Epilepsy

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Classification

- Generalized seizures

- Focal or partial seizures:

1. Focal seizures without loss of consciousness
2. Focal seizures with loss of consciousness
3. Focal seizures with secondary generalisation

Temporal Lobe

Seizures can start with aura in the form of automatism (hand or oral) and it can be reactive (drinking from a cup in the hand) or perseverative (continuation of complex act). This form is usually localised to the **temporal lobe**

Temporal lobe

- **Mesial temporal lobe epilepsy**

Typically begins with aura consisting of epigastric rising, fear, staring, oroalimantary automatism. Deporsanalisation, dream like state. De ja Vu,

Sometime olfactory hallucination or gustatory hallucination. There are sometime automatisms.

This may evolve to generalised tonic clonic seizures

The most common cause is **hippocampal sclerosis**

Focal seizures

Neocortical temporal cortex epilepsy

Auditory hallucination, vertigo, language disturbances, musicogenic seizures

Hypothalamus

- Laughter is:
  - Stereotyped
  - Brief (seconds)
  - Emotionless (no happiness)
- May include:Smiling, giggling, Autonomic signs (flushing, tachycardia)

Child appears to be “laughing for no reason

Most common hypothalamic hamartomas

Cingulate gyrus

- **Behavioral / Emotional**
- **Sudden fear, agitation, panic**
- **Aggressive or bizarre behavior**
- **Vocalization** (shouting, screaming)
- Laughing or crying (non-emotional)
- **Hypermotor activity**
- Thrashing, kicking, pelvic thrusting
- Sudden **running or climbing**
- Bilateral but often asymmetric movements

Frontal lobe

- Frontal seizures bizarre motor automatims, hypermotor activity, salivation, spitting
- Orbitofrontal cortex: complex motor automatism and olfactory hallucinations.
- Clonic, or Tonic clonic movement in primary motor area according to the motor homunculus involved.
- An example of partial seizures that affect the primary motor cortex is Jacksonian march

Focal seizures

- Fencers posture: asymmetric bilateral movement of the upper limbs: supplementary motor cortex. This can be bilateral without loss of consciousness
- Unilateral forced gaze and head deviation (versive seizures): premotor cortex and forntal eyes field.

Arm and facial seizures, with laryngeal movement, tachycardia, : contraateral insula

Tonic clonic seizures

- convulsive activity typically lasts < 1 minute.
- Mood changes can precede seizures by days
- Immediately pretonic-clonic phase: a few myoclonic jerks or brief clonic seizure activity; occasionally begins with forced eye and head deviation
- Tonic phase: contracture of the axial musculature with upward eye deviation, pupillary dilation, and forced expiration of air {epileptic cry}; usually involves some decerebrate posturing. Tongue and jaw muscle tonus causes perioral injury, typically lateral tongue biting. Frequently, patient becomes cyanotic, tachycardic, and hypertensive

Tonic clonic seziures

- Clonic phase: starts as low-amplitude, high-frequency (~ 8 Hz) convulsive movements of the extremities > the thorax and abdomen that progresses to high-amplitude, low-frequency (~ 4 Hz) movements. Development of atonia breaks the seizure and causes incontinence

Tonic clonic seizures

- Postictal phase: patient is poorly responsive and hypotonic; confusion and memory impairment may last a few minutes to hours, occasionally followed by psychiatric changes (depression, psychosis, anxiety, irritability) that can persist for about a day (a) Postictal phase involves generalized fatigue, soreness, and migrainous headaches

Tonic sezires

- diffuse contraction of the axial musculature, sometimes involving the proximal limbs or entire limbs;

It can be in the form of startle response that can follow a stimulus of any type

Atonic seizures

- Sudden loss of consciousness and muscle tone especially of the head. This can results in a drop attacks
- These are usually preceded by myoclonic jerks
- EEG may demonstrate polyspikes and waves during the ictal period and followed by generalised slowing of the central area

