up Ca ions is disturbed [9, 14], even that part of the Ca which participates in the formation of contraction under the original conditions is unable to be evacuated from the myoplasm down to its initial level after completion of the evoked contraction. This leads to the development of mechanical oscillations in accordance with mechanisms described for the myocardium of warm-blooded animals [6, 7].

LITERATURE CITED

1. K. Yu. Bogdanov, S. I. Zakharov, and L. V. Rozenshtraukh, Fiziol. Zh. SSSR, No. 6, 859 (1980).
2. E. G. Vornovitskii, A. A. Galfayan, A. N. Kaidash, et al., Byull. Éksp. Biol. Med., No. 7, 8 (1978).
3. E. G. Vornovitskii and E. Yu. Bychkova, Byull. Éksp. Biol. Med., No. 11, 7 (1980).
4. E. Bozler, Am. J. Physiol., 139, 477 (1943).
5. P. Braveny, J. Sumbera, and V. Kruta, Arch. Int. Physiol., 74, 169 (1966).
6. A. Fabiato and F. Fabiato, J. Physiol. (London), 249, 469 (1975).
7. A. Fabiato and F. Fabiato, Circulat. Res., 40, 119 (1977).
8. G. R. Ferrier, Circulat. Res., 41, 622 (1977).
9. S. Harigaya and A. Schwartz, Circulat. Res., 25, 781 (1969).
10. R. Jensen and B. Katzung, Nature, 217, 961 (1968).
11. R. Kaufman, A. Fleckenstein, and H. Antoni, Pflüg. Arch. Ges. Physiol., 278, 435 (1963).
12. J. Kedem, R. Jarom, J. Mahler, et al., Cardiovasc. Res., 6, 353 (1972).
13. D. Masher, Pflüg. Arch. Ges. Physiol., 323, 284 (1971).
14. Z. I. Penefsky et al., in: Myocardial Biology, Baltimore (1974), pp. 31-39.
15. M. Reiter, Arch. Pharmakol. Exp. Pathol., 242, 497 (1962).

INTRACEREBRAL PRESSURE AND EEG DURING MEASURED CHANGE OF THE CSF VOLUME

É. B. Sirovskii, E. I. Pal'tsev, and Ch. Yu. Kasumov

UDC 616.831-008.918+616.831-073.097]-02:616.832.9-008.811

KEY WORDS: changes in CSF volume; CSF pressure; intracerebral pressure; EEG; power spectrum of EEG.

As a rule the clinical state of neurosurgical patients and the effectiveness of their treatment are assessed on the basis of the results of a neurological examination, investigation of the state of the brain function by electroencephalography, and determination of the level of intracranial hypertension, assessed as changes in the CSF pressure (CSFP). Evidence has now been obtained that the CSFP is not identical with the pressure inside the brain tissue and does not always accurately reflect mechanical pressures developing in it [2, 3, 7, 8].

To study the biophysical characteristics of brain tissue methods based on a measured change in the CSF volume have been developed [4-6].

This paper describes the results of an investigation of the effect of a measured change in the CSF volume on the clinical state of the patient, the CSFP and the intracerebral interstitial fluid pressure (ICIFP), and on the EEG for the purpose of comparing the clinical and physiological data and of determining the advisability of an artificial change in the volume and pressure of the CSF in the acute postoperative period.

EXPERIMENTAL METHOD

Altogether 31 patients were studied after removal of supra- and subtentorial brain tumors (basal meningiomas in 14 patients, intracerebral tumors in 6, tumors in the region of the posterior cranial fossa in 11).

N. N. Burdenko Institute of Neurosurgery, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR V. S. Rusinov.)

Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 91, No. 6, pp. 661-663, June, 1981. Original article submitted February 12, 1981.

