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Review on Antiepileptic Drugs

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Abstract: *The **"Antiepileptic Drug"** discusses epilepsy and the role of medications in managing seizures. Epilepsy affects around 1% of the global population, and while antiepileptic drugs (AEDs) do not cure the condition, they control seizure frequency and severity. The text reviews the historical development of AEDs from early plant-based remedies to modern compounds and explains their mechanisms, primarily involving modulation of sodium and calcium channels and enhancement of GABAergic inhibition. It classifies AEDs into groups such as barbiturates, hydantoins, succinimides, benzodiazepines, and newer drugs like vigabatrin, tiagabine, and lamotrigine. The recent drug XEN1101 shows promising results for treatment-resistant focal epilepsy. Common side effects include dizziness, fatigue, and mood changes, pathophysiology and the sign and symptoms of that drug while precautions are needed when combining AEDs with alcohol or driving. The paper concluded that although newer drugs improve tolerance and safety, more effective and individualized therapies are still required for optimal epilepsy management.*

Keywords: Epilepsy, Seizure, Barbiturates, Hydantoins, Succinimides, Benzodiazepine

I. INTRODUCTION

Around 1% of the global population is affected by epilepsy, a brain disorder that can shorten lifespan. While seizures are a key symptom, the onset of epilepsy begins before the first seizure and can continue to progress after seizures start. Epilepsy presents with a variety of seizure types and syndromes, as well as accompanying conditions like anxiety, depression, and higher mortality rates.

AEDs do not have the ability to prevent or reverse drug-resistant epilepsy, address comorbidities, or provide a comprehensive approach to reducing the overall burden of the disease. It is concerning that there has been little advancement in improving seizure control in the past 40-50 years since carbamazepine and valproate were introduced. This lack of progress in developing more effective epilepsy treatments has led to disappointment among patients and doctors, making them less inclined to use newer, more expensive AEDs. Insurers are hesitant to Pay for pricier medications that do not offer significant advantages over cheaper generic options, and the pharmaceutical industry is losing interest in creating new compounds for epilepsy.[1]

Use of second-generation drugs in the pediatric population antiepileptic

Epilepsy is common in the pediatric population. Nine second-generation antiepileptic drugs have been approved in the US for use in epilepsy over the past 15 years: felbamate, gabapentin, lamotrigine, topiramate, tiagabine, levetiracetam, oxcarbazepine, zonisamide, and pregabalin. Their use in pediatric patients is fairly widespread, despite most of these agents not having US FDA indications for use. Felbamate and gabapentin were the first two second-generation antiepileptic drugs to be approved in the US. Felbamate use has been limited because of the occurrence of hepatotoxicity and aplastic anemia. Although gabapentin is a fairly well tolerated antiepileptic drug, its use has also been limited as a result of inconsistent efficacy and concern about seizure exacerbation. Lamotrigine and topiramate are broad-spectrum antiepileptic drugs with efficacy in a wide variety of seizure types. Both agents have some tolerability concerns: rash with lamotrigine and neuropsychiatric events with topiramate. There are very little data on tiagabine use in children, but this agent appears to be effective and to have a good tolerability profile. Levetiracetam is a second-generation antiepileptic agent that is available intravenously. Considering its good efficacy, fast onset of action, and low incidence of serious adverse effects, its use in the acute setting could potentially increase.[2]

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Medication and history

This information is provided as general information about the drug treatment of epilepsy. The information is not a substitute for proper medical advice. Consult your doctor for any questions or concerns you have about you or your child's medication. For urgent information about incorrect dosing or overdosage please contact your doctor, pharmacist or the Poisons Information Centre 13 11 26 Nationwide Australia.

Antiepileptic medications do not cure epilepsy, but rather attempt to prevent seizures. Strictly speaking, these medications are antiseizure or anticonvulsant, rather than antiepileptic. Antiepileptic medications do not alter the underlying problem predisposing to seizures. People with epilepsy are prescribed antiepileptic medications with the aim of decreasing the number, severity, and/or duration of seizures. While seizure freedom is the ideal outcome of treatment, seizures can still occur while taking antiepileptic medication.

https://www.rch.org.au/neurology/patient_information/antiepileptic_medications/#how-do-antiepileptic-medications-reach-nerve-cells

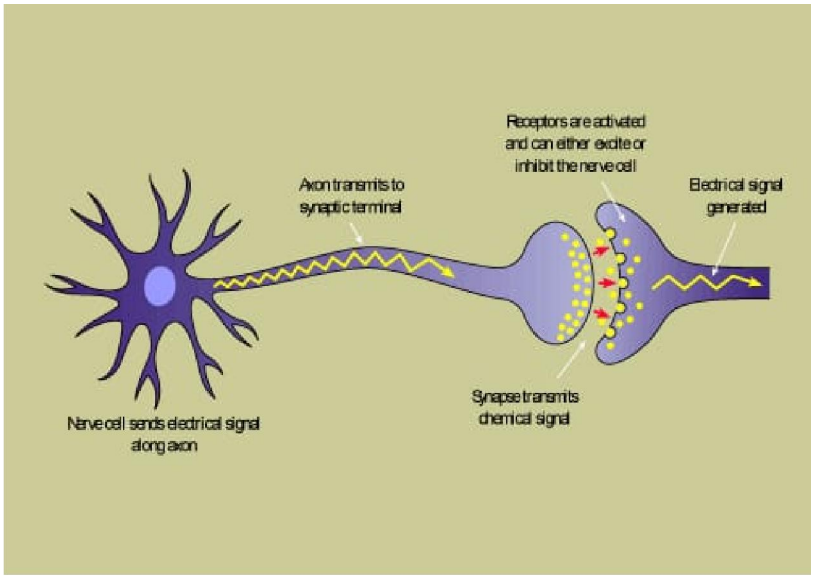

fig.1.1 medication for antiepileptic drug.

[3]

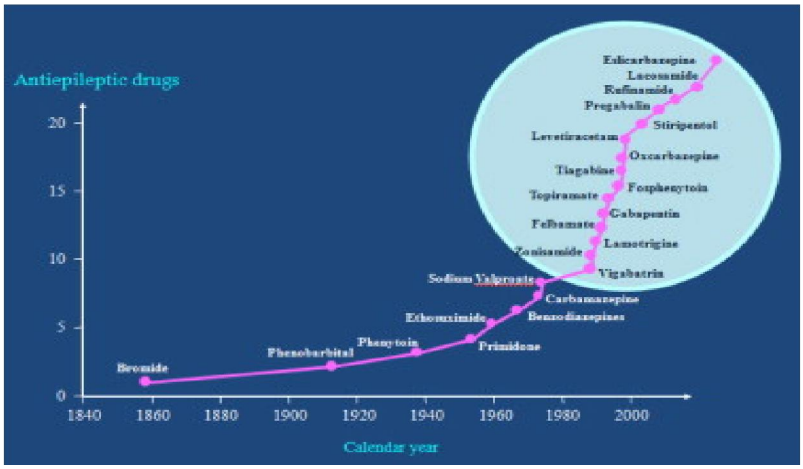

fig 1.2. History of antiepileptic drug [4]

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Modern era

The modern era of AED development began in 1975 when the National Institute of Neurological Disorders and Stroke in the United States established the Anticonvulsant Drug Development Programmed. More than 28,000 new chemical entities from academic and pharmaceutical chemists have since been screened, resulting in the licensing of an increasing list of AEDs (Fig. 1). Suitable compounds were identified by target-orientated design, structural modification of existing molecules and, most importantly, by laborious and systematic screening against a range of seizure models in rodents. The most commonly used tests include maximal electroshock, subcutaneous pentylenetetrazol, 6 Hertz test and a battery of other screening options.Interesting, many newly identified AEDs appear to have unique mechanisms of action despite demonstrating efficacy against a similar range of similar seizure models. They all have in common the ability to decrease neuronal excitation or increase neuronal inhibition by one or more of pharmacological processes, including modulation of voltage-gated cation channels, potentiation of GABA-eric activity, inhibition of glutamatergic processes and modification of neurotransmitter release.[5]

