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QUESTION 09

Describe the physiological basis for the mechanism of action of three commonly used anticonvulsant groups with examples

Seizures are a transient clinical manifestations that result from an episode of epileptic neuronal activity.

Epileptic neuronal activity is a specific dysfunction characterised by
- abnormal synchronisation
- excessive excitation and/or inadequate inhibition
- affecting variable populations of neurons

Decrease Seizure threshold	Increase Seizure threshold
Excitatory Post Synaptic Potentials	Inhibitory Post Synaptic Potentials
NMDA activity	GABA activity
Sodium influx	Potassium efflux
Calcium influx	Chloride influx
Paroxysmal depolarisations	Low pH

Anticonvulsants reduce seizure activity by
- reducing actions of threshold lowering agents and/or
- potentiating actions of threshold increasing agents
- they may act on multiple pathways, and many mechansims remain contraversial

Altering the sodium channels necessary for depolarisation and therefore AP generation
- this is the most common mechansim for anticonvulsants and contains most of the major drugs
- Phenytoin/Carbemazipene bind to inactive sodium channels, reducing likelyhood of AP

Increasing the activity of GABA system (major inhibitory system in CNS)
- Benzodiazepienes - enhance $GABA_A$ receptor affinity for GABA
- Vigabatrin - inhibits GABA tansaminase which breaks down GABA

Reducing the activity of NMDA system (major exitatory system in CNS)
- Toprimate - inhibits the action of kainite on NMDA receptors

Blocking Ca channels and thereby reducing neurotransmitter release
- Ethosuximide - blocks T-type calcium channels

Unknown - Levetiracetam