

HIGHLIGHTING INTRACRANIAL PRESSURE MONITORING IN PATIENTS WITH SEVERE ACUTE BRAIN TRAUMA

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SUMMARY - Intracranial pressure (ICP) monitoring was carried out in 100 patients with severe acute brain trauma, primarily by means of a subarachnoid catheter. Statistical associations were evaluated between maximum ICP values and: 1) Glasgow Coma Scale (GCS) scores; 2) findings on computed tomography (CT) scans of the head; and 3) mortality. A significant association was found between low GCS scores (3 to 5) and high ICP levels, as well as between focal lesions on CT scans and elevated ICP. Mortality was significantly higher in patients with ICP > 40 mm Hg than in those with ICP ≤ 20 mm Hg.

KEY WORDS: head injury, intracranial pressure, coma.

Ressaltando a monitorização da pressão intracraniana em pacientes com traumatismo cerebral agudo grave

RESUMO - Monitorização da pressão intracraniana (PIC) foi adotada em 100 pacientes com traumatismo cerebral agudo grave, usando-se preferencialmente um catéter subaracnóide. Associações estatísticas foram avaliadas entre valores máximos de PIC e : 1) número de pontos na Escala de Coma de Glasgow (ECG); 2) achados na tomografia computadorizada (TC) da cabeça; e 3) mortalidade. Encontrou-se associação significante entre baixo número de pontos (3 a 5) na ECG e PIC elevada, assim como entre lesões focais na TC e hipertensão intracraniana. A mortalidade foi significativamente maior em pacientes com PIC > 40 mm Hg do que naqueles com PIC ≤ 20 mm Hg.

PALAVRAS-CHAVE: traumatismo cerebral, pressão intracraniana, coma.

Continuous monitoring of intracranial pressure (ICP) in humans was first proposed by Guillaume and Janny¹⁰. This landmark contribution was followed by extensive work of Lundberg on the same subject¹¹, where ICP patterns were more thoroughly established. Post-traumatic intracranial hypertension has been associated with increased morbidity and mortality¹²⁻¹⁴. When elevated ICP occurs, cerebral blood flow may decrease⁷. Conversely, increases in cerebral blood flow (cerebral luxury perfusion) may also significantly contribute to relevant ICP increments^{4,5,17}. In addition to cerebral hemodynamic changes, brain edema may also play a role in the pathophysiology of intracranial hypertension^{6,9}.

Routine ICP monitoring has been adopted at our institution since 1989. Over the years, our preference has been for subarachnoid catheter monitoring. This technique appears to satisfactorily comply with clinical requirements, particularly because it is practical, inexpensive, and carries just a small risk of complications. The present work is an outgrowth of our experience. Attempts were

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made to delineate statistical associations between elevated ICP and other parameters related to the diagnosis and management of severe acute brain trauma.

CLINICAL MATERIAL AND METHODS

During the period from June 1989 to April 1992, one hundred patients were prospectively evaluated in our Intensive Care Unit (ICU). Age ranged from 11 to 70 years, with 81% of the patients in the range of 11 to 40 years. There were 81 males and 19 females. All patients were comatose, with Glasgow Coma Scale²⁰ (GCS) scores of 8 or less on admission to the ICU. Automobile accidents represented the most frequent cause of injury (54%), followed by motorcycle accidents (17%) and other causes (29%).

All patients underwent mechanical ventilation and ICP monitoring. The latter was by means of a subarachnoid catheter in 89 of the patients, or a subarachnoid bolt in the remaining 11. Placement of the ICP monitor was in the ICU, at bedside, in most patients. Only if acute surgical lesions were diagnosed, ICP monitors were placed in the operating room, immediately after intracranial surgery. The catheter was filled with a cephalosporin. As to complications of this technique, only one patient (1%) developed cerebrospinal fluid infection which responded to therapy.

For classification purposes, ICP was considered normal if ≤ 10 mm Hg, mildly elevated if from 11 to 20 mm Hg, moderately elevated if from 21 to 40 mm Hg, and markedly elevated if > 40 mm Hg. Intracranial hypertension was managed according to a cumulative protocol to maintain ICP below 20 mm Hg². According to Cruz protocol, in addition to therapeutic ICP normalization, we have also recently attempted to maintain normalized cerebral extraction of oxygen (CEO_2)³⁻⁵ (arteriojugular oxyhemoglobin saturation difference). This additional management modality is because CEO_2 normalization represents adequate therapeutic match between global cerebral consumption of oxygen and cerebral blood flow³⁻⁵.

Statistical associations were evaluated between ICP and : 1) GCS scores on admission to the ICU; 2) predominant findings on the admission computed tomography (CT) scan of the head; and 3) mortality. Statistically assessed ICP values were the highest documented throughout the acute phase. Chi-squared test (with Yates' correction if necessary) was used for statistical evaluation, and a p value < 0.05 was considered significant.