Records of medicinal plants and minerals date to ancient Chinese, Hindu, and Mediterranean civilizations. Ancient Greek physicians such as Galen used a variety of drugs in their profession. During the 16th century AD, after Western medicine began to recover from its long sleep during the Dark and Middle Ages, pharmaceutical practice began to develop rapidly. In 1546 the first pharmacopoeia (list of drugs and their preparation) appeared in Germany [6]

Development

This article provides a comprehensive narrative review of the history of antiepileptic drugs (AEDs) and their development over time. Firstly, it explores the significant role of serendipity in the discovery of essential AEDs that continue to be used today, such as phenobarbital and valproic acid. Subsequently, it delves into the historical progression of crucial preclinical models employed in the development of novel AEDs, including the maximal electroshock stimulation test, pentylenetetrazol-induced test, kindling models, and other animal models. Moving forward, a concise overview of the clinical advancement of major AEDs is provided, highlighting the initial milestones and the subsequent refinement of this process in recent decades, in line with the emergence of evidence-based medicine and the implementation of increasingly rigorous controlled clinical trials. Lastly, the article explores the contributions of artificial intelligence, while also offering recommendations and discussing future perspectives for the development of new AEDs [7]

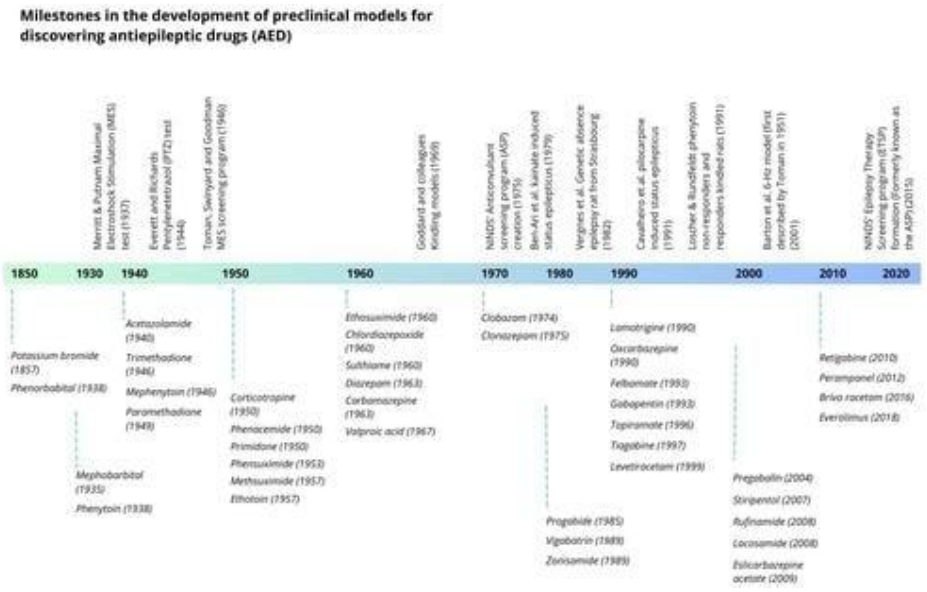

fig .1,3. Development of antiepileptic drug [8]

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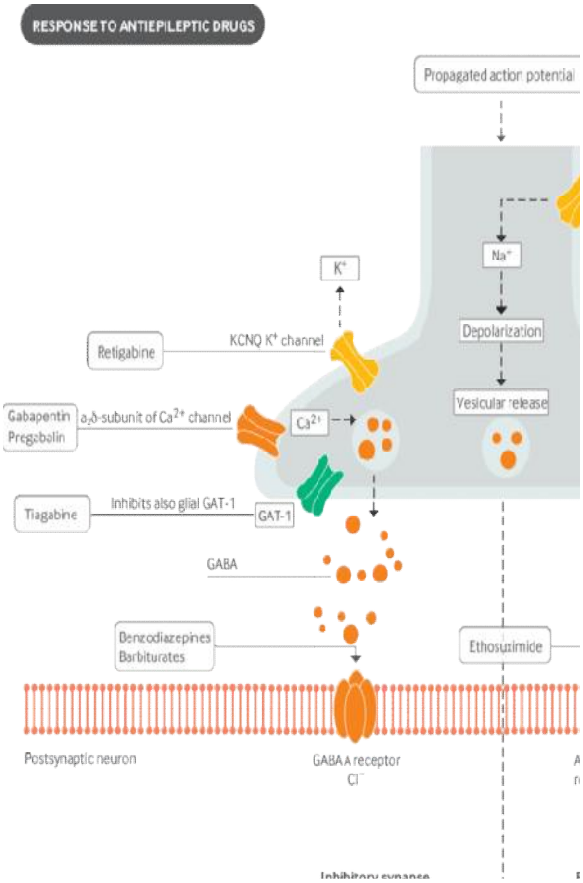
Mechanism of Action

Established antiepileptic drugs (AEDs) decrease membrane excitability by interacting with neurotransmitter receptors or ion channels. AEDs developed before 1980 appear to act on sodium channels, gamma-aminobutyric acid type A (GABAA) receptors, or calcium channels.

Benzodiazepines and barbiturates enhance GABAA receptor-mediated inhibition. Phenytoin (PHT), carbamazepine (CBZ), and possibly valproate (VPA) decreases high-frequency repetitive firing of action potentials by enhancing sodium-channel inactivation. Ethosuximide (ESM) and VPA reduce a low threshold (T-type) calcium-channel current. The mechanisms of action of the new AEDs are not fully established. Gabapentin (GBP) binds to a high-affinity site on neuronal membranes in a restricted regional distribution of the central nervous system.

This binding site may be related to a possible active transport process of GBP into neurons; however, this has not been proven, and the mechanism of action of GBP remains uncertain. Lamotrigine (LTG) decreases sustained high-frequency repetitive firing of voltage-dependent sodium action potentials that may result in a preferential decreased release of presynaptic glutamate.

The mechanism of action of oxcarbazepine (OCBZ) is not known; however, its similarity in structure and clinical efficacy to CBZ suggests that its mechanism of action may involve inhibition of sustained high-frequency repetitive firing of voltage-dependent sodium action potentials. Vigabatrin (VGB) irreversibly inhibits GABA transaminase, the enzyme that degrades GABA, thereby producing greater available pools of presynaptic GABA for release in central synapses. Increased activity of GABA at postsynaptic receptors may underline the clinical efficacy of VGB. [9]



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Classification of antiepileptic drug

