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Long-Term Intracranial Pressure Monitoring in Comatose Patients Suffering From Head Injuries. A Critical Survey

By

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Summary

On the basis of a series of 75 patients, the practical use of ICP continuous long-term recording in severe head injuries without mass lesions or remaining deeply comatose after surgical procedures is critically analyzed. ICP monitoring alone seems to be not essential for prognosis. Conversely it is of much greater use as a guide to management (respirator treatment, osmotics, CSF drainage). If the pros and cons of the procedure are carefully weighed, it would appear that, for the time being, ICP long-term monitoring is justified only in comatose patients on intensive care.

The practical use of continuous intracranial pressure (ICP) recording in head injuries is still controversial. In actual fact, ICP measuring by the intraventricular technique entails some real risks, and extradural recording is not entirely reliable and calls for a minor surgical procedure. The question remains as to whether the advantages obtained by measuring ICP continuously for several days counterbalance the drawbacks of this procedure.

To Fleisher *et al.* [13], who reported on the vast experience of Tindall's group, the answer is negative in the cases of severe head injuries without mass lesions, because intracranial hypertension was seen only in a minority of their patients and ventriculitis occurred in about 10% of patients monitored.

Intracranial pressure monitoring in head injury patients has already been studied thoroughly from different standpoints. However, surveying the body of the data of literature, we notice that the material reported on is not strictly homogeneous.

First of all, to make a prognostic assessment some authors con-

sider only admission pressure before any treatment [2], whereas others take into account pressure values in patients undergoing different treatments for keeping ICP as low as possible. Secondly, the pathological situations that are gathered in the same series are fundamentally different (mass lesions operated or not operated on, diffuse lesions, secondary complications, etc.). The time of recording is also different, and prolonged recordings are not always performed, and finally, except in a few series [2, 7, 31], the patients were partly breathing spontaneously and partly on respirator treatment.

Therefore it seemed worthwhile to us to study a relatively homogeneous series of patients suffering from severe head injuries, in whom prolonged ICP monitoring was carried out up to death or to a definite improvement. Early and long-term respirator treatment was undertaken. We have considered only patients without mass lesions, and a small group of patients operated on, who remained in a coma state after the operation. In the latter patients ICP was not recorded preoperatively.

We feel that it must be pointed out that we are studying ICP in patients on intensive care, for in untreated trauma patients the course of ICP is quite different.

What we have been recording in our patients is the result of trauma and its complications on one hand and of therapy on the other.

Material and Methods

ICP was monitored in 75 patients suffering from severe head injuries. All of them were comatose; all but one were intubated. Fifteen were operated on (two epidural haematomas, five acute subdural haematomas, eight cerebral lacerations), remaining in a coma state after the operation.

Forty patients exhibited bilateral decorticate or decerebrate posturing. Oculocephalic and oculo-vestibular reflexes were serially studied in 23 patients.

In 40 patients ICP recording started on admission within the first day of the head injury; however, all patients but one had already been intubated. In eight patients the ICP recording started on the second day and in two on the third day from head injury. In five patients ICP recording started later.

The time of recording ranged from a few hours to 34 days; the average time was 6.5 days per patient. ICP was recorded until the patients died or definitely improved with a stable baseline within normal limits.

In 66 patients ICP was measured by Lundberg's intraventricular technique (Statham P 23 Db transducer, Soxil, General Electric or Siemens amplifiers).

In nine patients with very small ventricles ICP was measured in the upper spinal space approached antero-laterally between C 1 and C 2, with the patient lying in the supine position. The needle instead of being advanced in the sagittal plane, as for performing percutaneous vertebral angiography, is directed medially. The intervertebral foramen can be easily entered and the theca tapped.

Speed chart ranged from 1 mm/sec to 2 cm/hour.

The patients were classified, according to ICP values, in three groups:

1° patients with ICP below 20 mm Hg,
2° patients with ICP ranging from 20 to 50 mm Hg,
3° patients with pressure above 50 mm Hg.

We considered the highest values of mean ICP, without taking into account occasional variations due to straining, coughing, bronchial aspiration, etc.

Early long-term respirator treatment was undertaken in 70 patients; in 27 ICP recording was started when they were breathing spontaneously.

In adults the volume of ventilation ranged from 10 to 15 l/m (30–40% O_2). Arterial PCO_2 was kept between 20 and 30 and arterial pH from 7.45 to 7.50.

Thirty-eight patients were given 10% intravenous glycerol in saline. Single doses ranged from 250 to 500 ml. In some patients glycerol was repeated, with up to 150 g of glycerol in 24 hours.

