**Features suggestive of epilepsy (MATIC)**

* **M**uscle twitches when on floor / **M**emory deficits
* **A**uras / **A**bsence attacks
* **T**ongue biting (ps normal faint can lead to tongue bite if they fall)
* Incontinence
* **C**onfusion(15 – 20mins post ictal)

**Differential diagnosis for blackouts**



**Most common causes:** vasovagal attacks (faints), epilepsy and cardiac arrhythmias

**First Aid Measures**

* ABC, Prevent injury, cushion the head, **if >5minutes call an ambulance**
* Look for medical bracelet or card
* Place in recovery position once seizure is open
* **Call the ambulance IF:** 1ST seizure, >5mins, another seizure without regaining consciousness, injured during seizure, urgent medical attention
* **Never restrain OR put anything in their mouth**

**Syncope (HAVOCSS)**

**Hypoglycaemia**

* Tremor, hunger, perspiration, light headedness or LOC

**Arrhythmias**

* E.g. Stoke Adams attack (transient arrhythmia causing reduced CO and LOC)
* May be palpitations warning an patient will have a slow or absent pulse
* Recovery within seconds, the patient flushes, pulse speeds up and patient regains consciousness

**Vasovagal syncope:**

* provoked by **emotion, pain, fear, standing too long**
* onset over seconds
* **REDUCED VENOUS RETURN TRIGGERS BARORECEPTOR REFLEX 🡪 INHIBITION OF SNS leading to syncope**
* Often preceded by **nausea, pallor, sweating** and **closing of the visual field.**
* **can not occur if lying down. Patient can remain unconscious for up to 2 minutes**
* **Reflex anoxic convulsions** may occur due to cerebral hypoperfusion
* **Uncommon:** urinary incontinence and fall causing tongue injury

**Orthostatic hypertension**

* **Drop of systolic upon standing of greater than 20mmHg**
* Inadequate vasomotor response
* common in
	+ Hypovolaemia 🡪 dehydration, diuresis, exsanguination, burns
	+ Addisons
	+ anti-hypertensive takers (CCB, BB, ACI)
	+ Tranquilisers/ LevoDOPA takers
	+ Diabetic autonomic neuropathy, Shy-Drager syndrome
* Venous pooling in legs made worse by heat, alcohol and antihypertensives
* Reduced venous return and end diastolic volume 🡪 reduced stroke volume and CO 🡪 transient drop in BP

**Carotid Sinus Syncope**

* Minimal contact i.e. shaving causes faint (hypersensitive baroreceptors causing excessive reflex bradycardia

**Situational Syncope**

* **Cataplexy (**laughter / intense emotion leads to syncope**)**
* **Cough syncope**
* **Effort syncope** i.e. on exercise – cardiac in origin (e.g. aortic stenosis, HOCM)
* **Micturition syncope** – men at night

**Structural**

* Aortic stenosis, HOCM, Atrial myxoma (tumour of the atrium)

**Fugor state**

* Loss of memory of events whilst acting normally – featureless history

**Mechanisms leading to seizures**

* Cellular
	+ Excitation (too much)
		- Ionic 🡪 altered RMP e.g. sodium, potassium or calcium
		- Neurotransmitter 🡪 glutamate
	+ Inhibition
		- Ionic 🡪 inward chloride, outward potassium
		- GABA Neurotransmitter
	+ Structural
		- Disorganised neurones 🡪 tumours, tuberous sclerosis

**Difference between epilepsy and acute provoked seizures**

**Epilepsy**

* A recurrent tendency to spontaneous, intermittent, abnormal electrical activity in part of the brain, manifest as seizures . Convulsions are the motor manifestations of this
* Causes include:
	+ Idopathic (most common)
	+ Structural: cortical scarring e.g. previous head trauma (>1 week after trauma)
	+ Developmental disturbance
	+ Space occupying lesions
	+ Stroke
	+ Genetic: tuberous sclerosis
	+ Sarcoid, SLE, PAN

**Acute Provoked Seizures**

* Occur in the presence of an external insult
	+ **Organic (Remember VITAMIN CDE)**
		- **Infection 🡪** encephalitis, meningitis
		- **Trauma**
		- **Metabolic 🡪** Hypoglycaemia, hyponatraemia
		- **Neoplasia (Tumours)**
		- **Drugs or toxins** e.g alcohol withdrawal
		- **Febrile convulsion**
	+ **Syncope**
	+ **Psychogenic**
		- Dissociative disorder, factitious disorder