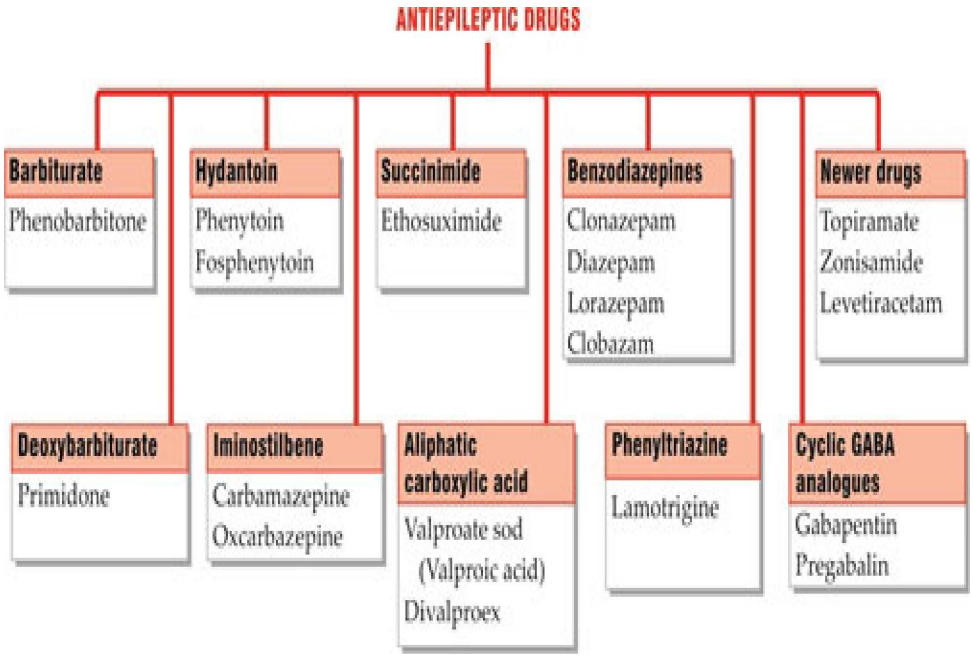
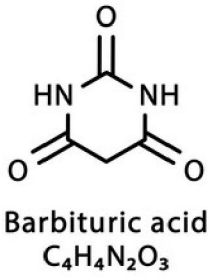


Fig.1.4. classification of antiepileptic drug.

1] Barbiturates: -

Barbiturates are medications that cause you to relax or feel drowsy. They can also stop or prevent convulsions and seizures. The most common uses are for anesthesia reasons, treating epilepsy and nonepileptic seizures, insomnia and other conditions. Barbiturates affect your brain by increasing a brain chemical called gamma-aminobutyric acid (GABA), which slows down the activity of your brain cells. [10]

Structure



Barbituric acid
$C_4H_4N_2O_3$

shutterstock.com · 2089927228

[11]

Mechanism of action

The primary mechanism of action of barbiturates is inhibition of the central nervous system. It causes central nervous system depression. This is brought about by stimulating the inhibitory neurotransmitter system in the brain called the [gamma]-aminobutyric acid (GABA) system. The GABA channel Is a Chloride channel that has five cells at its gate. When barbiturates bind to the GABA channel, they lead to prolonged opening of the channel letting in Chloride ions

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into the cells in the brain. This leads to increased negative charge and alters the voltage in the brain cells. This change in voltage makes the brain cells resistant to nerve impulses and thus depresses them.[12]

Examples: -

Butalbital: - This medication is part of many combination medications, including aspirin, acetaminophen, caffeine and codeine. Depending on the combination, it can treat migraines and tension headaches.

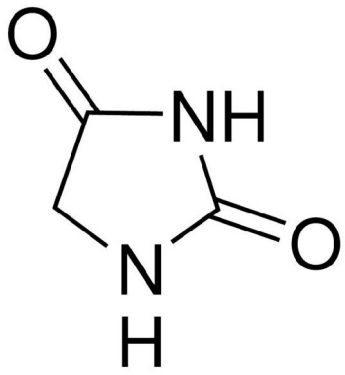
Methohexital: - This medication is useful for anesthesia in short diagnostic and treatment procedures. It's very helpful in procedures like electroconvulsive therapy.[13]

2. Hydantoin

Hydantoin anticonvulsants are structurally related to barbiturates. They have an allantoin heterocyclic base. Hydantoins slow the synaptic transmission by blocking sodium channels from recovering from the inactivated state, and inhibits neurons from firing. This stops the repeated excitation of cells that results in seizures. Hydantoin anticonvulsants are used to treat a wide range of seizures types.[14]

Structure: -

[15]



Mechanism of Action: -

A hydantoin-anticonvulsant that stabilizes neuronal membranes by decreasing sodium and calcium ion influx into the neurons. Also decreases post-tetanic potentiation and repetitive discharge. [16]

Examples

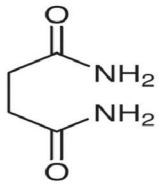
Phenytoin: -Phenytoin works by slowing down impulses in the brain that cause seizures. Phenytoin is used to control seizures.[17]

Sphenion: -Sphenion is an antiepileptic agent used for the management of generalized convulsive status epilepticus and prevention and treatment of seizures occurring during neurosurgery.[18]

3. **Succinimide: -**

Succinimide anticonvulsants are thought to increase the seizure threshold, inhibit T-type calcium channels and inhibit the three-cycle per second thalamic 'spike and wave' discharge in absence seizures. Succinimide anticonvulsants are mostly used to treat absence seizures.[19]

Structure :-



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[20]

Mechanism of action

Succinimides act on calcium 'T' channels to block voltage-dependent calcium conductance in thalamic neurons. Side effects of ethosuximide are either gastrointestinal (nausea, vomiting, and abdominal pain) or involve the central nervous system (CNS) (lethargy, dizziness, and ataxia).[21]

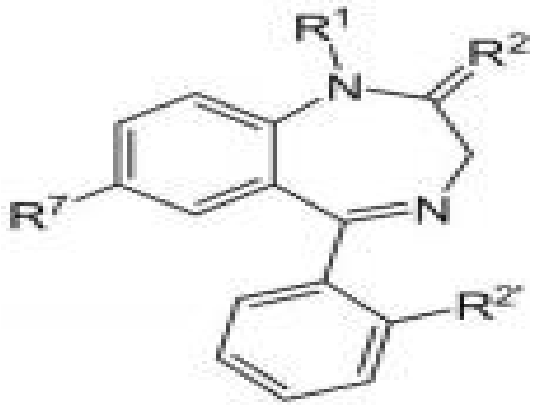
Examples

Ethosuximide remains a useful compound in pediatric practice in the treatment of absence seizures, but it does not appear to be effective against other seizure types. [22]

4. Benzodiazepines

Benzodiazepines are a class of medications that slow down activity in your brain and nervous system. They're most often used for treating anxiety and related mental health conditions, as well as brain-related conditions like seizures. These medications are tightly regulated and are only available with a prescription.[23]

Structure



[24]

Mechanism of action

The mechanism by which benzodiazepines, such as diazepam (DZP), enhance GABA receptor function has been termed allosteric. Allosteric, in this sense, refers to DZP binding at a site distinct from the agonist (GABA) binding site. Structure–function studies have indeed verified that the DZP binding site is distinct from the GABA binding site [25]

Examples

Lorazepam and diazepam are both FDA approved to treat anxiety and anxiety disorders, such as panic disorders and panic attacks. Like other benzodiazepines, lorazepam and diazepam can be used to treat seizure disorders, or epilepsy. They can also be used as a premedication before surgery for the purpose of sedation [26]

5. Iminostilbene

Iminostilbene is a chemical compound with two benzene rings fused to an azepine ring.[1] Many pharmaceuticals, such as carbamazepine, oxcarbazepine, and dopamine, are based on a dibenzoazepine structure.[27]

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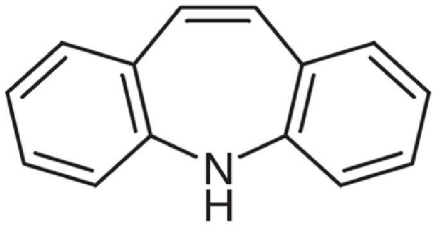
Structure:



[28]

Mechanism of Action: -

Iminostilbene is a sodium channel blocker binds preferentially to voltage-gated sodium channels in their inactive conformation, which prevents repetitive and sustained firing of an action potential.[29]

Examples

Carbamazepine (CBZ) is a carbamylated derivative of iminostilbene and is related structurally to the cyclic antidepressants. It is currently considered the drug of first choice for treatment of focal epilepsy and is also effective in the treatment of partial seizures.

