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Acute Intraoperative Intracranial Hypertension in Neurosurgical Patients:

Mechanical and Pharmacologic Factors

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Intracranial pressure (ICP) and cardiovascular changes were studied intraoperatively in eight neurosurgical patients undergoing craniotomy. Four patients with preoperative signs of increased ICP had acute elevations in ICP (34–88 torr). These occurred during intubation with thiopental and succinylcholine, initial exposure to halothane, application of a head clamp, and intracranial insertion of a saw guide. Some abrupt increases in ICP occurred without significant alterations in systemic arterial and central venous pressures or evidence of hypoxia or hypercarbia. In response to similar stimuli, patients presumed to have normal ICP's preoperatively had no marked changes in ICP. During administration of halothane, cerebral perfusion pressures associated with acute episodes of intracranial hypertension were less than or equal to 60 torr in the preoperatively-high-ICP group, while they remained above 80 torr in normal-ICP patients. (Key words: Intracranial pressure; Halothane; Tracheal intubation; Neurosurgery; Cerebral perfusion pressure.)

INITIAL CONCERN with increases in intracranial pressure (ICP) caused by administration of certain volatile anesthetics to neurosurgical patients[1,2] has directed attention away from additional intraoperative sources of acute increases of ICP. Investigators have found precipitous elevations in lumbar and ventricular cerebrospinal fluid (CSF) pressures during tracheal intubation.[3,4] Stephen felt that this CSF-pressure elevation was the result of hypoxia and hypercapnia that developed during difficult intubations.[3] Alexander and Lassen[5] recently suggested that arterial hypertension such as that accompanying endotracheal intubation[6,7] might induce rapid cerebral swelling in patients with brain tumors or acute cerebrovascular disease.

During the conduct of neurosurgical operations using halothane anesthesia and hypocarbia, we have studied the respective roles of arterial and central venous pressures and mechanical stimuli in the genesis of intraoperative intracranial hypertension. In addition to the previously reported ICP increases caused by halothane and intubation, we have identified other mechanical sources of ICP elevation during the performance of surgical operations.

Methods and Materials

Intracranial pressure, blood pressure (BP), and central venous pressure (CVP) were monitored continuously in eight patients undergoing craniotomy. The patients were divided into two groups. The first consisted of four patients presumed preoperatively to have increased ICP's (I-ICP) resulting from supratentorial intracranial tumors or obtundation and secondary obstructive hydrocephalus caused by recent subarachnoid hemorrhage. Each of these patients had one or more of the following signs or symptoms commonly associated with intracranial hypertension: headache; nuchal rigidity; decreased level of consciousness; papilledema; paresis of cranial nerve VI. The four patients in the second group were presumed to have normal preoperative intracranial pressures (N-ICP), based on the absence of the above-mentioned signs and symptoms, as well as on diagnostic knowledge of their intracranial diseases and/or recently-measured intracranial pressures. The N-ICP patients had either a small tumor causing a focal neurologic deficit or seizures, cortical atrophy with seizures, or subarachnoid hemorrhage after 72

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TABLE 1. Intracranial Pressures and Arterial Blood Pressures during Induction and Stabilization of Anesthesia in Neurosurgical Patients*

	Period 0 Preinduction		Period 1 Postinduction		Period 2 Laryngoscopy†		Period S_1 Pre-intubation		Period 3 Halothane		Period S_2 Pre-clamp		Period 4 Clamp		Period S_3 Pre-puncture		Period 5 Cuffle Insertion	
	ICP	BP	ICP	BP	ICP	BP	ICP	BP	ICP	BP	ICP	BP	ICP	BP	ICP	BP	ICP	BP
Increased ICP Group																		
Patient 1	0	122	6	103	32	142	20	125	47	110	16	108	56	136	26	80	82	100
Patient 2	2	121	4	110	32	162	8	119	38	100	15	96	46	114	16	101	40	108
Patient 3	14	66	13	68	33	76	11	68	44	56	18	68	61	110	18	68	26	82
Patient 4‡	31	120	20	50	54	124	10	50	27	62	27	51	33	75	14	66	20	62
Normal ICP Group																		
Patient 5	8	102	6	128	13	212	11	188	12	155	14	125	16	152	8	82	28	85
Patient 6	7	150	6	98	10	142	6	125	11	130	—	—	—	—	—	—	—	—
Patient 7	6	100	4	150	11	168	1	75	6	72	2	49	19	62	—	—	—	—
Patient 8‡	23	105	10	148	16	202	5	200	7	155	12	110	—	—	—	—	—	—

* Arithmetic means are computed; pressures are in torr.
† CSF drainage started at least 24 hours prior to induction of anesthesia (see Methods).

hours of ventriculostomy decompression with a normal sensorium.

