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)
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Fig. 2. Case 2. The response of intracranial pressure to assisted respirations with 40 per cent oxygen during 10 day period. The respirator was applied (up arrow) when intracranial pressure approached 50 mm. Hg or there was some evidence of neurological deterioration and was then removed (down arrow) when pressure had been reduced. Note fluctuations in systolic blood pressure which are not related to intracranial pressure.

Comatose. Mechanical ventilation with 40 per cent oxygen was begun. Pressure fell from 95 to 25 mm. Hg in 6 minutes, and she began to improve. However, the intracranial pressure soon rose again rapidly, despite continued respiratory assistance. Hyperventilation with 100 per cent oxygen was instituted. The pressure responded immediately but declined more slowly than before. At a level of 20 mm. Hg, approximately an hour after beginning hyperventilation, the respirator was switched to a normal pattern with 40 per cent oxygen. Intracranial tension again increased, and periodically during the next 24 hours it was necessary to use hyperventilation in order to control the pressure.

For 2 hours intracranial pressure approximated the mean blood pressure, and cerebral blood flow must have virtually ceased if the recorded pressures were correct. A bottle of saline, connected by a stopcock to the catheter, was slowly raised until the saline began to drip. The distance from the bottle to the external auditory meatus corresponded to the recorded intracranial pressure within 5 mm. Hg. We assumed that the blood pressure measurements, obtained with a standard brachial cuff, were inaccurate.

Fig. 2 illustrates the effect of respiratory assistance on intracranial pressure during the following days. The respirator was applied when the pressure reached 50 mm. Hg, or when it was warranted by the patient’s clinical condition. On each occasion intracranial tension fell to near normal levels, then increased again when the respirator was removed. Intracranial pressure gradually declined to normal, and on the 11th postoperative day, the catheter was removed.

One month following surgery the patient was alert, and well oriented, but with evidence of severe residual brain damage. The correct diagnosis is still in doubt.

Fig. 3 illustrates another patient in whom prompt changes in intracranial pressure were produced by alterations in respiratory function. Five hours following removal of an acoustic neurinoma, intracranial pressure rose to 50 mm. Hg, despite apparently adequate respiratory function, then gradually fell (A1) when respiratory assistance with 40 per cent oxygen and room air was given. Subsequently (B) removal of the respirator was accompanied by a marked rise in intracranial tension which immediately fell toward normal when respiratory assistance was given again. This emphasizes the effect of minimal depression of respiratory function on intracranial pressure and the prompt response to adequate ventilation which can be obtained.

In the following case, intracranial pressure
ceased to respond to respiratory control during the course of the gradual rise in intracranial tension.

Fig. 3. A. Fall in intracranial pressure produced by respiratory assistance with 40 per cent oxygen (1). B. Rapid changes in intracranial pressure when the respirator was removed (1 and 3) and reinstitution of respiratory assistance with 40 per cent oxygen (2 and 4). Time between triangles = 30 minutes.

Case 3. A 60-year-old man had had partial removal of a pituitary adenoma in 1949 and was asymptomatic until 1955 when he again developed progressive loss of vision. He was treated with radiation therapy with remission until the summer of 1964, a year prior to his last admission. At that time he began to complain of excessive tiredness and intermittent headache. For 6 months he had had progressive blurring of vision in both eyes. During the week prior to admission he had become markedly confused.

On examination his reactions were sluggish and disoriented, and he rarely responded to commands. The pupils were equal and reacted to light. Visual acuity was difficult to evaluate but was greatly reduced in both eyes, and the optic discs were pale.

X-rays of the skull showed complete destruction of the sella turcica and considerable enlargement of the sphenoid sinus. A bilateral carotid arteriogram revealed lateral displacement of the terminal carotid arteries; the first portions of the anterior cerebral arteries were greatly elevated.

At operation a large pituitary adenoma was found which extended into the anterior 3rd ventricle producing obstructive hydrocephalus. The bulk of the tumor was removed, and a catheter was placed in the right lateral ventricle for postoperative recording of intracranial pressure.

Postoperative course. Following surgery he responded only to painful stimulation, and within

Fig. 4. Case 3. Response of intracranial pressure (ICP) and blood pressure (SAP) to withdrawal (1st arrow) and reinstitution (2nd arrow) of respiratory assistance. Subsequently a spontaneous increase in intracranial and blood pressures occurred. An infusion of mannitol was begun (3rd arrow), and both pressures fell rapidly. Time between triangles = 2 hours.
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Fig. 5. Case 3. Prior to death intracranial pressure equals the mean blood pressure and varies directly with it. Time between triangles = 2 hours.

a few hours intracranial pressure had increased to 45 mm. Hg. A tracheostomy was performed, and hyperventilation as well as intermittent withdrawal of cerebrospinal fluid through the ventricular catheter caused a fall in intracranial pressure. However, these maneuvers became progressively less effective, and 24 hours following surgery re-exploration was performed.

