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Original articles

Monitoring in non-traumatic coma. Part I: invasive intracranial measurements

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SUMMARY The arterial blood pressure, intracranial pressure, and organ system failure scores were reviewed for 49 infants and children with non-traumatic coma from various causes. The neurological outcome was good in 21 patients, moderate in five, and poor in 23. There was no significant difference in maximum intracranial pressures between patients with a good outcome and those with a poor one, but patients with a poor outcome had significantly lower minimum cerebral perfusion pressures. During the period of admission 18 patients had cardiovascular failure, nine had renal failure, and two developed severe coagulopathy. Seventeen of the 19 patients in whom at least one of these systems failed died.

Our findings emphasise the diversity of illnesses associated with raised intracranial pressure in children and the number who develop multiple organ failure, and the values and limitations of using minimum cerebral perfusion pressure and the organ system failure scores as guides to severity of illness and prognosis.

In children and infants with acute non-traumatic cerebral insults, the intracranial pressure may be monitored invasively to give warning of impending brain tissue shifts[1] and to allow indirect assessment of cerebral perfusion. For clinical purposes the cerebral perfusion pressure was taken to be the difference between the mean arterial blood pressure and the mean intracranial pressure.

The value of reducing raised intracranial pressure and of maintaining an adequate cerebral perfusion pressure has been shown in studies of outcome in infections of the central nervous system, and cerebral ischaemia,[2] and Reye's syndrome.[3] In these, the minimum cerebral perfusion pressure was a better guide to prognosis than the peak or maximum intracranial pressure alone; a minimum cerebral perfusion pressure of about 40 mm Hg can be viewed as a watershed with poor neurological outcome below this pressure and likely good outcome above it.[3]

Overall severity and an estimate of probable outcome of the illness may also be assessed by scoring systems such as the clinical classification system[4] and the organ system failure criteria for infants and children.[5]

A review of a heterogeneous group of children with non-traumatic cerebral insults was undertaken to determine the influence of minimum cerebral perfusion pressure and maximum intracranial pressure on outcome, as well as the value of clinical severity scores in documenting progress and indicating outcome.

Patients and methods

In this retrospective study we have reviewed the experience in the paediatric intensive care unit at this hospital during the two years January 1985 to December 1986. Forty nine children presented with acute non-traumatic cerebral insults and had their intracranial pressures monitored as part of their management. There were 29 girls and 20 boys aged 0·06 to 13·06 years (median 3·3). Seventeen children had infections of the central nervous system, 14 had metabolic encephalopathies, seven had suffered ischaemic damage, and 11 had various other conditions (table 1).

The clinical classification grade[4] (table 2) assigned on admission and the observations and investigations made each day were reviewed, and all patients were categorised using the clinical classification system[4] and the organ system failure criteria for infants and children (table 3).[5] Data collected included seizures in the 24 hour period before or after admission and the presence of 'deep coma' (fixed dilated pupils, or a Glasgow coma score[6] of 5