In 26 patients different amounts of CSF were withdrawn through the ventricular catheter. Recently we have carried out semi-continuous or continuous drainages, measuring the quantity of CSF removed to keep ICP below 20 mm Hg.

Post-mortem examination was carried out in all the patients who died.

Results

The ICP course in head injuries is indeed very protean: some tracings have relatively stable baselines, others show minor or major pressure waves. There are G. Tindall's [34] pre-plateau waves and Lundberg's [25] true plateau waves. The latter, however, seem to be rather uncommon.

In some patients pressure is elevated on admission, decreasing thereafter but the contrary may also happen. In five cases we have observed very late ICP elevations, whose mechanism is probably manifold.

Save in extremely ill patients with the highest degree of hypertension (above 50 mm Hg) no correlation whatsoever was found between clinical condition and ICP values.

In nine deeply comatose patients, in whom we had not been able to tap the lateral ventricles, we recorded ICP from the upper spinal space at C 1–C 2. Spinal recordings prove reliable as long as the supratentorial pressure is freely transmitted through the tentorial notch and the foramen magnum. After ICP rises exceeding 70–80 mm Hg, pressure recorded from the spinal space becomes stable at a level of 30–50 mm Hg, and the Queckenstedt manoeuvre is blocked, the cerebral pulse being still transmitted. Subsequently, in the latest stages ICP drops again up to 15–20 mm Hg and the tracing becomes flat and pulseless. It must be remembered, however, that artificial ventilation was never stopped and therefore ICP was recorded in some cases after brain death. Extreme intracranial hypertension seems to be relatively uncommon in patients on intensive care.

In our material, except in patients with very high baselines and

vasoparalysis, ICP values alone are not a reliable guide to a prognostic assessment (Table 1). More parameters must be correlated: age, posturing, and oculo-vestibular reflexes. Posturing, with pressure exceeding 20 mm Hg, has a definitely bad prognostic significance; patients over 40 years with posturing never survived what-

Table 1. *ICP Values and Outcome: 75 Patients*

ICP values	Recovered	Severely disabled	Vegetative survival	Dead
0–20 mm Hg (24 patients)	5	3	—	17
20–50 mm Hg (49 patients)	9	5	2	24
50 mm Hg (9 patients)	—	—	—	10

Table 2. *ICP, Decerebrate Posturing and Outcome: 65 Patients*

ICP values	Alive	Vegetative survival	Dead
0–20 mm Hg			
Posturing (13 patients)	4		9
No posturing (12 patients)	3		9
20–50 mm Hg			
Posturing (24 patients)	5	2	15
No posturing (17 patients)	10		8

ever their pressures (Table 2–3). All the patients in whom oculocephalic and oculo-vestibular reflexes were tested had abnormal responses, so no conclusion can be drawn from this point of view (Table 4).

Seventy patients underwent respirator treatment early. In 27, ICP recording was started when they were still breathing spontaneously: all of them were already hyperventilating, and the gradients of $paCO_2$ between spontaneous and controlled ventilation were usually very small (2–5 torr). The same gradients were measured after the discontinuation of artificial ventilation.

ICP values before starting the artificial ventilation ranged from 10 to 20 mm Hg in 11 patients and from 20 to 50 mm Hg in 16 patients. In the latter group controlled ventilation was able to reduce ICP significantly only in 10 patients, but in 8 of them ICP rose again up to its initial values in a few hours. It is worth noting that four out of six patients in whom ICP was not reduced survived.

The effects of the discontinuation of respirator treatment were tested in 31 patients. After ventilation, $paCO_2$ gradients ranged between 2 and 5 torr in most patients.

Broader gradients up to 9–10 torr were quite rare, being observed

Table 3. *Age, ICP Values, Decerebrate Posturing and Outcome: 75 Patients*

Age	ICP	Posturing	Alive	Vegetative survival	Dead
0–20 years	0-20 mm Hg	+	2		2
		—	2		1
	20–50 mm Hg	+	4	1	4
		—	4		1
	> 50 mm Hg	+			6
20–40 years	0–20 mm Hg	+	1		1
		—	1		5
	20–50 mm Hg	+	2	1	6
		—	3		2
	> 50 mm Hg	+			1
over 40 years	0–20 mm Hg	+			4
		—	1		4
	20–50 mm Hg	+			7
		—	2		4
	> 50 mm Hg	+			3

Table 4. *Oculo-Vestibular Reflexes ICP and Outcome*

Tonic deviation 20 patients, no response 3 patients			
Tonic deviation	Alive	Vegetative survival	Dead
0–20 mm Hg	1		6
20–50 mm Hg	9	2	2
No response (frozen eyes)			
20–50 mm Hg	—	—	3

only in hypoventilating patients. In four patients $paCO_2$ gradients were not found on repeated examinations.

Artificial ventilation was never stopped when mean ICP exceeded 35 mm Hg. With baselines ranging from 20 to 35 mm Hg (15 patients) a steep rise in ICP took place in most patients; after the initial peak, ICP tended to decrease slowly reaching its initial

value in one to two hours. On some occasions, particularly in the first phase after the head injury, the baseline remained somewhat higher than before stopping ventilation. In four patients hyperventilating spontaneously, in whom there was no $paCO_2$ gradient, ICP remained unmodified.

When the baseline was within normal limits the ICP variations were rather unpredictable: ICP sometimes rose abruptly as in cases with higher pressure, but sometimes there was only slight, if any, modification in ICP values.

In 12 patients we have studied serially the responses to stopping artificial ventilation. We have noticed that with the same baseline and the same $paCO_2$ gradient the ICP elevation was small after glycerol infusion or CSF withdrawal, whilst some time later a very steep increase of ICP occurred. In the first days after head injuries ICP usually rose steeply up to very high values, whereas only slight variations were recorded some days later even with higher baselines.

In two patients who had undergone decompressive procedures only moderate increases in ICP were recorded.

Thirty-eight patients were given 10% glycerol. The drug was supplied in different ways: in single, repeated, and prolonged administrations.

The first 100 ml were always infused as quickly as possible (in about 10 minutes in most cases). We never infused less than 250 ml at a time in order to maintain the hypotensive effect from the first 100 ml as long as possible. Usually the effect of 250–500 ml of 10% glycerol lasts about four to six hours, but in some patients it is much shorter (one to two hours). In the latter cases repeated administrations are usually ineffective.

Slow administrations proved incapable of bringing ICP down significantly. We have also given 10% glycerol over prolonged periods: 1,000–1,500 ml a day in four to six doses. This procedure had some effect for two to three days at most; subsequently we had to try other procedures, mainly CSF withdrawal.

In five patients with early conjunctival oedema, urine hyperosmolality, and a tendency to widespread oedema, glycerol failed to modify ICP. Intracranial hypertension could be controlled only by reducing fluid intake (up to 500–1,000 ml/24 hours). Only one of these patients survived. Altogether glycerol proved to have some effectiveness in reducing high ICP in 26 patients: 14 survived. Two of the patients in the group where glycerol failed did survive but both remained severely disabled.

In 26 patients different amounts of CSF were withdrawn through

the ventricular catheter. In some cases in whom glycerol failed, CSF substraction was the only means for reducing ICP, but its effect in these cases were short-lasting. In nine cases we calculated the amount of CSF which had to be withdrawn to keep ICP within normal values: such amounts varied from 100 to 500 ml/24 hours. In six cases we left the drainage open, removing 250—500 ml per day.

In three patients the quantity of CSF drained decreased spontaneously in some days, but three patients continued to drain large amounts for several days. As CSF dynamics seemed to be clearly altered in the latter cases we inserted valves and all these patients recovered quickly.

In the patients draining the largest amounts of CSF the effects of glycerol were moderate and short-lasting.

At post-mortem, definite ventriculitis was seen in 2 patients out of 51 (4%).

Discussion

If admission pressure, or even better the pressure immediately after the head injury, is not recorded most of the highest values will be overlooked. Crockard *et al.* [6, 7], who were also able to measure ICP within the first hour after gunshot injuries, and D. Becker *et al.* [2], recorded the highest pressure in acute subdural haematomas and in some gunshot wounds. Such values seem to be rare in diffuse cerebral injuries.

The course of ICP in head injury patients on intensive care is unpredictable; ICP recording should be prolonged for a sufficient time, since, as Collice *et al.* [5], Rossanda *et al.* [31], and Enevoldsen *et al.* [12] pointed out, late ICP rises may sometimes occur, due to hyponatremia or pCO_2 elevations. In a few patients we have also observed these late elevations in ICP. These are likely to be related mainly to troubles with CSF dynamics and hydrosaline metabolism.

As regards the actual value of ICP for making a prognostic assessment in head injury patients, there are different opinions. Troupp an Vapalahti [37, 38, 39] found good correlation between ICP and outcome, whereas to Johnston *et al.* [21], Johnston and Jennett [19, 20], Jennett [18], Collice *et al.* [5], and Bruce *et al.* [3] this correlation seems to be poor. Everybody agrees that pressure exceeding 50 mm Hg always has an ominous prognosis, except in children. Cold *et al.* [4] state that a high level of pressure (above 30 mm Hg) during the first two days indicates a poor outlook. De Rougemont [8, 10] noticed that intracranial hypotension also presages a fatal outcome.

De Rougemont [8, 9] found that the ICP/SAP ratio is reliable for prognosis in head injuries, but Troupp *et al.* [36] stated that SAP is not

worth monitoring, for it does not contribute to a more accurate prognosis.

D. Becker *et al.* [2], considering only admission pressure, correlate ICP values, decerebrate and decorticate posturing, and oculocephalic reflexes. In their patients with mass lesions the ultimate results tend to correlate with decerebrate or decorticate posturing and oculo-cephalic reflexes, rather than with the absolute level of ICP. In diffuse cerebral injury, when admission pressure was normal, the result could be good or excellent, even with posturing, provided oculo-cephalic reflexes were intact. When ICP was elevated on admission the results were usually poor. When diffuse cerebral injury was not associated with posturing, all patients did well, even with high admission pressure.

As regards prognosis our findings can be summed up as follows: Baselines exceeding 50 mm Hg were always related to a fatal outcome. In these patients autoregulation was severely impaired, and in the last stages complete vasoparalysis took place, ICP following SAP passively. With a baseline up 50 mm Hg, ICP alone was not a reliable guide to prognosis. If high pressure was associated with decerebrate or decorticate posturing prognosis was poor. No patients over 40 years of age with posturing survived, no matter what their pressures were. Altogether, our results appear to be broadly in line with Becker's [2].

Before considering the role played by ICP monitoring as a guide to the management of head injuries, the question arises as to whether there is a critical level at which CPP is reduced and CBF impaired. Of course ICP should be brought down before reaching such a level.

Lundberg's [27] clinical observations, and Donaghy and Numoto's animal studies indicate that 35 mm Hg is the critical level. In animals studied through a cranial window this pressure produces occlusion of cortical veins. De Rougemont [8, 9] considers that the ICP/SAP ratio should be kept below 0.5. Rossanda [31] suggests that in clinical practice ICP calls for hypotensive therapy when the baseline reaches 25 mm Hg. Tindall *et al.* [35] arbitrarily indicate that a sustained ICP above 30 mm Hg needs specific treatment.

We think it is very difficult to talk in terms of critical levels, as factors underlying increased ICP may be more important than the level of pressure, as Jennett and Johnston [19] pointed out. It must be borne in mind, however, that intracranial hypertension itself may bring about widespread brain lesions [1, 15, 16]. Therefore it would seem logical that the ICP should be kept as low as possible. Nevertheless, raised ICP levels may be well tolerated by patients with localized mass lesions without brain shift or with CSF dynamics disturbances.

Conversely, the same values may by no means be tolerated if they are caused by diffuse lesions with widespread oedema and circulatory disturbances. We feel, therefore, that levels of ICP must be appraised only in the context of the whole clinical situation.

The actual value and the effects on ICP of respirator treatment in head injuries are still debated. Lundberg, Kjallquist, and Bien [26], in their classical monograph, have already noticed that ICP falls immediately after starting hyperventilation, but after some hours cerebral vessels adapt to the different arterial pCO_2 and ICP rises again, reaching its previous values. The same thing occurs when hyperventilation is interrupted: after an initial peak, ICP falls slowly, attaining its original baseline in some hours.

Recently Rowed *et al.* [32], in animal experiments and in patients, found that hyperventilation does not modify P/V relationships in intracranial hypertension, even if raised ICP is significantly reduced. Unlike Miller and Leech [24, 30], they think osmotics and corticoids have a beneficial effect on the P/V curve, rendering the intracranial content more tolerant to a volume addition. Dealing specifically with head injuries it must be remembered that almost all comatose patients in whom airway is free and respiratory function not impaired are already hyperventilating when they are still breathing spontaneously.

Gradients of pCO_2 between spontaneous and controlled breathing are very small, as Gordon [17], and Rossanda *et al.* [31] observed. In our experience we have observed that controlled ventilation is able to reduce ICP immediately in most patients, but after an initial drop ICP climbs again to its original values in one to two hours; only in the most severely ill patients with the most vigorous spontaneous hyperventilation or with vasoparalysis does ICP not change. Therefore, it seems to us that the beneficial effects of respirator treatment should not be considered only in terms of ICP reduction. In fact, artificial ventilation is sometimes able rapidly to improve the neurological condition in patients with intracranial pressures within normal limits.