RESULTS

There was a significant association between low GCS scores (3 to 5) and severe intracranial hypertension (Table 1). Likewise, there was a significant association between predominantly focal

Table 1. Intracranial pressure versus coma scores.

GCS	ICP			Total
	≤ 20	21-40	> 40	
3-5	14	12	24	50
6-8	20	18	12	50
Total	34	30	36	100

Distribution of the patients according to admission Glasgow Coma Scale (GCS) scores and maximum levels of intracranial pressure (ICP, in mm Hg). A significant difference was found ($p < 0.05$, chi-squared test).

Table 2. Intracranial pressure versus major CT findings.

Major CT findings	ICP			Total
	≤ 20	21-40	> 40	
Focal	22	25	32	79
Diffuse	12	5	4	21
Total	34	30	36	100

Distribution of the patients according to major findings on the admission computed tomography (CT) scans of the head and maximum levels of intracranial pressure (ICP, in mm Hg). A significant difference was found ($p < 0.04$, chi-squared test).

CT findings and markedly elevated ICP (Table 2). A significant association was also found between mortality and severe intracranial hypertension (Table 3).

Table 3. Intracranial pressure versus outcome.

Outcome	ICP			Total
	≤ 20	21-40	> 40	
Survival	30	22	10	62
Death	4	8	26	38
Total	34	30	36	100

Distribution of the patients according to two major outcome categories and maximum levels of intracranial pressure (ICP, in mm Hg). A significant difference was found ($p < 0.002$, chi-squared test).

DISCUSSION

ICP monitoring

Intracranial pressure monitoring in patients with acute traumatic coma has been advocated under a variety of circumstances. While Miller has previously proposed ICP monitoring in all comatose patients with acute brain injuries¹⁵, Cruz has recently reported ICP and CEO_2 monitoring in selected patients only, in whom the cerebrospinal fluid spaces were compromised. This excluded patients with normal CT findings, and those with pure diffuse axonal injury (without associated brain swelling)⁵.

ICP monitoring has also been addressed as a means of monitoring cerebral perfusion pressure (the difference between mean arterial pressure and intracranial pressure). In fact, this monitoring capability has been advocated as a guide for management of cerebral perfusion pressure, besides ICP^{14,15,18}.

Conversely, management of severely brain-injured patients has also been reported without ICP monitoring¹⁹. In this single report by Stuart and co-workers, the overall mortality rate was 34%. However, a comparative group of patients undergoing aggressive ICP monitoring and management was not evaluated, so that mortality rates were not thoroughly compared.

As to the monitoring technique, indications, limitations, and complications of different technical modalities have been previously addressed^{1,8,16,21}. Mollman and co-workers¹⁶ conducted a comparative study on ICP monitoring with a ventricular catheter, a subarachnoid catheter, and a subarachnoid bolt. In this study¹⁶, monitoring with a subarachnoid bolt tended to underestimate ICP increases, while no significant differences were found between ventricular and subarachnoid catheter monitoring. These findings add support to our routine adoption of subarachnoid catheter monitoring in most patients.

GCS scores and ICP

Our results disclosed a significant association between low GCS scores and markedly elevated ICP. These findings are in agreement with those of Yano and co-workers²¹. Whether elevated ICP contributed to neurologic impairment (low GCS scores) is not clear. In fact, according to our findings and those of others²¹, just associations were found. The latter, however, do not necessarily indicate cause-and-effect relationships.

CT findings and ICP

Our data revealed a tendency for patients with focal CT findings to have more pronounced ICP problems. This involved post-operative ICP recordings, and those in patients with non-surgical hemorrhagic contusions as well. It should be emphasized, however, that severe ICP problems may

also be found in patients with predominantly diffuse brain swelling⁴. Thus, our results do not rule out the relevance of ICP monitoring in patients with predominantly diffuse traumatic brain swelling; rather, they highlight the importance of ICP monitoring even in patients in whom acute intracranial hematomas have been emergently removed.

Mortality and ICP

Our findings of increased mortality in patients who developed markedly elevated ICP are in agreement with previous reports^{12,13,21}. Yano and co-workers²¹ have previously found a mortality rate of 93.4% in patients with ICP levels > 40 mm Hg (versus 72.2% in our series). These impressive figures have been found despite relatively sophisticated standards for intensive care and monitoring, and argue for additional research efforts on novel therapeutic modalities for adequate control of markedly elevated ICP in these critically ill patients.

CONCLUSIONS

From the above described, it is apparent that routine ICP monitoring in severely brain-injured patients should be taken into account, particularly in cases with CT scan evidence of compromised cerebrospinal fluid spaces. This would involve patients without surgical lesions, as well as selected cases in whom acute intracranial hematomas have been emergently removed.

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