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MEANS USED IN EPILEPSY (LITERATURE REVIEW).

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ABSTRACT

The mechanism of action of anticonvulsants has not been fully studied. These drugs mainly prevent the spread (generalization) of impulses in an epileptic center, because their use reduces the amount of neurotransmitters (glutamide) and increases the amount of neurotransmitters (GAMK) in the brain, reducing the transmission of impulses between neurons. Such changes in neurotransmitters reduce the sensitivity of neurons and prolong their refractory period. Of course, changes in the activity of ATPases, which control the exchange of ions in the nerve endings, are of particular importance. Antiepileptic drugs do not completely eliminate seizures, but due to regular and continuous consumption, they stop the exacerbation of the disease by reducing and reducing the attacks and ensure the ability of patients to work. But if you suddenly stop taking the drug and suddenly reduce the dose, there is a risk of worsening of the disease and turning into epileptic status.

The first drug with high efficiency is phenobarbital, but this drug induces sleep. Antitussive drug is used in rare cases where it does not induce sleep (mainly in large cases).

Key words

Diphenine, Hexamidine, Etaminal, Clonazepam, Carbamazepin, Trimetine, Ethosuxymide, Sodium volproate, Lamotrigine

Introduction. Myoclonus creates significant disability in patients. This symptom or sign can have many different etiologies, presentations, and pathophysiologic mechanisms. A complete evaluation of the etiology of myoclonus is essential to develop a treatment strategy. The best etiologic classification scheme is a modified version of that proposed by Marsden et al. Clinical neurophysiology evaluated by electromyography and electroencephalography in 1982 can be used to classify the pathophysiology of myoclonus using the neurophysiology classification scheme. If the etiology of the myoclonus cannot be eliminated or treated, symptomatic treatment of the myoclonus itself may be necessary. Unfortunately, there are few controlled studies on the treatment of myoclonus. Myoclonus treatment strategies are derived from the best categories from the neurophysiology

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classification scheme: 1) cortical, 2) cortical-subcortical, 3) subcortical-nonsegmental, 4) segmental, and 5) peripheral. The classification of cortical physiology is the most common. Levetiracetam is recommended as a first-line treatment for cortical myoclonus, but valproic acid and clonazepam are commonly used. Cortical-subcortical myoclonus is a physiology manifested by myoclonic seizures, such as primary epileptic myoclonus (eg, juvenile myoclonic epilepsy). Valproic acid has shown efficacy with other drugs that provide an adjunctive role in such epileptic syndromes. Clonazepam is used for subcortical-nonsegmental myoclonus, but depending on the syndrome, other treatments are used for this physiological type of myoclonus. Treatment of segmental myoclonus is difficult, but clonazepam and botulinum toxin are used. Botulinum toxin is used for focal examples of peripheral myoclonus. Treatment for myoclonus is usually ineffective and/or limited by side effects[1].

Anticonvulsants (also known as **antiepileptic drugs**, **antiseizure drugs**, or **anti-seizure medications** (**ASM**)) are a diverse group of pharmacological agents used in the treatment of epileptic seizures. Anticonvulsants are also increasingly being used in the treatment of bipolar disorder and borderline personality disorder, since many seem to act as mood stabilizers, and for the treatment of neuropathic pain. Anticonvulsants suppress the excessive rapid firing of neurons during seizures. Anticonvulsants also prevent the spread of the seizure within the brain.

Conventional antiepileptic drugs may block sodium channels or enhance γ-aminobutyric acid (GABA) function. Several antiepileptic drugs have multiple or uncertain mechanisms of action.