Second operation. The brain was moderately swollen, and there was necrosis of the tip of the right frontal lobe. The anterior half of the right frontal lobe was resected, and the bone flap was removed. A catheter was placed in the femoral artery for continuous recording of blood pressure.

Postoperative course. Following operation, intracranial pressure again rose but could be controlled by respiratory assistance and subsequently hypertonic mannitol (Fig. 4). However, intracranial tension eventually reached the level of the mean blood pressure and could not be reduced with hyperventilation or mannitol. Spontaneous respirations ceased, and norepinephrine was then required to maintain the blood pressure. Fig. 5 illustrates correspondence of the 2 pressures during alterations in the vasopressor infusion rate. The patient died on the 8th postoperative day, and permission for postmortem examination was refused.

Comment. The response of the blood pressure to rising intracranial pressure varied greatly. In Case 2 intracranial tension exceeded the mean blood pressure without initiating a vasopressor response, but in Case 3 the arterial pressure responded immediately to changes in intracranial pressure. In addition to the variability observed in the vasopressor threshold, marked spontaneous changes in blood pressure occurred, unrelated to intracranial tension, in patients without a prior history of arterial hypertension (Figs. 2 and 6).

One of the principal criteria established for the end stage of cerebral vasomotor paralysis in the monkey was the immediate rebound of the intracranial pressure to the level of the blood pressure following sudden evacuation of the balloon. Clinical confirmation of this
Spinal fluid protein obtained prior to admission was 20 mg. per cent. A vertebral arteriogram suggested a right cerebellar tumor.

Ventriculogram and craniotomy were planned, but on the evening prior to surgery she was found comatose. When examined 30 minutes previously, she had been neurologically unchanged; now she did not respond to painful stimulation, the pupils were dilated and fixed to light, and respirations had virtually ceased. She was intubated, placed on the respirator, and taken to the operating room. A needle attached to a transducer was inserted into the right lateral ventricle through a coronal burr hole. The opening pressure was 90 mm. Hg. Blood pressure at this time was 140/70. Forty ml. of fluid were removed in 5–10 ml. increments. After each evacuation the pressure fell to zero then gradually rose again. A catheter was inserted into the ventricle and another in the femoral artery for recording blood pressure. The patient was transferred to the intensive care unit.

Initial recordings showed the intracranial pressure higher than the diastolic blood pressure. When ventricular fluid was removed, the pressure fell, but immediately rose again to the arterial level. A needle was inserted into the lumbar subarachnoid space. Spinal fluid pressure was 21 mm. Hg at a time when intracranial pressure was 110 mm. Hg. Fig. 7 illustrates the effect of withdrawal of ventricular fluid on intracranial pressure. In 3 hours, 100 ml. ventricular fluid were removed, but there was no change in the neurological state.

Communication of pressure from the intra-
cranial space to the spinal compartment was restored by the injection of fluid into the lumbar subarachnoid space but the block occurred when the injection ceased (Fig. 8). Twenty-four hours after surgery the patient died. At postmortem examination a cystic astrocytoma was found in the right cerebellar hemisphere. A small amount of fluid remained in the ventricular system.

Discussion

Spontaneous variations in intracranial and lumbar subarachnoid pressure occur normally and are of no pathological significance. However, if there is a space-occupying intracranial mass, these fluctuations in pressure may be greatly exaggerated. Moreover, marked alterations in brain volume of unknown etiology have frequently been described and usually are manifested by a rapid change in cerebrospinal fluid pressure or sudden swelling of the brain at the time of craniotomy. This has been observed following severe head trauma, in patients with intracranial neoplasms, hydrocephalus, and pseudotumor cerebri. We have also seen rapid fluctuations in intracranial pressure from 10 to 90 mm. Hg in patients following spontaneous subarachnoid hemorrhage. Therefore, these pressure waves are not related to specific intracranial pathology, and the only common factor is a space-occupying intracranial mass or prior brain swelling.

Wolff and Forbes observed dilatation of cortical vessels in response to increased intracranial pressure, and Evans et al. have emphasized that cerebral vasodilatation increases cerebral blood volume and thereby causes a further rise in intracranial pressure. Thus a cycle is established. When vasodilatation increases blood flow, it also causes a secondary rise in intracranial pressure tending to further impede flow. If a systemic vasopressor response occurs, cerebral blood flow and blood volume rise again, and intracranial pressure continues to increase. This appears to be the origin of the pressure waves.