Table 1 *Details of 49 patients studied*

Case no	*Age (years)*	*Sex*	*Diagnosis*	*Cerebral perfusion pressure*	*Intracranial pressure*	*Seizures*	*Deep coma*	*First EEG*	*Worst EEG*	*Outcome*
Infections of the central nervous system (n=17):										
1	0·87	Female	*H influenzae* meningitis	55	10	Yes	No	Ib	—	Poor
2	1·64	Female	*H influenzae* meningitis	55	15	Yes	No	I	I	Good
3	3·11	Male	*H influenzae* meningitis	74	26	No	No	I	I	Good
4	1·57	Male	*H influenzae* meningitis	40	35	No	No	I	II	Moderate
5	0·06	Female	Group B streptococcal meningitis	F	F	Yes	Yes	c	IIc	Died
6	2·15	Female	Meningococcal meningitis	5	65	Yes	Yes	III	III	Died
7	3·82	Male	Tuberculous meningitis	F	F	Yes	No	I	I	Died
8	1·53	Female	Tuberculous meningitis	32	18	No	No	III	III	Died
9	4·95	Female	Measles encephalitis	42	33	Yes	No	Ib	Ib	Good
10	7·27	Male	Epstein-Barr virus encephalitis	62	30	Yes	No	I	I	Good
11	3·08	Male	Viral encephalitis	75	15	Yes	No	I	I	Good
12	10·70	Male	Viral encephalitis	68	30	Yes	No	I	I	Good
13	3·50	Female	Viral encephalitis	55	35	No	No	N	—	Good
14	1·33	Female	Viral encephalitis	41	33	Yes	No	I	I	Good
15	11·10	Male	Viral encephalitis	38	45	Yes	No	I	I	Died
16	13·10	Male	Viral encephalitis	65	20	Yes	No	I	I	Good
17	0·61	Male	Viral encephalitis	73	17	Yes	No	N	I	Good
Metabolic illnesses (n=14):										
18	7·15	Female	Hepatic failure (hepatitis A)	48	33	No	No	I	I	Good
19	12·30	Female	Diabetic ketoacidosis	60	43	No	No	I	I	Good
20	6·67	Male	Propionic acidaemia	42	12	No	No	N	—	Moderate
21	0·49	Female	Methylmalonic acidaemia	42	22	No	No	I	—	Good
22	7·12	Female	Maple syrup urine disease	30	14	No	No	I	III	Died
23	4·37	Female	Maple syrup urine disease	45	50	Yes	No	I	I	Good
24	3·30	Male	Type I GSD hypoglycaemia	50	48	Yes	No	a	I	Good
25	1·07	Female	Postoperative hypoglycaemia	F	F	Yes	Yes	Ib	IIb	Died
26	5·49	Male	Postoperative hypoglycaemia	40	54	No	Yes	I	III	Died
27	5·66	Female	Reye's syndrome like illness	19	67	No	Yes	II	III	Died
28	1·47	Male	Medium chain acyl COA DH deficiency	50	55	Yes	No	Ib	I	Moderate
29	8·60	Female	Reye's syndrome	15	30	Yes	Yes	I	III	Died
30	11·90	Female	Reye's syndrome	64	21	No	No	I	I	Good
31	0·51	Male	Reye's syndrome—chicken pox	31	47	Yes	Yes	c	IIb	Poor
Ischaemic illnesses (n=7):										
32	1·96	Male	Near drowning	44	26	No	Yes	I	I	Poor
33	7·77	Female	Near drowning	54	29	No	Yes	II	IIb	Died
34	8·85	Female	Near drowning	61	23	No	No	N	—	Good
35	2·50	Female	Cardiac arrest—epiglottitis	63	22	No	Yes	III	III	Died
36	1·56	Male	Cardiac arrest—25% burns	20	55	No	Yes	II	III	Died
37	0·35	Male	Sudden infant death syndrome —near miss	38	28	Yes	No	c	I	Moderate
38	0·14	Female	Intraventricular haemorrhage	F	F	Yes	No	Nb	—	Moderate
Other illnesses (n=11):										
39	3·23	Female	Polyarteritis nodosum	40	76	Yes	No	I	III	Died
40	0·65	Female	Encephalopathy of unknown aetiology	59	20	Yes	No	IIc	IIc	Poor
41	0·83	Female	Staphylococcal empyema	46	26	No	No	N	—	Good
42	3·72	Female	Hypothalamic astrocytoma	57	30	No	No	ND	ND	Good
43	5·53	Female	Ventriculoperitoneal shunt blockage	55	22	Yes	No	Ib	—	Good
44	11·80	Male	Acute hydrocephalus	57	43	No	No	N	I	Good
45	10·20	Male	Hypertensive encephalopathy	F	F	Yes	No	III	III	Died
46	1·70	Female	Subarachnoid haemorrhage	30	50	Yes	No	II	II	Poor
47	1·45	Male	Encephalopathy—neuroblastoma	62	45	Yes	No	Ib	III	Poor
48	8·45	Female	Haemolytic uraemic syndrome	25	41	Yes	No	c	IIc	Died
49	3·52	Female	?Haemolytic uraemic syndrome	45	10	Yes	Yes	I	III	Died

ND=not done; F=failed monitoring.

or less not attributable to drugs) on admission. Electroencephalograms were graded according to the criteria given in table 4. The time between the first sign of depressed consciousness and the initiation of intracranial monitoring was also noted for each patient.

The intracranial pressure was monitored using a subarachnoid screw (Codman ICP system) in 33 patients, a fluid filled intraventricular catheter in 13, and a subdural transducer (Gaeltec pressure transducer model ICT/b) in three. The subarachnoid screw was generally used in the older children, the limitation being the thickness of the skull vault. The youngest child monitored with this technique was 6 months old. The ventricular and subdural systems were used at all ages. Morbidity was low, with no haemorrhages, and only one case of infection (with the ventricular catheter). The median duration of monitoring was three days (range 0·5 to 16). Arterial blood pressure using a transduced intra-arterial cannula was shown on the same bedside monitor as the intracranial pressure (Hewlett Packard patient monitor 78353A or 78354A). Intracranial pressure and blood pressure measurements were charted half hourly, and during unstable periods such as acute rises in intracranial pressure or falls in blood pressure, or both. The highest intracranial pressure and lowest cerebral perfusion pressure measurements in the first 12 hours of monitoring, as well as the maximum intracranial pressure and minimum cerebral perfusion pressure for the period of monitoring, were recorded for each patient either from the half hourly measurements or from the measurements during the acute episodes if they lasted more than two minutes.