Preoperatively each patient received either no premedication or a small dose of pentobarbital with atropine. Through a twist-drill hole made using local anesthesia, a no. 14 French flexible catheter was introduced into a lateral cerebral ventricle. Small amounts of CSF, ranging from a few drops to 3–4 ml, were spilled as the ventricular tubing was inserted and connected to the pressure transducer. Two patients with subarachnoid hemorrhage (patients 4 and 8) came to the operating room with ventriculostomies already established. These were clamped approximately 75 minutes prior to induction of anesthesia.

Blood pressure was measured through an 18-gauge Long-Dwell catheter inserted into the radial artery. A 17-gauge Intracath catheter percutaneously placed into the superior vena cava or right atrium with radiographic confirmation was used for CVP measurement. The ICP zero reference was the midpoint of the cranial cavity, and the other pressures were referenced to heart level. There was little difference between these reference points, since all measurements were made with the patients supine. When mean pressures are reported in the text, they represent arithmetic means of systolic and diastolic pressures, and they are preceded by a capital M.

Arterial blood gases were measured prior to induction of anesthesia and thereafter intermittently. Shortly after intubation, end-tidal carbon dioxide (percentage) was monitored in most patients. Pressures and end-tidal CO_2's were recorded on a multichannel polygraph.

Patients were preoxygenated for 3 minutes and given 3 mg *d*-tubocurarine iv just prior to induction. Anesthesia was induced with thiopental (125–450 mg), followed by succinylcholine (60–80 mg). Immediately after tracheal intubation, ventilation was controlled and anesthesia maintained with 3 l nitrous oxide, 2 l oxygen, and 0.5 to 2.0 per cent halothane, administered through a recently-calibrated Fluotec vaporizer.

Results

The most interesting results were found during several periods coded and identified as follows:

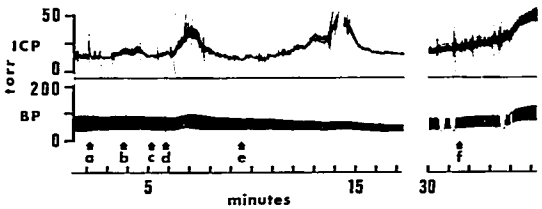

FIG. 1. Polygraph record of intracranial pressure (ICP) and blood pressure (BP) changes in a patient with high ICP prior to induction. Pressures are in torr. Event code: *a*, thiopental, 250 mg; *b*, thiopental, 200 mg; *c*, succinylcholine, 80 mg; *d*, laryngoscopy and intubation; *e*, begin 1.5 per cent halothane; *f*, application of head clamp. Time, in minutes, is counted from the administration of *d*-tubocurarine prior to induction of anesthesia.

0. Preinduction
1. Postinduction—immediately after administration of thiopental and succinylcholine
2. Maximal ICP response to laryngoscopy and intubation
S_1. Stable period prior to administration of halothane
3. Maximal ICP response to halothane
S_2. Stable period prior to application of head clamp
4. Maximal ICP response to application of the head clamp
S_3. Stable period preceding insertion of Gigli saw guide
5. Maximal ICP response to insertion of the saw guide

During the stable periods there was no change in the ICP or other variables monitored for at least 2 minutes. The head clamp used in period 4 has three prongs which are driven through the skin to seat in the outer table of the skull. The saw guide is a thin stainless steel strip which is passed over the dura mater between two burr holes.

Table 1 summarizes the mean changes in ICP and BP, during the periods defined above, for I-ICP and N-ICP patients. In the I-ICP group large increases in ICP and BP accompanied laryngoscopy and tracheal intubation. ICP elevations of the same magnitude occurred during the initial exposures to halothane. However, these ICP increases were accompanied by decreases in BP. Application of the head clamp and insertion of the saw guide caused increases of ICP which were associated with moderate or no increases in arterial pressure.

