Neurosurgical Clinic, Helsinki University Central Hospital, Helsinki, Finland

Diffuse Experimental Brain Injury: Methods, Histological Findings, and Changes in Intracranial Pressure and Blood Pressure

By

S. Valtonen and H. Troupp

With 5 Figures

Summary

This paper deals with the effects of a diffuse experimental brain injury in the rabbit. The injury was induced by injecting a small amount of olive oil into one internal carotid artery. Out of 27 experimental animals 12 died from the injury after a mean interval of 62 minutes; the remainder survived for at least this period with little or no rise in intracranial pressure. The animals which died from injury had a confluens sinuum pressure of 73 mm Hg and a confluens sinuum/arterial blood pressure ratio of 0.68. This ratio is higher than that seen after a severe local cold injury, but lower than that seen in connexion with hydrostatically raised intracranial pressure. These findings support the view that a large local injury reduces the power of the brain to withstand raised intracranial pressure, and that scattered small lesions are less detrimental than one large one. The confluens sinuum pressures recorded tally well with clinically recorded pressures. It may be assumed that this type of experimental injury resembles severe clinical injuries—brain contusions—to a considerable extent.

As intracranial pressure is an important factor in assessment of clinical brain injuries (Jennet 1973, Troupp 1967, Vapalahti 1970, Vapalahti and Troupp 1971) it has been extensively investigated experimentally as well as clinically. Several different methods of raising the intracranial pressure have been used (Berman et al. 1969, Kaste 1970, Kuurne 1971, Langfitt et al. 1964, Ommaya et al. 1964). The results have differed considerably; for instance, with a local cold lesion the animals died at much lower levels of intracranial pressure than when intracranial pressure was raised hydrostatically (Kuurne and Troupp 1972). It has also been suggested that the local cold lesion caused distortion of the brain stem and affected surrounding tissue.
In this paper, we are investigating the rise in intracranial pressure and its prognostic significance after a diffuse experimental brain injury. It is hoped that such a brain injury might resemble a clinical diffuse injury—a brain contusion—more than a local cold lesion or hydrostatically raised intracranial pressure.

For the injury, we chose a massive oil embolism, achieved by injecting a small amount of olive oil into the internal carotid artery of rabbits. Similar methods have been used previously for induction of oedema of the brain (Blinderman et al. 1962, Brock et al. 1972, Broman 1949, Edström and Essex 1956, Tzonos and Finck 1968).

**Material and Methods**

For the experiments rabbits breathing spontaneously were used. The mean weight of the animals was 3490 grams and the total number 27. The animals were anaesthetized with urethane injected into an auricular vein; atropine was also given at the beginning of the anaesthesia. The mean dose of urethane was $1.55 \pm 0.25$ g/kg and that of atropine $0.17 \pm 0.03$ mg/kg. For local anaesthesia 1–2% prilocaine was used.

A tracheostomy was performed and the common carotid arteries were exposed. A piece of thick surgical silk was loosely placed round the left common carotid artery; no more preparations were carried out on this side. On the right side both the common and the internal carotid arteries were cannulated. To prevent clot formation, 5000 I.U. of heparin was given intravenously at this stage. Then the animals were turned prone, the calvaria was exposed and the periosteum scraped off. The confluens sinuum was exposed with a dental drill, and a plastic tube was attached to it with isobutyl metacrylate. Three screws were driven into the skull, and cold-curing acrylate was used to fill in the bone defect and anchor the plastic tube firmly to the screws.

Blood pressure and confluens sinuum pressure were recorded with Statham P23 AA pressure transducers fed from an Advance Electronics PP 31 D.C. source. The outputs from the transducers were amplified in a Norma 709 D.C. amplifier and fed into a 3-channel Norma inkwriting recorder.

An injection of 0.05 ml olive oil together with 0.25 ml physiological saline into the right internal carotid artery caused the diffuse brain injury, while the left common carotid artery was kept closed. The amount of oil had been determined in preliminary experiments and was found not to be critical.

After the trauma the parameters mentioned were recorded until the animal suffered respiratory arrest or until it become clear that no rise in intracranial pressure was taking place. This meant monitoring for at least 50 minutes after the injury. All except two of the surviving animals then received 0.2 mg metaraminol bitartate and some of this group then had a rise in intracranial pressure.

The heads of the animals were fixed in formaldehyde solution for several weeks. The brains were then removed and inspected, and histological slides were made. These were stained with Sudan and haematoxylin-eosin stains and studied under a light microscope. The same procedure was followed with the lungs of three of the animals.