For this reason ICP values are not relevant for establishing the indications for respirator treatment. By contrast, once respirator treatment is started, ICP monitoring is very useful to show when artificial ventilation can be stopped safely. ICP monitoring is likewise very important as a guide to therapy with osmotics, so that patients with normal pressures are not given unnecessary drugs. In fact, very often clinical deterioration has no relationship with ICP, which may remain within normal limits.

The initial effect of osmotic infusions depends on the speed of infusion, its duration, and on the quantity given. Kuhner *et al.* [23]

showed that large amounts of Mannitol are unnecessary, Johnston *et al.* [22], and Becker *et al.* [2] found that the results of repeated and prolonged administrations are disappointing. In comatose patients suffering chiefly from head injuries Bruce *et al.* [3] showed that the infusion of Mannitol causes the biggest increase of CBF in patients in whom ICP reduction is less relevant. As Miller [28] points out, the oedema fluid removed by Mannitol would be replaced immediately by an equivalent increase of blood volume due to impaired autoregulation of cerebral vessels.

In our experience, glycerol infusion proved effective in decreasing to some extent ICP in 26 patients out of 38, but on many occasions the reduction in ICP was moderate and of short duration. In other patients glycerol was effective only in the first few days. In our patients, repeated and prolonged administrations gave unrewarding results. High pressures which are never influenced by glycerol seem to presage a poor outlook (only 2 out of 11 patients survived). Further thorough studies are needed to elucidate hydrosaline metabolism disturbances occurring in some patients in whom glycerol is ineffective. In these patients, as in the inappropiate ADH secretion syndrome (Fox *et al.* [14]), only the reduction of fluid intake proved effective.

Unfortunately in our patients only a few routine clinical data are available and an exhaustive study of water balance was not performed.

In many patients CSF withdrawal is the only way of reducing high ICP. In some cases with high brain compliance ICP drops steeply after the removal of only a small amount of CSF, whereas in others considerable quantities must be removed to restore a normal baseline. Continuous drainage set at a level of 15 mm Hg was undertaken in six patients who continued to drain large amounts (250 to 500 ml) for several days. Three of these patients underwent CSF shunt procedures and recovered quickly. This would indicate that the disturbances of CSF dynamics are not rare even in the very early phase of head injuries, as Enevoldsen *et al.* [12] have recently observed.

Conclusions

It would be unwise to draw too definite conclusions from one single parameter (ICP), which is the result of many factors. Thus, ICP must always be correlated with other data, both clinical and instrumental. On the whole, ICP monitoring appears not to be essential for prognosis in head injury patients, although it may be

of some practical use if correlated with clinical data. When high ICP is not modified by therapy the outlook is usually poor.

By contrast, ICP monitoring is much more valuable as a guide to management. While the indications for artificial ventilation cannot be stated only in terms of ICP values, its discontinuation may entail some risks if ICP is not monitored. Conversely, if ICP does not vary significantly after stopping the ventilator, respirator treatment can be stopped safely and ultimately abandoned if clinical condition and paO_2 are satisfactory.

As there is no parallel between ICP levels and clinical conditions, osmotics can properly be infused only when ICP is monitored. ICP monitoring also showed that massive doses as well as repeated and prolonged administrations are usually ineffective and may produce untoward effects.

Generally speaking, fluid intake can also be regulated on the basis of ICP values. As Shenkin *et al.* [33] pointed out, restriction of fluid intake may play a relevant role in prevention and treatment of brain oedema.

Furthermore, ICP study enables us to detect CSF dynamics disturbances early and to manage them properly.

After considering the advantages of long-term ICP recording we must also take into account the far from negligible risks of this procedure.

It is indeed still very difficult to establish whether ICP monitoring is really worthwile in head injuries without mass lesions. We would say that for the time being it seems to be suitable only for comatose and dangerously ill patients on intensive care. In these cases, we feel, the advantages appear to outweigh the disadvantages. ICP monitoring, however, is likely to have a much broader range of applications in severely injured but non-comatose patients, to indicate complications, and to guide therapy, provided that safer procedures are available.

Preoperative ICP recording in head injuries in patients with well-defined mass lesions or brain shift is quite a different problem that must be dealt with separately.

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