These patients were managed similarly using standard nursing and therapeutic measures.[7 8] Specific nursing care included attention to body position with head tilt up at 25–30°, the avoidance of neck compression, and the maintenance of normal core temperature. Factors known to precipitate episodes of raised intracranial pressure such as endotracheal suction, physiotherapy, movement, and chest radiographs were anticipated and treated, first with hyperventilation, then with thiopentone. The aim of treatment was to reduce the intracranial pressure to 15 mm Hg or less and to maintain the cerebral perfusion pressure above 50 mm Hg. The protocol

Table 2 *Clinical classification system*[4]

Class I	Patient not requiring intensive care
Class II	Patient requiring overnight observation
Class III	Physiologically stable patient requiring intensive nursing and monitoring
Class IV	Physiologically unstable patient requiring intensive care from nurses and doctors and frequent reassessment and adjustment of treatment

Table 3 *Criteria for organ system failure for infants and children.*[5] *Failure for a system defined by meeting at least one of the criteria*

System and measurement	*Criteria*
Cardiovascular:	
Systolic blood pressure	<40 mm Hg* <50 mm Hg†
Heart rate	<50 or >220/minute* <40 or >200/minute†
Serum pH	<7·2 with normal $PaCO_2$
Cardiac arrest	
Continuous infusion of inotropic agent to maintain blood pressure or cardiac output, or both	
Respiratory:	
Respiratory rate	>90/minute* >70/minute†
$PaCO_2$	>8·6 kPa
PaO_2	<5·3 kPa in the absence of cyanotic heart disease
PaO_2 (mm Hg) divided by the oxygen tension in inspired air	<200 in the absence of cyanotic heart disease
Mechanical ventilation for longer than 24 hours postoperatively	
Neurological:	
Glasgow coma score[6]	<5
Fixed dilated pupils	
Haematological:	
Haemoglobin concentration	<50 g/l
White cell count	$<3{\cdot}0\times10^9$/l
Platelet count	$<20\times10^9$/l
Renal:	
Serum urea concentration	>18 mmol/l
Serum creatinine concentration	≥180 mmol/l in the absence of pre-existing disease
Dialysis	

*=less than 1 year old; †=1 year old or more.

Table 4 *Criteria for grading of electroencephalograms*

Predominant activity:	
N	— Normal or borderline for age and state
I	— Slow activity (0·5–3 cycles/second) or periods of low amplitude (<50 μV), or both, of less than five seconds duration
II	— Generalised low voltage activity (<50 μV)
III	— Electrocerebral silence (isoelectric)
Discharges:	
a	— Continuous and unchanging (status epilepticus)
b	— Isolated (focal or multifocal)
c	— 'Electrical storms' (runs of discharges changing in amplitude, rate, and morphology with shifting focal distribution)

included mechanical hyperventilation (the $PaCO_2$ being lowered to 3·3–4·0 kPa), paralysis, and sedation with pancuronium, diazepam, and morphine (each 0·1 mg/kg/dose), phenobarbitone (standard dose for age and weight), osmotherapy (to 300–310 mmol/l) with intravenous mannitol and frusemide, and drainage of cerebrospinal fluid through a ventricular catheter. Patients with labile raised intracranial pressure who did not respond to these measures were given thiopentone, either intermittently or by infusion. Restoration of a satisfactory cerebral perfusion pressure was achieved by increasing the mean systemic arterial blood pressure with infusions of colloid or continuous infusions of dopamine, or both. Fifteen patients required thiopentone, and six required dopamine.

Outcome of the survivors was assessed from outpatient reviews at this hospital or from questionnaires completed at referring units. The median time of assessment of the survivors was 8·0 months (range 1–24) after the initial illness. Neurological outcome was graded using a simplification of the outcome scale proposed by Seshia *et al* (table 5).[9]

Statistical analyses were by Student's *t* test, the Mann-Whitney U test, and Levin and Bertell's estimate of population attributable risk applicable to retrospective studies.[10]

Results

Twenty six of the 49 patients had a good or moderate neurological outcome, and 17 died during the acute illness. The remaining six children had severe neurological deficits.

