Pediatric Epilepsy

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Definitions

• Seizure:
  – “The action of capturing someone or something using force”
  – A sudden disruption of brain’s normal electrical activity accompanied by altered consciousness or other neurological or behavioral manifestations
Definitions

• Epilepsy
  – A neurological disorder in which a person has repeated seizures over time

Introduction

• Epilepsy affects 1-2% of the population
• 1/10 Americans will have a seizure at some point
• At least 200,000 have at least one seizure/month
• 25% of all epilepsy cases develop before the age of 5 years
• About 125,000 new cases diagnosed each year
Main Seizure types

- Generalized seizures:
  - Abnormal electrical activity exists in all regions of the brain at the same time

- Partial (Focal) seizures:
  - Seizure onset is in one part of the brain—also called the ‘epileptic focus’

Tools used in the diagnosis

- History and Physical !!
- EEG
- MRI (High resolution is preferred)
- PET scan
Major Seizure Types

• GTC
  – Loud cry at onset
  – Stiffening of all extremities
  – Followed by rhythmic jerking of all extremities
  – LOC
  – Tongue bite, incontinence
  – Sleepiness afterwards

Major Seizure Types

• Absence seizures
  – Sudden loss of awareness
  – Brief duration
  – High frequency
  – No post ictal confusion
Major Seizure Types

• Myoclonic seizures
  – Brief, sometimes violent jerking of trunk or extremities
  – May have multifocal jerks
  – No LOC if brief
  – May have repeated myoclonic jerks

Major Seizure Types

• Atonic seizure
  – Sudden loss of tone
  – May or may not be associated with a fall
  – No LOC if brief
  – May be preceded by a myoclonic jerk
Pediatric Epileptic Syndromes

Pediatric Epileptic syndromes

• Epilepsy syndrome
  – A cluster of signs and symptoms regularly occurring together
  – must involve more than just the seizure type; thus frontal lobe seizures, for instance, do not constitute a syndrome

• Epileptic encephalopathy
  – the epileptic processes itself contributes to the disturbance in cerebral function

• Benign epilepsy syndrome
  – characterized by seizures that are easily treated, or require no treatment and remit without sequelae
Pediatric epileptic syndromes

- Benign familial and non-familial neonatal seizures
- Symptomatic neonatal seizures
- Severe neonatal seizures
- Infantile spasms and West syndrome
- Malignant migrating seizures in infancy
- Benign myoclonic epilepsy in infancy
- Severe myoclonic epilepsy in infancy
- Malignant astatic epilepsy
- Lennox-Gastaut syndrome
- Epileptic status in non-progressive encephalopathies
- Febrile seizures
- Idiopathic or benign epilepsies in childhood
- Idiopathic localization related epilepsies in infants and young children
- Epilepsy with centro-temporal spikes
- Idiopathic childhood occipital epilepsies
- Idiopathic childhood epilepsy in infants
- ESES syndrome
- Electrical Status Epilepticus during slow-wave sleep
- Childhood absence epilepsy
- Myoclonic absences
- Reflex epilepsies
- Juvenile absence epilepsy
- Juvenile myoclonic epilepsy
- Progressive myoclonic epilepsies
- Epilepsies and chromosomal abnormalities
- Rasmussen syndrome

Epileptic syndromes in:

- **Neonates**
  - Early Infantile Epileptic Encephalopathy
  - Early Myoclonic Encephalopathy

- **Infancy & early childhood**
  - Infantile spasms
  - Lennox-Gastaut Syndrome
  - MAE (Doose Syndrome)
  - Landau-Kleffner Syndrome (LKS)

- **Childhood**
  - Typical absence seizures
  - Absence with myoclonus
  - Benign rolandic epilepsy

- **Adolescence**
  - Juvenile myoclonic epilepsy
Neonates

Awake & Asleep
Ohtahara syndrome (EIEE)

- Newborns in the first three months (typically within 10 days of life)
- Affects boys more than girls
- Seizure: primarily tonic, less often - partial or myoclonic
- Common etiologies: metabolic disorders, structural damage, unknown