In response to all stimuli except passage of the saw guide (data available for one patient only) the maximal increases in ICP in the N-ICP group were much smaller. In the N-ICP group, despite wide fluctuations in BP, mean ICP remained within −15 to +10 torr of the preinduction value prior to introduction of the saw guide. During the same period the mean changes in CSF pressures in the I-ICP group ranged from −3 to +65 torr. BP changes in the two groups were similar.

In figure 1, three typical ICP responses associated with various intraoperative stimuli are shown on the polygraph record of an I-ICP patient. During intubation (fig. 1, *d–e*) MICP

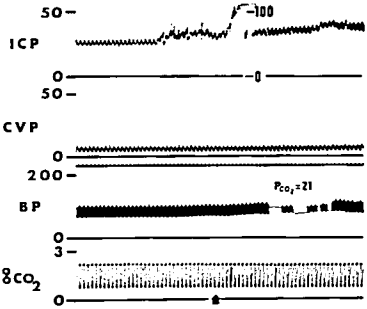

FIG. 2. Polygraph record of intracranial pressure, central venous pressure (CVP), arterial blood pressure, and end-tidal CO_2 (per cent) in a patient with a high preinduction ICP. The arrow indicates insertion of the saw guide after 85 minutes of anesthesia. The guide was immediately withdrawn. Pressures are in torr. Note the scale change for ICP necessitated by the great increase (from 50 torr to 100 torr, full scale). The center channel indicates time marks at 5-second intervals.

increased by 20 torr, while MBP went up only 8 torr. (In the other three I-ICP patients the increases in MBP ranged from 40 to 50 torr with intubation. In the N-ICP group MBP increased 18–84 torr during intubation.) A second ICP peak occurred shortly after the administration of 1.5 per cent halothane (fig. 1*e*) and was accompanied by a 12-torr decrease in MBP. The peak pressure decreased somewhat more rapidly in this patient than in some others, but in every patient whose ICP increased with halothane, the pressure slowly decreased spontaneously while halothane administration continued. The third ICP peak response in figure 1 resulted from application of the head clamp (*f*), and appeared after 20 minutes of halothane–nitrous oxide–oxygen anesthesia. In this instance (fig. 1*b*) an initial small increase in ICP preceded an increase in BP which was accompanied later by a much greater increase in ICP. In every patient this increase in ICP with placement of the head clamp spontaneously decreased slowly if no additional measures to produce more rapid decompression were taken.

Another typical ICP response in an I-ICP patient is shown in figure 2. Introduction of the saw guide (fig. 2, *arrow*) evoked a small initial increase in ICP, which was followed by a much greater increase in ICP and a small increase in blood pressure. Despite immediate removal of the guide, the pressure remained elevated, and shortly thereafter the surgeon requested that CSF be withdrawn from the lateral ventricle to permit safer opening of the dura mater.

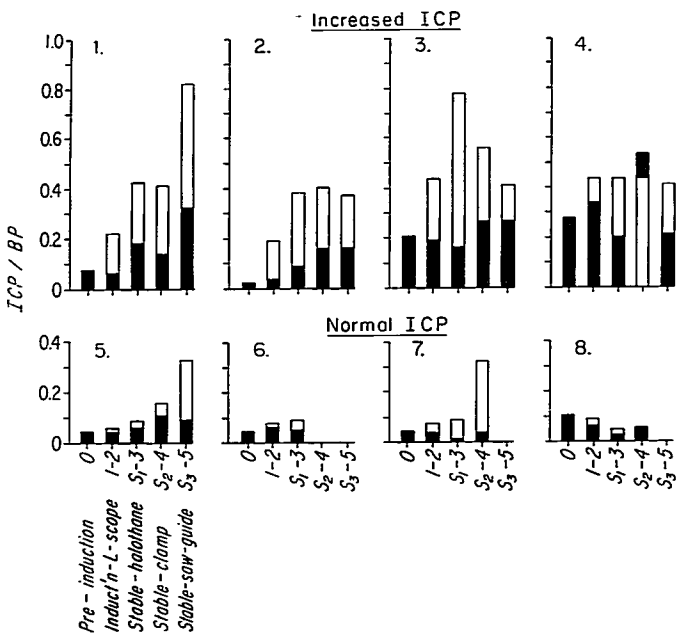

FIG. 3. Intracranial pressure/blood pressure ratios for patients with increased and normal ICP's prior to operation. The solid bars indicate a prevailing stable condition and the open bars denote the ICP/BP relationship during an ICP peak response. Further elaboration in text. ("L-scope" is laryngoscopy.)