Table 5 *Neurological outcome in survivors. The worse group is selected if there is a dissociation between the neurological and developmental states*

Group	*Neurological state*	*Development (handicap)*
Good:	Normal; seizures if present well (100%) controlled	None
	or	
	Minimal alteration of tone or deep reflexes; isolated cranial nerve palsy; mild weakness (grade 4) or ataxia; seizures if present fairly well controlled (99–75%)	Mild
Moderate:	Moderate weakness (grade 3) or ataxia; behaviour disturbance; cranial nerves severely affected; seizures moderately controlled (74–50%)	Moderate
Poor:	Severe weakness (<grade 3) or ataxia; tetraplegia; uncontrolled seizures (<50%)	Severe

INTRACRANIAL PRESSURE AND CEREBRAL PERFUSION PRESSURE MEASUREMENTS

All patients had received some treatment to reduce intracranial pressure before monitoring and the institution of full neurointensive care. When the patients who had good outcomes were compared with those who had poor outcomes, the interval between the first sign of depressed consciousness and the initiation of monitoring was significantly shorter in those who had a good outcome, with the respective range (median) being 0·0–1·0 (0·5) days and 0·5–5·0 (1·0) days, respectively, $p<0.05$. Complete data about monitoring were available in 44 patients, 40 of whom had intracranial pressures of 15 mm Hg or more at some time during monitoring.

In the first 12 hours of monitoring there was no difference between the highest intracranial pressure and lowest cerebral perfusion pressure in those with good, and those with poor outcomes. The means (SD) in the two groups were 22·1 (8·0) mm Hg and 27·6 (15·7) mm Hg (intracranial pressure) and 65·1 (11·8) mm Hg and 56·3 (22·2) mm Hg (cerebral perfusion pressure). For the duration of monitoring,

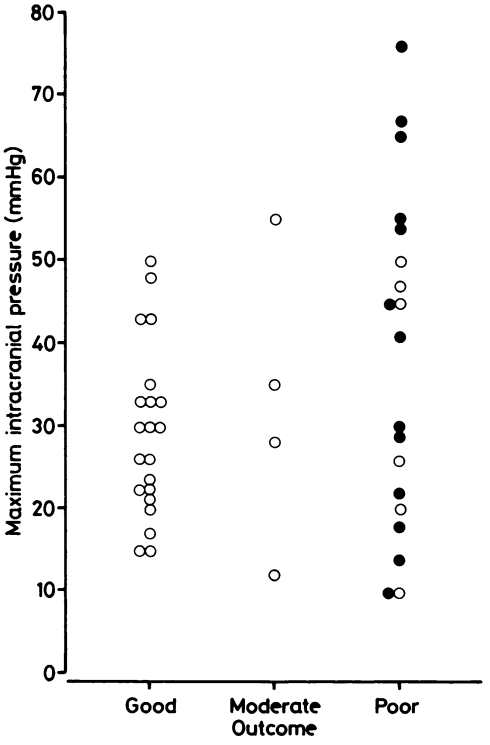

Fig 1 *Maximum intracranial pressure for the period of monitoring, and outcome in 44 patients with complete data; ●=patients who died during the acute illness.*

however, though the maximum intracranial pressure was similar in the two groups—29·3 (10·3) mm Hg and 38·1 (20·1) mm Hg (fig 1)—lower minimum cerebral perfusion pressures were found in those who had poor outcomes, 56·9 (10·6) mm Hg and 37·2 (16·6) mm Hg, respectively, $p<0·001$ (fig 2).

All patients who had good outcomes had minimum cerebral perfusion pressures that did not go below 40 mm Hg. Fourteen patients had perfusion pressures of 40 mm Hg or less, 12 of whom had a poor, and two a moderate outcome. There were nine children with minimum cerebral perfusion pressures in the range 38–42 mm Hg whose outcomes were distributed equally among the three groups (fig 2).

