

Intracranial Pressure is a Better Predictor of Mortality than Cerebral Perfusion Pressure

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ABSTRACT

Objective: To evaluate whether elevated intracranial pressure (ICP) or depressed cerebral perfusion pressure (CPP) is a better predictor of intracranial compartment syndrome and long-term functional outcomes in blunt traumatic brain injury.

Methods: This was a retrospective evaluation of data collected on 203 patients with blunt traumatic brain injury who were admitted to Miami Valley Hospital, a Level I trauma center, over a 2 years period, whose initial hospital management required an intracranial pressure monitor. Serial measurements of ICP and CPP were recorded during the patients' hospital stay. These patients were then evaluated at 3,6,12 and 24 months post-injury to assess their outcome based on functional status, as defined by death vegetative state, severe disability, moderate disability and good recovery.

Results: Utilizing an ICP cut-off value of 25 or greater and a CPP value of less than 60 at any point during the patients' hospital course, ICP elevation consistently correlated with a higher percentage of deaths and persistent vegetative state than a depression in CPP value. Outcomes as measured by severe or moderate disability were similar in both groups. However, neither measure approached statistical significance.

Conclusion: ICP appears to be a better predictor of intracranial compartment syndrome and extent of brain injury, predicting better than CPP values, the outcome of death or persistent vegetative state. This may help to predict prognosis, change management strategies and guide discussions with family, especially in the early phase of injury.

Keywords: Traumatic brain injury (TBI), Cerebral perfusion pressure (CPP), Intracranial pressure (ICP).

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RESUMEN

Objetivos: Evaluar si la elevación de la presión intracraneal (PIC) o la disminución de la presión de perfusión cerebral (PPC) es un mejor predictor de un síndrome compartimental intracraneano y los resultados funcionales a largo plazo en el trauma craneoencefálico contuso.

Métodos: Este fue un estudio retrospectivo de los datos recolectados de 203 pacientes con trauma craneoencefálico contuso que fueron admitidos al Hospital del Valle en Miami, un centro de trauma nivel I, a lo largo de un período de 2 años, cuyo manejo hospitalario inicial requirió un monitor de presión intracraneal. Determinaciones seriadas de la PIC y la PPC se registraron durante la estancia hospitalaria de los pacientes. Luego estos pacientes fueron evaluados a los 3,6,12 y 24 meses

después de la lesión para evaluar los resultados basándose en el estado funcional, tal como se define por el estado de muerte vegetativo, discapacidad grave, discapacidad moderada y buena recuperación.

Resultados: Utilizando un valor de corte de ICP de 25 o mayor y un valor CPP menor de 60 en cualquier momento durante la estancia hospitalaria del paciente, la elevación de la PIC siempre correlaciono con un mayor porcentaje de muertes y el estado vegetativo persistente que en cuanto a una disminución en el valor de la PPC. Los resultados, medidos como discapacidad grave o moderada, fueron similar en ambos grupos. Sin embargo, ninguna de las medidas se acercó a la significación estadística.

Conclusión: La PIC parece ser un mejor predictor para el síndrome de compartimiento intracraneal y extensión de la lesión cerebral, prediciendo mejor que los valores del PPC, el resultado de la muerte el estado vegetativo persistente. Esto puede ayudar a predecir el pronóstico, cambiar estrategias de manejo y guiar las discusiones con la familia, especialmente en la fase temprana de la lesión.

Palabras clave: Presión intracraneal (PIC), Presión de perfusión cerebral (PPC), Síndrome de compartimiento intracraneano, Síndrome hipertensión intracraneana, Trauma craneoencefálico (TEC).

INTRODUCTION

One of the most controversial areas of traumatic brain injury critical care is the management of cerebral perfusion pressure (CPP). CPP is the difference between the mean arterial pressure (MAP) and the intracranial pressure (ICP).¹ When pressure autoregulation is impaired and when CPP is below the lower limit of pressure autoregulation, cerebral blood flow (CBF) is dependent on CPP.²⁻⁴ It is important to emphasize that the controversial issue is not hypotension because overwhelming evidence from numerous clinical studies shows that hypotension has adverse consequences for the patient with TBI.²⁻⁴ Rather, the key controversial issues are what is the minimum level of CPP that is adequate for a brain-injured patient. Does increasing CPP beyond the minimum level, which provides adequate perfusion of the brain, have an additional beneficial therapeutic effect? Does increased levels of CPP provide any advantage over current management?⁵

In the United States, there has been a push toward protocol driven care.⁶ Our center is no different with a CPP protocol used in all patients with an intracranial pressure monitor (ICP). At times, however, in an attempt to provide adequate CPP levels mean arterial blood pressure were being

driven to abnormal levels. We hypothesized that increasing mean arterial pressure beyond normal values does not in fact increase CBF but may in fact be detrimental. The CPP is also a calculated number developed from two independent variables MAP and ICP. We felt that in patients with high ICP pushing CPP through increasing MAP would have at least no effect and may instead be deleterious. To answer this question we embarked on a study to determine if CPP or ICP is a better predictor of death.

