CEREBROVASCULAR AUTOREGULATION AND REACTIVITY IN PRIMARY NEUROPATHIC ORTHOSTATIC HYPOTENSION.

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The primary neuropathic orthostatic hypotension provides an unique model of cerebrovascular autonomic denervation for the experimental study of the mechanisms which control the autoregulation of the CBF in man.

The published observations (Skinhøj et al., 1971; Gotoh et al., 1971; Caronna and Plum, 1972; Meyer et al., 1973) show various effects of that disease on the autoregulation of CBF. These differences may be imputed, in part to various degrees of severity of the autonomic nervous damage and, in part, to methodological discrepancies.

The studied patient is a 75 years old woman with a typical history of progressive autonomic failure and of other signs of degenerative neurological affection, evoking a Shy and Drager syndrome. The tests of autonomic nervous system function permit to localize the main lesion at the preganglionic efference level.

The rCBF of 50 regions of the left hemisphere are measured by the 2 min.-initial slope index of the regional 133-Xenon clearance curves after injection of 5 mCi of this substance into the internal carotid artery, and using a gamma-camera. Caution is taken to avoid any count rate saturation of the detector. The initial slope index is automatically computed, using the least-square method . The half 90% confidence interval of the monoexponential slope estimation is computed and expressed in the same units as the CBF. The arterial pCO2 and the heart level corrected mean arterial blood pressure (MABP) are measured in the internal carotid artery.

The mean rCBF is 48.2 ml/min.100gr at rest and in the horizontal position, with a MABP of 140 mmHg and a paCO2 of 38 mmHg;

its distribution is normal.

Hyperventilation lowers the paCO2 to 28 mmHg and the mean CBF

to 41.2 ml/min.100gr, without any alteration in MABP.

Tilting-up induces an immediate arterial hypotension in the patient; rCBF measurement are performed at different levels of MABP (see fig.), after 5 min. of steady state of the pressure; they reveal a partial loss of autoregulation, with a point of breaking between 120 and 100 mmHg of MABP.

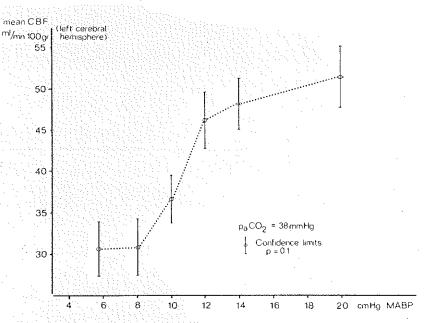
The IV infusion of phenylephrin raises the MABP up to 200 mm Hg, but does not significantly modify the CBF nor its distribution

in the studied cerebral hemisphere.

Active flexion and extension movements of the right wrist and forearm, in the horizontal position and in normocapnia, are accompanied by the appearance of a relative hyperemia at the left fron-

torolandic cerebral convexity.

The presyncope state at MABP = 57 mmHg is characterized by a significant hyperemia of the prefrontal region and by a paradoxal stabilisation of the CBF values (see fig.): the distribution pattern of rCBF shows a deep fall of perfusion in the posterior regions of the cerebral hemisphere (mean rCBF of the posterior half hemisphere = 23.1 ml/min.100gr), whereas the anterior regions are in a relative hyperemia, with regard to the value of CBF expected from the slope of the curve (see fig.) (mean rCBF of the anterior half hemisphere = 38.1 ml/min.100gr). In this very peculiar condition of presyncope and anxiety, a partial compensattion of the autoregulation loss appears in the frontal region.



The mean left hemispheric rCBF of a patient with a Shy and Drager syndrome, related to the MABP; ×, at rest in the horizontal position; ⋄, during IV infusion of phenylephrin; ⋄, at different levels of orthostatic arterial hypotension.

In conclusion, this investigation of the rCBF regulation in one case of primary neuropathic orthostatic hypotension reveals that the degeneration of the autonomic innervation of the vessels does not induce a complete loss of autoregulation of the CBF and does not alter the reactivity of the cerebral vessels to the paCO2 and to the level of the local cerebral metabolism. This fact agrees with the study of Skinhøj et al. (1971), using a similar method of rCBF measurement. It is presumable that the other authors observe a complete loss of autoregulation in analogous cases because they induce an abrupt orthostatic hypotension and measure the instantaneous alteration in CBF by means of the determination of the cerebral arteriovenous difference in pO2.

The study offers an unique example of competition between the ischemia due to the loss of autoregulation and the relative hyperemia in relation with a local raise of the cerebral activity.

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