In seven patients who had poor outcomes, cerebral perfusion pressure during the period of monitoring was always greater than 42 mm Hg and we therefore reviewed the clinical features of these patients more fully. In two (cases 1 and 49) the maximum intracranial pressure during monitoring was below 15 mm Hg. Case 1 had had an episode of status epilepticus lasting at least one hour two days before monitoring. The clinical course of case 49 before monitoring was complicated by disseminated intravascular coagulation, hypoglycaemia, renal failure, and cardiac arrest. The remaining five patients with poor outcomes despite good cerebral perfusion pressures had maximum intracranial pressures above 15 mm Hg (range 20–45). Three of these (cases 32, 33, and 35) had been resuscitated after cardiorespiratory arrest before monitoring. Of the other two children, one (case 40) had had two diagnostic lumbar punctures with neurological deterioration after each, the second being performed 24 hours before admission. The other (case 47) had a maximum intracranial pressure of 45 mm Hg and a minimum cerebral perfusion pressure of 62 mm Hg. This apparently adequate cerebral perfusion pressure therefore reflected systemic hypertension rather than the intracranial state.

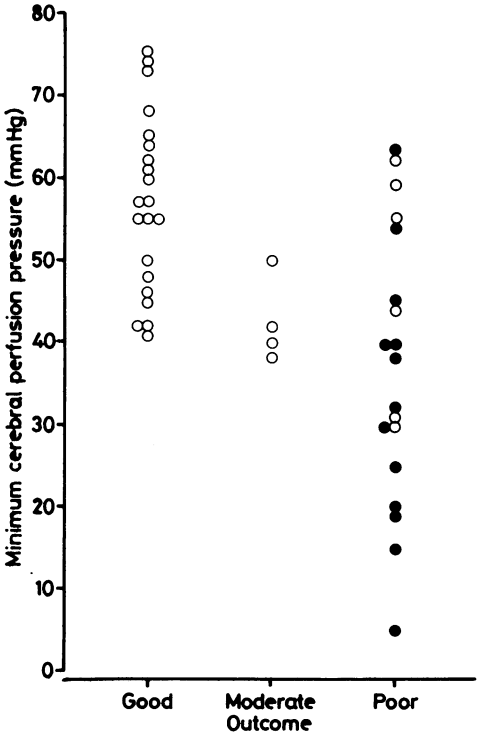

Fig 2 *Minimum cerebral perfusion pressure for the period of monitoring, and outcome in 44 patients with complete data. ●=patients who died during the acute illness.*

In summary, 12 of the 14 patients with minimum cerebral perfusion pressures of 40 mm Hg or less had poor outcomes compared with seven out of 30 patients with minimum cerebral perfusion pressures greater than 40 mm Hg. Review of the age, sex ratio, interval between the first sign of depressed consciousness and initiation of monitoring, diagnostic categories, and proportion with seizures either before or after admission, were similar when the patients with minimum cerebral perfusion pressures of 40 mm Hg or less were compared with those with minimum cerebral perfusion pressures greater than 40 mm Hg (table 6). Using Levin and Bertell's estimate of population attributable risk applicable to a retrospective study,[10] the estimated risk of poor outcome attributable to a minimum cerebral perfusion pressure of 40 mm Hg or less is $R_A=0·6$ (95% confidence interval 0·26 to 0·78), so assuming low cerebral perfusion pressure to be a major determinant of poor outcome, if minimum cerebral perfusion pressures of 40 mm Hg or less could be avoided, poor outcome could be reduced by between 26 and 78%.

SCORING SYSTEMS

On admission 44 patients (90%) were in the most severe category of the clinical classification system. The remaining five patients were in class III. The mortality during the acute illness of patients who were in class IV on admission was 36% (16 of 44).

Sedation, paralysis, and ventilation meant that the criteria for failure in the neurological system in the organ systems failure scoring system could not be assessed adequately and consistently in all patients for each day of admission. Those in deep coma, however, could be identified. On admission there were 12 such patients and all had poor outcomes. Measurement of intracranial pressure

Table 6 *Comparison of patients whose minimum cerebral perfusion pressures were >40 with those ≤40 mm Hg*

Details of patients	*Minimum cerebral perfusion pressure*	
	>40 mm Hg (n=30)	*≤40 mm Hg (n=14)*
Sex ratio (male:female)	13:17	6:8
Age (years):		
Range	0·49–13·06	0·35–11·1
Median	3·5	2·69
Diagnostic group:		
Infection of the central nervous system	11	4
Metabolic illness	8	5
Ischaemic illness	4	2
Other illness	7	3
Time from first sign of depressed level of consciousness to initiation of monitoring (days):		
Range	0–5·0	0·5–4
Median	1·0	1·0
Patients with seizures	16	8
Outcome:		
Good	21	—
Moderate	2	2
Poor	7	12

was unsuccessful in two of the 12, and in the other 10 the range and median of the cerebral perfusion pressure were 5–63 and 36 mm Hg, respectively.