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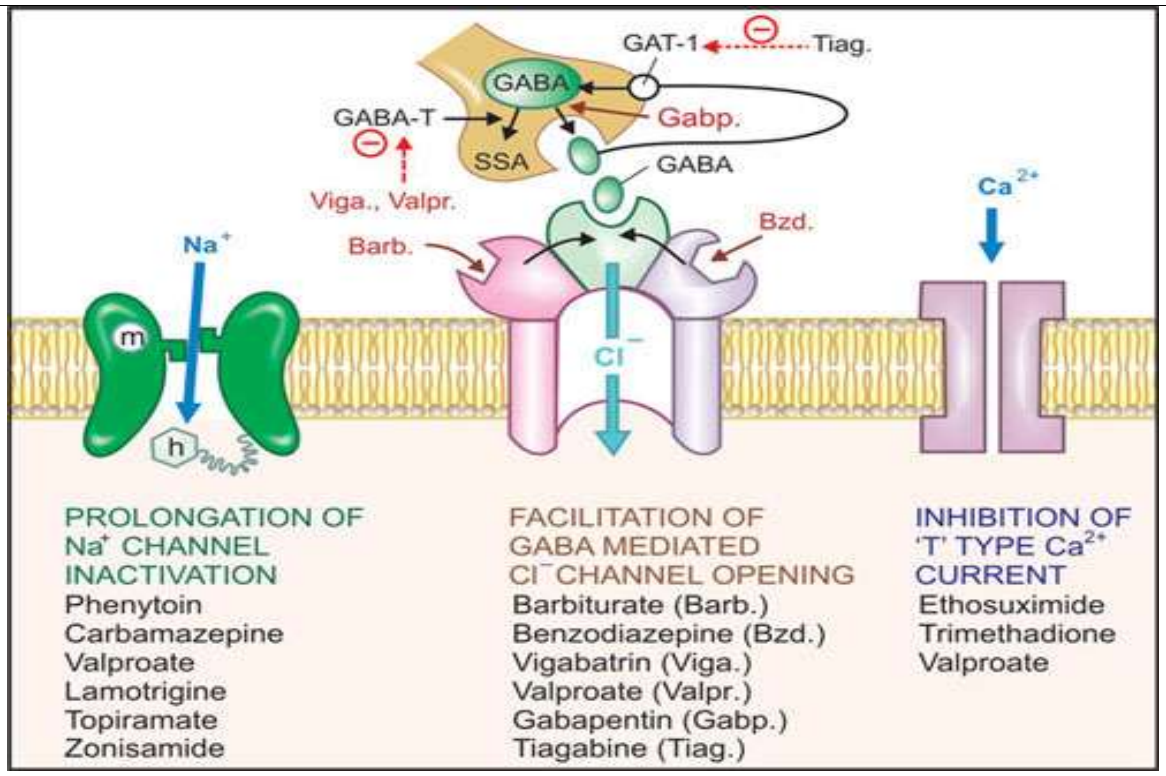

Next to the voltage-gated sodium channels and components of the GABA system, their targets include $GABA_A$ receptors, the GABA transporter type 1, and GABA transaminase. Additional targets include voltage-gated calcium channels, SV2A, and α2δ. By blocking sodium or calcium channels, antiepileptic drugs reduce the release of excitatory glutamate, whose release is considered to be elevated in epilepsy, but also that of GABA. This is probably a side effect or even the actual mechanism of action for some antiepileptic drugs, since GABA can itself, directly or indirectly, act proconvulsively. Another potential target of antiepileptic drugs is the peroxisome proliferator-activated receptor alpha.

Some anticonvulsants have shown antiepileptogenic effects in animal models of epilepsy. That is, they either prevent the development of epilepsy or can halt or reverse the progression of epilepsy. However, no drug has been shown in human trials to prevent epileptogenesis (the development of epilepsy in an individual at risk, such as after a head injury)[.2] A review of more than 200 studies over the past three decades found that anticonvulsants were effective in healthy people, in patients with and without epilepsy, and with and without brain damage. multifaceted psychological effects on patients are analyzed and summarized. The following results are mainly shown: 1. Carbamazepine: can have a positive effect on "expansive" behavior and mood in about 50-60% of patients. Cognitive and psychomotor indicators are almost unchanged. 2. Valproinate: negative mental effects can rarely be observed for a long time (exception: reversible encephalopathy). May have carbamazepine-like effects on behavior. 3. Phenytoin: It has a negative effect on cognitive and psychomotor indicators. The effect on

