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Treatment of Intracranial Hypertension

Analysis of 105 Consecutive, Continuous Recordings of Intracranial Pressure

By

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Summary

One hundred and five consecutive recordings of intracranial pressure (ICP) in 95 patients over a three-year period, using a Scott cannula inserted through a burr hole or a twist drill hole into the anterior horn of the lateral ventricle, represent the patient material for this report. The clinical diagnoses were head injury [32], intracranial tumour [31], aneurysm and arteriovenous malformation [18], brain swelling secondary to systemic disease [8], and brain swelling of unknown etiology [6]. ICP exceeded 20 mm/Hg in 86 of the recordings (maximum 110 mm/Hg). Hypertonic mannitol was administered 73 times in 48 patients. ICP was reduced 10% or more (mean 52%) in all but three administrations. The effect of hyperventilation was tested in 50 trials in 34 patients. ICP was reduced 10% or more (mean 47%) in 34 trials. The mean time to maximum reduction of ICP was eight minutes, and ICP returned to control almost immediately after cessation of hyperventilation. Hypothermia was studied in 40 trials in 40 patients. ICP was reduced 10% or more (mean 51%) in half the patients. The infection rate was 6.3% in this four-hospital setting, but four of the six infections were in one hospital. If this hospital is excluded, the infection rate is 3.1%.

Introduction

The principal reason for continuously recording intracranial pressure (ICP) is to provide information that will permit better management of the individual patient with intracranial hypertension [8, 15, 28]. ICP is now monitored frequently in neurosurgical practice [4, 5, 7, 8, 19], because it is evident that increased ICP is a common cause of neurological death [6, 11, 12, 17, 25] and numerous methods for treating intracranial hypertension are available [7, 8]. This report contains an analysis of 105 consecutive, continuous recordings of ICP in 95 patients using an intraventricular cannula. The two major pur-

poses of the report are to describe the responses of ICP to therapy in patients with diverse intracranial pathology and to report the incidence of complications in a four-hospital setting in which the patients were managed by many attending and resident neurosurgeons.

Materials and Methods

The 95 patients were studied during a three-year period. They ranged in age from 1 to 73 years, 60 males and 35 females. The clinical diagnoses are listed in Tab. 1. The indications for recording ICP were clinical evidence of a severe brain insult (*e.g.*, head injury, subarachnoid hemorrhage), cerebral swelling secondary to systemic disease or of undetermined etiology, and clinical evidence of postoperative intracranial hypertension in patients following craniotomies. Seventy patients were stuporous or in a coma at the time of the recording.

ICP was recorded with a Scott cannula inserted through a burr hole or a twist drill hole 2 cm lateral to the midline at the level of the coronal suture into a lateral ventricle, the right frontal horn in 86 patients and the left frontal horn in eight patients. The right occipital horn was used in one patient. The Scott cannula was connected by sterile intravenous tubing to a Statham transducer model B 23 D, then to a stripchart recorder for continuous display of ICP. The transducer was calibrated at frequent intervals to adjust for drift as necessary. The transducer was wrapped in a sterile towel and pinned to the sheet at the level of the patient's occiput so head and transducer moved together during changes in the vertical position of the patient's head. Therefore, the zero reference point was at or near the tip of the occipital lobe. The recording time ranged from a few hours to 16 days with a mean of 3.8 days.

On completion of the recording CSF from the tubing and/or Scott cannula was obtained for routine culture and sensitivities. Some of the patients in this series had measurements of regional cerebral blood flow (rCBF) and the cerebral metabolic rate of O_2 utilization ($CMRO_2$). Twenty-two of these patients have been reported in a separate communication [1].

Results

ICP ranged from 0 to 110 mm/Hg. In 86 patients ICP exceeded 20 mm/Hg one or more times during the course of the recordings, excluding transient ICP changes secondary to movement of the patient, suction, and straining (Tab. 2). The plateau waves of Lundberg [15] were observed in 19 recordings, and B waves also were observed in 19 recordings.