6. Newer drugs

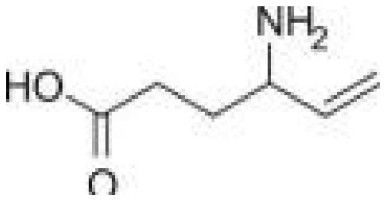
Examples

Vigabatrin: -

Vigabatrin is a medication used in the management and treatment of infantile spasms and refractory complex partial seizures. It is in the anti-epileptic class of medications.[30]

Mechanism of action: Vigabatrin is an irreversible inhibitor of gamma-amino-butyric acid transaminase (GABAT), an enzyme that degrades GABA. It is structurally the same as GABA with an extra vinyl group. Given this fact, it acts as a substrate for GABA-T, setting GABA free in the synaptic cleft. The concentration of GABA, a neuro-inhibitory transmitter, increases in the brain, terminating seizure activity. Apart from inhibiting GABA-T, vigabatrin prevents neuronal uptake of GABA and stimulates its release into the synapse. Some studies show that vigabatrin enhances the action of the inhibitory neurotransmitter glutamine, which researchers believe adds to its anticonvulsant effect.[31]

Structure



[32]

2.Tiagabine

Tiagabine is an anticonvulsant medication produced by Cephalon that is used in the treatment of epilepsy. The drug is also used off-label in the treatment of anxiety disorders and panic disorder. [33]

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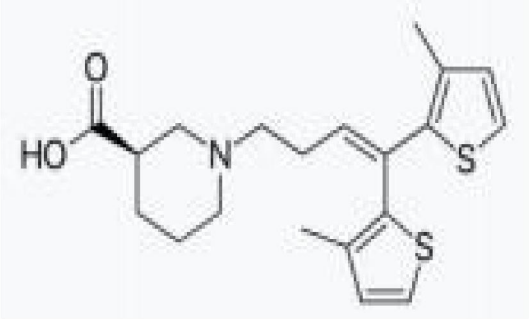
Mechanism of Action: -

The precise mechanism by which Tiagabine exerts its antiseizure effect is unknown, although it is believed to be related to its ability to enhance the activity of gamma aminobutyric acid (GABA), the major inhibitory neurotransmitter in the central nervous system. Tiagabine binds to recognition sites associated with the GABA uptake carrier. It is thought that, by this action, Tiagabine blocks GABA uptake into presynaptic neurons, permitting more GABA to be available for receptor binding on the surfaces of postsynaptic [34]

Structure

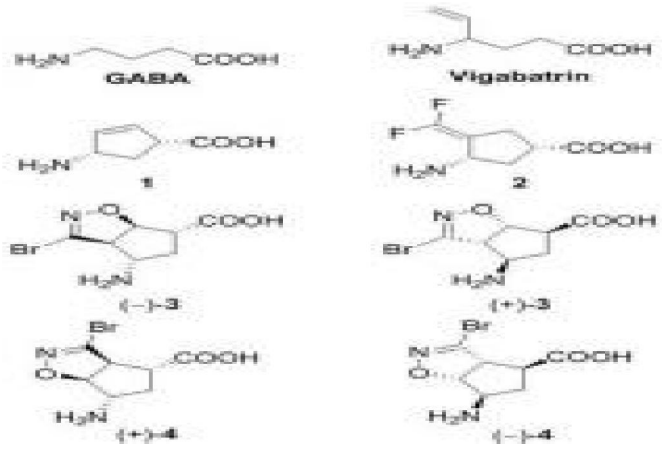


[35]

7. Cyclic GABA analogues

A GABA analogue is a compound which is an analogue or derivative of the neurotransmitter gamma-Aminobutyric acid (GABA) (the IUPAC of which is 4-aminobutanoic acid). Many GABA analogues are used as drugs, especially as anticonvulsants, sedatives, and anxiolytics.[36]

Structure



[37]

Mechanism of action

Gabapentin, a cyclic analogue of GABA, acts by enhancing GABA synthesis and also by decreasing neuronal calcium influx via a specific subunit of voltage-dependent calcium channels. Topiramate acts, in part, via an action on a novel site of the GABAA receptor.[38].

Example: -

Pregabalin and gabapentin are used to treat neuropathic pain (pain caused by an abnormality of, or damage to, the nerves) and epilepsy. They may be used alone or in combination with other medicines.[39]

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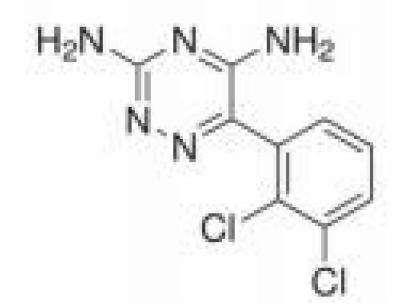
8] Phenyl triazine:

Phenyl triazines are a class of molecules containing a phenyl group and a triazine group. These molecules are pharmacologically important.[40]

Structure



[41]

• Mechanism of Action: -

Lamotrigine likely acts by inhibiting sodium currents by selective binding to the inactive sodium channel, suppressing the release of the excitatory amino acid, glutamate. The mechanism of action of lamotrigine in reducing anticonvulsant activity is likely the same in managing bipolar disorder.[42]

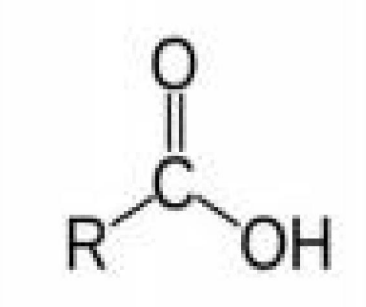
• Examples: -

Lamotrigine: - Lamotrigine is an antiepileptic drug belonging in the phenyl triazine class. It is used in the treatment of both epilepsy and as a mood stabilizer in bipolar disorder.[43]

9] Aliphatic Carboxylic Acid: -

Aliphatic acids, also known as aliphatic carboxylic acids, are organic compounds containing a carboxyl functional group (-COOH) attached to an aliphatic carbon chain. The term “aliphatic” refers to organic compounds that do not contain aromatic (benzene ring) structures [44]

Structure



[45]

• Mechanism of Action: -.

In general, carboxylic acids undergo a nucleophilic substitution reaction where the nucleophile (-OH) is substituted by another nucleophile (Nu). The carbonyl group (C=O) gets polarized (i.e. there is a charge separation), since oxygen is more electronegative than carbon and pulls the electron density towards itself.[46]

• Examples: -

Valproate: - Valproate is indicated for epilepsy only, whereas its derivative divalproex (DVP) and valpromide (VPM) are indicated for bipolar disorders only. DVP is composed of sodium valproate and valproic acid (VA) in a 1:1 molar ratio and VPM is a prodrug completely hydrolyzed in the gastric tract VA. [47]

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Formulation and dosage form of drugs