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Ohtahara syndrome

- EEG: burst suppression
- Treatment: Antiepileptic drugs or corticosteroids occasionally helpful
- Severely progressive course
  - Frequent seizures
  - Accompanied by physical and mental retardation
  - May progress to other syndromes: West and LGS
Early Myoclonic Encephalopathy

- Onset: within first month of life
- Myoclonic jerks -> partial seizures/massive myoclonus/IS/tonic seizures
- Delayed milestones, hypotonia, progressive cerebral atrophy

Ohthara vs EME

- Etiology - structural brain lesions are most probable
- Seizure: tonic spasms
- EEG: burst-suppression (BS) in both waking and sleeping states
- BS evolves to hypsarrhythmia around 3-4 months of age, and sometimes further to diffuse slow spike-waves
- Course: evolution to West syndrome, and further to LGS
- Etiology - non-structural/metabolic disorders
- Seizure - myoclonia and frequent partial motor seizures
- EEG - BS more apparent in sleep
- BS may persist up to late childhood after a transient evolution to hypsarrhythmia in the middle to late infancy
- Course: may have a transient phase of West syndrome.
Infants

30 microvolts
Hypsarrhythmia

- High amplitude asynchronous slow waves intermixed with multifocal spikes
- Appears in sleep first, and later in wakefulness
- Sleep may show a burst-suppression like pattern
- Is an interictal pattern
Infantile Spasms

- Peak frequency is from 4 to 9 months
- Boys > girls
- Resistant to treatment
- Brief flexion or rarely extension of extremities, and appear in clusters
- Multiple etiologies
- In 30%, no specific etiology is found
- Main treatment options include ACTH and vigabatrin. VPA and clonazepam may be considered
Lennox-Gastaut Syndrome

- Age of onset between 3-5 years
- Have tonic, myoclonic, atonic, atypical absence seizures
- Mental retardation
- Irregular, slow, spike pattern in wakefulness and generalized fast in sleep
- Intractable seizures
- VPA, RUF, TPM, LTG, ZNS, VNS or ketogenic diet may help in some cases
Children
Absence Epilepsy

- Prevalence: 2% to 8%
- Age of onset= 4-10 years with peak at 5-7 years
- Typically ♀ > ♂
- Genetically determined
- + family history of seizures
- Average seizure duration → 4-20 seconds
Absence Epilepsy

- Seizures are of:
  - Short duration
  - Abrupt onset
  - Abrupt termination
  - Impairment of consciousness
  - High frequency
- EEG shows ~3.5-4 Hz at onset, with 0.5-1 Hz at the end
- HV precipitates attacks in 95-100% of untreated patients

Absence Epilepsy

- Good prognosis if no other seizure types are present
- First line agents are:
  - Ethosuximide
  - Valproic acid
Juvenile Absence Epilepsy

- Age at onset—9-13 years
- Typically ♀ = ♂
- (+) Genetic factors
- Average seizure duration 4-30 seconds
- ~80% will have GTCS and ~15-25% will have myoclonic jerks

Juvenile Absence Epilepsy

- Absence seizure are less frequent than CAE
- EEG shows 3-4 Hz generalized SPW
- HV brings out seizures in almost all patients
- Life long disorder with seizure control in majority of patients
- First line agent is VPA
<table>
<thead>
<tr>
<th>CAE</th>
<th>JAE</th>
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<tbody>
<tr>
<td>Onset: 4-8yrs</td>
<td>Onset: 9-13yrs</td>
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<tr>
<td>Seizure – absence</td>
<td>Seizure – less</td>
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<tr>
<td></td>
<td>frequent absence</td>
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<td>with longer duration,</td>
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<td>GTCs-typically in</td>
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<td>am, rarely-</td>
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<tr>
<td></td>
<td>myoclonic jerks</td>
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<tr>
<td>EEG: 3 Hz sp/w</td>
<td>EEG: 3-4 Hz sp/w</td>
</tr>
<tr>
<td>Prognosis: good</td>
<td>Prognosis: typically</td>
</tr>
<tr>
<td></td>
<td>not outgrown</td>
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</tbody>
</table>
Juvenile Myoclonic Epilepsy

- Mean age of onset is 14 years
- $\frac{3}{2} > \frac{1}{2}$
- $\frac{1}{3}$ have family history of epilepsy
- Accounts for 5-10% of all epilepsies
- 3 main seizure types
  - Myoclonic seizures — in almost all patients
  - GTCs — 80-90%
  - Absence seizures — 15-30%

