ELECTROENCEPHALOGRAPHIC AND RHEOENCEPHALOGRAPHIC CHANGES IN SUBARACHNOID HEMORRHAGES

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Spontaneous, non-traumatic subarachnoid hemorrhages have been observed as a complication in a variety of brain diseases. Magee (17) found the presence of a brain aneurysm, assumed as a causative agent of subarachnoid hemorrhage, in 43 out of a total of 58 cases with lethal outcome. The difficulty to explain the pathogenesis and to determine the site of bleeding is emphasized in the works of Bogolepov (1), Konovalov (7), Lerman (8), Bodechtel (11), Binnie and Margerison (12, 13), Symon and Bes (18) and Symonds (19), who failed to discover invariably reliable clinical and paraclinical signs in the patients studied by them. Angiographic studies show aneurysmal dilatations in 5—30 per cent of the patients with subarachnoid hemorrhages — Basset and co-authors (1953), Olivecrona (1953) (cited by 12), whereas post-mortem studies — in 21 to 33 per cent — Riggs, Rupp (1943), King, Sade and Campony (1954) (cited by 12, 13). Accumulation of a more extensive information on the issue is badly needed to determine the localization and cause of subarachnoid hemorrhages and accordingly, to broaden the scope of operative management in this particular type of cerebral vascular diseases.

Woodhall, Lowenbach — 1945 (cited by 12), and thereafter many authors as Borodula (2), Egorova (3), Zhirmunskaya (4), Zlotnik (5), Konovalov (6, 7), Lerman (8), Hubenov (9), Yarrulin (10), Clifford (14) and Hermann, Pia (15) have drawn attention to the possibility to lateralize the ruptured aneurysm by means of EEG and REG methods. In the acute stage of subarachnoid hemorrhage this is not always realizable because of the developing hematoma, and vascular spasm produced by the ruptured vessel.

It is the purpose of the present research to try to discover some etiopathogenetic factors, and to supplement with EEG and REG criteria the clinical and angiographic study for topography of bleeding and degree of involvement of the brain parenchyma determination in spontaneous, non-traumatic hemorrhages.

A series of forty patients with subarachnoid hemorrhages, proved clinically and paraclinically, were studied. The angiographic examinations showed aneurysmal dilatation of the arterial vessel in 14 cases, while in 12 the aneurysm was established pathoanatomically.

The clinical signs in all patients are characterized by anamnestic data such as frequent headache of at least one year duration, loss of consciousness for periods ranging from several minutes to 1—2 days, vomiting, meningoradicular stimulation syndrome, rarely generalized convulsions, speech derangements and discrete pyramidal signs. In the acute stage of the di-

sease, the lumbar liquor of the patients under study was sanguineous. Since the clinical examination in 26 patients failed to provide sufficient evidence as to the side of the ruptured vessel owing to a prevailing symptomatics of elevated intracranial pressure, EEG and REG studies, as well as carotid angiography were performed.

Aneurysmal dilatations were discovered in five patients only when the angiographic method was used. Atherosclerotic changes of the brain vessels, associated with arterial hypertension, were considered as an etiological factor in the remaining 14 patients with subarachnoid hemorrhages, chronic alcohol intoxication — in three cases, and probable congenital defect of the vascular wall — in four.

The EEG study conducted in all patients within three days after the onset of the vascular accident, disclosed the following results: against the background of irregular, low to middle voltage alpha rhythm, diffuse slow-wave alterations of theta and middle-voltage single waves were observed, having frontal or fronto-temporal localization in 14 patients, parieto-occipito-temporal localization — in 4, and lateralization of the pathological changes with focus formation — in five. The remainder (17 patients) displayed pathological changes in the EEG, consisting in excitation manifestations through acute waves, peaks, tapered theta waves with synchronization in short-term orders. Similar changes were detected in patients sustaining hemorrhages with frontobasal or para-truncus localization, confirmed clinically and pathoanatomically. The stimulation manifestations outlined were intensified in the course of functional tests. Recordings, repeated within ten days, showed with greater precision the localization of the pathological focus.

In the course of REG study, gross bilateral changes were established in the architectonics of the pulse tracing. In twenty cases, the peaks of the latter were slightly tapered or denser, while the polydicrotic waves disclosed proximal positioning, and in isolated cases, the dicrotic tooth exceeded the height of the systolic peak. In three patients additional waves were discovered in the isoline area (venous waves). Against the background thus outlined, both in the global and regional lead, a moderately manifested REG asymmetry persisted. On the side of the lowered amplitude, the morphological changes of the pulse curve were more clearcut. In six patients the peaks of the REG tracing were rounded, while the polydicrotic waves were with flattened out tops. The pulse curve amplitude was slightly lowered bilaterally, and on this background a moderately pronounced REG asymmetry was visually recorded.

The REG data demonstrate a total increase of the vascular tone at an obvious predomination of the latter in some of the regional leads. Many authors (Borodulya (2), Konovalov (7), Lerman (8), Hubenov (9), Yarullin (10), Herman and Pia (15), Kreindler, Hornet and Nissimi (16), attribute the decrease of the blood-brain index in restricted subarachnoid hemorrhages to the presence of a reflectory brain vessels' spasm, caused by blood extravasation into the vascular reflexogenic zones. The permanent spasm of cerebral arteries in rupture of brain anurysms, according to data submitted by Konovalov, 1965 (7), is due not as much to mechanical compression of

the arteries by the cerebral extravasate, as to their active contraction. Continuous spasm of the major and minor cerebral vessels has been also described by Zlotnik (5), Lerman (8), Hermann and Pia (15) and others.

Researches into spontaneous non-traumatic subarachnoid hemorrhages focus the attention chiefly to clinical symptoms, to changes in the cerebrospinal fluid and to intracranial aneurysms, considered as the most frequent cause for their appearance — Bodechtel (11), Magee (17), Symonds (19). The failure to find evidence of a similar etiological factor in all the cases, and more particularly in younger patients, makes mandatory the search for additional causing agents, such as alcohol intoxication, inflammatory diseases of the brain, and congenital insufficiency of the vascular wall. Electroencephalographic and rhecencephalographic studies, in conjunction with the angiographic finding, could give us valuable information as to the side of ruptured aneurysm, degree of lesion of the brain parenchyma, vascular hemodynamic disturbances, and rather rarely, about the topography and extent of vascular malformation.

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ЭЛЕКТРОЭНЦЕФАЛОГРАФИЧЕСКИЕ И РЕОЭНЦЕФАЛОГРАФИЧЕСКИЕ ИЗМЕНЕНИЯ ПРИ СУБАРАХНОИДАЛЬНЫХ КРОВОИЗЛИЯНИЯХ

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РЕЗЮМЕ

Авторы исследовали 40 больных со спонтанными, нетравматическими субарахноидальными кровоизлияниями. Проведенные ангиографические исследования позволили установить у 14 из них аневризматическое расширение. Отсутствие во всех случаях доказательств о наличии подобного этиологического момента требует поиска иных причинных факторов, как хроническая алкогольная интоксикация, воспалительные заболевания головного мозга, врожденная малоценность сосудистой стенки и др. Электроэнцефалографические и реоэнцефалографические исследования, прибавленные к ангиографическим находкам могут сориентировать нас в отношении стороны с которой находится разорвавшийся сосуд, степени поражения паренхимы мозга, сосудистых гемодинамических нарушений и реже — топики и величины порока развития сосуда.