Drug / Indication	Formulations	Dosing	Pharmacokinetic Data	Initial Target Range
Carbamazepine (Tegretol®, Tegretol-XR®, Carbitrol®,) Indicated for complex partial seizures, generalized tonic-clonic seizures, mixed seizure patterns or other partial or generalized seizures. Not indicated for Absence seizures.	*Chewable tabs*: 100 mg *Tablets*: 200 mg *XR tabs*: 100, 200, and 400 mg *Extended-release caps:* 200 and 300 mg *Suspension*: 100 mg/5ml Peak plasma concentrations of carbamazepine epoxide are achieved in 2 hours or less for suspension, whereas enteric-coated tablets are slower on average 5–7 hours.	<6 yrs: Tablets, suspension: Start 5mg/kg PO daily divided BID-QID, increase every 5–7 days to a max of 35 mg/kg/day 6–12 yrs: Tablets, suspensions, XR tabs: Start 10mg/kg PO daily divided BID-QID, increasing by 100 mg every 7 days to a max of 1000 mg/day Over 12 yrs: Tablets, suspensions XR tabs: Start at 200 mg PO BID, then increase by 200 mg every 7 days. Max: 1000 mg/24h (12–15 yrs), 1200 mg/24h (>15 yrs), 1600 mg/24h (adults, in rare instances)	**Half-life:** Initially range from 25–65 hrs, decreasing to 12–17 hrs on repeated doses **Bioavailability:** 89% **Plasma Protein Binding:** 70–80%	4–12 µg/ml
Clonazepam (Klonopin®) Indicated for use alone or as an adjunct in the treatment of Lennox-Gastaut syndrome, akinetic and myoclonic seizures; also used in refractory absence seizures.	*Tablets:* 0.5, 1 and 2 mg Peak plasma concentrations achieved usually in 1 to 4 hours.	Infants and Children: (<10 yrs of age or < 30 kg) Initial dose between 0.01 and 0.03 mg/kg/day divided BID-TID, then increase by 0.25–0.5 mg every 3 days. Maintenance dose range between 0.1 to 0.2 mg/kg. Adults: (> 10 yrs or > 30 kg) Initial dose should not exceed 1.5 mg/day divided into 3 doses, increase by 0.5–1 mg every 3 days to a max dose of 20 mg/day.	**Half-life:** 30–40 hrs **Bioavailability:** 90% **Plasma Protein Binding:** 85%	20–75µg/L
Diazepam (Valium®, Diastat®) Indicated as adjunct in convulsive disorders, such as status epilepticus.	*Tablets:* 2, 5, and 10 mg *Oral Solution:* 5 mg/5ml and 10 mg/10ml *IV:* 5 mg/ml *Intensol* (Concentrate solution): 5 mg/ml *Diastat®* Rectal administration: 2.5, 5, 10, and 15 mg fixed unit-doses Peak plasma concentrations are achieved in 30 to 90 minutes for tablets and rectal formulations.	**Status epilepticus:** Infants to 5 yrs: IV: 0.05–0.3 mg/kg/dose given over 2–3 min every 15–30 min to a max of 5 mg; may repeat in 2–4 hrs as needed. > 5 yrs: IV: 0.05–0.3 mg/kg/dose given over 2–3 min every 15–30 min to a max of 10 mg; may repeat in 2–4 hrs as needed. Adults: IV: 5–10 mg Q10–20 min, up to 30 mg in 8-hr period, may repeat in 2–4 hrs as necessary Intensol® (Intranasally): 2 mg doses have been used Diastat® (Rectal administration): 2–5 yrs: 0.5 mg/kg 6–11 yrs: 0.3 mg/kg 12 yrs and adults: 0.2 mg/kg A second dose may be required and given 4–12 hrs after the first **Other:** Children >6 months: PO: 1 to 2 1/2 mg, 1–2 times daily, increase as tolerated Adults: PO: 2–10 mg, 2–4 times a day Geriatrics: PO: 2–2 ½ mg QD-BID initially, increase gradually as needed	**Half-life:** 46–71 hrs IV: 14.4 ± 7.0 hrs Intranasal: 17.8 ± 15.5 hrs **Bioavailability:** 90% **Plasma Protein Binding:** 95–98%	0.2–1.5 µg/ml
Ethosuximide (Zarontin®) Indicated for the control of absence seizures.	*Capsules:* 250 mg *Syrup:* 250 mg/5ml Peak plasma concentrations achieved in 1 to 4 hours for capsules, whereas syrup is faster.	3–6 yrs: Start 7.5 mg/kg PO BID, increase every 4–7 days to maintenance dose of 15–40 mg/kg/day in 2 divided doses 6 yrs and older: Start 250 mg BID, increase by 250 mg every 4–7 days to a maintenance dose of 20–40 mg/kg/day in 2 divided doses.	**Half-life:** Children: 30 hrs Adults: 50–60 hrs **Bioavailability:** 100% **Plasma Protein Binding:** <10%	40–100 µg/ml

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		maintenance dose of 20–40 mg/kg/day in 2 divided doses. Optimal dose for most pediatric patients is 20 mg/kg/day Dosages exceeding 1.5 g daily in divided doses may be required.	**Binding:** <10%	
Felbamate (Felbatol®) Indicated for partial seizures with and without generalization in adults and for the treatment of partial and generalized seizures associated with Lennox-Gastaut syndrome in children.	*Tablets:* 400 and 600 mg *Suspension:* 600 mg/5ml Peak plasma concentrations achieved in about 3 hours.	Children with Lennox-Gastaut syndrome (Ages 2–14 yrs): Adjunctive therapy: 15 mg/kg/day divided 3–4 times daily up to 45 mg/kg/day. Some children may require higher doses. ≥14 yrs and adults: Monotherapy and adjunctive therapy: 1200 mg/day divided 3–4 times daily to a maintenance dose of 2400 mg/day. Max dose is 3600 mg/day Geriatrics: Use caution in dosage selection due to decreased hepatic, renal or cardiac function.	**Half-life:** 20–30 hrs **Bioavailability:** 90% **Plasma Protein Binding:** 22–25%	40–100 µg/ml
Gabapentin (Neurontin®) Indicated as adjunctive therapy in adults for the treatment of partial seizures with and without secondary generalization	*Capsules*: 100, 300, and 400 mg *Tablets:* 600 and 800 mg Peak plasma concentrations achieved within 2 to 3 hours.	Pediatric dosing: Start 10–20 mg/kg/day, increase by 10 mg/kg/day to a maintenance does of 30 mg/kg/day. Max is 50–100 mg/kg/day in refractory patients. > 12 yrs and adults: Start 300 mg PO X 1 day 1, increase by 300 mg PO BID on day 2 and increase by 300 mg PO TID on day 3. Effective dose is 900 to 1800 mg/day given in divided doses TID to a maximum of 3600 mg/day. The maximum time between doses in the TID schedule should not exceed 12 hours. Benign Rolandic Epilepsy: 23–35 mg/kg/day Geriatrics and Renal patients: Reduction of dose maybe required in patients with age-related compromised renal function **CrCl (ml/min) Dose** > 60 400 mg TID 30–60 300 mg BID 15–30 300 mg QD <15 300 mg QOD	**Half-life:** 5–7 hrs **Bioavailability:** 60% **Plasma Protein Binding:** < 3% *Saturable absorption	4–16 µg/ml
Lamotrigine (Lamictal®) Indicated as adjunctive therapy in adults with partial seizures as well as generalized seizures of Lennox-Gastaut Syndrome	*Dispersible Tablets:* 5 and 25 mg *Tablets:* 25, 100, 150, and 200 mg **Both formulations are found to be equivalent whether administered dispersed in water, chewed and swallowed, or swallowed as whole. Peak plasma concentration achieved within 2 to 3 hours.	Adding to a regimen containing VPA in 2–12 yrs: Start 0.15 mg/kg/day in divided QD-BID X 2 wks, then increase to 0.3 mg/kg daily divided QD-BID x 2 wks, then increase every 1–2 wks by 0.3 mg/kg/day to a usual maintenance dose of 1–5 mg/kg/day. Max is 200 mg/day divided QD-BID. Adding to an enzyme-inducing regimen without VPA in 2–12 yrs: Start 0.6 mg/kg PO daily divided QD-BID in 2 divided doses X 2 wks, then increase to 1.2 mg/kg PO daily divided QD-BID X 2 wks, then increase every 1–2 weeks to maintenance of 5–15 mg/kg/day. Max is 400 mg/day in 2 divided doses Adding to a regimen containing VPA in >12 yrs: Start 25 mg QOD X 2 wks, then increase to 25 mg QD X 2 wks. Increase by 25–50 mg/day every 1–2 weeks until usual maintenance dose of 100–400 mg/day in 1–2	**Half-life:** Ages 10 mo-5.3 yrs: 7.7–44.9 hrs Ages 5–11 yrs: 7.0–65.8 hrs Adults: 14.4–70.3 hrs **Depends if patient is on an inducing or inhibiting anticonvulsant **Bioavailability:** 98% **Plasma Protein Binding:** 55%	2–20 µg/ml