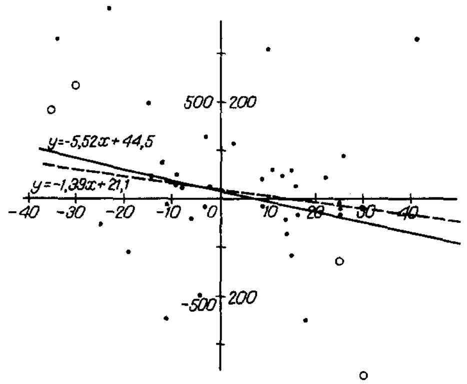

Fig. 1

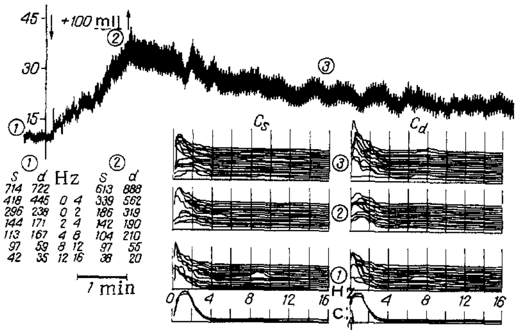

Fig. 2

Fig. 1. Changes in total power of EEG (MΣ) and power of frequencies composing EEG within the 8-17 Hz range (Mα) as a function of changes in CSF pressure and volume. Origin of coordinates corresponds to mean initial values of test parameters, negative values denote a decrease compared with initial levels. Continuous line – regression for dependence of MΣ on CSFP, broken line – regression for dependence of M on CSFP. Ordinate, pressure (in mm Hg); abscissa, values on left – MΣ, on right – Mα (in μV).

Fig. 2. Dynamics of EEG during measured change in ventricular CSFP in a patient after removal of a glial tumor of the left parieto-temporal region. Top trace shows changes in CSFP. Ordinate, pressure (in mm Hg). Bottom: right – graph of change in EEG spectrum before (1), during (2), and after (3) a measured increase in CSFP; left – quantitative assessment of total power (in μW^2) of EEG and its frequency ranges per minute (in μV^2) before (1) and during (2) elevation of CSFP; d – for right, s – for left cerebral hemisphere. 1, 2, 3) Marks corresponding to times of recording EEG. C) calibration.

The postoperative condition of the patients, assessed by means of the 15-point Glasgow coma scale [9], ranged from compensated (15 points) to comatose (6 points).

To monitor CSFP continuously, the lateral ventricle was catheterized and the catheter connected hydraulically to an Elema (Sweden) measuring system. The biophysical properties of the brain were studied by the method of making a measured change in the CSF volume [1, 4]. The CSF was first withdrawn from the ventricular system, after which the same volume (4-10 ml) of physiological saline was injected, at a constant rate. Meanwhile the curve of the change in ICIFP was recorded.

The local ICIFP was measured continuously by means of a specially made catheter with double microperforated wall, which was buried in the brain tissue to a depth of 1.5-2 cm at the end of the operation [2].

Parallel recordings of the EEG from 10 surface electrodes, arranged in accordance with the 10-20% scheme, were made on a 12-channel polyphysiograph (from OTE Biomedica, Italy). A continuous graph of the change in the EEG spectrum with time and a quantitative estimate of changes in its component frequencies, and also numerical values of the total power of the EEG and of its separate frequency ranges per minute were obtained from two EEG derivations (C_s-C_d) by means of a Berg–Fourier analyzer (from OTE Biomedica).

EXPERIMENTAL RESULTS

Assessment of the clinical state after 87 tests of measured change in the CSF volume showed that in 76 cases the patient did not react to the investigation. They showed no increase in either local or general cerebral neurological symptoms. In 11 cases at the height of CSF hyper- or, less frequently, hypotension the patients developed a feeling of discomfort, which they could not describe exactly. They complained of worsening of headache, dizziness, nausea, numbness in the limbs, and a feeling of heat. In some patients a change of 8-10 ml in the CSF volume, with the associated fall of 25-40 mm Hz in the CSFP took place without any subjective sensations, whereas in others, a change of 2-4 ml in CSF volume and a fall of 10-20 mm Hg in the CSFP gave rise to a state of discomfort.

