NEUROPAT. POL., 1982, 20, 3—4
PL ISSN 0028-3894

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UNCOUPLING BETWEEN RECOVERY OF CORTICAL BIOELECTRIC ACTIVITY AND PROGRESSION OF MORPHOLOGICAL ALTERATIONS AFTER CEREBRAL ISCHEMIA IN GERBILS

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The aim of the study was to evaluate recovery of the cortical bioelectric activity (ECoG) after 30 min bilateral occlusion of the common carotid artery in gerbils, and to analyse the causes of its secondary decline.

MATERIAL AND METHODS

The experiments were carried out in 22 adult Mongolian gerbils under intraperitoneal pentobarbital anesthesia (70 mg/kg). Two groups of animals were studied — 10 with pathophysiological control and survival time up to 9 hrs, and 12 without above control to assess progression of morphological alterations. All surgical procedures were performed using 12.5 × binocular operating microscope. In animals from the first group electrocorticogram — mono-polar and bipolar leads, electrocardiogram — second limbic lead and blood pressure from the femoral artery were continuously recorded. An anterior midline cervical incision was made and both common carotid arteries were separated free from accompanying structures. Bilateral common carotid artery occlusion of 30 min duration was produced with aneurysm clips. Animals were tracheotomised, and during the period of respiratory disturbances (12—13 min after occlusion) supportive controlled ventilation was applied. Cerebral bioelectric activity was evaluated on the basis of 5 grade score. Postmortem intra-aortic dye injection was performed to demonstrate communications between vertebro-basilar and carotid arterial systems. Animals from the second group were sacrificed every

hour after ischemia in groups of two up to 6 hrs, perfused with saline, and fixed with formaline. Routine histological staining (hematoxylin--eosin, Klüver-Barrera) was applied.

RESULTS

After common carotid arteries occlusion blood pressure increased around 40 mm Hg and in a majority of cases it was never below the control values till the end of cerebral ischemia. During the whole ischemic period the bioelectric silence was recorded from the cerebral cortex. Between 10 and 12 min of cerebral ischemia the respiratory disturbances developed and it was necessary to employ supportive controlled ventilations. Since 10—15 min of cerebral ischemia progressing bradycardia developed which was moderate at the end of ischemic period. Release of the common carotid arteries occlusion produced always a rapid drop of blood pressure which later on normalized, however, in 3 cases pharmacological support was necessary. Recovery of ECoG took place between 50 min and 3 hrs after the end of cerebral ischemia starting from the low frequency high voltage bioelectric activity reaching in two animals the control tracing. However, in 8 animals it never reached the control values being 25—50% lower as compared to the control amplitude and frequency. After this period secondary slow, progressive decline of ECoG appeared, and animals survived less than 9 hrs.

The morphological alterations were symmetrical and appeared already 1 h after ischemia. They were characterized by vacuolization of neuropil with generally unchanged neurons. Since 2—3 h after ischemia progression of alterations was distinct and characterized by tissue rarefaction and disseminated degenerative changes i.e. ischemic cells in neocortex and hippocampus. Since 4 h after ischemia, typical edematous alterations appeared in the white matter accompanied by demarcated necrotic areas in the grey structures. Focal necrosis in the neocortex and hippocampus (H2 sector) with fully developed edematous features of the white matter were evident in the 6th hour after ischemia.

Postmortem intra-aortic dye injection revealed in all animals very small vessels linking the basilar artery with the posterior cerebral arteries, providing insufficient communication between the vertebro--basilar and carotid circulations.

DISCUSSION

The results confirmed our former studies (Pluta, Kapuściński 1980) and those of other authors (Hossmann, Zimmerman 1974) that spontaneous bioelectric activity may recover even after 30 min of cerebral

ischemia in normothermic conditions. In a majority of cases the cerebral bioelectric activity progressively deteriorates leading finally to the brain death. Abnormalities in brain circulation that occur during the recovery period may play an important role in the pathomechanism of irreversible tissue lesions (Mossakowski 1978). The above data clearly show that during recovery of ECoG with tendency toward normalization, considerable progression of structural alterations exists with dominance of cytotoxic edema. They suppress the bioelectric activity for the second time leading to brain death. After longer period of cerebral ischemia even normalization of ECoG seems to be of no prognostic value. The widespread microscopic damage can be predicted by quite different sets of clinical criteria for brain death (Black 1978). The clinical evaluation considering the duration of cerebral ischemia seems to be the crucial survival prognostic factor.

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ROZPRZĘŻENIE POMIĘDZY POWROTEM KOROWEJ AKTYWNOŚCI BIOELEKTRYCZNEJ I POSTĘPEM ZMIAN MORFOLOGICZNYCH PO NIEDOKRWIENIU MÓZGU U CHOMIKÓW MONGOLSKICH

Streszczenie

W znieczuleniu pentobarbitalem u 22 chomików mongolskich wykonano 30-minutowe niedokrwienie mózgu przez obustronne zamknięcie tętnicy szyjnej wspólnej. Podczas niedokrwienia i w okresie poischemicznym u zwierząt oceniono zmiany ciśnienia krwi, czynności oddechowej i serca oraz aktywności bioelektrycznej kory mózgu. Pośmiertnie oceniono zmiany morfologiczne w mózgu. Spontaniczna czynność bioelektryczna kory mózgu powracała pomiędzy 50 min i 3 godz. po zakończeniu niedokrwienia mózgu, osiągając w niektórych przypadkach kontrolną amplitudę i częstotliwość zapisu. Podczas powrotu czynności bioelektrycznej mózgu z tendencją w kierunku normalizacji obserwowano progresję zmian strukturalnych z dominowaniem obrzęku cytotoksycznego.

Rozwój zmian strukturalnych powodował pogorszenie czynności bioelektrycznej prowadząc do śmierci mózgu. Wyniki badań wskazują, że kliniczny pogląd biorący pod uwagę czas trwania niedokrwienia mózgu wydaje się być uzasadniony i jest głównym czynnikiem prognostycznym przeżycia.

ДЕЗОРГАНИЗАЦИЯ МЕЖДУ ВОЗВРАЩЕНИЕМ БИОЭЛЕКТРИЧЕСКОЙ КОРКОВОЙ АКТИВНОСТИ И ПРОГРЕССОМ МОРФОЛОГИЧЕСКИХ ИЗМЕНЕНИЙ ПОСЛЕ ИШЕМИИ ГОЛОВНОГО МОЗГА У МОНГОЛЬСКИХ ХОМЯКОВ

Резюме

В анестезии пентобарбиталом у 22 монгольских хомяков авторы произвели 30-минутную ишемию головного мозга при помощи двустороннего закрытия общей сонной артерии. Во время ишемии и в постишемическом периоде они оценили у животных изменения давления крови, дыхательной функции и деятельности сердца, биоэлектрической активности мозговой коры, а также морфологические изменения. Спонтанная биоэлектрическая функция мозговой коры возвращалась между 50 мин. и 3 час. после окончания ишемии головного мозга, достигая в некоторых случаях контрольную амплитуду и частоту записи. Во время возвращения биоэлектрической функции головного мозга со стремлением в направлении нормализации авторы наблюдали прогрессию структуральных изменений с доминированием цитотоксического отека.

Развитие структуральных изменений вызывало ухудшение биоэлектрической функции и приводило к смерти головного мозга. Результаты исследований показывают, что клиническое мнение, принимающее во внимание продолжительность ишемии головного мозга кажется быть обоснованным и является главным прогностическим фактором переживания.

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