Acute Brain Swelling in Neurosurgical Patients*

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In previous investigations of the pathophysiology of intracranial hypertension and cerebral swelling, we recorded intracranial pressure, cerebral blood flow, and several additional variables during expansion of a supratentorial balloon in the monkey. Among the phenomena observed were recurrent, marked rises in intracranial pressure. These pressure waves occurred spontaneously, were readily produced by hypoxia and hypercapnia, and could be aborted by hyperventilation with high oxygen mixtures. As pressure waves continued, the brain swelled, intracranial pressure ultimately rose to that of arterial pressure, and cerebral blood flow ceased.

There is evidence that the intermittent brain swelling responsible for the periods of marked intracranial hypertension is due to cerebrovascular dilatation. We also believe that the end stage of the process, when intracranial pressure equals the arterial pressure and varies directly with it, is partly due to paralysis of cerebral vaso-constrictor tone. The purpose of this report is to present clinical confirmation of these earlier observations.

The cerebral swelling which occurs following craniotomy frequently develops during the first 24 hours. If brain damage has not been severe, the patient improves rapidly following surgery. Rarely the initial postoperative improvement is followed by abrupt deterioration and death, which is often attributed to a major vascular accident. However, this can be caused by acute brain swelling.

Case 1. A 47-year-old woman had total removal of a large acoustic neurinoma. There was no significant technical difficulty. During the first hour after surgery she awoke and was well oriented although her reactions were still slow. The only neurological deficit noted at this time was paralysis of the right facial nerve. Breathing was shallow but effortless, and the trachea contained few secretions. An hour later she began to complain of severe headache and became duller. Blood pressure had risen from 130/70 to 170/100. A lumbar puncture was performed, and the pressure was more than 600 mm. CSF. Approximately 15 ml of spinal fluid was removed. The closing pressure was 100 mm. CSF; the level of consciousness improved, and she said that the headache had subsided. Blood pressure also returned to normal. However, 15 minutes later she became quite restless, again complaining of severe headache, and within 10 minutes was comatose with decerebrate posturing on painful stimulation. The pupils were dilated and failed to respond to light, and marked respiratory depression was now evident. Blood pressure was 240/130. Endotracheal intubation was performed promptly and hyperventilation begun with 100 per cent oxygen. Within 5 minutes she began to improve. At the end of 30 minutes the pupils were normal, the patient was responding to command, and blood pressure was 160/90. She subsequently recovered, and although she is able to walk unaided and care for her daily needs, residual ataxia of the right arm and leg has thus far prevented her from returning to work.

Comment. The increased intracranial pressure immediately following surgery and the subsequent correlation of marked arterial hypertension with neurological signs indicative of brain stem compression suggested the same pressure wave observed in the animal experiments. The prompt response to hyperventilation strengthened the hypothesis that brain swelling under these circumstances was due to acute cerebrovascular dilatation.

On the basis of this and similar clinical observations, a technique was developed for continuous measurement of intracranial pressure following craniotomy. At the end of the operation a No. 10 French catheter was inserted into the subdural space, beneath intact bone, or into a lateral ventricle if hydrocephalus was present. It was brought out through a stab wound adjacent to the incision and tightly sutured to the scalp. When the patient had been returned to the

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intensive care unit, the catheter was attached to a transducer and pressure continuously observed on a polygraph. We have in this manner recorded intracranial pressure in 41 postoperative craniotomy patients. This report is concerned with patients who developed severe intracranial hypertension during their postoperative course.

We began with the assumption that the brain swelling following craniotomy is at least in part due to cerebrovascular dilatation. In Case 1, intracranial pressure had increased rapidly during the postoperative period despite apparently adequate respiratory function and the process was rapidly reversed by hyperoxygenation. Therefore, in all patients in whom intracranial pressure was recorded, the endotracheal tube was left in place following surgery, and respirations were assisted with a Bird respirator and 40 per cent oxygen and room air. When the patient would no longer tolerate the endotracheal tube it was removed. If intracranial pressure exceeded 50 mm. Hg and was accompanied by evidence of neurological deterioration, the patient was either reintubated or a tracheostomy was performed. Forty per cent oxygen and room air and 100 per cent oxygen, with and without hyperventilation, have been used in an attempt to control the pressure. This method of treatment has been supplemented with intravenous infusion of hypertonic solutions in some patients.

The following case illustrates the rapid relief of acute intracranial hypertension which can be achieved with controlled respiration.

Case 2. A 22-year-old woman was admitted with marked papilledema, progressive visual loss, and bilateral 6th nerve palsies. An EEG showed mild diffuse slowing, and a bilateral carotid arteriogram was normal. A pneumoencephalogram was planned but visual acuity began to decrease rapidly. A provisional diagnosis of pseudotumor cerebri had been made, and an emergency right subtemporal decompression was performed at 8:00 p.m. on the 4th hospital day. The dura was surprisingly lax, and pressure recorded from a catheter inserted into the subdural space over the frontal lobe was 10 mm. Hg.

Postoperative course. The patient was returned to the intensive care unit. When the catheter was again attached to the transducer, pressure was nearly 50 mm. Hg but fell to normal during the next 3 hours. The endotracheal tube was removed at this time. At 4:00 a.m. the next day, pressure began to rise again, and an hour later had reached 50 mm. Hg (Fig. 1). The patient's reactions became dull; she had a left hemiparesis and respiratory depression. A tracheostomy was performed, but by the time it was completed the intracranial pressure had risen to the mean blood pressure level of 95 mm. Hg, and she was

![Fig. 1. Case 2. Blood pressure and pulse rate during spontaneous increases in intracranial pressure. The initial drop in pressure (1st arrow) was produced by normal ventilation with 40 per cent oxygen. The second pressure wave declined more slowly despite hyperventilation with 100 per cent oxygen (2nd arrow). Pressures are in mm./Hg.](image-url)