

Trigeminal Nerve at the Level of Exit Cranial Openings in the Pathogenesis of Classical Trigeminal Neuralgia

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Relevance. It leads to mental and physical exhaustion of a person, deprives him of normal work and personal life, often disabling him. Two anatomical structures are involved in the neurovascular conflict (NVK) in classical HTN: the trigeminal nerve root (CTN) and the artery (more often the superior cerebellar artery (BMA), less often the inferior anterior cerebellar artery (NPMA) and the basilar artery (BA). The occurrence of NVK occurs with a critical convergence of these structures and an increase in the traumatic effect of the artery on the CTN. The force of the artery hitting the CTN is subject to the physical laws of hydrodynamics. In turn, endothelial dysfunction contributes to atherosclerotic vascular modification with increased rigidity of the vascular wall. The existing methods of treating HTN reflect the evolution of ideas about its etiology and pathogenesis. The lack of a unified understanding of the pathogenesis of NTN underlies the continued widespread use of destructive surgical interventions and insufficiently effective drug 4 treatment methods. Among the conservative methods of treatment of classical HTN, first-line drugs are anticonvulsants and, above all, carbamazepine, which suppresses cortical and stem foci of sensitization. In parallel with the increase in tolerance to the drug, the number of adverse events also increases (Garcia J.B. et al., 2010; Techasatian L. et al., 2015; Nasir S.A., et al. 2017). If drug therapy is ineffective, surgical treatment methods are used. Microvascular decompression (MVD) of the trigeminal nerve root is considered the "gold standard" among them (da Silva O.T. et al., 2016; Arnone G.D. et al., 2017; Otani N. et al., 2017). The Ministry of Internal Affairs ensures the complete elimination of pain syndrome, however, the percentage of relapses of the disease varies widely. According to F. Raymond, J.R. Sekula, M.M. Edward et al. (2011), the recurrence rate after MIA was 28%. Mortality ranges from 0.5 to 2% and averages 1.4% (Nurmikko T.J., Eldridge P.R., 2001; Tronnier V.M., Rasche D. et al., 2002; Sandell T., Eide P.K., 2010, etc.). The main cause of death is a violation of blood circulation in the brain stem (Spatz A.L. et al., 2007). In addition, damage to adjacent nerve structures occurs with varying frequency: IV, VI, VII and VIII nerves. In this regard, it is urgent to develop new effective methods of pharmacotherapy of classical HTN, allowing to relieve pain syndrome, prevent the development of the above-described complications and increase the duration of remission (Donahue J.H. et al., 2017). The degree of elaboration of the research topic In the literature available to us, we have not found works devoted to the study of the state of elastic properties of the arterial wall in patients with classical. The analysis did not reveal any articles or dissertations on the problem raised in our scientific work. To date, no studies have been conducted reflecting the condition of blood vessels in patients with classical HTN and the involvement of endothelial dysfunction in the pathogenesis of this disease. This implies insufficient knowledge of this topic, despite the pathogenetic significance of the vascular component in the formation of neurovascular conflict.

In patients receiving medication, the pulse wave before treatment in the first subgroup type A averaged 46.9%, type B 24.2%, type C 28.9%, in the second subgroup type A averaged 45.3%, type B 31.1%, type C 23.6%, in the third subgroup type A B The average was 53.1%, type B 23.9%, type C 22.9%. In the group of surgical patients in the pulse wave before treatment, the Av type averaged 57.8%, type B 17.6%, type C 24.3%. Deviations in the parameters of pulse wave types were observed both in the group of patients suffering from isolated classical HTN, and in the group with classical HTN and concomitant pathology, and differences in the average values in these groups before treatment turned out to be statistically insignificant ($p > 0.05$). After drug treatment, the frequency of pain attacks decreased, and differences in the frequency distribution before and after conservative therapy turned out to be statistically significant with a confidence level of $p < 0.05$). Angioscanning differences in the

average values of vascular stiffness before and two months after treatment using scheme 3, which included taking L-arginine, in the direction of improving its indicator turned out to be statistically significant ($p=0.001474$). At the same time, a statistically significant improvement in stiffness using scheme 3 was observed both in the group of patients suffering from isolated classical HTN ($p=0.007661$) and in the group of patients with HTN and concomitant pathology ($p=0.027709$). In 18 indicators of vascular stiffness two months after therapy, according to schemes 1 and 2, statistically insignificant changes in average values were revealed ($p=0.850619$ and $p=0.818295$, respectively), since these methods of drug treatment do not affect the elastic properties of the arteries. Positive dynamics in the indicators of pulse wave types was also observed in the third subgroup of patients who received complex treatment, including the administration of L-arginine. Patients showed a tendency to decrease the value of the pulse curve type A, increase the value of the curve type B and curve type C, which, in turn, indicates an improvement in the elasticity of arterial vessels. Using drug therapy according to scheme 3, two months after the treatment, a statistically significant ($p=0.003305$) change in the structure of the pulse wave was observed: the content of type A decreased from 53.1% to 33.7%, and the presence of type C increased from 22.9% to 44.6%. Statistically significant differences in the average composition of the pulse wave before and two months after treatment according to scheme 3 were noted both in the group of patients suffering from isolated classical HTN ($p=0.003872$) and in the group of patients with HTN and concomitant diseases ($p=0.00009$). After treatment, type A in the pulse wave averaged 46.3%, type B 25.6%, type C 26.9%. The difference in the average composition of the pulse wave in this group of patients before and two months after treatment is statistically insignificant ($p=0.95090$). In the second subgroup of patients (who did not receive L-arginine) in the pulse wave after treatment, type A averaged 38.5%, type B 29.2%, type C 32.3%. The difference in the average composition of the pulse wave before treatment and two months after therapy in this subgroup turned out to be statistically insignificant ($p=0.37331$). The absence of a positive trend in the pulse wave graph using schemes 1 and 2 also indicates the absence of influence of these treatment methods on the elastic properties of the arteries.