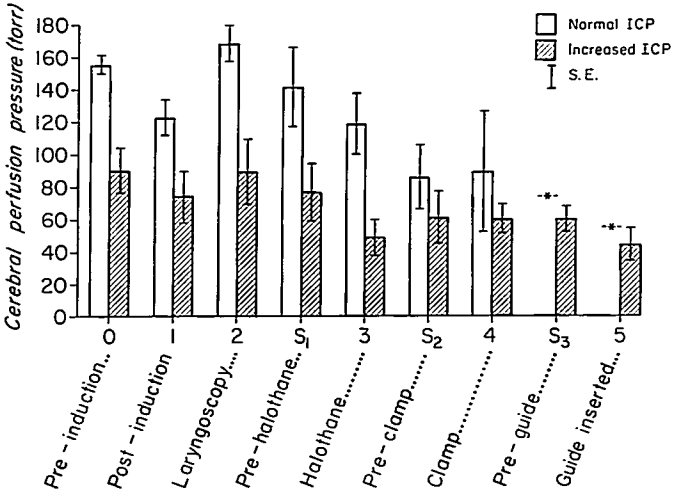

FIG. 4. Mean cerebral perfusion pressures (±SE) computed for the preinduction and peak ICP response periods in the increased-ICP and normal-ICP groups. An asterisk indicates values for one patient.

CVP was recorded continuously in six patients, three each in the I-ICP and N-ICP groups. Aside from small fluctuations of less than 4 torr during intubation, no CVP changes accompanied the maximal ICP responses described above. All intubations were quickly and easily performed except in one patient in the I-ICP group. Blood taken from this patient during the intubation ICP peak had a Pa_{CO_2} of 30 torr and a Pa_{O_2} of 286 torr. Arterial blood-gas samples taken intermittently from other patients during periods of acutely elevated CSF pressures had Pa_{O_2}'s greater than 85 torr and Pa_{CO_2}'s in the range of 21 to 36 torr. End-tidal CO_2 measurement in six of the eight patients after intubation demonstrated no acute changes associated with sudden alterations in intracranial tension.

Discussion

Differences between I-ICP and N-ICP Patients

Representative ICP measurements were not obtained in our patients prior to commencement of anesthesia, owing to CSF loss when the ventricle was cannulated or to periods of controlled CSF drainage preoperatively. Because of this, preinduction ICP's of the two groups of patients were similar. However, their ICP responses to subsequent stimuli were different. Factors which may have been responsible for these different responses include presence of a space-occupying lesion, propensity to accumulate cerebral edema, and abnormal cerebrovascular responses.

Initially, a slowly-expanding intracranial-mass lesion will have little effect on ICP, because CSF is expressed from the cranial cavity into the more-distensible spinal-arachnoid space.[8] This mechanism provides an important source of volume compensation for the expanding intracranial mass. When this means of pressure buffering is exhausted, any additional small increment in intracranial volume will produce a substantial increase in ICP. This ability for spatial compensation is attenuated in patients who have signs of increased ICP.

Since alterations in tumor-tissue mass and CSF volume occur slowly, changes in other intracranial compartments may form the basis of the rapid, partially-reversible elevations of

ICP observed in our patients. Risberg has demonstrated alterations in cerebral blood volume paralleling acute changes in ICP in patients with brain tumors.[9] Abnormal cerebrovascular reactivity has been demonstrated in ischemic brain tissue and in areas surrounding brain tumors.[5] In these areas, flow is pressure-dependent, and the autoregulatory capacity is lost.[5, 10, 11] Presumably, sudden increases in blood pressure could increase the blood volume and flow in these vessels, and thereby increase ICP.[5] Schutta has shown experimentally that arterial hypertension in this situation can lead to acute cerebral edema and herniation of intracranial tissue within 2 minutes.[12] He suggests that loss of tone in the larger vessels permits transmission of high arterial pressures into the cerebral capillary bed, leading to increases in extracellular space and swelling of neuronal elements. Therefore, increases in blood volume and/or brain water content may have contributed to the intracranial hypertension in our patients.