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behavior is opposite. 4. Phenobarbitone and Primidone: various adverse effects on performance and behavior seemed to be proven, especially in children and adolescents as "extended" disorders. 5. Ethosuximide: In addition to drug-specific provocation of psychotic disorders, it has both positive and negative effects on behavior and cognitive functions. 6. The psychoactive effects of other anticonvulsants such as benzodiazepines, Sultiam, and Feneturide are briefly described. 7. Polytherapy: has a negative effect on mental functions. Finally, the results are discussed in terms of their clinical relevance[3]. Seizure-epilepsy is a common disease, which develops suddenly, unexpectedly, and is accompanied by various types of attacks: Grand convulsions This attack begins with an aura and quickly progresses to exertion. Patient falls down. After exertion, the patient becomes weak and falls asleep. Can't remember what happened when you wake up (amnesia).

Psychotic attacks are disorders of the patient's behavior, which he is not aware of

Sometimes there are unreasonable actions, but there is no effort.

Small convulsions - short-term loss of consciousness, some groups of muscles in the face and other parts of the body may twitch

Short-term contraction of muscles in the case of myoclonus swelling) is observed..

Epileptic status is considered a severe condition in the course of the disease, there are many (up to 200-300) consecutive, rapidly recurring seizures (convulsions) in one day. Patients do not have time to regain consciousness. It can lead to death (due to weakening of the heart muscle, development of pulmonary edema and paralysis of breathing). But most patients survive.

Antitussives are effective in preventing and reducing outbreaks. The disease does not disappear completely. This group of tools does not give the same result in all manifestations of the disease. Therefore, different means are used for each form of the disease.

Treatment of epilepsy takes many years. Therefore, the tools used in them must meet the following requirements: should have high activity and long-lasting effect;

They should be well absorbed from MIT; they should also give good results in various types of seizures; there should be little or no side effects. Because patients accept it every day, for years; these tools do not need to accumulate in the body, to learn and develop dependence on them; they should be less toxic, the width of the therapeutic effect should be large.

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Group name	Drugs included
AP	Risperidone, Quetiapine, Olanzapine, Aripiprazole, Amisulpride, Paliperidone, Ziprasidone, Haloperidol
CLZ	Clozapine
AD	Escitalopram, Sertraline, Paroxetine, Fluoxetine, Bupropion, Venlafaxine, Trazodone
AED	Valproate, Carbamazepine, Lamotrigine, Topiramate, Levetiracetam
Li	Lithium
BDZ	Lorazepam, Clonazepam, Alprazolam, Diazepam
ZPD*	Zolpidem
AC*	Benztropine, Trihexyphenidyl
PPL*	Propranolol

A drug that fully meets all these requirements and gives good results in all attacks of the disease has not been created yet[4,5,6,7,8,9,10].

The purpose of the work: to provide general knowledge about anticonvulsant drugs and their use. To give a classification of anticonvulsant drugs. To provide an understanding of the main effect of anticonvulsants. Understanding the mechanisms of action of anticonvulsant drugs. To educate about the side effects of anticonvulsant drugs. To educate about the instructions for use and contraindications of anticonvulsant drugs.