**Pathophysiology of Syncope**

* Baroreceptors in the **carotid sinus & aortic arch** sense pressure
* **If pressure increases** 🡪 NTS 🡪 Rostro-ventrolateral medulla (S) 🡪 nucleus ambiguous (PS)
	+ INHIBIT SYMPATHETIC output and INCREASE PARASYMPATHETIC
	+ Vasodilation
* So increased BP reduces sympathetic tone
* Decreased pressure increases Sympathetic activity

**Starlings Law:** stroke volume increases in response to an increase in end diastolic volume, if all other factors are kept constant (e.g. contractility)

**Medications**

**Generally only prescribed after the second seizure**

|  |  |  |  |
| --- | --- | --- | --- |
| Drug | MoA | Pharmacokinetics | SEs |
| **Carbamazepine** | Membrane stabiliser | **LIVER ENZYME INDUCING**Taken **BD**, initial low dose | Dose related → giddiness, nausea, drowsiness, diplopia **(confused and dizzy going around that maze)**Allergic → leucopenia, rashes |
| **Valproate** | Unknown | Taken **BD/OD**Blood levels no use | (**VALPROATTTE**) → **a**ppetite increase, **l**iver failure, **p**ancreatitis, **r**eversible hair loss, **o**edema, **a**taxia, **t**eratogenicity, **t**remor, **t**hrombocytopenia, **e**ncephalopathy |
| **Phenytoin** | Membrane stabiliser | **LIVER ENZYME INDUCING**Taken OD**Blood monitoring useful** | Dose → drowsiness, ataxia, nystagmus **(Cerebellar symptoms)**Allergic → rash, lymphadenopathyChronic use → **g**um **h**ypertrophy, **a**cne, hirsutism, folate deficiency**Phenytoins Cerebellum Lacks Folate HAG** |
| **Lamotrigine** | Membrane stabiliser | Dosing depends on other anticonvulsants | Dose → nausea, dizziness, tremor, **headache**Allergic → rash, fever, arthralgia, lymphadenopathy, Stevens-Johnson syndrome(**FLARS**) |

**\*Valproate:** causes dysmorphic facial features, congenital abnormalities, learning difficulties 🡪 **5mg folic acid daily** 🡪 **PREGNANT WOMEN SHOULD SWITCH TO LAMOTRIGINE if possible**

**Focal Seizures**

**Temporal lobe (AUTOMAN)**

* **Automatisms, uncal (smell), terror, out of body experience, memory phenomena, abdominal sensation, not speaking properly.**
* Almost any symptom can arise
* **Automatisms:**
	+ Simple actions like lip smacking
	+ Manual: fiddling, grabbing
	+ Complex: singing, kissing, driving
* **Uncal** 🡪 olfactory hallucination
* **Hippocampal involvement 🡪** emotion (**T**error) / derealisation (**O**ut of body experience)
* **Memory phenomena**
	+ **Déjà vu (**sense of familiarity**)**
	+ **Jamais vu** (sense of unfamiliarity)
* Rising **Abdominal sensation**
* **Dysphasia (Not speaking properly)**

**Frontal Lobe**

* **Motor features** 🡪 posturing, peddling movements of the legs
* **Jacksonian March 🡪** a spreading focal motor seizure often with retained awareness (commonly affects the thumb and face)
* **Motor arrest**
* **Behavioural disturbance** (may be misdiagnosed as psychogenic)
* **Dysphasia**

**Parietal Lobe**

* Sensory disturbance (e.g. tingling, numbness)

**Occipital Lobe**

* Visual phenomenon

**Classification of epilepsies**

**Partial 🡪 onset localised to a focal area of the brain**

* **Simple 🡪** consciousness unimpaired
* **Complex 🡪** consciousness impaired
* **Secondary generalised**

**Generalised**

**Primary Generalised Seizures**

**Primary generalised (A C+/-T MA)**

**Absence (Petit mal)**

* Often in childhood , 90 – 95% seizure free by threshold
* Typically brief (<10s) pauses
* May present as a decline in school performance
* EEG – Bilateral symmetrical 3Hz spike wave pattern.
* **Ethosuximide (or Sodium Valproate)**

**Tonic-clonic (Grand mal)**

* May begin with an epileptic cry
* Limbs stiffen (tonic) then jerk (clonic)
* First line **Sodium Valproate**

**Tonic:** Limb stiffening only

**Clonic:** Jerky movements only

**Myoclonic**

* Sudden jerk of a limb, face or trunk
* Patient may be thrown to the ground suddenly
* **1st Sodium Valproate, 2nd Clonazepam/Lamotrigine**