Evaluation of specific modes of treatment for intracranial hypertension is difficult in very ill patients. Spontaneous changes in vital signs are common, and therapeutic maneuvers such as tracheal toilet and turning the patient alter ICP. Methods of treatment for increased ICP that act quickly (and ICP returns rapidly toward control values when the treatment is terminated) can be evaluated more accurately in the individual patient than treatment modalities that

are slower in onset and act for a longer period of time. In this series, 58 patients received various doses of dexamethasone on to which other methods of treatment were superimposed. We have been unable to evaluate the effects of steroids in this group of patients because of the superimposition of other methods of treatment.

Table 1. *Clinical Groups*

	Number	Associated hydrocephalus
Intracranial tumors	31	17
Supratentorial	18	6
Infratentorial	13	11
Head injury	32	4
Vascular disease (aneurysms and arteriovenous malformations)	18	4
Brain swelling secondary to systemic disease	8	0
Brain swelling of unknown etiology	6	0
Total	105	25

Table 2. *ICP in 105 Consecutive Continuous Recordings in 95 Patients*

	Head injury	Intracranial tumors	Vascular disease	Brain swelling secondary to systemic disease	Brain swelling of unknown etiology	Total
ICP over 20 mm/Hg	30	27	17	6	6	86
ICP below 20 mm/Hg	6	8	4	1	0	19

In 40 patients intermittent drainage of CSF through the ventricular cannula was performed on 46 occasions. ICP fell immediately in all cases, confirming the well-established clinical experience that the most effective means of rapidly reducing ICP is withdrawal of a few milliliters of CSF. In several patients with rapidly rising ICP and concomitant clinical decompensation, this was a life-saving procedure [10]. However, in patients with markedly elevated baseline ICP, intracranial tension tended to return rapidly to its previous level after withdrawal of CSF or ventricular drainage, and other methods of treatment were required. Furthermore, too vigorous or prolonged withdrawal of CSF occasionally resulted in ventricular collapse and

obstruction of the cannula. The recording was no longer available when it was needed most.

In the studies of hypertonic mannitol [8, 14, 16, 26, 29], hyperventilation [2, 8, 13, 20, 23], and hypothermia [8, 21, 22, 24], a reduction in ICP of 10% of control or more was defined as an effect of the agent. Hypertonic mannitol was administered as a bolus (0.18–2.5 gm/kg of a 25%

Table 3. *Clinical Diagnoses and Responses to Mannitol in 44 Patients—73 Administrations; Dose: 0.18–2.5 gm/kg*

	Head injury	Intracranial tumors	Vascular disease	Brain swelling secondary to systemic disease	Brain swelling of unknown etiology	Total
Response	27	13	7	11	9	67
No response	1	1	0	0	1	3
Undetermined	0	0	1	2	0	3

Table 4. *Characteristics of Responses to Mannitol in 38 Patients—67 Administrations*

ICP Reduction

Range: 10–98%
Mean: 51.9%

Time to Maximum Reduction

Range: 20–360 minutes
Mean: 88 minutes

Time to Return to Control

Range: 45 minutes–11 hours
Mean: 210 minutes

solution in 2–10 minutes) 73 times in 44 patients. A reduction in ICP occurred 67 times, with a range of 10–98% and mean reduction in ICP of 52%. The response by disease categories are listed in Tab. 3. The time from completion of the bolus injection to the maximum response of ICP was 20 to 360 minutes with a mean of 88 minutes. The time from completion of the bolus injection to return of ICP to the control value was 45 minutes to 11 hours with a mean of 210 minutes (Tab. 4). Among the 6 administrations in which no response was obtained, three could not be evaluated properly because of the introduction of other variables. On patient subsequently

responded to a larger dose, and one patient had responded to five previous doses of mannitol. Rebound of ICP to 10% or more above control values occurred in only three patients (Tab. 5). Thus, hypertonic mannitol almost always reduced increased ICP irrespective of the intracranial pathology. The amplitude and length of the response varied greatly; as a rule, the higher the initial ICP, the shorter was