Interictal EEG shows 3-6 Hz spike/polyspike-and-wave discharges
Myoclonic jerks show polyspike-and-wave discharges
Photo-paroxysmal response is seen in with polyspikes in ~33%
Juvenile Myoclonic Epilepsy

- First line agent: Valproate
- Other options include: LTG, LEV, TPM
- 80-90% can be well controlled on treatment
- General thought is that JME is for life
Benign Rolandic Epilepsy

- Most common focal idiopathic epilepsy of childhood
- (+) genetic predisposition
- 10-13% will only have one seizure
- 65-70% will have seizures in sleep or upon awakening
- 10-20% with seizures only in wakefulness

Benign Rolandic Epilepsy

- Hemifacial clonic seizures: brief, usually nocturnal, and occasionally generalized
- Somatosensory symptoms such as paresthesias
- Age of onset: between 3-14 years
- Boys affected more than girls
- Normal neurological exam
- Responds well to medications
Benign Rolandic Epilepsy

- Normal development
- No abnormality on imaging
- EEG: sleep activation of independent, high-voltage centro-temporal spikes and slow waves
- Complete remission by the mid-teenage years in more than 90%

Benign Occipital epilepsy

- Seizure: visual symptoms, may be followed by hemi-clonic seizures or automatisms
- Visual symptoms:
  - transient, partial, or complete loss of vision
  - elementary or complex visual hallucinations
  - visual illusions (eg, micropsia, metamorphosis)
- EEG: occipital sp/w in trains that block with eye opening
- Frequent migrainous headaches
**Panayiotopoulos syndrome**

- Subset of BOE
- Onset 4-8 yr-old
- Seizures: during sleep, vomiting w/ eye deviation, may last >30min
- Other autonomic symptoms: color change (especially pallor), flushing, cyanosis
- EEG – variable epileptiform discharges though tend to be posteriorly dominant

**Landau-Kleffner Syndrome**

- Unknown cause
- Onset between ages 2-8
- Sudden progressive language regression w/ verbal auditory agnosia
  - Unable to comprehend their own name or recognize common sounds from the environment (e.g., fire engine, doorbell)
- Occasional complete loss of speech w/ deterioration of behavior
  - May be mistaken for deafness, autism or a developmental language disorder
Landau-Kleffner Syndrome (LKS)

- Seizures - focal or generalized
- Common EEG pattern: continuous posterior temporal spikes often during sleep
- Treatment: anti-epileptic seizure medications such as VPA and/or steroids
- Surgical treatment with sub-pial transections
- Seizures remit by teenage years
- Persistent language/behavioral deficits in 50%
Continuous Slow Spike and Wave in Slow Sleep

- Child with partial seizures has cognitive deterioration
- EEG:
  - Generalized sp/w in >85% of slow-wave sleep
  - Awake EEG - focal or generalized epileptiform discharges during wakefulness
  - Abnormalities persist or stop unexpectedly
  - Cognitive problems may persist

Severe Myoclonic Epilepsy in Infancy (SMEI, Dravet syndrome)

- Occurs in the first year of life in previously healthy children
- Seizure: prolonged and repeated febrile and afebrile generalized or unilateral convulsive
- Course: emergence of cognitive deterioration, interictal myoclonus, clumsiness, ataxia
- Subsequent marked slowing / stagnation of psychomotor development, accompanied by psychotic or autistic traits and hyperactivity between 1-4 years
- De novo mutations of the SCN1A gene in one third
Severe Myoclonic Epilepsy in Infancy (SMEI, Dravet syndrome)

- SCN1A positive in about 70-80%
- Seizure control does not guarantee improvement in cognition
- Seizures are usually refractory to most medications
- Avoid AEDs used for partial epilepsies (OXC, CBZ, PHT etc)

Severe Myoclonic Epilepsy in Infancy (SMEI, Dravet syndrome)

- AEDs that seem to help:
  - VPA
  - Clobazam
  - Stiripentol (Not available in the US)
  - Ethosuximide
  - Ketogenic diet
Febrile Seizures