Analysis Of The Work The mechanism of action of anticonvulsants has not been fully studied. These drugs mainly prevent the spread (generalization) of impulses in an epileptic center, because their use reduces the amount of neurotransmitters (glutamide) and increases the amount of neurotransmitters (GLMK, gdiin) in the brain, reducing the transmission of impulses between neurons. Such changes in neurotransmitters reduce the sensitivity of neurons and prolong their refractory period. Of course, changes in the activity of ATPases, which control the exchange of ions in the nerve endings, are of particular importance. Antiepileptic drugs do not completely eliminate seizures, but due to regular and continuous consumption, they stop the exacerbation of the disease by reducing and reducing the attacks and ensure the ability of patients to work. But if you suddenly stop taking the drug and suddenly reduce the dose, there is a risk of worsening of the disease and turning into epileptic status The first drug with high efficiency is phenobarbital, but this drug induces sleep. Antitussive drug is used in rare cases where it does not induce sleep (mainly in large cases).

DIPHENIN: is a product of hydantoin, which increases the release of Na* ions from neurons and reduces their excitability. It increases the amount of GAMK in the brain. Well absorbed from MIT. Increases the activity of MOS in the liver. The effect is about 20-30 hours. It accumulates in the body less than phenobarbital. He

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doesn't sleep. Has an antiarrhythmic effect (especially in arrhythmias caused by cardiac glycosides).

It is used in severe convulsions, psychomotor attacks and status epilepticus.

Adverse effects: dizziness, body temperature increase, ataxia, nystagmus, tremor, allergic reactions, respiratory disorders, dyspepsia, hirsutism, hyperplasia of the gum is observed in many cases. It cannot be used during pregnancy, liver, kidney and heart failure, cachexia **HEXAMIDIN** is a pyrimidine derivative. To phenobarbital in chemical structure

similar. It is less effective than phenobarbital, but it is less toxic and less sedating. Absorbed quickly from MIT. Effect7-14 hours. It is broken down in the liver. 20% feconverts to nobarbital, 40% is excreted unchanged in the urine. MOS activity index strengthens. Effective in major seizures, effective in psychotic attacks less expensive.

Side effects: ataxia, drowsiness, nausea, headache, dermatitis, leukopenia. lymphocytosis, anemia. The amount of folic acid is reduced. With continued use, it induces mental changes. It is impossible to use in diseases of the liver, kidney and hematopoietic organs, in pregnancy.

ETAMINAL.- a product of barbituric acid, which gradually turns into phenobarbigal in the body. It does not differ from pheobarbital in its anticonvulsant activity. but compared to it, it is less toxic, does not cause drowsiness, is well absorbed from MIT. Patients responded well. It gives a good result in all types of epilepsy with convulsions, as well as in preventing attacks without convulsions. Increases the activity of MOS. It has a hypobilirubinemic, antioxidant, antihypoxant effect. Increases bile secretion.

Unpleasant effects develop very rarely. Cannot be used in decompensated heart, kidney and liver failure.

CHLOROCONE-chloropropionamide product. Used in severe convulsions. Strong anti-aging effect. Patients respond well.

Adverse effects: dyspepsia, dizziness, impaired metabolism, liver and kidney. Control is needed when shooting, especially when shooting from a distance.

Clonazepam is a benzodiazepine product similar to nitrazepam in its chemical structure. It has sedative, muscle relaxant, anxiolytic and antispasmodic effects, the antispasmodic effect is stronger than that of other benzodiazepines. MIT is quickly absorbed. Effect 18-20 hours. Excreted in urine. Adults are used in small and large convulsions of cholera. This drug generally gives good results in all types of seizures.

Adverse effects: ataxia. dyspepsia, irritability, irritability, irritability. Can not be used during pregnancy, breastfeeding, transport drivers, myasthenia