It can accordingly be suggested that in cases when a drop of CSFP caused slight worsening of the clinical state it was not due to the hypo- or hypertension as such, but to inability of the CNS to adapt itself quickly to the fall of intracranial pressure.

Changes in ICIFP arising in response to a measured change in CSF volume were not directly dependent on the change in CSFP, but were ambiguous. With a fall in CSF volume, leading to a fall of 6-23 mm Hg in CSFP, in 55% of cases the local ICIFP rose by 0.6-3 mm Hg.

With an increase in CSF volume, raising the CSFP by 15-40 mm Hg, in 37% of the cases ICIFP was virtually unchanged, or it changed by not more than 0.5 mm Hg, in 34% of the cases ICIFP fell by 0.6-4.5 mm Hg, and in 29% it increased by 0.6-3 mm Hg.

During a measured change in CSFP of between 5 and 50 mm Hg, ICIFP may therefore remain unchanged. In cases when ICIFP did change, these changes were very small (under 10% of the changes in CSFP), and were not always in the same direction as the changes in CSFP. Hence it follows that the response of ICIFP is not a simple reflection of changes in CSFP but is brought about indirectly through a number of factors which require special study.

The reaction of the EEG to a measured change in CSF volume and pressure also was ambiguous. Changes in the total power of the EEG and the power of the α-rhythm in response to a rise and fall of CSFP, obtained in nine patients, are shown graphically in Fig. 1. As this figure shows, during a measured change in CSFP the total power of the EEG could either increase or decrease. In 60% of cases, however, the following trend was observed in the EEG changes: A fall in CSFP led to an increase in the power of the EEG whereas a rise in CSFP caused the power to fall. This relationship is represented on the graph by a linear regression line. Meanwhile the changes in the power of the EEG were very small, not more than 8% of its initial value. In 40% of the cases a fall in CSFP led to a decrease in power of the EEG, and an increase in CSFP led to a corresponding increase in power. Changes in the EEG in this direction were even less marked and did not exceed 3.5% of the initial power. The degree of the reactive changes of the EEG in response to an artificial change in CSFP was more marked in patients in whom the course of the postoperative period was complicated and, in particular in comatose patients.

An example of a very small change in the EEG spectrum in response to an increase in CSFP is shown in Fig. 2. It shows that the power spectra before, during and after an increase in CSF volume and CSFP do not differ significantly from one another and that maximal power is located in the low frequency range. Quantitative analysis of the power spectrum revealed asymmetry of the reaction of the EEG to an increase in CSFP. For instance, in the "sound" hemisphere the total power of the EEG decreased, whereas in the affected hemisphere it increased.

A measured change in CSFP thus gives rise to short-term changes in the EEG in different directions. These results suggest that an artificial change in CSFP is treated by the CNS as a nonspecific extremal stimulus, the response to which usually depends on the initial state of the system.

LITERATURE CITED

1. E. I. Paltsev and É. B. Sirovskii, in: Biomechanics of the Circulation, Respiration, and Biological Tissues [in Russian], Riga (1981), pp. 25-27.
2. É. B. Sirovskii and E. I. Paltsev, Vopr. Neirokhir., No. 5, 8 (1976).
3. É. B. Sirovskii, in: Abstracts of Proceedings of the Second All-Union Congress of Neurosurgeons [in Russian], Moscow (1976), pp. 157-159.
4. E. I. Pal'tsev (Paltsev) and É. B. Sirovskii (Sirovsky), in: Intracranial Pressure IV, New York (1980), pp. 250-255.
5. A. Marmarou, K. Shulman and J. La Morgese, in: Intracranial Pressure II, Berlin (1975), p. 86.
6. D. Miller, J. Caribi and J. Picard, Arch. Neurol. (Chicago), 28, 265 (1973).
7. K. Shulman, A. Marmarou, and S. Weitz, in: Intracranial Pressure II (Berlin), 1975, p. 221.
8. A. Schettini, L. McKay, J. Mahid, et al., Anesthesiology, 36, 4 (1972).
9. G. Teasdale, Nursing Times, 71, 914 (1975).