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BASIC SEDATIVES

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Abstract. This article discusses sedatives that have a general calming effect on the central nervous system, reducing the severity of reactions to external stimuli and daytime activity. Medicines in this group enhance inhibition processes and weaken excitation processes in the cerebral cortex, potentiate the effects of hypnotics, analgesics and other neurotropic sedatives, facilitate the onset of natural sleep without causing muscle relaxation, ataxia, mental and physical dependence.

Keywords: analgesic, hypnotic, sedatives, anxiolytics.

The name of sedatives comes from the Latin word *sedatio* - “calm”.

Despite the variety of modern anxiolytics, doctors continue to prescribe sedatives for the treatment of anxiety in outpatient practice due to their good tolerability and the absence of significant side effects.

Normally, higher nervous activity occurs under conditions of equilibrium between the processes of excitation and inhibition. The performance of the cerebral cortex depends on the strength and mobility of these processes. Exceeding the natural limit of strength, balance and mobility leads to anxiety. As a rule, anxiety appears against the background of a weakening of the inhibition process or an intensification of the excitation process.

Sodium and potassium bromides have been known to medicine since 1826. The active component is bromine anion. Non-dissociating organic bromine compounds do not have sedative properties.

Bromine salts are hygroscopic powders; when taken orally, they have a strong irritating effect on the intestinal mucosa, so they are used in the form of solutions or mixtures with starch mucus.

The mechanism of the sedative effect of bromides was established by Ivan Petrovich Pavlov and his students using the method of conditioned reflexes. In the doctoral dissertation of employee I.P. Pavlova, I.P. Nikiforovsky (1910), it was shown that bromides accelerate the extinction of conditioned reflexes based on the process of excitation, facilitate the development of inhibitory conditioned reflexes, and improve the differentiation of excitatory and inhibitory stimuli. Thus, bromides enhance inhibition in the cerebral cortex.

Further research by I.P. Pavlov to determine the mechanism of the sedative effect of bromides were carried out on dogs affected by the flood in Leningrad in 1924. In animals rescued from water-filled premises, firmly developed conditioned reflexes were impaired, the differentiation of inhibitory and excitatory stimuli disappeared (inhibitory stimuli began to cause excitation) . Treatment of dogs with bromides quickly restored the original conditioned reflex activity. The optimal therapeutic effect was observed when bromides were used only in individual doses. With a strong type of higher nervous activity, large doses were required, with a weak type, small doses.

Probably, bromine anions displace chlorine anions from the intercellular fluid in the brain. When the blood is saturated with bromides at approximately 60% of the total chloride content, deep depression of the respiratory center occurs.

Bromides in large doses, enhancing inhibition in the motor areas of the cerebral cortex, have an anticonvulsant effect in epilepsy. In toxic doses, bromine salts cause sleep and coma.

Soluble bromides are well absorbed from the intestine, create high concentrations in the blood and intercellular fluid, and penetrate cells poorly. The concentration of bromine anion in the brain is 3-4 times less than in the blood.

Bromides accumulate significantly. With a single administration of sodium bromide to dogs in an average dose, the concentration of bromine in the blood remained increased by 2.5-3 times after 12 days and decreased only after 20 days. Bromides are excreted by the kidneys. Reabsorption of bromine and chlorine anions in the renal tubules occurs according to a competitive principle. A small part of the dose of bromides is released from the body by the lacrimal, sweat, bronchial, and salivary glands, and due to the irritating effect, the secretory function of the glands is enhanced.

In medical practice, preparations of valerian, motherwort, skullcap and passionflower are used.

Valerian officinalis (*Valeriana officinalis*, named after the locality of Valeria in Pannonia on the territory of modern Hungary) contains 0.5-2% essential oil (borneol ester of isovaleric acid) in its roots and rhizomes. The plant also contains alkaloids, saponins, valeroside glycosides, and valeric acid.

The effect of valerian preparations on higher nervous activity has been known since ancient times. The Greek physician Dioscorides (1st century AD) in his essay "On Medicines" described valerian as a remedy that can "control thoughts." In the Middle Ages, valerian was considered a medicine that brought complacency, harmony and tranquility.

Valerian preparations have a local, reflex and resorptive effect. The reflexes are caused by the taste and smell of valerian, which stimulate the endings of sensory nerves in the oral and nasal cavities.

Valerian preparations weaken convulsions caused in experimental animals by strychnine and brucine. They have a neuroprotective and nootropic effect, facilitate falling asleep, prolong sleep, improve its quality, potentiate the effect of hypnotics, antipsychotics and anxiolytics.

Valerian preparations enhance the processes of inhibition and excitation in the cerebral cortex; their effect can be compared with the effect of a combination of bromides and the psychostimulant caffeine. In large doses, valerian

preparations inhibit the reticular formation of the midbrain. The effect of valerian preparations on the central nervous system is due to an increase in the functions of inhibitory neurotransmitters. Biologically active substances of valerian are agonists of A1 receptors of adenosine and benzodiazepine receptors, stimulate the release and suppress the neuronal uptake of GABA.

Passionflower, or meat-red passionflower (*Passiflora incarnata*), in the form of a liquid extract and tincture prepared from the herb of this plant, has a sedative effect in cases of anxiety and insomnia.

References

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