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		divided doses. Adding to an enzyme-inducing regimen without VPA in >12 yrs: Start 50 mg/day X 2 wks, then increase to 100 mg/day in 2 divided doses X 2 wks. Increase by 100 mg/day every 1–2 wks until usual maintenance dose of 300–500 mg/day BID. Doses as high as 700 mg/day divided BID have been used.		
Levetiracetam Keppra®	*Tablets:* 250, 500 and 750 mg Peak plasma concentration within by 1.5 hrs.	Children: One study used 13–30 mg/kg in 5–12 yo. Data suggested children metabolized drug more quickly than adults. Adults: Start 500 mg PO every 12h, increase by 1000 mg/day every 2 wks to a maintenance dose between 500–1500 mg PO Q12H. Max dose is 3000 mg/day. Renal dosing: Adults: CrCl 30–50: 250–750 mg Q12H 10–30: 250–500 mg Q12H <10: 50–100 mg Q24H	**Half-life:** 6–8 hrs **Bioavailability:** 100% **Plasma Protein Binding:** <10%	20–60 µg/ml
Lorazepam (Ativan®) Indicated as adjunctive therapy for convulsive disorders, specifically Status Epilepticus.	*Tablets* : 0.5, 1, and 2 mg *IV:* 2 mg/ml *Tubex Blunt Pointe* (sterile cartridge units): 1 mg/0.5ml, 2 mg/ml, and 4 mg/ml *Intensol* (concentrate solution): 2 mg/ml and 4 mg/ml Peak plasma concentration achieved 1.5 to 2 hours.	Status Epilepticus: Neonates: 0.05 mg/kg IV over 2–5 minutes; may repeat in 10–15 min Infants and children: 0.1 mg/kg slow IV over 2–5 min, do not exceed 4 mg/single dose; may repeat second dose of 0.05 mg/kg slow IV in 10–15 min if needed Adolescents: 0.07 mg/kg slow IV over 2–5 min; max dose of 4 mg/dose; may repeat in 10–15 min Adults: 4 mg/dose given slowly over 2–5 min; may repeat in 10–15 min; max dose per 12-hour period is 8 mg Geriatrics: (PO) Initial dose of 1–2 mg/day in divided doses, adjust as needed and as tolerated	**Half-life:** 12 hrs **Bioavailability:** 90% **Plasma Protein Binding:** 85%	20–30µg/L
Midazolam (Versed®) Indicated for use in infants with status epilepticus refractory to other treatments.	*IV*: 1 mg/ml and 5 mg/ml *Oral solution:* 2 mg/ml	6 months to <6 yrs: Syrup: May require higher than usual dose up to 1.0 mg/kg, max dose of 20 mg 6 yrs to <16 yrs: Syrup: single dose of 0.25 to 0.5 mg/kg, max dose of 20 mg Status Epilepticus refractory to standard therapy: (IV) Infants >2 months and children: Loading dose of 0.15 mg/kg followed by a continuous infusion of 1 mcg/kg/min, range of 1–18 mcg/kg/min	**Half-life:** PO: 2.2–6.8 hrs IV: 2.9–4.5 hrs **Bioavailability:** >90% **Plasma Protein Binding:** 97%	Not routinely monitored
Oxcarbazepine (Trileptal®)	Tablet: 150, 300, 600 mg Peak plasma concentration within 4 hours	Children: Doses Start 8–10 mg/kg, do not exceed 600 mg/day in divided doses. Target dose should be achieved over a 2 week period and is dependent on patient weight. 20–29 kg: 900 mg/day 29.1–39 kg: 1200 mg/day >39 kg: 1800 mg/day Ranging from 20–75 mg/kg/day have been used Adults: 600–3000 mg/day divided BID	**Half-life:** 3 to 13 hrs **Bioavailability:** 96% **Plasma Protein Binding:** 33%	5–50 (MHD) µg/ml
Phenobarbital (Luminal®)	*Tablets:* 15, 16, 30, 32, 60, 65, and 100 mg *Oral Powder Capsules:* 15 mg	Status Epilepticus: (IV) Pediatric dose: Start 10–20 mg/kg IV X 1. Then 5–10	**Half-life:** Adults: 53–118 hrs (mean 78 hrs)	Infants and children: 15–30 µg/ml

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Indicated for generalized and partial seizures.	*Oral Powder Capsules:* 16 mg *Elixir:* 15 mg/5ml and 20 mg/5ml *IV:* 30 mg/ml, 60 mg/ml, 65 mg/ml, 130 mg/ml Peak plasma concentration is about 2 hours after an oral dose.	mg/kg IV X 1. Then 5–10 mg/kg IV Q 15–30 min. Max 40 mg/kg. Neonates: Loading dose of 15–20 mg/kg in a single or divided dose Infants, children, and adults: 15–18 mg/kg in a single or divided doses; usual max loading dose is 20 mg/kg Maintenance dose: (IV or PO) (usually starts 12 hrs after the loading dose) Neonates: 3–4 mg/kg/day QD, may increase to 5 mg/kg/day if needed Infants: 5–6 mg/kg/day in 1–2 divided doses Children and adults: 1–5 yrs: 6–8 mg/kg/day in 1–2 divided doses 5–12 yrs: 4–6 mg/kg/day in 1–2 divided doses >12 yrs and adults: 1–3 mg/kg/day in 1–2 divided doses Seizure disorder: 2 months: 3–5 mg/kg PO/IV daily divided QD-BID 2 mo- 2 yrs: 5–8 mg/kg PO/IV daily divided QD-BID > 2 yrs: 3–5 mg/kg PO/IV daily divided QD-BID **Dose should also be reduced with renal impairment or hepatic disease.	(mean 79 hrs) Children and newborns (<48 hrs old): 60–180 hrs (mean 110 hrs) **Bioavailability:** 70–90% **Plasma Protein Binding:**20–45%	µg/ml Adults: 10–40 µg/ml
Phenytoin (Dilantin®) Indicated for the control of generalized tonic clonic and complex partial seizures. Prevention and treatment of seizures occurring during or following neurosurgery.	*Infatabs (chewable)*: 50 mg *Suspension:* 125 mg/5ml *Extended-release capsule:*30 and 100 mg *IV:* 50 mg/ml Peak plasma concentra Tion achieved for extended-release capsule is between 4–12 hours and for chewable table and suspension ranges between 2–3 hours. **Kapseals and IV are formulated with the sodium salt of phenytoin. Suspension and infatabs are formulated with the free acid form. There is approximately an 8% increase in the drug content with the free acid form over the sodium salt. Dosage adjustments and serum level monitoring may be necessary.	Pediatrics: Seizure disorder: Neonates: Start 15–20 mg/kg IV X 1 or 3 divided oral doses given 2–4 hrs apart. Maintenance 5–8 mg/kg/day IV/PO divided BID-TID. Begin maintenance dose 12 hrs after loading dose. Infants and children: Start 10–20 mg/kg IV X 1 or 3 divided oral doses given 2–4 hrs apart. Decrease by 1–2 mg/kg/day for every 2–3 yrs of age. Begin maintenance dose 12 hr after loading dose. Maintenance dose is 5–10 mg/kg/day IV/PO divided BID-TID. >6 yrs and adolescents: For all formulations: May require the minimum adult dose of 300 mg/day Adults: Infatabs: Maintenance is usually 300 – 400 mg/day divided BID-TID. Max is 600 mg/day. Extended-release: See dose above Suspension: See dose above Status Epilepticus (IV): Neonates: Loading dose 15–20 mg/kg in single or divided dose. Begin maintenance dose 12 hrs after: 5 mg/kg/day in 2 divided doses, usual 5– 8 mg/kg/day in 2 divided doses, some patients may require dosing Q8hrs Infants and children: Loading dose 15–18 mg/kg in a single or divided dose, maintenance dose of 5 mg/kg/day in 2–3 divided doses Usual doses: 0.5–3 yrs: 8–10 mg/kg/day 4–6 yrs: 7.5–9 mg/kg/day 7–9 yrs: 7–8 mg/kg/day	**Half-life:** Oral: 7–42 hrs (average 22 hrs) Infatabs: 7–29 hrs (average 14 hrs) **Bioavailability:** 100% **Plasma Protein Binding:** 90–95%	5–25 µg/ml