Table 5. *Rebound of ICP Following Hypertonic Mannitol*

Patient	Diagnosis	Control	ICP (mm/Hg) Maximum response	Rebound
CL_2	Postoperative meningioma	44	28	60
DB_1	Postoperative meningioma	50	38	60
RF_2	Cerebral infarction	50	18	70

Table 6. *Clinical Diagnoses and Responses to Hyperventilation in 34 Patients—50 Trials*

	Head injury	Intracranial tumors	Vascular disease	Brain swelling secondary to systemic disease	Brain swelling of unknown etiology	Total
Response	9	14	3	5	3	34
No response	4	1	4	1	0	10
Undetermined	2			3	1	6

the response to mannitol. We do not have sufficient data to comment on refractoriness to repeated administrations in the same patient.

Hyperventilation was studied in 50 trials in 34 patients who had an endotracheal tube or tracheostomy in place, by increasing the rate and depth of respiration. ICP was reduced by 10 to 80% with a mean of 46.6% in 34 of the 50 trials. In ten trials there was no detectable reduction in ICP, and in six trials the response could not be determined because of straining or another variable that precluded evaluation (Tab. 6). The time from initiation of hyperventilation to maximum reduction of ICP ranged from 2 to 30 minutes with a mean of 7.6 minutes. After cessation of hyperventilation ICP returned to control values in less than five minutes in all cases. In 14 trials the patients were maintained on hyperventilation after a demonstrated reduction in ICP. In nine of the 14 trials ICP returned to the control value in 12 to 80 minutes despite continued hyperventilation at the same rate and volume that reduced ICP in the first place. In the

remaining five trials ICP was reduced for periods ranging from two to 30 hours (Tab. 7). In contrast, to mannitol, which reduced ICP in nearly all cases irrespective of the intracranial pathology, hyperventilation had a selective effect. Patients with severe brain damage from vascular accidents and head injuries responded less well than patients with intracranial tumours and diffuse brain swelling (Tab. 6).

Table 7. *Characteristics of Responses to Hyperventilation in 34 Patients—50 Trials*

ICP Reduction
Range: 10–80%
Mean: 46.6%
Time to Maximum Reduction
Range: 2–30 minutes
Mean: 7.6 minutes
Return of ICP to Control with Continued Hyperventilation
9 trials
Range: 12–80 minutes
Mean: 37.6 minutes
Sustained Reduction of ICP with Continued Hyperventilation
5 trials
Range: 2–30 hours
Mean: 10.8 hours

Hypothermia was induced in 40 trials in 40 patients by placing the patient between thermal blankets, with or without the addition of ice bags between the legs and beneath the arms. Mild hypothermia (32–36 °C) decreased ICP by 10% or more in 13 of 32 trials. There was no response in 13, and in six trials the results were indeterminate because of the introduction of other variables (Tab. 8). The mean reduction of ICP in the 13 patients was 51%, and the time from induction of hypothermia to maximum ICP response was 240 to 720 minutes with a mean of 516 minutes (Tab. 9). Moderate hypothermia (27–31 °C) was used in eight trials, in addition to the 32 trials of mild hypothermia; that is, body temperature was reduced into this range by intent. A reduction in ICP was obtained in four trials, in two trials there was no response, and two trials were indeterminate (Tab. 8). The mean reduction in ICP was about the same as with mild hypothermia (Tab. 9). The times to maximum reduction

Table 8. *Clinical Diagnoses and Responses to Hypothermia in 40 Patients—40 Trials*

	Head injury	Intracranial tumors	Vascular disease	Brain swelling secondary to systemic disease	Brain swelling of unknown etiology	Total
Mild Hypothermia (32 to 36 °C) 32 patients:						
Response	4	5	0	2	2	13
No response	4	4	4	0	1	13
Undetermined	3	3	0	0	0	6
Moderate Hypothermia (27 to 31 °C) 8 patients:						
Response	2	0	0	2	0	4
No response	1	0	0	1	0	2
Undetermined	1	0	0	0	1	2

