Proceedings of the Society of British Neurological Surgeons

The 80th Meeting of the Society of British Neurological Surgeons was held in Cambridge on 18-20 June 1970 as a joint meeting with the Deutsche Gesellschaft für Neurochirurgie.

HEAD INJURIES IN THE 1970S

WALPOLE LEWIN (Cambridge) stated that newer diagnostic methods might allow a better understanding of the several complications that followed head injury. Illustrations of these methods and their limitations were given. He expressed the view that the emphasis in the immediate future might well rest on the recognition that after head injury a dynamic process was set in train with resultant secondary effects, some of which could be prevented and others corrected.

INTERRELATIONSHIP OF RESPIRATORY DISTURBANCES AND CHANGES IN BLOOD AND CSF GASES AFTER SEVERE HEAD INJURY

R. A. FROWEIN and A. KARIMI-NEJAD (Köln) reported that their clinical observations and analysis of blood and CSF gases—even with a free airway—had demonstrated two forms of respiratory insufficiency after head injury or raised intracranial pressure. The first was depressed respiration—namely, alveolar hypoventilation with arterial hypoxia, hypercapnia, depressed pH, and partly decreased base excess. The other resulted in hyperventilation with hypoxia but with arterial hypocapnia, normal or increased pH, and in contrast CSF acidosis. They had observed the same changes in meningitis and in acute cerebrovascular disturbances. They suggested that the cause of arterial hypoxia in this hyperventilation syndrome might be a functional intrapulmonary ventilation perfusion inequality.

Both groups of respiratory disturbances led to hypoxia and both, therefore, required respiratory therapy. In depressed respiration the indication for assisted ventilation was usually based on the increased PaCO_2 . In hyperventilation, however, the decreased PaCO_2 indicated hypoxia and represented complex central as well as pulmonary disturbances. Treatment was not only by neuroplegics but, if the respiratory rate rose higher than 30/min, was also by assisted controlled ventilation—that is, by IPPB and slight negative expiratory pressure. The effect of assisted ventilation could be demonstrated by continuous blood gas monitoring. The results obtained in 381 patients were encouraging, even in the older age groups, but they showed the limitations of treatment in cases in which serious aspiration had occurred initially.

ELECTROLYTES, FLUIDS, AND ENERGY METABOLISM IN TRAUMATIC BRAIN OEDEMA

H. J. REULEN, M. SAMII, K. FENSKE, and K. SCHÜRMANN (Mainz) described a systematic analysis of the different factors contributing to the development of cerebral oedema as a basis of treatment of this serious complication of severe head injury. Focal cortical oedema was produced in dogs by the application of cold as described by Klatzo. The oedematous areas were outlined by vital staining by Geigy blue dye. Twenty-four hours after the infliction of the lesion, extracellular space water, electrolytes, phosphates, and some of the metabolites of glycolysis were measured in stained and unstained areas of the injured hemisphere and in tissue from the contralateral uninjured hemisphere. The cold injury produced a sharply limited necrosis of the superficial cortex, surrounded by oedematous tissue which had taken up the blue dye. In the stained cortex adjacent to the lesion a slight increase in the water, sodium, and chloride content was found but there was a much greater increase in the subjacent deeply stained white matter.

The authors developed a method of estimating the cerebral extracellular space in order to distinguish between intracellular and extracellular accumulation of fluid. This depended upon achieving the same concentration of extracellular indicator (radioactive labelled sodium-thiosulphate) in both serum and CSF by parallel intravenous and ventriculo-cisternal infusions. An increased extracellular space, compared with normal tissue, was found in both damaged cortex and white matter and was two to three times greater in the latter.

Study of the distribution of electrolytes between extra- and intracellular spaces showed that intracellular Na and Cl increased in oedematous grey and white matter while K decreased. A relationship between an increase of intracellular Na and intracellular water was then demonstrated leading to the conclusion that the increase of intracellular sodium content was one factor responsible for cellular swelling.

Results of the analysis of intermediate metabolites of glycolysis revealed a disturbance between energy production and consumption in oedematous tissue. Anaerobic ATP production by means of an accelerated rate of glycolysis appeared insufficient to meet cellular energy requirements and the energy reserves of the affected