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TREATMENT OF ACUTE MIDBRAIN SYNDROME AND THE APALLIC SYNDROME

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During the very first days after onset of a traumatic coma it is difficult or impossible to identify the future state of a patient. All possibilities of treatment (surgical and conservative) should be applied. However, there is little evidence that brain edema can be sucessfully treated with drugs. Artificial ventilation and perfect body position appear to be the most important measures in the treatment of the acute midbrain syndrome. The development of an apallic syndrome is seen after deep stages of traumatic midbrain syndrome and is characterized by the onset of overactivity of the sympathic nervous system, which is clinically easily recognized by a sudden onset of tachycardia. Without special treatment the sympathetic catabolic drive leads to marasm and severe peripheral and central nervous system lesions will appear. Even high caloric nutrition can not stop this development. The plasma level of norepinephrine is extremly high in these patients. Treatment with beta blocking agents and debrisoquine supresses the catabolic drive and allows a normocaloric nutrition. In cases with no or poor increase of human growth (HGH) after aginin stimulation HGH will be of additional value in the therapy of the disturbed metabolism in apallic patients.

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