIA.
- **Benign paroxysmal vertigo.**
- **Episodic phenomena in sleep:**
  - Benign nocturnal myoclonus
  - REM parasomnia
    - RBD
    - Sleep paralysis.
  - NREM parasomnias
    - Night terror
    - Somnambulism
    - Periodic leg movements of sleep.
- **Psychic experiences:**
  - Fugue states
  - Episodic dyscontrol
  - Aggressive or vocal out burst.

B. Incorrect seizure classification (partial or generalized).

Correct classification of seizure types is of utmost importance for the correct choice of drugs. Certain pitfalls can occur in the identification of certain seizure types; for example, it is sometimes difficult to differentiate complex partial seizures from absence attacks. Table 4.1 shows the clinical points to differentiate the two.

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Table 4.1: Differentiating features between absences and complex partial seizures (CPS).

	Absences	*CPS*
Age of onset	Childhood	Any age
Duration of attack	short (< 30 s)	Longer
Awareness of attack	often not	Usually
Clinical features	Slight	Aura, automatism
Post-ictal	None	Confusion
Frequency	Many	Infrequent
Effect of HV	Marked increase	None

C. Pitfalls in EEG interpretation.

- *Lack of properly taken EEG:* Some of the seizures are precipitated by sleep, some on sleep deprivation, and some by photic stimulation and hyperventilation. Generally, hyperventilation will precipitate epileptic discharges in generalized epilepsy like absences and photic in JME and absences with eye lid myoclonia. Sleep can activate spike discharges in partial epilepsies like BRE , temporal lobe or frontal lobe epilepsy as well as in conditions like LKS and CSWS. Scotopic response favors occipital lobe epilepsy. Sleep deprivation can precipitate EEG discharges in JME.
- *Errors in EEG interpretation:*
  - Over interpretation of physiological variants and nonspecific sharp-waveforms may mislead the diagnosis of epilepsy.
  - Fragments of generalized discharges-misinterpreted as focal abnormalities.
  - Rapid secondary generalization in extratemporal epilepsy may be mistaken as primary generalized epilepsy.

Prolonged video-EEG recording is extremely helpful tool in the evaluation of medically intractable seizure patients and can be performed as an outpatient or inpatient basis. Inpatient monitoring is often necessary when AEDs are withdrawn to enhance the chance of recording a seizure during monitoring. A typical duration of inpatient monitoring is 3 to 5 days.

D. Failure to identify a seizure syndrome or causative condition.

A failure classify the seizure type correctly might result in the wrong choice of drug and medically refractory seizures (for example treating complex partial seizures as absence seizures). Another example is missing a diagnosis of juvenile myoclonic epilepsy, by not asking the history of prior myoclonus.

Errors in Management

- **Wrong drug for the seizure type or seizure syndrome.**

In the drug selection, it is very important to remember that wrong selection of AEDs can worsen seizure: CBZ may increase atypical absence, absence or even sometimes convulsive seizure especially among patients who have the Lennox-Gastaut syndrome. If ethosuximide is inappropriately prescribed to a patient with complex partial seizure, patient may remain vulnerable to continuing seizure activity. Phenobarbitone and

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carbamazepine may increase absence, atonic and myoclonic seizures. Concurrent use of clonazepam with valproate may exacerbate absence status epilepticus.

One may also note that sudden withdrawal of phenobarbitone from epileptic patient may lead to exacerbation of seizures and sometimes develop into status epilepticus.

- **Inadequate dose of medication.**

Here one should be aware of certain pharmacokinetic principles. One should not judge the efficacy of a drug and increase or decrease the dose of a drug until steady state of the drug is reached. Steady state refers to the time taken for a stable drug level to reach after each dose administration.

In order to achieve a new steady state serum concentration, 5 half lives must elapse after the initiation of drug therapy. Therapeutic efficacy of a drug can be assessed only after this state has been reached. Too often, an AED is discarded as ineffective because inadequate time has been allowed to build up a steady state concentration. Similarly the dosage of a drug is sometimes increased too rapidly seeing no response. This may result in the accumulation of toxic concentration of the drug.

As already told, the time taken for the steady state to achieve for a particular drug is dependent on the half life, i.e. 5 half lives.

The half life and the time taken for the steady state to reach are given in Table 4.2.

Table 4.2: Time to achieve the steady state for AEDs.