Relation of ICP and BP Alterations

Simultaneous increases in ICP and BP occurred with intubation in our patients, and either no change or a comparatively smaller increase in blood pressure was associated with the other acute increases of ICP. The relationship between BP and ICP is illustrated in figure 3. Assuming a normal mean blood pressure of 90–100 torr and a normal maximal ICP of 10 torr, a "normal" ICP/BP value would be 0.1 or less. In figure 3 the relative increases in the ICP/BP ratio in the I-ICP patients in stable periods (solid bars) and during acute increases of ICP (open bars) greatly exceeded the 0.1 value. Although relative increases in the ratio occurred in the N-ICP group, the values were not much greater than the normal ratio value. The higher stable-period ratios for the I-ICP patients indicate a progressive relative increase in ICP as the operation progressed.

Cerebral Perfusion Pressure

As ICP increases, mean cerebral perfusion pressure (CPP = BP − ICP) may decrease. Uneven intracranial-tissue pressure gradients may lead to even greater focal decreases in tissue perfusion pressures.[5] The potential for cerebral ischemia is further enhanced by anesthetic depression of blood pressure.[13] Johnston and co-workers suggest that CPP may actually be more important than the actual ICP during acute episodes of intracranial hypertension.[14] Figure 4 shows selected mean CPP's obtained in our patients. CPP improved during intubation owing to the associated arterial hypertension. After the administration of halothane, CPP's in both groups of patients decreased. The post-halothane decreases in CPP in the I-ICP group were greater because their ICP's were higher. In the absence of measurements of cerebral blood flow, the information provided by CPP is, at best, only suggestive.

Etiology of the Effects of Halothane and of Mechanical Factors on ICP

The ICP response to halothane is believed to be the result of the direct vasodilatory action of the volatile anesthetic, and can occur as BP decreases.[15, 16] Etiologic analysis of intracranial hypertension produced by the head clamp and saw guide is more complex, for, although the blood pressure increased, the absolute increases in ICP in three of the I-ICP patients were greater than the increases in arterial pressure. The increase in systemic arterial pressure with head clamping indicated that nociceptive reflexes were still active and that light anesthesia could be involved in this ICP increase. Insertion of the saw guide resulted in an immediate increase in ICP in every I-ICP patient and in the one N-ICP patient studied. This was later followed either by no change in blood pressure or by a minor increase. It was possible that the guide depressed the dura mater and created a significant mass effect which led to acute intracranial hypertension. However, the ICP increase was maintained after withdrawal of the guide. This substantial fixed increase in ICP may represent a variation of the Cushing response.[17, 18] The dura is known to be richly innervated with sensory fibers, and dural stimulation by the guide could activate a primary intracranial vascular reflex, leading to increased ICP.[19, 20] No confirmation of the existence of this mechanism or of the relative importance of passive and active intracranial vascular responses in the generation of clamp-induced and saw-guide-induced ICP elevations could be established from our data.

Clinical Recommendations

The practical lessons we have learned from our studies suggest several approaches to the anesthetic management of craniotomies. Preoperatively, it is important to identify patients with high intracranial pressures, large mass lesions, or obstructive hydrocephalus. Patients with normal CSF dynamics present no special problems. Reduction of cerebral edema prior to operation can be obtained by the use of osmotic dehydrating agents and potent diuretics.[21] Increased anesthetic depth prior to intubation may attenuate the cardiovascular response, leading to hypertension. Volatile anesthetic agents should not be used to attain deeper anesthesia, since they lead to increases in intracranial blood volume. Rather, agents such as thiopental,[22] fentanyl,[23] and droperidol[23] could be employed, since they are potentially cerebrovasoconstricting. The use of phentolamine as a means to modify the arterial response has also been suggested.[7] Endotracheal topical anesthesia[6] may help, but should be used only after adequate depths of anesthesia have been obtained, since laryngoscopy alone can cause arterial hypertension in the lightly-anesthetized patient. Selective application of monitoring of ventricular CSF pressure and drainage may further optimize chances for a successful neurosurgical operation on the high-risk patient.

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