ANTI-EPILEPTICS

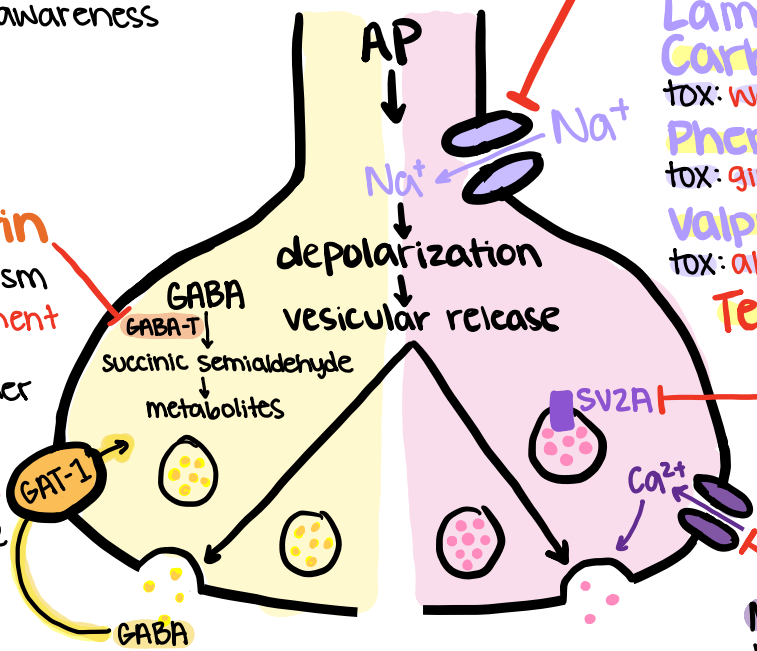
Normal: ordered, nonsynchronous firing

Seizure: disordered, synchronus, and rhythmic firing of populations of brain neurons

Epilepsy: periodic, unpredictable occurrence of seizures

- **PARTIAL** (↓aura)
  - Simple → conscious
  - Complex → loss of consciousness
- **GENERALIZED:**
  - **Tonic-Clonic:** stiffening, then spasming of limbs/face
    - **Tonic:** ↑muscle tone
    - **Atonic:** abrupt loss of muscle tone
    - **Myoclonic:** rapid, brief muscle contractions
  - **Absence:** lapses in awareness

Mutations in neuron ion channels can promote aberrant depolarization, and initiate seizures



VG Na^+ channel blockers

MOA: Slow the reset of Na^+ channels to resting state to stop AP propagation

- **Lamotrigine**
- **Carbamazepine** → CYP inducer
  - tox: water retention, hyponatremia
- **Phenytoin** → CYP inducer.
  - tox: gingival hyperplasia. zero-order kinetics
- **Valproate** → CYP inhibitor
  - tox: alopecia, weight gain

Teratogenicity

Vigabatrin

MOA: inhibits metabolism

tox: progressive, permanent bilateral vision loss

- used only if failed other therapies

Tiagaban

MOA: inhibits reuptake

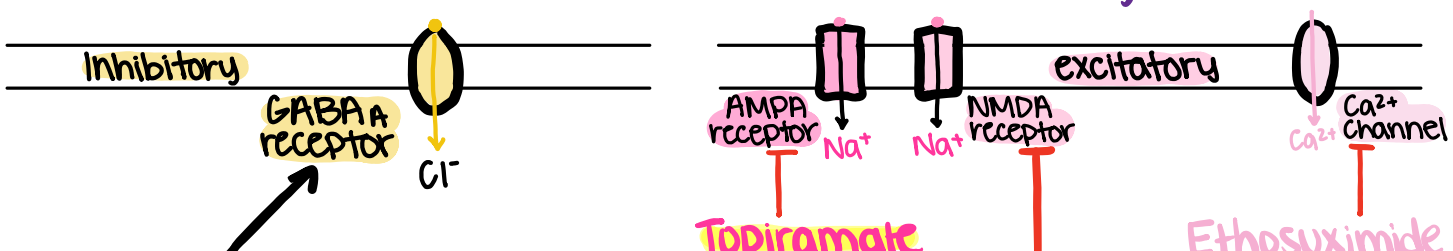
Levetiracetum

MOA: inhibits glutamine release by blocking a vesicle fusion protein

VG Ca^{2+} channel blockers

MOA: ↓Ca^{2+} influx and NT release from presynaptic neuron

- **Gabapentin**
- **Pregablin**



GABA-R positive Modulators

MOA: ↑Cl^- influx and post-synaptic hyperpolarization

- **Benzodiazepines**
  - **Clonazepam**
  - **Diazepam** (status epilepticus)
- **Barbituates**
  - **Phenobarbital**

Topiramate

tox: weight loss, renal calculi (stones)

Felbamate

tox: aplastic anemia, acute liver failure

Ethosuximide

MOA: Slow Ca^{2+}-induced depolarization

- Indicated for **absence seizures**

Common toxicities

- **Neurologic** - sedation, dizzy, fatigue, ataxia, visual disturbance
- **GI** - nausea / vomiting