Drug	*Half-life*	*Time to reach steady state*
Phenytoin	15–30 hours	5–15 days
Phenobarbitone	46–136 hours	14–21 days
Carbamazepine	11–17 hours	3–5 days
Valproate	6–18 hours	2–4 days
Lamotrigine	10–15 hours	5–15 days
Topiramate	20–14 hours	5 days
Levetiracetam	7–8 hours	2–3 days
Gabapentin	5–7 hours	1–2 days

Incorrect interpretation of blood levels may be problematic and result in inappropriate medication adjustments. The most common error is dose reduction due to 'elevated levels': Since the pharmacodynamics properties varies from person to person, it is better to continue the medication in the same dosage, unless it has produced side effects. Another common error is the misinterpretation of *nontrough* (or random) drug levels. Correct interpretation of `therapeutic ranges' requires *trough* drug levels obtained just before the next dose.

- **Poor medication compliance:** Poor compliance is a common cause of pseudo-intractability. This may relate to inadequate patient education, failure to establish a good relationship with the patient, or a complex dosing schedule. Drug blood levels may be helpful in the detection of noncompliance, but correct interpretation is needed.

  Poor medication compliance is due to following factors:

  - *Inadequate patient education:* Epilepsy is still shrouded by many myths and misconceptions. The treatment of epilepsy begins with appropriate counseling for the patient and the family. This is first step in eliminating the misconceptions

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concerning epilepsy. The patient should be clearly told about the need medications in the appropriate dose, and point that medications have to be taken for a prolonged period of time should be explained.
  - Too frequent or complex of dosing schedule
  - Intolerable adverse effects of the medication

- **Failure to recognize the etiology:** Another serious mistake is a failure to recognize an underlying disorder responsible for the intractability, such as a brain tumor, or infection. It is also important to inquire about possible triggering factors, such as alcohol, stimulants (caffeine and illicit drugs), medications and sleep deficiencies. As told earlier, certain childhood epileptic syndrome like West syndrome, Lennox-Gastaut syndrome, progressive myoclonic epilepsy, are very resistant to treatment.
- **True pharmacological intractability**.

What to Do in Case of True Refractory Epilepsy?

Having corrected all the causes of pseudorefractorines of seizures, if the patient is still having seizures, the patient should be referred to a higher center for considering epilepsy surgery or methods of treatment like vagal stimulation.

The basic idea is to correctly localize a surgically remediable lesion in the brain which is responsible for the resistant seizures. Partial seizures constitute the majority of resistant epilepsies.

A comprehensive presurgical evaluation is necessary to pinpoint the seizure onset site as well as to identify the risk of morbidity. These evaluations typically include ictal video-EEG recording, neuropsychological and speech-language studies, and brain imaging. Video-EEG monitoring was found to be appropriate in differentiating epileptic from non-epileptic seizures, to distinguish between primarily generalized and partial onset seizures, and to determine seizure onset localization and lateralization. Chronic intracranial EEG monitoring with the use of subdural grids or depth electrodes may be useful for localization of the epileptogenic zone.

Imaging modalities done for patients with medically refractory partial epilepsy include magnetic resonance imaging (MRI), computed tomography (CT), positron-emission tomography (PET) and single-photon emission computed tomography (SPECT).

Surgical Treatment

In medically intractable partial seizures, surgical resection of the focal epileptogenic cortex is the most effective method to render the patients free from seizure. Ideal candidates are those patients who have seizures emanating from a localized region of the brain that can be resected without neurologic morbidity.

The surgical therapy of epilepsy should not be considered as the last resort when all combinations of AEDs have been tried. For example, temporal lobe epilepsy due to mesiotemporal sclerosis typically has a poor prognosis with AED therapy; but has an excellent prognosis with anterior temporal lobectomy. The goals of the surgical treatment are to eliminate seizures, reduce AED toxicity and avoid neurologic morbidity.

The anterior temporal lobectomy is the most common surgical procedure performed for medically refractory epilepsy. About 60 to 80% of medically intractable patients are rendered seizure-free after temporal lobectomy.[23,24]

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In addition about 10 to 20% of patients have a substantial reduction in seizure activity following this surgery. Those patients who remain seizure-free for 2 to 3 years after surgery can be considered for AED withdrawal. The operative mortality rate for surgical treatment of epilepsy is approximately 1 in 400[25] The potential morbidity depends on the localization of the epileptogenic zone. The surgical outcome is not as favorable for cortical regions outside of the temporal lobes.

When to Refer a Medically Refractory Patient?

If a patient continues to have seizures despite treatment with two monotherapy and AED pushed to maximum tolerable dosage, then consider for referring the patient to a comprehensive epilepsy program. It should be kept in mind that longer the duration of intractable seizures, the greater the risks for psychosocial deterioration, loss of development, and secondary epileptogenesis.

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