

Gründig E, Gerstenbrand F, Maida E, Binder H, Hawrylewicz A.

Über die Anwendung von Guanidin-Hydrochlorid in der Behandlung von degenerativen Nerven- und Muskelerkrankungen. II. Aminosäureanalysen des Liquor cerebrospinalis und des Serums; Verlaufskontrollen

[The application of guanidine hydrochloride to the treatment of degenerative nervous and muscular diseases. II. Amino acid analysis of the CSF and serum: follow-up determinations (author's transl)]

Abstract

The amino acid composition was determined in the CSF and serum of 19 patients with amyotrophic lateral sclerosis or with other degenerative anterior cell lesions, of 6 patients with degenerative diseases of the spino-cerebellar system, as well as of 4 patients with dystrophic muscle diseases. The amino acid patterns, as well as the clinical status, were controlled continuously during therapy with guanidine hydrochloride. The following results were obtained: In 14 patients with amyotrophic lateral sclerosis the concentration of tau, ser, asn, glu, gln, gly, ala and met in the CSF was lower than in the controls, but not with respect to the serum. In the 6 patients with anterior horn cell lesions the CSF was continuously controlled and the clinical status correlated with the amino acid pattern. On successful therapy the CSF amino acid pattern returned to normal. When progression occurred the amino acid pattern relapsed again into an abnormal pattern. At a late state of the disease guanidine hydrochloride was ineffective. In 3 patients with Friedreich's disease the CSF values of tyr and phe were low, whereas try and orn were elevated. A similar tendency was found in serum, too. After guanidine hydrochloride therapy the clinical status, as well as the concentration of tyr, phe and try, returned to normal, but orn values increased continuously in CSF and serum. In 1 patient with the Roussy Levi syndrome and in 2 patients with olivo-ponto-cerebellar atrophy no changes were found in the CSF. In 3 patients with Erb's muscular dystrophy or with dystrophic myotonia, no clinical or biochemical (CSF) response to guanidine hydrochloride was seen. One might assume that the degenerative diseases of the nervous system are connected with disturbances in most transport systems. Therefore, changes in cellular metabolism and in the neurotransmitter systems must exist. The effect of guanidine hydrochloride is not specific; it causes a general increase in membrane permeability.

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Wien Klin Wochenschr. 1978 Jan 20;90(2):48-56.

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