• Common, 3% of all children
• 6 months to 5 years, peak 20 months
• Complex/ Atypical
  – Focal
  – Duration > 15 minutes
  – More than 1 seizure during 24 hrs

• Differentiate from seizures with meningoencephalitis

Febrile Seizures
risk for recurrence

• 1/3 have 2nd seizure
• 1/2 of these have further seizures
• Risk for recurrence:
  – Increased with
    • seizure onset < 13 months
    • positive family history
    • Seizure with lower grade fever
  – Not increased with a complex seizure
Febrile Seizures
risk for epilepsy

- Complex febrile seizures
- Abnormal/ delayed development
- Family history of epilepsy

GEFS+

- Febrile seizures persist beyond 6 years of age or have associated afebrile tonic-clonic seizures
- 30% may have other epilepsy syndromes - absence, myoclonic, akinetic
- Self-limited, with seizure resolution by mid-adolescence
- Family members may have Dravets or Doose syndromes
- Autosomal dominant
- Typical course: febrile seizures in childhood → quiescent interval of many years → onset of different seizure type
Myoclonic-Astatic epilepsy
Doose syndrome

- Onset: 7mths - 6 yrs
- Normal development until seizure onset
- First seizure – typically febrile
- Subsequent symmetric myoclonic jerks followed by absent muscle tone resulting in head nods or severe falls
Myoclonic-Astatic epilepsy
Doose syndrome

- Less frequent: absence, tonic, repetitive myoclonic jerks resembling clonic-tonic-clonic seizures
- EEG-generalized spike or polyspike-wave discharges and theta frequencies in the parietal regions
- Genetics—probably polygenic
  - Found in some GEFS+ families
- Prognosis: variable, at least 50% go into remission, most with normal or slightly decreased cognition

Anti-Epileptic Drugs (AEDs)
General rules for starting AEDs

- Start with a single agent
  - Reduced toxicity
  - No interactions
  - Better compliance
  - Easy to control side effects

Side Effects

- Understanding adverse effects has an important role in deciding optimal therapy
- Certain adverse effects may be age dependent
- Side effect profile in children cannot be assumed from adult data
- In the past 15-20 years, newer medications have increased the treatment options for children
When to start treatment?

- 30-40% rate of seizure recurrence after single GTC
- Treatment is indicated when:
  - Recurrence is likely
  - Frequent seizures
  - “Dangerous seizures”
  - Abnormal EEG
  - Focal structural lesion
  - Strong family history of epilepsy

How to choose an AED?

- Things to consider:
  - Age of patient
  - Seizure type (Epilepsy syndrome)
  - Side effect profile
  - Available formulations
  - Drug interactions
  - Cost
Anti-Epileptic Medications

Drugs for Epilepsy

- phenobarbital 1912
- mephtobarbital (Mebaral) 1935
- phenytoin (Dilantin) 1938
- trimethadione (Tridione) 1946
- mephenytoin (Mesantoin) 1947
- paramethadione (Paradione) 1949
- phenytoine (Thiantoin)* 1950
- phenacemide (Phenurone) 1951
- metharbital (Gemonil)* 1952
- benzchorpropamid (Hibicon)* 1952
- phensuximide (Milontin) 1953
- primidone (Mysoline) 1954
- methsuximide (Celontin) 1957
- Ethotoim (Peganone) 1957
- aminoglutethimide (Eliptin)* 1960
- ethosuximide (Zarontin) 1960
- diazepam (Valium) 1968
- carbamazepine (Tegretol) 1974
- clonazepam (Klonopin) 1975
- valproate (Depakene) 1978
- clorazepate (Tranxene) 1981
- felbamat (Felbatol) 1993
- gabapentin (Neurontin) 1993
- lamotrigine (Lamictal) 1994
- fosphenytoin (Cerebyx) 1996
- topiramate (Topamax) 1996
- tiagabine (Gabitril) 1997
- levetiracetam (Keppra) 1999
- zonisamide (Zonegran) 2000
- oxcarbazepine (Trileptal) 2000
- pregabalin (Lyrica) 2005

*withdrawn from the market
### AEDs

#### Partial seizures
- carbamazepine
- phenytoin
- gabapentin
- tiagabine
- oxcarbazepine
- pregabalin

#### Broad spectrum
- felbamate
- lamotrigine
- topiramate
- zonisamide
- levetiracetam

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### Most commonly used AEDs in pediatrics
- Levetiracetam
- Oxcarbazepine
- Valproic acid
- Topiramate
- Vigabatrin
- Lamotrigine
- Zonisamide
- Ethosuximide
- Rufinamide
- Felbamate
- Phenobarbital
Side Effects

- Sedation
- GI upset
- Rash (Lamotrigine)
- Interactions with other medications
- Behavioral changes (Levetiracetam)
- Renal stones (Zonisamide, topiramate)
- Metabolic disturbances (Oxcarbazepine, topiramate)

General stuff about AEDs
Anti-seizure drugs:  
Half-life

- Long (over 24 hours):  
  phenobarbital, phenytoin, lamotrigine, zonisamide
- Medium (12-24 hours):  
  valproic acid, lamotrigine, topiramate
- Short (<12 hours):  
  carbamazepine, gabapentin, oxcarbazepine, tiagabine, levetiracetam, pregabalin

Anti-seizure drugs:  
Titration time

- Begin full dose:  
  phenobarbital, phenytoin, gabapentin, levetiracetam, zonisamide, pregabalin
- 1-2 weeks:  
  valproic acid, carbamazepine, oxcarbazepine
- Over 4 weeks:  
  tiagabine, lamotrigine, topiramate
Anti-seizure drugs: Hepatic metabolism

- Inducers: phenobarbital, phenytoin, carbamazepine
  mild: topiramate, oxcarbazepine
- Inhibitor: valproic acid
- None: gabapentin, lamotrigine, tiagabine, levetiracetam, zonisamide, pregabalin

### AED Interactions

<table>
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<tr>
<th>AED</th>
<th>Other AEDs affected</th>
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<td>CBZ, TPM, OXC, LTG, ZNS</td>
<td>Theophylline, warfarin, digoxin, OC</td>
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<tr>
<td>PB</td>
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<td>Theophylline, warfarin, digoxin, OC</td>
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<tr>
<td>Mild inducers</td>
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<td>ZNS</td>
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Indications for plasma drug levels

- Narrow therapeutic range
- Documentation of patient compliance
- Lack of efficacy/presence of toxicity
- Suspicion of drug interactions

“Routine” is NOT an indication

Important message…

- Remember, you can always start with a medication that is broad spectrum and has fewer side effects…you can seldom go wrong with your choice!
Status Epilepticus (SE)

- Is a medical emergency
- Seizure lasting longer than 30 minutes, or 2 or more seizures without complete recovery in between
- Refractory SE refers to SE that does not respond to first or second line AEDs
Status Epilepticus (SE)

- Convulsive status epilepticus is associated with epilepsy and cognitive and behavior impairments later in life
- Goal of treatment is to minimize the duration of seizures to reduce adverse outcomes
- More than 80% of children have febrile or other identifiable etiology

SE and mortality

- Factors that determine mortality
  - Duration of SE
  - Late initiation of effective treatment
  - Etiology of SE
  - Presence of identifiable CNS process
  - Age

Towne, Epilepsia 1994
Principles of Management

- Effective treatment requires early and robust pharmacological intervention
- 4 phases of convulsive status epilepticus management are recognized:
  - Pre hospital
  - Treatment in the ER
  - Second line treatment after failure of 1st line agents
  - Continuous infusion

Overview of AEDs used for SE

- Management of SE may be divided into:
  - First line therapy: Benzodiazepines
  - Second line therapy: Phenytoin
  - Third line therapy: ?
- The real challenge is to find the appropriate agent to control seizures after first and second line therapies have failed (refractory status)
- No clear standards are available for 3rd, 4th or 5th line medications
Lorazepam 0.1 mg/kg IV x1
Repeat lorazepam 0.1 mg/kg IV x1
Fosphenytoin 20mg/kg IV x1
Additional fosphenytoin 10mg/kg IV x1

REFRACTORY STATUS EPILEPTICUS

Midazolam bolus (0.2mg/kg) followed by continuous infusion 0.1 mg/kg/hr
Titrate infusion rate until seizures stop or 2mg/kg/hr is reached