**Atonic :** loss of muscle tone casing a fall

**Status Epilepticus**

* **>30 minutes of continuous seizure activity / Repetitive seizures without regaining consciousness**
* **1st stage (0 – 10 minutes): Oxygen** – during inter-ictal period insert an airway, position in **semi-prone position** with the head down to prevent aspiration. **Establish IV access** and **note the time**
	+ **Early status (0-10mins)**
		- **LORAZEPAM IV BOLUS 2mg/min** – **4mg dose** // **Diazepam 10mg IV // Buccal Midazolam**
		- **2nd dose of benzodiazepine** may be repeated **ONCE** within 24h (WRITE UP 2 doses on ON STAT DOSE rather than PRN part of the drug chart) – write ‘for convulsions >5min
		- Give usual anti-epileptics if already on treatment
* **2nd stage (0 – 30 minutes): monitoring of temp, cardiac, resp and BP.** Consider possibility of **non epileptic status**. If there is any suggestion of **alcohol abuse/impaired nutrition,** give **thiamine** as **IV Pabrinex BEFORE glucose (Keep it PG). 100ml of 10% glucose if hypoglycaemic** and then **10%glucose at 100ml/hr.**
	+ **Emergency investigations: Venous Blood count, clotting, glucose, urea, sodium, potassium, calcium, LFT and anticonvulsant drug levels, save 5ml blood and 50ml urine for toxicology. CXR to evaluate possible aspiration**
	+ **Established status (10 – 30 mins)**
		- **Valproate/ Leviteracetam/ IV Phenytoin 20mg/kg @ 50mg/min - contraindicated:** significant hypotension, bradycardia, heartblock, porphyria, generalised epilepsy (history of myoclonus or typical absence seizures), overdose of recreational drugs/antidepressants (**use valproate/levetiracetam instead**)
* **3rd stage (0 – 60mins) :** Establish aetiology and consider need for **urgent CT** (NO previous epilepsy history, new focal neurology), **alert anaesthetist** and **ICU.** Consider **pressor therapy** if needed
* **4th Stage (30 – 90mins)**
	+ Refractory status: Transfer to ICU, establish ICU monitoring and EEG if available (anaesthetise for 12-24h after last clinical / electrographic seizure then taper dose. **Propofol 1-2mg/kg bolus then 2-10mg/kg/hr –** Risk of infusion syndrome increases with duration of therapy. **Midazolam 0.1 -0.2mg/kg bolus/ Thiopental sodium** also an option
* Consider maintenance anti-epileptic medication on the adult neurology team
* Check if any **pre-hospital benzodiazepines** have been given
	+ **If 2 adequate doses have been given and seizures have recurred within 24h 🡪2nd line status treatment**

**REMEMBER TO DO A PREGNANCY TEST**

**Diagnosis and Investigations in Epilepsy**

* **Prospective recording of the events/ video**
* **EEG** – useful where there is a history suggestive of epilepsy – can be abnormal in 1% of the general population
* **ECG**
* **MRI** especially in focal epilepsy
* **Blood testing** for comorbidities or provocation e.g. FBC (infection), lactate, glucose, electrolytes, toxicology
* While investigating **advised not to drive**

**Driving and Social issues in epilepsy**

* If unprovoked /isolated seizure – **6 months off**
* People with epilepsy may only drive after a period of **1 year seizure free (if >5 years seizure free, a til 70 licence usually restored)**
* More stringent if HGV driver
* Sleep epilepsy 🡪 if seizures have only occurred during sleep for **>3 years** a patient may drive
* Withdrawal of epilepsy medication – should not drive whilst anti-epilepsy medication is being withdrawn and for 6 months after the last dose



**Features suggestive of non-epileptic seizures**

|  |  |
| --- | --- |
| **Clinical features** | **History** |
| * Ability of observer to modify the patients motor activity
* Asynchronous limb movements
* Avoidance behaviour during seizure
* Change in symptoms
* Closed eyes during seizure
* Ictal crying
* Biting the **tip of the tongue** rather than the side
* Fluctuation of symptoms
* Pelvic movements (especially forward thrusting)
* Resisted eyelid opening
* Seizures provoked by suggestion
* Side to side head movements
 | * Associated psychiatric history → especially BPD, depression, PTSD, dissociative disorder
* Flurries of seizures leading to A&E admission
* High seizure frequency
* Hx of sexual or physical abuse
* Lack or concern or over concerned
* No history of injury from seizure
* No response to AEDs or paradoxical increase
* Personal, family, or professional experience with epilepsy
* Seizures never occur whilst alone
 |