pared with the control. The changes in LPO intensity were the most pronounced 7 and 14 days after CCI, and therefore the antioxidant activity of carnosine was studied at these times. On day 7 and 14 after CCI erythrocyte resistance to hydrogen peroxide was decreased 120% and the content of TBA-active products was increased 34%. Substantial (55%) increase in the brain content of endogenous peroxides and Schiff bases was recorded 14 days after CCI. A substantial increase (3-fold above the control) in the content of TBA-active products was recorded 7 days after CCI. Carnosine administered to rats at a daily dose of 20 mg/kg (intraperitoneally) throughout the entire post-traumatic period had noticeable and diverse effects on LPO. On day 7 the effect of carnosine on free-radical oxidation was the same as that observed after CCI, whereas on day 14 LPO was markedly inactivated. Specifically, peroxide resistance to erythrocytes increased by 60% compared with that recorded in CCI, while the level of TBA-active products and the intensity of blood SCL were close to the control value. The brain contents of endogenous peroxides, Schiff bases, and TBA-active products were markedly reduced, exceeding the control values only by 15-20%. Thus, when administered after CCI, carnosine partially, and in some cases completely, normalized LPO parameters, which allows us to regard the compound (providing that broader studies are carried out) as a potential candidate for use in neurosurgery to stimulate the antioxidant resistance of the organism during the post-trauma period.

Relationship Between the Kinetics of Lipid Peroxidation and Autoimmune Reactions after Craniocerebral Injury

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Activation of lipid peroxidation (LPO) is known to be accompanied by changes in the immune response. For example, it was found that the adaptive LPO reduction at high altitudes is attended by inhibition of the immune response. In contrast, after adrenalectomy and under the influence of immunostimulatory agents the intensity of LPO increases, which stimulates antibody production.

In our studies we attempted to establish a relationship between LPO intensity and the autoimmune response after craniocerebral injury (CCI). We roceeded from the fact that a closed CCI is accompanied a pronounced LPO activation in the blood and, secondarily, in the brain of experimental animals. Destruction and necrosis of brain tissues induce a process of autoimmunization to the host brain antigens, the extent of this reaction characterizing the severity of CCI. If there is a relationship between the studied processes, the administration of ionol (120 mg/kg, three times a day) or bemegride (100 mg/kg/day for 30 days) to CCI rats (120 days after trauma), should, by normalizing LPO, produce an indirect effect on the production of autoantibodies.

Ionol partially lowered LPO intensity in intact rats. The LPO intensity was evaluated by spontaneous chemiluminescence and the blood and brain contents of TBA-active products. Bemegride and CCI (to a greater degree) activated the free-radical processes, which were markedly inhibited in the case of combined loads with antioxidant.

The autoimmune response was evaluated by the titer of antibodies against neurospecific enolase (NE), glial protein (GP), and brain antigen (BA). A nonuniform immunological reaction was noted when ionol or bemegride was injected into intact rats. The titer of anti-NE antibodies was lowered substantially, but there was no change in the response to BA. Ionol had no effect on the level of anti-GP antibodies, bemegride increased it by 21%. As a result of CCI, the titers of anti-NE and anti-BA antibodies increased by 21 and 38%, respectively, while that of anti-GP antibodies decreased. The changes in the antibody titers (particularly of anti-BA antibodies) were more pronounced in CCI rats. Ionol significantly lowered the antibody titers in CCI rats. This effect was observed after the combined administration of antioxidant and bemegride.

Taken together, our findings indicate that an increase or decrease in LPO intensity is accompanied by inhibition or stimulation of antibody production, i.e., it facilitates the autoimmune response to brain antigens. For the same reason we may surmise that directed antioxidant inhibition of LPO may be a reliable means of reducing the severity of autoimmune aggression.