Myoclonic seizures

- Shock like movement of one muscle or a group of muscles.
- Irregular and can be singular or repetitive.
- It affects the eyelid, facial muscles, upper limbs ad lower limbs
- EEG: Polyspikes and waves

Juvenile Myoclonic epilepsy

- Symptoms: seizures involve bilateral but asymmetric flexor movements of the upper extremities or rarely of the lower extremities that develop after awakening
- usually no loss of consciousness
- Other types of seizures commonly exist such as absence and tonic clonic
- Photic stimulation provokes discharges

Juvenile Myoclonic epilepsy

- interictal EEG demonstrates bursts of bilateral, symmetric 3.5– 6-Hz spike-and-wave and polyspike discharges; ictal EEG exhibits diffuse polyspike activity followed by 1– 3-Hz slow waves
- Pathophysiology: genetic, but usually with complex inheritance pattern
- Treatment: valproate > lamotrigine, levetiracetam, topiramate, zonisamide

Absence epilpsy

- Symptoms: 5– 10-second long unresponsive staring spells ± rhythmic facial movements or picking behaviours; does not have an aura or postictal state
- Childhood-onset form is usually self-limited, whereas the juvenile-onset form is more likely to persist into adulthood
- autosomal dominant inheritance
- slow-wave discharges on EEG may relate to cyclic activity of T-type calcium channels and repolarizing potassium currents in the reticular thalamic nucleus

Absence epilpsy

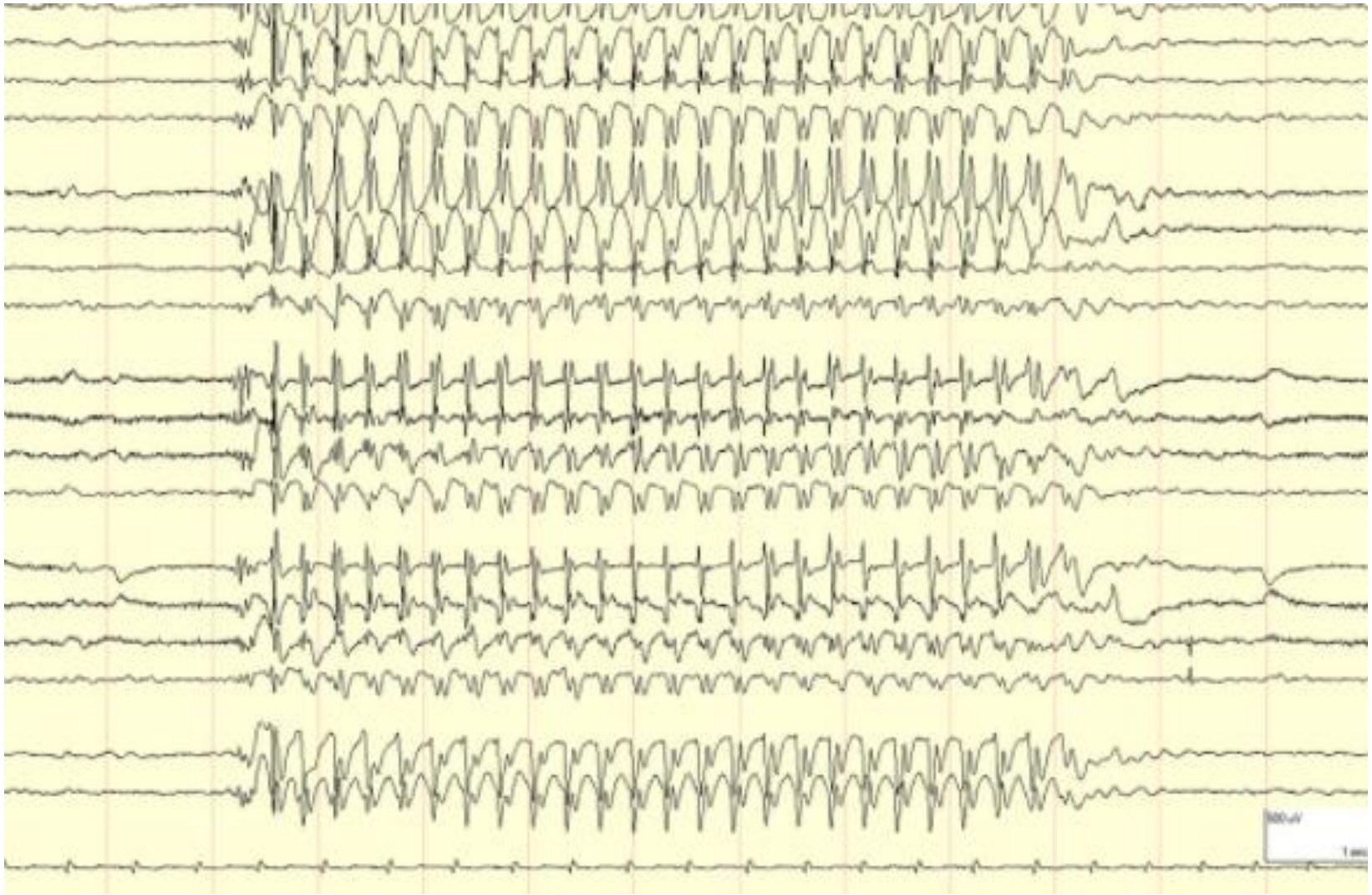
- Occur on a daily basis in the childhood-onset form, more infrequently in the juvenile-onset form
- 50% also have generalized tonic-clonic seizures, more commonly in the juvenile-onset form; these are infrequent and can be well controlled with medication
- Diagnostic testing: EEG demonstrates bilateral spike-and-wave complexes at 3 Hz that are reliably activated by hyperventilation.