Pressure phenomena similar to those observed in the present series have been described by Lundberg in patients with space-occupying lesions. He recorded intraventricular pressure continuously prior to surgery; increases in pressure to 80–100 mm. Hg followed by a spontaneous fall to normal levels were frequently observed.

Pressure waves probably account for the intermittent, often severe, neurological signs and symptoms which may occur in patients with chronic space-occupying lesions, and even result in acute death if the lesion is not removed. We believe that this is due to a rise in intracranial pressure sufficient to produce a critical reduction in cerebral blood flow and that this is the most likely explanation for the rapid change in Cases 2 and 4. However, in addition to diffuse cerebral ischemia,
transventricular herniation and brain stem compression also play major roles.

In the absence of prior intracranial abnormality, the vasodilatation required to produce severe intracranial hypertension must be quite marked; the speed of the phenomenon is also important. Since the intracranial space is enclosed by bone and filled to capacity, a slight increase in volume produces an enormous rise in pressure. Normally, an increase or decrease in cerebral blood volume is accompanied by an opposite adjustment in cerebrospinal fluid volume. This results in damping of the effects of blood volume changes on intracranial pressure. Also, as a lesion expands within the intracranial space, spatial compensation occurs largely through a reduction in cerebrospinal fluid volume. When this displaceable fluid has been reduced to a minimum, any slight additional increase in the volume of either the brain or a space-occupying mass causes a disproportionate increase in intracranial tension.12 At such a time changes in the volume of the cerebrovascular bed cause alterations in intracranial pressure that are much greater than those normally observed. This accounts for the marked sensitivity of intracranial dynamics to relatively minor alterations in the O₂ and the CO₂ content of the blood, when a mass lesion or pre-existent brain swelling is present.

Our results emphasize the importance of adequate respiratory exchange in postcraniotomy patients. Lundberg et al.17 made the same observations in preparative patients with space-occupying lesions. Because of the marked sensitivity of intracranial pressure to moderate degrees of hypoxia and hypercapnia, patients require tracheostomy, or endotracheal intubation and respiratory assistance whenever there is evidence of progressive neurological deterioration. Tracheostomy is most frequently performed when there is evidence of obstruction of the airway. If the airway is patent, tracheostomy alone is of no value, and the process can be reversed only by assisting respiratory function. This type of ventilatory abnormality is frequently encountered in brain damaged patients.

The enormous swelling of the brain which may occur rapidly during craniotomy when the airway becomes obstructed, is undoubt-
edly due in part to hypoxia and hypercapnia. However, the expansion of brain volume and increase in intracranial pressure normally produced by hypercapnia are minimal compared to the swelling of the brain which may be observed at surgery. Furthermore, obstruction of the airway does not invariably result in brain swelling, and at times the anesthesiologist is able to convince the surgeon that the airway is indeed patent. This suggests that factors other than CO₂ retention may contribute to brain swelling. Cairns4 discussed the possible role of vascular factors in cerebral swelling and referred to the sudden increase in brain bulk which occasionally occurs during marked retraction over a deep seated tumor in the total absence of any evidence of obstruction of the airway.

Rapid, generalized swelling of the brain was produced by LeBeau and Bonvallet15 with lesions in the midbrain. Obrador and Pi-Suñer18 frequently obtained acute brain swelling by placing lesions in the floor of the 4th ventricle, and attributed the swelling to acute vascular dilatation. Evans and Scheinker6 examined the brains of patients with post-traumatic cerebral swelling and concluded that severe brain injury causes vasoparalysis of capillaries and venules and an accompanying increase in venous pressure. Subsequently, Scheinker9 stated that the histological signs of vasoparalysis represent a stage in cerebral swelling which immediately precedes the most advanced stage, cerebral liquifaction. Thus, there is evidence that acute brain damage, and perhaps more specifically injury to the brain stem, results in a rapid diminution in cerebral vasoconstrictor tone. Although the cerebrovascular congestion and intracranial hypertension which result may be augmented by hypoxia and hypercapnia, the suddenness of the vasodilatation suggests that these factors do not contribute to the initial swelling.

Summary

After major intracranial surgery, the brain may swell sufficiently to raise intracranial pressure to the level of the blood pressure and stop cerebral blood flow. This usually develops during the 1st day and occasionally occurs acutely, within a few hours after surgery.

In order to clarify certain clinical aspects
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of this problem we have left catheters in the intracranial space of a series of post-craniotomy patients and correlated the continuous pressure recording with coincident clinical or blood gas changes. In almost all cases post-operative intracranial hypertension could be prevented by appropriate respiratory control.

The results of this clinical investigation are closely related to our previous studies on monkeys indicating that brain compression causes cerebrovascular dilatation and ultimately cerebral vasomotor paralysis.

References