In patients of the surgical group, differences in angioscan results before and two months after the MIA turned out to be statistically insignificant. The average vascular stiffness in surgical patients before and after MVD was $7.66\% \pm 3.74\%$ and $9.06\% \pm 3.80\%$ ($p=0.343253$). In the surgical group of patients after MVD, a change in the structure of the pulse wave was observed: the content of type A decreased from 57.8% to 46.7%, the content of type B increased from 17.6% to 25.6%, and the presence of type C increased from 24.3% to 27.8%. However, these indicators turned out to be statistically insignificant ($p=0.23531$). The indicators of the average composition of the pulse wave before and after the MIA both in the group of patients suffering from isolated classical HTN ($p=0.26034$) and in the group of patients with HTN and concomitant pathology, including those accompanied by vascular damage, turned out to be statistically insignificant ($p=0.20219$). Pharmacotherapy according to scheme 3 turned out to be the most effective. Thus, the level of significance of differences in clinical parameters after treatment between a subgroup of patients who received drug therapy according to scheme 3 and a subgroup of patients who received treatment according to scheme 1 (control) was: according to the frequency of pain attacks $p=0.000007$, according to VAS $p=0.000001$, BNI scale $p=0.000002$, night sleep assessment questionnaire $p=0.000413$, Hamilton scale $p=0.006653$. Comparing the results of treatment according to scheme 3 with the results of treatment according to scheme 2, statistically significant differences were observed in such indicators as: YOUR ($p=0.036135$), the night sleep assessment questionnaire ($p=0.019746$), the Hamilton scale ($p=0.031163$). The results of drug treatment according to the scheme of 3 patients with complete remission of pain syndrome were also compared with a group of 20 operated patients according to the same indicators. Pain syndrome was relieved in both groups by the time of discharge. In addition, night sleep scores and Hamilton Depression Scale scores improved. With the use of scheme 3, an increase in the effectiveness of treatment of classical HTN was achieved by increasing the duration of remission (recurrence during the first year after treatment was not observed in 92.3% of patients). **CONCLUSIONS** 1. In the formation of neurovascular conflict and the occurrence of classical NT, it is not the size of the exit cranial openings that is significant, but a decrease or loss of elasticity of the artery wall involved in the

conflict, which is confirmed by an increase in the level of endothelin-1 (on average $0.95 \pm 0.19 \text{ fmol/ml}$), total cholesterol (on average $6.21 \pm 0.16 \text{ mmol/l}$) and the results angioscanning (increased arterial stiffness (on average $5.44\% \pm 1.09\%$), predominance of pulse wave type A (on average $59.75\% \pm 4.53\%$)) in patients with classical hypertension. 21 2. In patients with classical HTN, before treatment, there were no deviations in homocysteine (on average $9.29 \pm 0.38 \text{ mmol/l}$) and cytokines (IL-1 β (on average $3.27 \pm 0.11 \text{ pg/ml}$), IL-8 (on average $13.91 \pm 0.47 \text{ pg/ml}$), TNF- α (on average $5.30 \pm 0.18 \text{ pg/ml}$)). After treatment, they were statistically insignificant ($p > 0.05$). Two schemes of pathogenetic therapy of classical HTN have been developed: using L-lysine escinate (scheme 2) and additional use of L-arginine. The use of scheme 2 made it possible to reduce the duration of inpatient treatment by an average of 7 days and increase the duration of achieved remission to an average of 9.8 months. The best results were obtained using scheme 3, which allowed to increase the duration of the achieved remission to an average of 11 months. 4. A decrease in the severity of endothelial dysfunction and an improvement in the elastic properties of the arthritic wall were achieved in patients with classical HTN treated according to scheme 3, which was confirmed by a decrease in endothelin-1 ($p = 0.000132$), total cholesterol ($p = 0.000060$) and angioscanning results (decrease in arterial stiffness ($p = 0.001474$), decrease in prevalence).

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