NICOTINTHERAPY

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The purpose of the scientific work: currently increasing memory(amnesia), Alzheimer's, dementia are aimed at preventing diseases. It is recommended for use only in chronic non-smoking patients. Chronic smoking leads to memory impairment.

Dementia is a term that describes several diseases that negatively affect memory, thinking and the ability to perform daily activities.Dementia develops over time. This mainly affects older people, but not all people develop dementia with age. Factors that increase the risk of developing dementia include: chronic smoking, depression, increased age (65), hypertension, increased sugar levels. Continuing to develop leads to Alzheimer's.

Alzheimer's is a progressive disease that is accompanied by neuronal degeneration. It is recommended as soon as the memory slows down. The neuron degenerates into protein (APP) accumulates in the cerebral cortex. The cortex is atrophied. Outside the senile Body, A B-amyloid accumulates.Hyperphosphiration is observed in the Axon, followed by an axonal transport disorder. The ego of the brain expands. Cognitive, motor, sensory disturbances occur.

Conducting nicotine therapy is the Prevention of neuronal degeneration. 1hafta is aimed at limiting nicotine 2 times. Should be given at 0.6 or 08 mg. Importantly, the patient should not be a chronic smoker. Because in order to avoid getting used to it again, this re-learning, on the other hand, can make the neuron die worse. Nicotine norm in human blood: walks 0-18 mg/mL in non-smokers,18-200ng/ml in passive smokers, 200 mg higher in active smokers. 50-100mg kills a person. From 1 cigarette, 1.1 to 1.8 mg of nicotine is released. Active smoking people are constantly activated sympathetic nerve. This in turn leads to a narrowing of the blood vessels, an acceleration of heart war. The first person who smokes a cigarette activates the parasympathetic nerve, and then the sympathetic nerve is permanently activated if the uham becomes chronic.In terms of its effect on neurons-increases the activity of the brain and impulse-conducting neurons. Leads to an increase in acetylcholine, an increase in acetylcholine in turn delays neuronal degeneration informational memory increases, attention is increased. Helps a person retain a lot of memory.Leads to an increase in hormones (adrenaline, dopamine) human activity increases. Accelerates nuclear formation in the impulse tract in synapses-enhances cognitive, motor, sensory transition. Nicotine can be used as an antidepressant, an anxiolytic. Helps a person to get out of deppression. It can be used to help with memory retardation. A certain amount of time increases memory gain, fixation,increased attention. It can also modulate the intestinal microbiota by regulating stress-related enzymes that

oxidize intestinal tissues, altering intestinal mucosa, expressing intestinal solid compound proteins, and altering the local acid/base balance in the colon through the direct toxic effects of countless compounds in tobacco. The psychogenic effect of chronic smoking is associated with an increase in regular smoking dopamine. An increase in dopamine, on the other hand, leads to a rapid excitation of emocyanal neurons located in the middle brain. This causes a behavioral disorder in the person. In a person, affective (in a state that is not in itself) leads to an increase in states. A constant increase in activityinj, on the other hand, leads to the subsequent occurrence of unexplained lesions. Chronic smokers are under constant stress, causing them to become irritable to little. And then to memory impairment. Inability to control activity and emotion psychatric disease can lead to the development of neuroses. The neurological effect of chronic smoking is as I mentioned constant stress, as well.In humanneyrons, it leads to the fact that the progroidmoddanitigrolysis occurs. The Tigroid substance is made up of a granular endiplasmatic mesh. It carries out protein synthesis in itself. The nerve tissue feeds on aminicislotes. The destruction of Det by tigrolysis leads to the fact that the nerve tissue is not nourished, that is, atrophy. Constant nicotine consumption due to exposure to synapses leads to changes in synapses. According to studies, presynaptic proteins in the rat affect presyntics by reducing their expressiveness (synaptophysin, synapsin-1), but do not affect the postsynaptic (PDS95) protein, while 205,235, and 404 epitopes in the hippocampus also show decreased acetylangan-tubulindarage and increased phosphorylated-tauni. (APP)amyloid precursor protein proliferation, B-amyloid CA3, has been seen to increase in dentate gyrus zones. Large amounts of nicotine cause the brain to change its structure. Regular increase in acetylcholine can lead to internal necrosis of the tissue. When the patient loses motor movement, sudden fasciculation is signaled by the development of internal necrosis. The sign darkens the surface of the skin, movement restriction.An increase in the impulse space between the neuromuscular causes internal necrosis. Neurons themselves produce acetylcholine via Acetylcholinethtransferase by adding choline with acetylcholinism. It can also be caused by heavy ingestion of latratoxin, pesticides, sussinilxolins.

In order for memory not to weaken, it is necessary first of all to start correcting sleep patterns.Because not getting enough sleep leads to brain aging.Reduce sugar levels, avoid chronic smoking, improve lifestyle, give up thinking, throw away harmful habits. Instead of them, it is necessary to put the reading system for one time, that is, for a comfortable time, sleep in time, engage in games that strengthen memory.

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