Table 9. *Characteristics of Responses to Hypothermia in 40 Patients—40 Trials*

Mild Hypothermia (32 to 36 °C) 13 Responses

ICP Reduction

Range: 33–77%
Mean: 50.7%

Time to Maximum Reduction

Range: 240–720 minutes
Mean: 516 minutes

Moderate Hypothermia (27 to 31 °C) 4 Responses

ICP Reduction

Range: 25–80%
Mean: 44.5%

Time to Maximum Reduction

Range: 120–180 minutes
Mean: 150 minutes

in ICP listed in Tab. 9 have little meaning, compared to the response times of ICP to hypertonic mannitol and hyperventilation, because the decrease in ICP paralleled the decrease in body temperature in nearly all cases. The time to maximum reduction of ICP was shorter with moderate compared to mild hypothermia, because body temperature was reduced as rapidly as possible in the former patients.

There were 46 deaths among the 95 patients, a demonstration of the severity of the illnesses in this series. Autopsies were performed in 20 patients. Along the track of the Scott cannula intracerebral and subdural hematomas of a few milliliters in volume and of no clinical significance were present in three and two patients respec-

Table 10. *Positive Cultures from 155 CSF Samples from 89 Recordings*

Organism	Number	Clinical infection
Proteus	1	Ventriculitis
Pseudomonas	2	(Same patient as above)
Staphylococcus coag. neg.	11	2—ventriculitis, 1—meningitis
Staphylococcus aureus	1	Subdural empyema
Corynebacterium species	1	None
Anaerobic streptococcus	1	None
Klebsiella	1	Ventriculitis and meningitis

tively. In 155 cultured samples of CSF from 89 recordings there were positive cultures from the CSF tubing and intraventricular cannula in 18. In eight cases the cultured organisms were grown from the tubing and intraventricular cannula, in eight from the intraventricular cannula alone, and in two from the CSF tubing alone. Six patients (5.7% of 105 recordings) manifested clinical signs of infection. The infection appeared to contribute to the death of three of the six patients. However, four infections and two of the deaths occurred in one of the four hospitals. If this hospital is excluded, the infection rate is 3.1%. The contributions to mortality in the three patients were ventriculitis, ventriculitis and meningitis, and subdural empyema. Tab. 10 lists the organisms cultured and the nature of the clinical infections.

In a group of 29 patients with continuous recording of ICP for five days or longer, positive cultures were obtained in eight (27.6%), and there were two clinical infections (6.9%). In the remaining 66 patients, ICP was recorded for a few hours through four days. There were ten positive cultures (13.2%) and four infections (5.3%). Therefore, the risk of intracranial contamination, if not clinical infection, appears to increase with the length of the recording time.

Discussion

We have used continuous recording of ICP mainly in two categories of patients, those who are comatose following an acute brain insult and following craniotomy for intracranial tumours and vascular lesions. In the latter group of patients, ICP is recorded postoperatively if the patient's neurological status was poor prior to surgery or if there is evidence at the time of operation that postoperative brain swelling might be a problem.

Five methods of therapy for intracranial hypertension were used in this series of patients. The effects of dexamethasone could not be evaluated properly, because none of the patients received dexamethasone alone. Withdrawal of CSF through the ventricular cannula is always the most rapid means of reducing ICP. It must be used sparingly if at all in patients with normal or small ventricles for fear of obstruction of the tip of the cannula by the ventricular wall. In this circumstance it is necessary to inject a small amount of fluid to clear the cannula, increasing the risk of infection and the risk of inducing a plateau wave. To avoid the former, we are now employing a millipore filter next to the monitoring device [5]. Withdrawal of CSF (and continuous ventricular drainage) is most effective in patients with hydrocephalus irrespective of the cause. During prolonged periods of drainage, computerized tomography of the brain has been of value in assessing ventricular size and anticipating the need for other forms of therapy to treat brain swelling [3].