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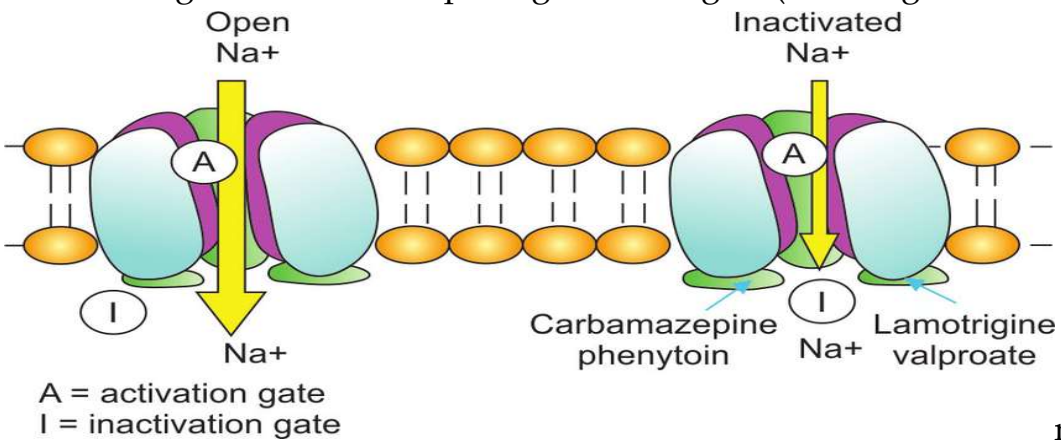
Carbamazepine is a derivative of dibenzazepine. Against hard work. has an analgesic, moderate aitidipressant, activating effect. Blocks the Na channel in nerve cell membranes. reduces the activity of excitatory neurotransmitters (glutamate). GAMK enhances sexual processes. All from MIT absorbed at the same rate in patients. The effect is 10-20 hours. Carbamazepine can be used together with phenobarbital, hexamidine and other antiepileptic drugs. Patients responded well. Due to its antidepressant effect, it improves the mood of patients. Patients remain communicative and active. Carbamazepine is effective in all types of epileptic attacks. The drug is effective in repelling chronic ogres (m-n, trigeminal neuralgia).



not

Side effects: dyspepsia, drowsiness, headache, dizziness. accommodation disorder. When allergic reactions, leukopenia, thrombocytopenia develop, treatment with this drug is stopped.

Transport drivers cannot work while taking this medicine. It is not possible to accept alcohol.

TRIMETIN_ oxazolidine product. It is an especially effective drug in minor convulsions and mental equivalents of epilepsy. Orca brain reduces the instability of neurons by weakening polysynaptic reflexes. The drug reduces the amount of sodium ions in neurons, is quickly absorbed from MIT. But it is slowly removed from the body. The effect is 12-24 hours. Accumulation is possible. Dehydration effect is less than that of carbamazepine. Trimethine, like ethosuxemide, suppresses the function of T-type calcium channels and does not introduce calcium ions into neurons.

Side effects: Inability to look at light. hemorrhoids. allergic dermatitis. aplastic anemia, agranulocytosis, liver and kidney dysfunction. When treated with trimethine, large seizures of epilepsy can develop, so phenobarbital is given. Has a teratogenic effect. Application is limited for now..

ETHOSUXYMID is used in the same way as trimethin, but without it. Effective in small seizures of epilepsy. It easily passes through the placental barrier and is excreted in milk. Therefore, it cannot be used by pregnant and lactating