All patients were ventilated during the course of their illnesses. A score for the respiratory system did not differentiate between those with true respiratory failure and those who were ventilated solely for treatment. The organ system failure scoring was therefore limited to the cardiovascular, renal, and haematological systems.

At the end of the first day of admission nine of the 49 patients had failure of at least one of these systems. During the period of admission the cardiovascular system failed in 18 patients, the renal system in nine, and the haematological system in two. Nineteen patients (39%) had at least one of the systems fail at some time during their admission, and of these 17 had poor outcomes.

Discussion

The diversity of illnesses associated with raised intracranial pressure has been recognised before,[1] and this review not only confirms this but also emphasises that many of these children have multiple organ failure. This finding is important, and contributes to the high mortality and morbidity.

The maximum intracranial pressure is an unreliable guide to prognosis as the difference between the groups with good and poor outcomes was not clinically relevant. Indeed, intracranial pressures greater than 40 mm Hg were found in three patients whose eventual outcomes were good. All children with intracranial pressures above 55 mm Hg, however, had poor outcomes (fig 1).

These data confirm previous reports[3] that the cerebral perfusion pressure is a better guide than the intracranial pressure to eventual neurological outcome, and values around 40 mm Hg seem to be critical. It must be noted, however, that in this series the values in the first 12 hours of monitoring were not significantly different between those with good and those with poor outcomes.

Despite the observation that none of the patients with a cerebral perfusion pressure below 40 mm Hg had a good outcome, there were patients with apparently adequate cerebral perfusion pressure who had poor outcomes. In those in whom there had been a delay between the initial cerebral damage and monitoring, and those in whom the damage was due to hypoxia or complex pathophysiology, the eventual outcome may have reflected the severity of the cerebral damage that occurred before monitoring.[11] In addition, patients with raised intracranial pressure and acute systemic hypertension had cerebral perfusion pressures that reflected the degree of hypertension rather than the intracranial state. It must also be emphasised that there were nine patients who had minimum cerebral perfusion pressures in the range 38–42 mm Hg. Neurological outcome in these patients was distributed equally in the good, moderate, and poor groups. This suggests that prognosis based on cerebral perfusion pressure alone is unreliable when values are in this borderline range.

This study has taken no account of time spent at different cerebral perfusion pressures or of the responsiveness of intracranial pressure to treatment. It may be that the differences in outcome could be further explained by these factors. Despite their limitations these retrospective data strongly suggest that neurological outcome in comatose children may be improved if cerebral perfusion pressure can be maintained above 40 mm Hg. This could be achieved by earlier recognition of children at risk of raised intracranial pressure, and rapid initiation of adequate monitoring in order to prevent episodes of poor cerebral perfusion.

Intracranial measurements can give an indication of intracranial fluid and tissue dynamics for the period of monitoring. They give no indication, however, of the severity of the cerebral insult and of cerebral function before and during monitoring. Non-invasive measurement of cerebral function in the paralysed and ventilated state may provide an

improved guide to prognosis, and the role of electroencephalography in these patients has been reviewed.[11]

The clinical classification system[4] was helpful in classifying patients broadly with respect to need on admission to the intensive care unit. Most, however, were in class IV and those most severely affected were not specifically identified. The organ system failure criteria for infants and children[5] was not entirely successful. It was limited because of the difficulty of neurological assessment for the purpose of comparative scoring in patients with changing degrees of paralysis and sedation, and differing degrees of cerebral damage. All the patients identified with either fixed dilated pupils or a Glasgow coma score less than 5 not attributable to drugs, however, had a poor outcome. The role of clinical neurophysiology in this context has also been discussed.[11] Furthermore, the finding of respiratory failure did not differentiate primary lung problems, true central respiratory depression and paralysis, or sedation and ventilation as part of treatment. Despite these limitations, failure in at least one of the systemic systems (cardiovascular, renal, or haematological) led to a more precise identification of those likely to have poor outcome. This was also a simple means of documenting deterioration or the need for increased support during the acute illness.

Though the early recognition of raised intracranial pressure is fraught with problems, we conclude that all patients with non-traumatic cerebral insult complicated by raised intracranial pressure or at risk of developing it, should be considered for measurement of intracranial pressure and invasive arterial monitoring at an early stage of their illnesses in order to prevent periods of inadequate cerebral perfusion pressure. In addition, the use of a limited organ system failure assessment provides valuable information both as a guide to prognosis and as a more objective assessment of severity.

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