Pentobarbital 5mg/kg bolus, followed by infusion at 1mg/kg/hr
Titrate until seizures stop or 3mg/kg/hr

Non-Pharmacologic Treatments
Ketogenic Diet

• About 20-30% of individuals will develop medically refractory epilepsy
• For this patient population, diet therapy can be highly efficacious
• It may be used in any age group, from infants to adults

Ketogenic Diet

• High fat, low protein and very low carbs
• Brain is forced to use ketones (from fats)
• This prevents the brain from using glucose as a source of energy
Ketogenic Diet

• Comprises of 1g/kg of protein and 5-10g/day of carbs, with the remaining calories (~75% of daily allowance) coming from long chain triglycerides

Ketogenic Diet

• The mechanism of action is not fully elucidated
• The thought is that ketosis can, through different metabolic pathways increase GABA concentration
Ketogenic Diet

**Table 1. Epilepsy syndromes and conditions in which the KD has been reported as particularly beneficial**

Probable benefit (at least two publications)
- Glucose transporter protein 1 (GLUT-1) deficiency
- Pyruvate dehydrogenase deficiency (PDHD)
- Myoclonic-astatic epilepsy (Doose syndrome)
- Tuberosclerosis complex
- Rett syndrome
- Severe myoclonic epilepsy of infancy (Dravet syndrome)
- Infantile spasms
- Children receiving only formula (infants or enterally fed patients)

Suggestion of benefit (one case report or series)
- Selected mitochondrial disorders
- Glycogenosis type V
- Landau-Kleffner syndrome
- Lafora body disease
- Subacute sclerosing panencephalitis (SSPE)

**Table 2. Contraindications to the use of the KD**

**Absolute**
- Carnitine deficiency (primary)
- Carnitine palmitoyltransferase (CPT) I or II deficiency
- Carnitine translocase deficiency
- β-oxidation defects
  - Medium-chain acyl dehydrogenase deficiency (MCAD)
  - Long-chain acyl dehydrogenase deficiency (LCAD)
  - Short-chain acyl dehydrogenase deficiency (SCAD)
  - Long-chain 3-hydroxyacyl-CoA deficiency
  - Medium-chain 3-hydroxyacyl-CoA deficiency
- Pyruvate carboxylase deficiency
- Porphyria

**Relative**
- Inability to maintain adequate nutrition
- Surgical focus identified by neuroimaging and video EEG monitoring
- Parent or caregiver noncompliance
Ketogenic Diet

- Most effective for refractory myoclonic epilepsies
- 1/3 will become seizure free, 1/3 will have significant benefit, and another 1/3 will have no change, or will worsen
- These include SMEI, and Doose syndrome

Common side effects
- Lack of weight gain
- Acidosis
- Constipation
- Kidney stones
- Growth inhibition
- Hyperlipedemia

Less common side effects
Ketogenic Diet

- Usually continued for as long as it is beneficial
- Typically it is kept for 1-2 years
- The diet is tapered over several months

Vagus Nerve Stimulator (VNS)
What is VNS?

- A generator is attached to a bipolar lead
- Interrogation and programming are done using a wand and handheld computer
What is VNS?

- Implantation is performed under general anesthesia.
- The lead is attached to the vagus nerve, and the generator is placed in the anterior chest.

Relevant anatomy and physiology

- Vagus nerve is generally a parasympathetic efferent nerve.
- ~80% of fibers provide the brain with visceral information for head, neck, thorax, and abdomen.
- Right vagus innervates the cardiac atria, while left vagus innervates the cardiac ventricles.
Relevant anatomy and physiology

- Vagus innervation of ventricles is less dense than that of the atria, fewer cardiac side effects are seen

How does it work?

