Introduction:
Depending on the nature of the brain injury and the severity of the victims, mortality in traumatic brain injury (TBI) ranges from 5 to 65% [1]. One of the targets for pathogenetic influence on the course of TBI is the use of pharmacological agents that are able to counteract the negative effects of excess concentrations of glucocorticoids on brain.

Methods:
The therapeutic effect of new pharmacological derivative 1-adamantylethoxy-3-morpholino-2-propanol hydrochloride (ademol) in rats with TBI was evaluated for 8 days. The pseudoperated animals and control group received 0.9% NaCl solution and the comparison group received amantadine sulfate. Cortisol levels were used to determine the efficacy of the test drugs in TBI.

Results:
In rats treated with ademol, the level of cortisol in the blood ranged from 179 to 188 ng/ml (P5-P95) and was 2.58-fold lower (p<0.05) compared to control pathology group on the 8 day of therapy. Instead, the effect of amantadine sulfate on the level of cortisol in the blood was significantly less than that of ademol. The concentration of cortisol in rats with amantadine sulfate in the blood ranged from 271-280 ng/ml (P5-P95), was 1.73 times lower (p<0.05), compared with the control pathology group, and by 49.2% (p<0.05) exceeded the corresponding value in animals treated with ademol.

Conclusion:
Therapeutic treatment of rats with severe TBI with a solution of ademol, preferably better than rats in the group with 0.9% NaCl and amantadine sulfate protect the brain from the formation of steroid neurotoxicity by cortisol (p<0.05).

References: