Acta Neurochirurgica 52, 249—263 (1980)

ACTA
NEUROCHIRURGICA

Neurosurgery Division, Regional General Hospital and Department of Pathology, University of Ancona, Italy

Intracranial Hypertension in Severe Head Injuries

By

I. Papo, G. Caruselli, M. Scarpelli, and **A. Luongo**

Summary

Long-term ICP monitoring was carried out in a series of 124 patients with severe head injuries admitted to the Intensive Care Unit. Forty-nine percent of patients were admitted within six hours of injury. Most of them were referred by Community Hospitals. Only patients with diffuse brain lesions or patients operated on for mass lesions and remaining in a coma state after operation are taken into account. Altogether, 46 patients survived, but 15 of them remained severely disabled or in a vegetative state, and 78 died. Twenty-four percent of the whole series succumbed to fulminationg intracranial hypertension. The average survival in this group was 5.1 days. Twenty-nine percent died after exhibiting different levels of intracranial hypertension ranging from 20 to 50 mm Hg. In this group the role of extracerebral complications as a cause of death should not be underestimated. Death caused by cerebral lesions with ICP not exceeding 15 mm Hg was exceedingly rare in the first 72 hours. Normal or fairly raised ICP does not rule out the risk of devastating intracranial hypertension: reliable and harmless P/V tests are needed. All patients who survived after showing sustained intracranial hypertension exceeding 50 mm Hg were under 20 years of age. In the present series the results of treatment of intracranial hypertension were, on the whole, rather disappointing.

Keywords: Head injuries; traumatic coma; intracranial pressure; intracranial hypertension.

Intracranial pressure (ICP) monitoring in head injuries has been extensively studied in the past decade, either to establish its practical value for therapy or as a prognostic criterion. Both problems are still being debated, mainly because long-term ICP monitoring entails some drawbacks [15].

The goal of the present report is to assess the role played by intracranial hypertension in determining mortality and morbidity in a series of 124 patients.

Miller *et al.* [13] have pointed out the significance of intracranial hypertension in a large clinical series. They have demonstrated that

0001-6268/80/0052/0249/$ 03.00

a more aggressive therapeutic policy for controlling intracranial hypertension, both surgically and medically, leads to a significant decrease in the overall mortality rate due to head injuries [2, 13].

In addition, several studies, some of which have been carried out in cooperation between different centres, have quantified outcome to ascertain whether therapies can significantly modify mortality and morbidity rate in such patients [9, 10, 12, 17]. Some authors have taken into account particular groups of patients children [5–7], or decerebrating patients [4, 14]. Comparison of reported results may entail difficulties on account of different ratings of coma used by neurosurgeons to define their patients. Langfitt [11] has advocated the adoption of the Glasgow Coma Scale (GCS) [18] and the Glasgow outcome assessment [8]. On these grounds, we have classified our patients in the same way, even though we do not entirely agree with the GCS, which disregards some important parameters such as pupil light reaction. As a matter of fact, in patients with no eye opening, no verbal responses, and decerebrate posturing, all of whom have a score of four points, outcome may be substantially different dependent upon whether pupil light reaction is still present or not. As far back as 1964 Bozza Marrubini [3] classified as coma 5 a level of coma in which the patients exhibit bilateral decerebrate posturing with preserved light reaction. Such a level of coma may be still reversible, while coma 6 (decerebrate posturing with no light reaction the *coma carus* of French authors) is irreversible after a few minutes of an acute compression. Even Teasdale *et al.* [19] agree that data concerning pupil light reaction must be included better to define decerebrate patients who presently score four points on GCS.

Our series differs in some respect from most series reported in literature. We have included only comatose patients admitted to our Intensive Care Unit (ICU). Most patients scoring on admission more than seven points as well as those patients with intracranial masses, chiefly epidural haematomas, who rapidly improved after removing the acute compression, have been excluded. Non-reacting patients with fixed pupils (three points) and patients lying in *coma dépassé* who were not admitted to the ICU were also excluded. On the whole, our report deals with patients with diffuse brain lesions or patients operated on for intracranial masses and remaining in a coma state after surgery. It conforms more closely to a subgroup of the patients reported by Jennett *et al.* [10]. Accordingly, as D. Becker [1] points out, we think that it would be unwise to try to derive conclusions concerning outcome of severely injured patients in general from this and other series which are the result of a selection according to particular criteria.

Clinical Material and Methods

Overall Data

In a five year period from 1974 to 1979, 1127 head injury patients were admitted to our Department, and 148 (13%) were operated on for mass lesions. The overall mortality was 23%, while mortality rate in the patients operated on was 33%. As in all series the highest mortality rate was observed in acute subdural haematomas (60%).

Criteria for Selecting Patients

On admission, all patients first undergo resuscitation procedures (intubation, assisted ventilation when necessary, infusion of fluids and blood, gastric drainage, etc.). Subsequently, non-invasive diagnostic procedures are immediately carried out (CT Scan, Echoencephalography). All patients harbouring intracranial masses suitable for surgical treatment are operated upon at once, with no additional monitoring.

In the ICU long-term ICP monitoring is set up in the following cases:

a) Patients with diffuse brain lesions who are still comatose after preliminary resuscitation in the emergency room. In most patients ICP recording is started as soon as they are admitted to the ICU. In a minority, monitoring is instituted when the clinical situation begins to deteriorate.

b) Patients with mass lesions who remain in a coma state after surgery.

ICP is recorded only in individual cases:

a) In non-reacting patients with bilateral dilated unresponsive pupils with no mass lesions and who show no improvement after emergency resuscitation.

b) In patients with too severe extracerebral lesions.

c) In patients with milder disturbances of consciousness.

d) In patients who improve quickly after surgery or preliminary resuscitation.

According to the above quoted criteria long-term ICP monitoring was performed in 124 cases. Other non-comatose patients with intracerebral mass lesions or CSF dynamics disturbances or both, who were monitored to get additional information for making surgical decisions, are not included in the present study. Of the 124 patients monitored 61 (49%) were admitted within 6 hours of injury. In 49 (39%) ICP recording was commenced on admission. Even in this group, the vast majority of the patients were referred to us by Community Hospitals; only a few were admitted directly to our Department from the site of the accident.

In 116 patients (93.5%) ICP monitoring was set up within 72 hours of injury. In 114 patients ICP was measured by Lundberg's intraventricular technique, and in the remaining 10 in the upper spinal space. The average duration of ICP recording was 6.5 days. All patients but two underwent respirator treatment: $PaCO_2$ was kept between 20 and 30 (25 ± 3.5) Torr. PaO_2 in all patients with unimpaired ventilation was >100 Torr. pH ranged from 7.40 to 7.55.

Osmotic drugs (Mannitol and Glycerol) were given in keeping with ICP values. Fluid intake restriction was carried out in seven patients. Barbiturates (3–5 mg/kg/24 h) were given recently in 17 patients. Steroids in either standard or high dosages were given only in the first phase of our experience. Since no obvious benefit in reducing elevated ICP and in improving clinical condition was observed and, as the incidence of complications (gastric haemorrhage, hyperglycaemia, hyperosmolality) appeared to be far from negligible, corticoids are no longer used. Therefore, the last 96 patients did not receive steroids.

CSF was withdrawn in 31 patients, and continuous ventricular drainage was set up in 9 patients, 4 of whom eventually underwent permanent surgical shunting.

In assessing ICP the highest values were taken into account; pressure elevations due to coughing, straining, or suction have been disregarded, as well as isolated and unexplained pressure waves and peaks.

Coma rating was assessed on admission. In some patients who deteriorated shortly afterwards, both admission and later scores are considered.

Summary of Patients

Overall Data

32 patients (26%) were found to have intracranial mass lesions (19 brain lacerations or intracerebral haematomas, or both, 9 acute subdural haematomas and 4 epidural haematomas). Surgical de-

Table 1. *Age of Patients*

Years	No. of patients
0–10	15
11–20	36
21–30	18
31–40	17
41–50	9
51–60	13
61–70	10
71–80	6

Table 2. *Coma Rating (Glasgow Coma Scale) on Admission*

Coma score points	No. of patients
4 to 5 points	84
6 to 7 points	26
Over 7 points	14

compression was carried out in 25 patients: 7 brain lacerations were not operated upon. 92 patients had diffuse lesions.

The age of the whole group is summarized in Table 1; the average age is 32 years.

Table 2 gives the coma rating on admission. It is worth noting that eight patients scoring six to seven points and three patients scoring more than seven points on admission deteriorated shortly afterwards to four points. Only one of these patients recovered.

Table 3 describes the incidence of bilateral decerebrate posturing and pupillary unresponsiveness.

Caloric vestibular stimulation was tested in 40 decerebrating patients. The results of this test were never normal: tonic deviation was observed in 34 patients, and no response in the remainder.

Table 3. *Clinical Features*

	On admission	Late deterior.	Total
Bilat. decerebrate post.	70 (56%)	11 (9%)	81 (65%)
Bilat. dil. unresp. pupils	23 (19%)	3 (2.5%)	26 (21.5%)

Table 4. *Quality of Outcome in the Survivors*

	g.r./m.d.	s.d./v.s.	Total
Mass lesions	8	4	12
Diffuse lesions	23	11	34

Table 5. *Late Extracerebral Complications as Cause of Death*

Pulmonary embolism	3
Renal failure	3
Pyogenic meningitis	2
Bronchopneumonia	1
Tracheal haemorrhage	1
Gastric haemorrhage	1
Sepsis	1

Overall Outcome

Forty-six patients (37%) survived, and 78 (63%) died. Of the survivors nine were operated on (four brain lacerations or intracerebral haematomas, or both, three acute subdural haematomas, and two epidural haematomas). Four additional patients with brain lacerations treated conservatively survived as well. Of the 33 survivors with diffuse lesions 2 were shunted early for CSF dynamics disturbances.

The quality of outcome in the whole group of the survivors is analysed in Table 4.

Of the 78 patients who died, 15 succumbed in the first 72 hours, 51 died 4 to 35 days (average 11.5) after injury and, finally, late extracerebral complications were responsible for death in the last

12 patients (average survival 31.5 days). Altogether 16 patients were operated on for mass lesions (7 brain lacerations or intracerebral hematomas, or both, six acute subdural haematomas, and two epidural haematomas). Three patients with brain lacerations treated conservatively died, two of them from late extracerebral complications.

Late extracerebral complications that caused death are listed in Table 5.

Incidence of Intracranial Hypertension

The overall values of ICP in the 114 in whom it was recorded intraventricularly are described in Table 6.

Table 6. *Highest Ventricular Pressure (114 Patients)*

Highest ICP value	No. of patients
0–20 mm Hg	30
20–50 mm Hg	59
Over 50 mm Hg	25

All the patients in whom ICP was recorded in the upper spinal space died (7 out of 10 within 72 hours). As far as spinal subarachnoid ICP measurement is concerned, three stages can be distinguished:

1. ICP is very high, approaching the levels that are measurable in the supratentorial compartment (60 to 70 mm Hg or even more). In this stage there is still communication between the supratentorial and the infratentorial compartment.

2. There is a clear-cut gradient between the two compartments. Brain tissue becomes increasingly impacted into the tentorial notch, so there is no longer communication between the posterior fossa and the supratentorial space. Infratentorial pressure ranges between 30 and 50 mm Hg, and pulsation is still clearly visible. Queckestedt's test is negative.

3. The tracing becomes flat and pulseless. Spinal pressure ranges between 10 and 15 mm Hg, which is the pressure in the isolated subarachnoid spinal space. In fact in this stage the tonsils become impacted into the foramen magnum, sealing the spinal space. This picture usually corresponds to brain death.

On admission two of our patients were in stage one and eight in stage two. In the terminal stage all exhibited the third type of tracing.

Results of P/V Tests

P/V relationship was studied by Miller's technique (introduction of a bolus of 1 to 2 ml of fluid into the ventricle) or by measuring the pressure response after discontinuing artificial ventilation (Papo and Caruselli 1978). When both tests were performed in the same patients, they yielded parallel results (Table 7). Obviously whenever, after discontinuing artificial ventilation there were no changes in $paCO_2$, ICP also remained unmodified.

Table 7. *Results of Discontinuing Artificial Ventilation*

No.	Age	Lesion	ICP	P/V test	Discont. vent.		Outcome
			level	Δ P	Δ $PaCO_2$	Δ P	
1	14	diffuse	10–15	+ 20	+ 3 torr	+ 40	died
2	42	ac. sub. haem.	20	+ 20	+ 4 torr	+ 40	died
3	16	br. lac. flap. removed	18	+ 2–3	+ 4.5 torr	+ 8	recov.
4	23	diffuse	25	+ 7	+ 3 torr	+ 15	died
5	23	diffuse	20	+ 2	+ 4 torr	+ 10	died
6	10	diffuse	18	+ 1	0	0	recov.
7	29	diffuse	18	+ 8	+ 6 torr	+ 20	veg. surv.
8	74	diffuse	10	+ 4	+ 5 torr	+ 10	died
9	61	ac. sub. haem.	35	+ 3	+ 2.5 torr	+ 8	died
10	20	diffuse	25	+ 20	+ 3 torr	+ 50	died

Pressure response after switching off the ventilator was tested in 41 patients (18 survived and 23 died).

Sixteen patients showed very sharp responses with high pressure peaks (over 20 mm Hg) and no spontaneous compensation. In this group six patients died. It is noteworthy that in five patients (one severely disabled and four dead) ICP baseline never exceeded 20 mm Hg. Minor pressure peaks, not exceeding 20 mm Hg, and spontaneous compensation were seen in 12 patients (5 survived and 7 died). Finally, in 13 patients (4 survivors and 9 dead) there were neither $paCO_2$ nor ICP changes.

Factors Influencing Outcome

Age: The correlation age/outcome is analysed in Table 8. We see that in the patients under 40 years of age, survival rate is 47% (good recovery 31% and severe disability/vegetative 16%), while the over 40 years survival rate is only 13% (good recovery 10%). Furthermore, the two oldest patients who made a good recovery scored more than seven points on admission.

It is worth noting that two children who remained severely disabled had sustained intracranial hypertension exceeding 50 mm Hg. The average age is 25.5 in the survivors, 38.7 years in the patients dying in the first 72 hours, 32.2 years in those who died later, and finally 48.1 years in the patients who succumbed to late extracerebral complications.

Table 8. *Age Versus Outcome*

Years	g.r./m.d.	s.d./v.s.	Death	Total
0–10	4	5	6	15
11–20	14	2	20	36
21–30	4	4	11	19
31–40	5	3	8	16
	27 (31%)	14 (16%)	45 (53%)	86
41–50	—	—	9	9
51–60	1	1	11	13
61–70	1	—	9	10
71–80	2 *	—	4	6
	4 (10%)	1 (3%)	33 (87%)	38

* More than 7 points on GCS.

Coma Scoring

Coma scoring *versus* outcome is described in Table 9, in contrast to Table 2, which defines the level of coma on admission. Eleven patients who had exhibited less severe disturbances of consciousness but deteriorated shortly afterwards despite active therapy are included in the first group (four to five points).

Extracerebral complications are responsible for death in eight out of nine patients scoring more than five points. In the last patient of this group a misdiagnosed mass lesion gave rise to a late acute intracranial hypertension. The patient had been previously monitored for five days: ICP never exceeded 15–20 mm Hg, and for this reason ICP recording had been discontinued two days prior to the sudden outburst of intracranial hypertension.

Brain-Stem Signs

Of the 95 patients scoring 4 to 5 points on GCS, 82 exhibited bilateral decerebrate posturing and 13 flexor responses. In the whole group mortality rate was 73%. Of the patients with flexor responses

2 made a good recovery, 1 remained severely disabled and 10 died (77%), whereas in the group of decerebrating patients 12 made a satisfactory recovery (15%), 11 remained severely disabled (13%), and 59 died (72%). Consequently, in our series, there was no significantly different outcome in deeply comatose patients, whether decerebrating or not. Bilateral dilated unresponsive pupils were observed in 26 patients—in 22 on admission and in the remaining 4 during peaks of intracranial hypertension. Twenty-two patients

Table 9. *Coma Score Versus Outcome*

Coma score	g.r./m.d.	s.d./v.s.	Death	Total
4 to 5 points	14 (15%)	12 (12%)	69 * (73%)	95
6 to 7 points	11 (65%)	2 (12%)	4 (23%)	17
Over 7 points	6 (50%)	1 (18%)	5 ** (42%)	12
	31 (25%)	15 (12%)	78 (63%)	124

* On admission 8 patients scored 6 to 7 points and 2 over 7 points.
** Dead of late extracerebral complications.

died, and four survived, three of them being severely disabled. The survivors deserve some comments: the first case was a boy of 16 years of age, in whom pupillary unresponsiveness lasted only a few minutes before the removal of an epidural haematoma. The second case concerns a boy of 10 years of age, in whom fixed mydriasis observed on admission was reverted by infusing Mannitol rapidly. The third patient, a 41-year-old man, harboured a huge haematoma in the basal ganglia: three days after injury ICP, whose baseline had ranged from 35 to 45 mm Hg, suddenly rose to 80 mm Hg. At that moment the pupils became dilated and unresponsive. This condition was reversed by infusing Mannitol rapidly, and then a bilateral subtemporal decompression was carried out right away. After surgery ICP dropped to 20 mm Hg and never rose thereafter, but no obvious clinical improvement ensued, for the patient remained in vegetative state. The last patient was a five-year-old boy who showed several episods of fixed mydriasis, reversed by Mannitol infusion. The patient was then given barbiturates (five mg/kg) for seven days, together with Mannitol. Continuous VD was also instituted. Intracranial hypertension was controlled, but the patient remained decorticate. All patients with sustained pupillary unresponsiveness died. In our series, therefore, *coma carus* or Bozza Marrubini's coma six always proved irreversible.

Vestibular responses were of little practical use in our patients. As a matter of fact, they were tested only in 40 decerebrating patients, and never yielded normal responses. All patients with "frozen eyes" died. Fourteen out of 34 patients with tonic deviation survived.

Intracranial Pressure

The ICP values *versus* outcome are reported in Table 10. On the whole, 29 patients (24%) died of uncontrollable intracranial hypertension: in 19, ICP was recorded in the lateral ventricle and in the remainder in the upper spinal space. The average survival in the whole group was 5.3 days. Only one patient died within 72 hours,

Table 10. *VFP Versus Outcome (114 Patients)*

Highest VFP	g.r./m.d.	s.d./v.s.	Death	Total
0–20 mm Hg	15 (50%)	3 (10%)	12 (40%)	30
20–50 mm Hg	13 (22%)	9 (15%)	37 (67%)	59
Over 50 mm Hg	34 (12.5%)	3 (12.5%)	19 (75%)	25

with ICP never exceeding 15 mm Hg. He was brought to our Department in deep coma with bilateral decerebrate posturing and bilaterally unresponsive pupils.

Six patients with sustained intracranial hypertension exceeding 50 mm Hg survived. They were boys of 5, 5, 9, 10, 14, and 19 years of age.

Three patients in whom barbiturate treatment was instituted early, made a satisfactory recovery, and three remained severely disabled.

We should say, however, that in all the survivors intracranial hypertension reached its peak beyond 50 mm Hg at least 72 hours after injury, whereas admission pressure within 12 hours of trauma was definitely lower, ranging from 20 to 35 mm Hg. Conversely, all patients admitted in the first 12 hours after injury with ICP exceeding 50 mm Hg died, most of them within 72 hours.

In 59 patients (48%) different levels of elevated ICP ranging from 20 to 50 mm Hg were observed. Twenty-three patients survived, and 36 died. In most patients who eventually died, ICP was not elevated when recording was started, but rose in the following days despite active therapy. Eight patients of this group died of late extracerebral complications.

Finally, ICP did not exceed 20 mm Hg in 30 patients (26%). Eighteen patients survived, and 12 died (4 from late extracerebral

complications). It must be acknowledged, nonetheless, that if we look at the criterion for assessing the lowest limit of intracranial hypertension suggested by D. Miller *et al.* (1977), we notice that only in eight patients (four survivors and four dead) did ICP never exceed 15 mm Hg. Thus, even in this group 73% of patients exhibited some degree of intracranial hypertension. Moreover, five patients in whom the ICP baseline remained below 20 mm Hg showed extremely high brain elastance on P/V tests. To evaluate the quality of survival, coma rating and ventricular fluid pressure (VFP) have been correlated in Table 11.

Table 11. *VFP and Coma Versus Outcome in the Survivors*

	g.r./m.d.	s.d./v.s.	Total
VFP 0–20 mm Hg			
4 to 5 points	9	1	10
6 to 7 points	5	1	6
Over 7 points	1	1	2
	15 (83%)	3 (17%)	18
VFP 20–50 mm Hg			
4 to 5 points	2	8	10
6 to 7 points	6	1	7
Over 7 points	5	—	5
	13 (59%)	9 (41%)	22
VFP over 50 mm Hg	3	3	6

Discussion

In assessing clinically the series of head injury patients, several parameters must be considered. The first one is a proper coma rating. The main criticism to be raised against the existing Coma Scales is that they do not take the factor "time" into due account. As a matter of fact, in none of the series reported in the literature is it clearly specified how long the patients remained in a given coma level. To the Glasgow group [10] a patient must remain in a coma state for at least six hours to be included in a series of severely injured cases. Nevertheless, no more data are supplied to define the duration of the most severe degree of impairment of consciousness, which represents the basic criterion for classifying the patients.

As far as outcome is concerned, a short-lasting decerebrate posturing, or even a flaccid coma with dilated unresponsive pupils before surgical removal of intracranial masses, as well as transient conditions reversed by omotic infusion, adequate ventilation, or CSF withdrawal, have nothing to do with the same sustained conditions when emergency procedures have already failed. On these grounds, all overall surveys of miscellaneous series are necessarily misleading to some extent. Consequently, intensive care series, consisting chiefly of patients with well-defined and stabilized coma are quite different from emergency room series, in which several patients suitable for successful salvage procedures are included.

The same criticism holds true for ICP as well: pressure waves or short-lasting plateaus of hypertension, or both, are by no means comparable to sustained baselines at the same levels.

Moreover, the significance of severe intracranial hypertension is quite different according to whether it occurs just in the first hours after injury or in a later stage. In fact, the patients with no operable mass lesions and no respiratory disturbances who very early exhibit intracranial hypertension, exceeding 50 mm Hg, are likely to have irreversible lesions whereby most of them die in the first days. In such patients anti-hypertensive therapy is usually ineffective.

By contrast, if severe intracranial hypertension takes place later, after two three, or more days, the management and in particular barbiturates can often reduce raised ICP significantly even though outcome may be poor.

For the foregoing reasons, all the "homogeneous group of patients" are in reality much less homogeneous than they should be. Notwithstanding this, the mortality rate in our clinical material appears to be broadly in line with other reports. In our decerebrating patients mortality was 72%: it was 76% in Pagni's series [14] and 72% in Bricolo's [4]. In the last of Jennett's cooperative studies [9] the mortality rate for decerebrating patients is 75%. Twenty-four per cent of our patients died rapidly from uncontrollable intracranial hypertension. In the Richmond series [13] 22 patients out of 160 (14%) succumbed to fulminating intracranial hypertension.

If ICP monitoring is carried out long enough, we see that death caused by cerebral lesions without intracranial hypertension is very rare, and exceedingly rare if Miller's [13] criterion for evaluating the upper limits of normal ICP (10 mm Hg) is adopted. Early death with normal intracranial pressure is quite exceptional (only one patient in our material). It should not be overlooked that, in patients never exhibiting definitely elevated ICP, extracerebral complications are likely to play the most important role as the ultimate cause of

death. Furthermore, on the basis of the data supplied by P/V tests, it can be inferred that a good number of patients who are considered to have normal pressure have actually very high brain elastance. We share, therefore, Miller's [13] opinion that in several normotensive patients there is an impending risk of devastating intracranial hypertension that cannot be predicted by monitoring resting ICP. For this reason, new more precise and less dangerous P/V tests should be investigated, as Miller *et al.* [13] point out.

Forty-eight per cent of our patients showed different levels of intracranial hypertension ranging from 20 to 50 mm Hg. Thirty-six patients (29%) of the whole series) died. The role of intracranial hypertension in determining progressive deterioration should not be underestimated. Nevertheless, intracranial hypertension which often appears some days after injury and tends to rise despite therapy, might also be the result of increasing brain hypoxia brought about by severe widespread lesions. Extracerebral complications are also likely to play a definite role as additional causes, both of mortality and morbidity.

As regards outcome in the survivors, we see that functional results were significantly worse in the patients in deep coma with elevated ICP. Most comatose patients with normal or slightly raised ICP, but high elastance on P/V tests, remained severely disabled as well. Conversely, the significance of elevated ICP in patients with milder disturbances of consciousness did not appear so obvious. In fact, a number of patients tolerated high levels of intracranial hypertension surprisingly well. Some patients with large brain lacerations or intracerebral haematomas, or both, who are not included in the present report, showed incredible values of ICP (up to 60–70 mm Hg) with minor disturbances of consciousness. Intracranial hypertension abated immediately after limited surgical procedures, and all patients made a satisfactory recovery.

Consequently, it would appear quite clear, as previously stated by several authors, that the significance and prognostic value of intracranial hypertension are substantially different depending on whether the patients have well-circumscribed mass lesions or CSF dynamics disorders, or both, or widespread lesions associated with impaired functioning of the diencephalon and the brain stem. In the former patients the direct management, either surgical or medical, of intracranial hypertension, which comes to the foreground as the most important factor of morbidity, is usually successful. In the latter, instead, the ultimate outcome appears to depend to a greater extent on the lesions of the nervous parenchyma which are rendered more severe by coexisting intracranial hypertension.

From the therapeutic standpoint the results achieved in our series cannot be considered very impressive, inasmuch as only 26% of patients made a satisfactory recovery. Of course, better nursing would have prevented many extracerebral complications. From the most recent section of our series, however, it would appear that barbiturates prove definitely more effective in controlling acute intracranial hypertension than do previous therapies. It would seem rather unlikely that in an intensive care series like ours, consisting mainly of "second-hand" patients lying in deep stabilized coma, the results of therapy aiming at controlling intracranial hypertension can be improved significantly.

In actual fact, although all therapies may prolong survival, the overall mortality and morbidity cannot be reduced substantially if most patient continue to be referred to neurosurgical departments too late or after improper primary management. Even barbiturates may be of very little use if given to patients with uncontrolled long-standing intracranial hypertension and complicated brain lesions. By contrast, the results of therapy, either medical or surgical, can be improved to a great extent by preventing or properly managing early complications (hypoxia, hypercarbia, systemic hypotension, etc.) and by detecting acute intracranial hypertension in time.

The excellent results achieved by the Richmond group [2, 13], San Diego group [12], and Philadelphia group [5, 6] clearly demonstrate the paramount importance of a timely aggressive treatment of intracranial hypertension in head injuries.

To conclude, we firmly believe that the correct management of severe head injuries is above all a matter of adequate social organization, whereby the proper therapy can be commenced at the site of the accident.

References

1. Becker, D. P., Comment on Jennett's paper [9].
2. Becker, D. P., Miller, J. D., Ward, J. D., Greenberg, R. P., Young, H. F., Sakalas, R., The outcome of severe head injury with early diagnosis and intensive management. J. Neurosurg. *47* (1977), 491—502.
3. Bozza Marrubini, M. L., Resuscitation treatment of the different degree of unconsciousness. Acta Neurochir. (Wien) *12* (1964), 352—365.
4. Bricolo, A., Turazzi, S., Alexandre, A., Rizzuto, N., Decerebrate rigidity in acute head injuries. J. Neurosurg. *47* (1977), 680—698.
5. Bruce, D., Raphaely, R. C., Goldberg, A. I., Zimmerman, R. A., Bilaniuk, I. T., Schut, L., Kuhl, D. E., Pathophysiology, treatment and outcome following severe head injury in children. Child's Brain *5* (1979), 174—191.
6. Bruce, D., Schut, L., Bruno, L. A., Wood, J. A., Sutton, L. N., Outcome following severe head injuries in children. J. Neurosurg. *48* (1978), 679—688.
7. Gruskievicz, J., Doron, Y., Peyser, E., Recovery from severe cranio-cerebral injury and brain-stem lesion in childhood. Surg. Neurol. *1* (1973), 197—201.

8. Jennett, B., Bond, M., Assessment of outcome after severe brain damage. A practical scale. Lancet *1* (1975), 480—484.
9. Jennett, B., Teasdale, G., Braakman, R., Minderhoud, J., Heiden, J., Kurze, T., Prognosis of patients with severe head injuries. Neurosurgery *4* (1979), 283—289.
10. Jennett, B., Teasdale, G., Gailbraith, Pickard, J., Grant, H., Braakman, R., Avezat, C., Maas, A. I., Minderhoud, J., Vecht, C. J., Heiden, J., Small, R., Caton, W., Kurze, T., Severe head injuries in three countries. J. Neurol. Neurosurg. Psychiat. *40* (1977), 291—298.
11. Langfitt, T. W., Measuring the outcome from head injuries. J. Neurosurg. *48* (1978), 673—678.
12. Marshall, L. F., Smith, R. W., Shapiro, H. M., The outcome with aggressive treatment in severe head injuries. Part I: The significance of intracranial pressure monitoring. J. Neurosurg. *50* (1979), 20—25.
13. Miller, J. D., Becker, D. P., Ward, J. D., Sullivan, H. G., Adams, W. E., Significance of intracranial hypertension in severe head injuries. J. Neurosurg. *47* (1977), 503—516.
14. Pagni, C. A., The prognosis of head injured patients in a state of coma with decerebrated posture. Analysis of 471 cases. J. Neurosurg. Sci. *17* (1973), 289—295.
15. Papo, I., Caruselli, G., Long-term intracranial pressure monitoring in comatose patients suffering from head injuries. Acta Neurochir. (Wien) *39* (1977), 187—200.
16. Papo, I., Caruselli, G., The effect on intracranial pressure of stopping controlled ventilation in patients with head injuries. Neurochirurgia *21* (1978), 157—163.
17. Pazzaglia, P., Frank, G., Frank, F., Gaist, G., Clinical course and prognosis of acute traumatic coma. J. Neurol. Neurosurg. Psychiat. *38* (1975), 149—154.
18. Teasdale, G., Jennett, B., Assessment of coma and impaired consciousness. Lancet *II* (1974), 81—84.
19. Teasdale, G., Murray, G., Parker, L., Jennett, B., Adding up the Glasgow Coma Score. Acta Neurochir. (Wien) Suppl. *28* (1979), 13—16.

Authors' address: I. Papo, M.D., Neurosurgery Division Regional General Hospital, I-60100 Ancona, Italy.