treatment

- Treatment:

1. ethosuximide, valproate, acetazolamide
2. Avoid carbamazepine, phenytoin, and gabapentin, which have all been found to increase seizure frequency and may induce absence status epilepticus

Absence epilepsy



Rasmussen syndrome

- focal motor seizures that spread to contiguous muscle groups; 60% develop focal status epilepticus {epilepsia partialis continua} before the disease remits after 2– 10 years
- inflammatory degeneration of the cortex and supratentorial subcortical structures inflammatory degeneration
- It does not involve the contralateral hemisphere or posterior fossa structures (

Rasmussen syndrome

- EEG demonstrates continuous focal spike discharges that spread to contiguous areas of cortex and eventually to mirror foci on the contralateral hemisphere
- Neuroimaging: may be normal initially, but hemispheric atrophy and ipsilateral hydrocephalus ex vacuo develop within 6 months
- Treatment: refractory to antiepileptic medications; limited benefit from immunosuppressive agents; early hemispherectomy can be curative

Antiepileptic drugs

- Predictive factors for successful seizure remission

1. A single type of seizure
2. Had no seizures for > 2 years
3. A normal neurological exam and IQ
4. A normalized EEG on antiepileptic treatment

Epilpesy treatment

- Management of medication failure

1. If maximal doses of an antiepileptic drug (AED) fail to control the seizures, there is only a 10% likelihood of developing control over seizures with a second AED
2. There is < 5% likelihood of developing control over seizures with a third AED or by using multiple AEDs after failing a second AED
3. 25% of patients with chronic refractory seizures are eventually found to have the wrong diagnosis

AED prophylaxis

- Prophylactic AED use in patients with another neurological disorder, but without seizures, is usually not recommended

1. Brain tumor: prophylaxis does not seem to reduce development of seizures in patients with primary or metastatic tumors
2. Stroke: not indicated for ischemic stroke, although AEDs are routinely used short-term in subarachnoid hemorrhage patients because of the fear of increased intracranial pressure that accompanies seizures
3. Severe head trauma : (prolonged loss of consciousness, amnesia, depressed skull fracture, contusion, or hematoma) can be treated with acute prophylaxis limited to 1 week