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		7–9 yrs: 7–8 mg/kg/day 10–16 yrs: 6–7 mg/kg/day Adults: Loading dose 15–18 mg/kg in a single or divided doses, maintenance dose of 300 mg/day or 4–6 mg/kg/day in 2–3 divided doses		
Tiagabine (Gabatril®) Indicated as adjunctive therapy in adults and children 12 years and older in the treatment of partial seizures.	*Tablets:* 2, 4, 12, 16, and 20 mg Peak plasma concentration obtained in 30 minutes.	12–18 yrs: Start 1–3 mg/kg/day PO QHS X 1 wk, increase to 8 mg PO QD on wk 2, then increase by 4–8 mg/wk to a max of 32 mg/day. Total daily dose should be given in divided doses of 2–4 times/day Adults: Initiate at 4 mg QD, increase PRN to max of 56 mg/day divided 2–4 times/day Geriatrics: See above adult dosing	**Half-life:**7–9 hrs **Bioavailability:**90% **Plasma Protein Binding:**95%	5–70 ng/ml
Topiramate (Topamax) Indicated as adjunctive therapy for adults and pediatric patients ages 2–16 years with partial onset seizures or primary generalized tonic-clonic seizures.	*Tablets:* 25, 100, and 200 mg *Sprinkle capsules:* 15 and 25 mg Peak plasma concentration achieved between 2 to 4 hours.	2–16 yrs: Start 1–3 mg/kg/day PO QHS X 1 wk. Titrate by 1–3 mg/kg/day divided bid at 1–2 wk intervals. Recommended total daily dose as adjunctive is 5–9 mg/kg/day in 2 divided doses, titrate up to optimal clinical response. Max 60 mg/kg/d Primary generalized tonic-clonic seizures: slower initial titration rate, dose of 6 mg/kg/day to be reached at 8 weeks Infantile Spasms: Up to 24 mg/kg/day has been used 17 yrs and adults: Initiate at 50 mg/day, daily maintenance dose as adjunctive therapy is 400 mg/day in 2 divided doses. Daily doses above 1600 mg have not been studied **Primary generalized tonic-clonic seizures: slower initial titration rate, maintenance dose should be reached at the end of 8 weeks Geriatrics: Reduce the usual adult dose by half if CrCl < 70 ml/min	**Half-life:** 21 hrs **Bioavailability:** 80% **Plasma Protein Binding:** 13–17%	2–25 µg/ml
Valproate/divalproex sodium (Depakote , Depakene) Indicated for use as monotherapy and adjunctive therapy in the treatment of simple and complex absence seizures. Adjunctive therapy in patients with multiple seizure types which includes absence seizures.	*Delayed-release tablets:* 125, 250, and 500 mg *Extended-release:* 500 mg *Sprinkle capsules:* 125 mg *Capsules:* 250 mg *IV:* 100 mg/ml *Syrup:* 250 mg/5ml Peak plasma concentration for syrup, capsule and tablet are achieved in less than 2 hrs, whereas for enteric-coated tablets is slower (3 to 8 hrs).	Children and adults: (IV) Total daily IV dose is equivalent to the total daily oral dose; IV dose should be divided every 6 hours; switch patients to oral as soon as clinically possible. IV has not been studied for >14 days. Initial dose of 10–15 mg/kg/day in 1–3 divided doses, increase by 5–10 mg/kg/day every 7 days to maintenance dose of 30–60 mg/kg/day in 2–3 divided doses. Max dose is 60 mg/kg/day. (Divalproex can be given BID) Depakene (valproic acid, capsules and syrup): Initial dose of 5–10 mg/kg/day until seizures are controlled. Max dose is 60 mg/kg/day. (If total daily dose exceeds 250 mg, should be given in divided doses).	**Half-life:** Newborns (1st week of life): 30–60 hrs Newborns <10 days: 10–67 hrs Children > 2 months: 7–13 hrs 2–14 yrs: 3.5–20 hrs Adults: 8–17 hrs **Bioavailability:** Approximately 100% (oral and IV are equivalent) **Plasma Protein Binding:** 80–90%	50–150 µg/ml

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		Depakote (divalproex sodium) sprinkle capsules: Monotherapy: Initial dose of 10–15 mg/kg/day. Max dose is 60 mg/kg Adjunctive: May be added at a dosage of 10–15 mg/kg/day. Max daily dose is 60mg/kg. (Doses exceeding 250 mg must be in divided doses). Depakote (divalproex sodium) delayed-release tablets: Monotherapy and adjunctive therapy: 10–15 mg/kg/day. Max daily dose is 60 mg/kg		
Vigabatrin Sabril® Infantile spasms* Partial seizures* * not FDA approved	Tablet: 500 mg Peak plasma concentration achieved in 1 hr.	Infantile Spasms: 40–100 mg/kg/day PO Adult: 500 mg PO Q12H, increase by 500 mg/day to a max of 4 g/day.	**Half-life:** 6–8 hrs **Bioavailability:** 50–75% **Plasma Protein Binding:** Negligible	Serum concentration not related to pharmacological effect
Zonisamide (Zonegran®)	Capsule: 100 mg Peak plasma concentration between 2 – 6 hrs.	Children: 2–4 mg/kg/day initially, increase up to a max of 4–12 mg/kg/day Adult: Start 100 –200 mg/day, gradually increase to 200–400 mg/day up to a maximum dose of 600 mg/day in divided doses.	**Half-life:** 50 – 70 hrs **Bioavailabiity:** 82 – 88% **Plasma Protein Binding:** 40–60%	10–40 µg/ml

Pathophysiology:-

Epileptic seizures are characterized by hypersynchronous and sudden bursts of electrical activity within groups of hyperexcitable neurons. These events occur when there is a transient imbalance between neuronal excitation and inhibition. The abnormal firing of action potentials typically begins in a localized area of the cortex or hippocampus and can subsequently propagate to other cortical and subcortical regions

The clinical manifestations of a seizure depend on factors such as the site of initiation (the seizure focus), the duration, and the electrographic pattern of the abnormal hypersynchronous discharges Within this focal region, seizure generation is primarily associated with enhanced excitatory signaling due to overactive glutamatergic transmission and dysfunction of ligand- or voltage-gated sodium and calcium channels. Additionally, reduced inhibitory control—mainly resulting from impaired γ-aminobutyric acid (GABA)-mediated neurotransmission and disturbances in extracellular potassium regulation—also contributes to seizure activity