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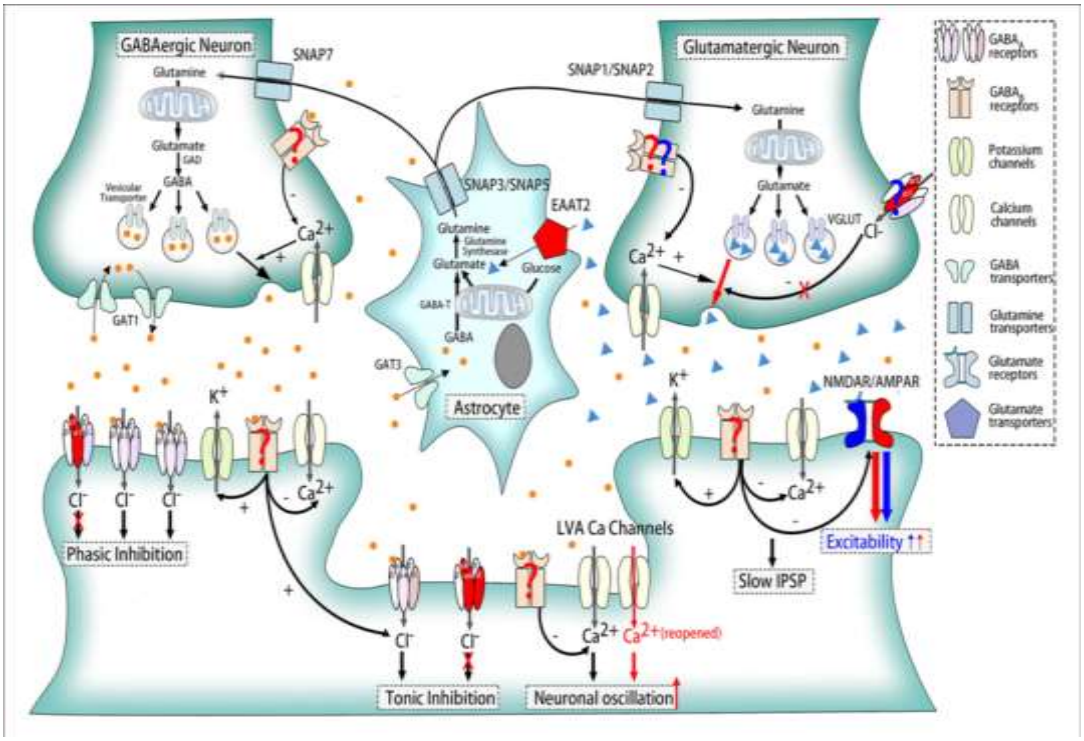
women. Develops symptoms of parkinsonism. Sometimes it causes unpleasant effects typical of trimethin, but they rarely develop.

SODIUM VOLPROATE_: A powerful anticonvulsant. Great and small, as well as myoclonus, gives good results in epileptic attacks. Mechanism of action: reduces the activity of GAMK-transaminase, as it activates glutamate decarboxylase and enhances RLMK synthesis. as a result, GAMK increases in the brain. This, in turn, reduces the excitability of the movement zones of the brain and reduces their readiness for tapas. Well absorbed from MIT.



The effect is 8 hours. Excreted in the urine-Tutkanokka is used in combination with other drugs, but can increase their unpleasant effects. Under the influence of sodium volproate, the mental state and mood of patients improves. an anxiolytic effect develops. But it does not relax the skeletal muscles, does not have a sedative effect. It has an anti-inflammatory effect.

Unpleasant effect. dyspepsia, allergy, loss of appetite (absorpsia) or loss of appetite, drowsiness, hair loss with prolonged use. disrupts liver, pancreas functions. Reduces the rate of mineral coagulation. It is forbidden to drink alcohol, it is forbidden to use during pregnancy and breastfeeding, because it has a teratogenic effect.

Lamotrigine is a powerful drug in the treatment of epilepsy.

Mechanism of action: Blocks channels of Na ions. It reduces the release of excitatory amino acids in the presynaptic membrane. Fully absorbed from MIT. The maximum concentration in the blood occurs after 2-2.5 hours. The effect is continuous, the effect lasts 24-30 hours. 55% of the absorbed drug is bound to mine oxides. About 65% is broken down in the liver and excreted in the urine in the form

of glucuronide. Therefore, when giving the drug, it is necessary to know the state of kidney and liver functions well.

Application: minor and major seizures, psychomotor epilepsy.

Adverse effects: dizziness, ataxia, diplopia, dyspepsia, allergy.

CONCLUSION: Anticonvulsant drugs are widely used in various fields of clinical medicine (neurology, therapy, psychiatry, pediatrics, etc.), therefore anticonvulsant and parkinsonism drugs are necessary for the body, in research and in the work of a general practitioner.

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