Hypertonic mannitol reduced ICP in nearly all patients, but there was great variability among the patients in the times from administration to the maximum response of ICP and the times for return of ICP to control values. Among the variables that might influence the responses of ICP to hypertonic mannitol are the dose and the rate of administration of the agent and the volume of brain with disrupted blood-brain barrier. Our patients received a wide range of doses (0.18–2.5 gm/kg) over periods of time that varied by five fold. Therefore, we were not able to define a dose/effect relationship. Hypertonic agents reduce ICP by reduction of the fluid content of the brain. The effect is dependent upon an intact blood-brain barrier. It follows that hypertonic mannitol will reduce fluid volume in edematous brain with an intact blood-brain barrier but will not do so if the barrier is defective, because the mannitol rapidly enters the brain parenchyma [18]. Irrespective of the status of the blood-brain barrier in edematous brain, hypertonic mannitol dehydrates normal brain and thereby reduces ICP [14, 16]. The data from this series of patients demonstrate that in nearly every case there was a sufficient

area of intact blood-brain barrier to reduce the fluid content of the brain.

Hyperventilation was less effective in reducing ICP than hypertonic mannitol. In three of four patients with severe vascular insults of the brain and in four of nine patients with severe head injuries, there was no response of ICP to hyperventilation. These results suggest the presence of vasomotor paralysis of the cerebral vessels [9]. Normally, hypocarbia constricts cerebral vessels resulting in decreased CBF and decreased cerebral blood volume [20]; the decrease in cerebral blood volume is responsible for the decrease in ICP. Vasomotor paralysis is defined in part as loss of the response of cerebral vessels to a change in $PaCO_2$. Thus, one may infer that hyperventilation failed to reduce ICP in the patients with severe brain insults because the cerebral vessels were paralyzed and no longer responded to changes in $PaCO_2$ [9].

The return of ICP to control in nine of 14 trials during continued hyperventilation at the same rate and depth can be explained by the refractoriness of cerebral vessels to persistent hypocarbia. ICP tends to return to control at a constant, reduced $PaCO_2$ [20]. In some patients, however, it was possible to control ICP with hyperventilation for many hours suggesting that hyperventilation may reduce ICP by some mechanism other than cerebral vasoconstriction.

The data on hypothermia are more difficult to interpret than the results from treatment of ICP by withdrawal of CSF from the cannula, hypertonic mannitol and hyperventilation. Hypothermia is in the same class with steroid treatment for intracranial hypertension, because the reduction of ICP is slower than with the other methods of therapy. Some of the patients in this series were restless (including decerebration) with intermittent fluctuations in vital signs that induced frequent and some times large changes in ICP. Therefore, there was difficulty in maintaining the type of steady state that is required to evaluate a therapeutic method that acts slowly. Despite these methodological restrictions, hypothermia was effective in reducing ICP in nearly half the patients, and the amount of reduction of ICP was comparable to the results obtained with hypertonic mannitol and hyperventilation. There was little difference between the effects of mild and moderate hypothermia; but the time taken to induce hypothermia, and therefore the time to maximum reduction of ICP, varied so greatly that additional studies will be required in order to correlate properly reduction in body temperature with reduction in ICP.

In none of the 95 patients was there clinical or post-mortem evidence of a significant intracranial hematoma or other brain

damage secondary to insertion of the Scott cannula. The incidence of intracranial infections in this series of patients is high. However, most of the infections occurred in one hospital, and shortly after completion of this series of patients, the neurosurgical service in that hospital was closed for several reasons. Excluding the one hospital, the adjusted rate of infection (3.1%) is still higher than the experience of Lundberg and colleagues (1.1%) [27] and may be attributable to the management of the patients by many personnel in multiple settings. Currently ICP is recorded continuously only in a neurosurgical and a pediatric intensive care unit. Our experience with infections emphasizes the necessity of meticulous management of patients with indwelling intracranial contamination remains, in our opinion, the advantages of continuous monitoring of the responses of ICP to therapy in very ill patients outweigh the risks of intracranial infection in these patients.

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