Epileptogenesis refers to the progressive alterations in neuronal excitability that culminate in spontaneous and recurrent seizures]. Multiple mechanisms influence neuronal excitability, including changes in neurotransmitter and neuromodulator systems, ion channel and receptor function, neurogenesis, inflammation, apoptotic pathways, gene and protein expression, and astrocytic activity.[48]

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Sign and symptoms :-

Numerous adverse effects are erratic and unrelated to the dosage or blood level of the medicine. Another name for them might be "idiosyncratic" side effects. These may consist of:

-Rash
-Problems with the liver or pancreas
-A serious drop in the number of white blood cells in your body (needed to fight infection)
-A serious drop in the number of platelets in your body (needed to control bleeding)
-Aplastic anemia (severe damage to bone marrow so blood cells aren't produced normally)
-Liver failure.[49]

Discussion: -

In cases where standard therapies fail, a medication called XEN1101 reduces seizure frequency by more than 50% in some patients and sometimes eliminates them altogether, a new study shows. Unlike several treatments that must be started at low doses and slowly ramped up, the new drug can safety be taken at its most effective dose from the start, the authors say. Focal seizures, the most common type seen in epilepsy, occur when nerve cells in a particular brain region send out a sudden, excessive burst of electrical signals. Along with seizures, this uncontrolled activity can lead to abnormal behavior, periods of lost awareness, and mood changes. While many available therapies control or reduce seizures, they fail to stop seizures in about one-third of patients and may cause harsh side effects, experts say.

Led by researchers at NYU Grossman School of Medicine, a new clinical trial found that patients who added XEN1101 to their current antiseizure treatments saw a 33% to 53% drop in monthly seizures, depending on their dose. By contrast, those given a placebo had on average 18% fewer seizures during the treatment phase of the trial, which lasted eight weeks. Most patients then volunteered to extend the trial, with about 18% of those treated with the new drug remaining entirely seizure free after six months, and about 11% having no seizures after a year or longer.

"Our findings show that XEN1101 may offer a swift, safe, and effective way to treat focal epilepsy," said study lead author, neurologist Jacqueline French, MD. "These promising results offer hope for those who have struggled for decades to get their symptoms under control. "For the study, which included 285 men and women with epilepsy and ran from January 2019 to September 2021, the research team recruited adults with epilepsy who had already tried and stopped taking an average of six drugs that failed to treat their focal seizures.

Patients in the trial had to have experienced at least four episodes a month despite ongoing treatment to qualify. The patients were randomly provided either a daily oral capsule of XEN1101 (in doses of 10 milligrams, 20 milligrams, or 25 milligrams) or an inert placebo tablet that appeared identical to the real drug. Among the results, the trial revealed no signs of dangerous side effects such as heart problems, allergic reactions, or concerning skin discolorations.

However, French says that the research team plans to expand the number of patients exposed to the drug and monitor for potential issues that could arise in the long term, or include specific groups of people, such as pregnant women. In addition, the team also intends to explore XEN1101 for other types of seizures, including those that broadly affect the brain at the same time (generalized seizures).

"Our study highlights the importance of finding as many therapeutic options as possible for those who suffer from seizures," says French. "Since everyone responds differently, treating epilepsy cannot be a one-size-fits-all approach." [50]

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Standard (first generation)	Newer (second generation)
Carbamazepine (Tegretol)	Acetazolamide (Diamox)
Clonazepam (Klonopin)	Clobazam (Onfi)
Chlorazepate (Tranxene)	Ezogabine (Potiga)
Diazepam (Valium)	Felbamate (Felbatol)
Divalproex sodium (Depakote)	Fosphenytoin (Cerebyx)
Ethosuximide (Zarontin)	Lacosamide (Vimpat)
Ethotoin (Peganone)	Lamotrigine (Lamictal)
Lorazepam (Ativan)	Levetiracetam (Keppra)
Mephobarbital (Mebaral)	Oxcarbazepine (Trileptal)
Methsuximide (Celontin)	Parampanel (Fycompa)
Nitrazepam (Mogadon)	Pregabalin (Lyrica)
Phenobarbital (Gardinal)	Progabide (Gabrene)
Phenytoin (Dilantin)	Rufinamide (Banzel)
Primidone (Mysoline)	Tiagabine (Gabitril)
Valproic acid (Depakene)	Topiramate (Topamax)
	Vigabatrin (Sabril)
	Zonisamide (Zonegran)

Fig.1.5 newer antiepileptic drugs

Side effects

All medications have the possibility of causing side effects. There are three main types of antiepileptic medication side effects.

Some mild side effects are common when starting antiepileptic medications, particularly if the dose is increased rapidly. These include nausea, abdominal pain, dizziness, sleepiness, irritability, anxiety or mood changes. These are usually not serious, but may worry some people and should be discussed with your doctor or pharmacist. Your doctor may slow the rate at which the medication is being introduced or may cut back another antiepileptic medication with which it may be interacting.

Some side effects are common to antiepileptic medications when prescribed at too great a dose and are similar to being "drunk" e.g. Unsteadiness, poor concentration, sleepiness, double vision, vomiting, tremor. It is important to report these side effects to your doctor immediately if they occur.

Some side effects are peculiar to individual medications and only occur in some people e.g. Rash, blood problems, liver problems, severe behaviour disturbance, worsening of seizure control. There are some patients or situations in which there may be increased risk of such side effects with a certain medication.

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Narrow-Spectrum AEDs:	Broad-Spectrum AEDs:
– phenytoin (Dilantin)	– valproic acid (Depakote)
– phenobarbital	– lamotrigine (Lamictal)
– carbamazepine (Tegretol)	– topiramate (Topamax)
– oxcarbazepine (Trileptal)	– zonisamide (Zonegran)
– gabapentin (Neurontin)	– levetiracetam (Keppra)
– pregabalin (Lyrica)	– clonazepam (Klonopin)
– lacosamide (Vimpat)	– rufinamide (Banzel
– vigabatrin (Sabril)	

Fig.1.6.side effects of antiepileptic drug.

There are two main categories of anti-epileptic drugs: narrow-spectrum ones that treat a few specific types of seizures, like partial seizures, and broad-spectrum ones that can treat both partial seizures and grand mal or myoclonic seizures (those characterized by muscle contractions during which a patient remains conscious). Most of the drug's use similar mechanisms to decrease brain activity that leads to seizures and have similar side effects.[51]

Precautions

Effects on ability to drive and use machines. Some antiepileptic medications can cause drowsiness, sleepiness, incoordination and slowed reaction time, especially when the medication is being introduced or the dose is being increased. Effectsof individuals should be assessed prior to driving or using machinery. Effects when taken with alcohol. If alcohol is taken in combination with antiepileptic medication, extra sedative effects can occur. Also, alcohol increases the likelihood of seizures.[52]

II. CONCLUSION

Epilepsy is a common and serious medical condition which mainly treated by general practitioners. The development of new antiepileptic drugs has broadened treatment options and provided significant benefits to patients who experience negative effects or frequent seizures despite using traditional drugs. Clinical trials have shown that the new medications are effective and well-tolerated. While there is no proof that the new drugs are more effective, Research indicates that they have a wider range of benefits, fewer interactions with other drugs, and are generally better tolerated by patients. As more research is conducted and experience with these drugs grows, more advantages may come to light.We come on conclusion find out or develop such medicine treatment who have high therapeutic index & less adverse drug reaction So further investigation needed.

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