- It is also postulated that VNS exerts its effects by modulation of chemicals affecting cerebral cortex and hippocampus
- VNS also modifies cerebral electrical activity through thalamocortical pathways, but the exact mechanism of action is not known
Potential side effects

- Most common side effects seen are:
  - Hoarseness
  - Cough
  - ‘Sigh’ like deep breaths
- These side effects may be alleviated by changing the settings
- Relief is usually immediate

Epilepsy Surgery
Epilepsy surgery

- Focal epilepsy/generalized epilepsy
- MRI and EEG and other testing must show concordant findings for high success rates
- Epilepsy has to be refractory
- Results in improvement in QOL, cognition,
- 2 main procedures:
  - Single stage procedure
  - 2 stage procedure

Pre-Surgical Workup

- VEEG
- High resolution MRI
- PET scan
- Ictal SPECT
- WADA or fMRI
Surgical Evaluation

- **One step**
  - A single epileptogenic lesion is identified
  - Neurosurgeon resects the lesion (and the surrounding tissue)

- **2-step**
  - For better localization, strips, grids or depths are placed in the brain
  - With hardware in place, patient is monitored in EMU to record seizures
  - Cortical mapping
  - Resection
Epilepsy surgery

- Focal cases are lesional
- Generalized could benefit from procedures like CC to prevent falls
- For lesional temporal cases, post-surgical seizure freedom rates could be as high as 70-80%!
- Extra temporal could have variable rates that depend on the size of the lesion

Epilepsy surgery

- The goals of the surgery are to render a patient seizure free, or to significantly decrease the number of seizures
- Removal of the surgical focus without resultant neurological deficits
- Improvement in quality of life
Seizure precautions

• “Would you hurt yourself if you lost consciousness doing an activity?”
• Should kids with epilepsy swim?  
  – YES!
• Play sports?  
  – YES!

Seizure precautions  
(Elements that worsen sz)

• Drugs—Yes! Marijuana is a drug!
• Certain medications  
  – Benadryl, Meropenem, Imipenem
• Sleep deprivation
• Febrile illnesses
• Stresses on the body—as in surgical procedures
• Medication non-adherence!!!
What to do when witnessing a seizure

- Make sure the patient is safe
- DO NOT place anything in mouth (at least not through the teeth!)
- Roll patient to side
- Put head of bed down
- One eye on the watch/clock
- Document the sequence of events

Rescue Medications

- Medications meant to abort seizures, thereby preventing:
  - Status epilepticus
  - Brain damage
  - Injuries
  - Hospitalizations
Rescue Medications

- Are benzodiazepines
  - Diazepam
  - Lorazepam
  - Clonazepam
  - Midazolam

Rescue Medications

- May also use these meds to break seizure clusters
- Precautions when using these medications include:
  - Sedation
  - Respiratory depression (if high or multiple doses are given)
  - Psychosis or hyperactivity is also rarely seen
Outcomes

- Outcomes depend on multiple factors that include:
  - Etiology
  - Comorbidities
  - Epileptic syndromes
  - Family history

Driving and Epilepsy
Driving and Epilepsy

- Driving a car is critical to employment, self-esteem and socialization
- Seizures while driving pose the risk of a crash
- The risk depends on certain factors like seizure frequency
- Therefore, individuals with controlled epilepsy may drive with legal restrictions

Driving and Epilepsy

- There is no clear scientific evidence to define these restrictions
- These restrictions and rules vary among states
- The rules in general limit licensing individuals at highest risk for seizures while driving
Driving and Epilepsy

• The primary standard for determining that risk is the seizure free interval
• Some states make it mandatory for the physicians to report names of patients with seizures to the D(B)MV.
Driving and Epilepsy

• As a medical professional, you need to:
  – Explain the state specific laws
    • www.epilepsyfoundation.org
  – Make clear documentation in the patient record of the discussion
  – Provide alternates to driving (Public transportation)
  – Encourage strict medication compliance

One last thing…
The Bottom Line!

- Pediatric epilepsy is very different from adult onset epilepsy
- Medication choice and dosage depends on the type of epilepsy
- Good history, MRI, EEG are extremely helpful for a correct diagnosis
- This helps in choosing the right medication

The Bottom Line!

- For medically intractable cases, think about non-pharmacologic options (KD, VNS, surgery)
- Must address precautions (Driving, swimming, sports etc)
- Outcomes vary depending on the syndrome or type of epilepsy
The Bottom Line

• Seizures come in all different shades
• In pediatric epilepsy, we look at the bigger picture and not just seizures
• Accurate diagnosis is of paramount importance
• Treatment is specific for seizure types
• Use of non-pharmacologic means to treat epilepsy

THANK YOU!