METHODS

We performed a retrospective analysis of prospectively compiled traumatic brain injury database at a single level one-trauma center. Institutional review board permission was obtained prior to data collection. Data collected included demographic data, which included: Age, gender and race. Prehospital data including glasgow coma scale (GCS) EMS, Paralytic drug usage by EMS was collected. Initial resuscitation (ER) data collected included GCS ER, paralytic drug usage ER, lowest SaO₂, pupillary response, systolic blood pressure lowest ER. Intensive care unit (ICU) data collected comprised; ICP > 20 hours, ICP > 25 hours, Mannitol high 10 ICU days, length of stay (los) ICU, length of stay (LOS) total, mortality, injury severity score (ISS), abbreviated injury score head (AIS), outcome 3 months, outcome 6 months, outcome 12 months, outcome 24 months, hours until death, death within 48 hours, ventilator days, pneumonia, red blood cell transfusion, craniotomy, hours CPP < 60, basal cisterns midbrain, midline shift foramen, SAH basal cisterns, Intraventricular hemorrhage, multiple Parenchymal lesions, ICP > 25. Data was analysis by Chi-square and students t-test where appropriate.

RESULTS

The database included 203 patients who were available for analysis. The average age of the patients in this study was 45-year-old. The patients, in this study, where predominately male (76%) and chiefly Caucasian (87.7%). The average glasgow coma scale (GCS) at initial resuscitation was five with an average injury severity score (ISS) of 24. The abbreviated injury severity (AIS) for neurotrauma was four indicating traumatic brain injury was the most important injury in most of the patients in this study. The systolic blood pressure (SBP) averaged 112 systolic. Systolic blood pressure in this range would indicative adequate perfusion at the time of assessment (Table 1).

Intracranial pressure (ICP) greater than 25 mm Hg is generally accepted as representing the pressure which when reached requires some type of intervention. In patients who were found to have an ICP of greater than 25 on initial bolt

Table 1: Patient demographics and scores with blunt traumatic brain injury

<i>ICP > 25 two category</i>		<i>Mean</i>
Age	0 hours	45 ± 22
	1 or more hours	35 ± 18
GCS EMS	0 hours	4 ± 3
	1 or more hours	4 ± 2
GCS ER	0 hours	4 ± 2
	1 or more hours	4 ± 2
Lowest SaO ₂	0 hours	96 ± 4
	1 or more hours	96 ± 12
SBP lowest ER	0 hours	112 ± 27
	1 or more hours	105 ± 29
Injury severity score	0 hours	24 ± 8
	1 or more hours	26 ± 9
Abbrev injury score head	0 hours	4 ± 0.5
	1 or more hours	4 ± 0.5
Hours til death	0 hours	124 ± 22
	1 or more hours	137 ± 82
Ventilator days	0 hours	8 ± 10
	1 or more hours	13 ± 10
CPP < 60 hours	0 hours	9 ± 13
	1 or more hours	19 ± 18
<i>Gender</i>		
	<i>Frequency</i>	<i>Percentage</i>
Male	156	76.8
Female	47	23.2
Total	203	100
<i>Race</i>		
	<i>Frequency</i>	<i>Percentage</i>
White	178	87.7
African American	22	10.8
Other	3	1.5
Total	203	100

placement the mortality rate was 29%. In patients who had ICP pressures of 25 or greater for up to 10 hours, the mortality was 21%. For patients with ICP above 25 for 11 to 50 hours the mortality rate was 50%. When ICP was elevated greater than 25 for more than 50 hours the mortality rate was 74%. (Table 2).

For cerebral perfusion pressure (CPP), there is no accepted length of time for study. This analysis we look at total time using time below CCP of 60 for 14 hours as our cut off. Comparisons where made of those patients who had a total time of CCP less than 60 less than 14 hours vs greater than 14 hours total time. This was done to separate early deaths from those who a more prolonged course. CPP of less than 60 was accurate in predicting death in the early 30% of the time. When CPP was less than 60 for greater than 14 hours the prediction of mortality was only 41% (Table 3)

Statistical analysis failed to show any significance however the trends would indicate that ICP is a better predictor of mortality than CPP (Tables 4 and 5).

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Table 2: ICP > 25 outcomes

			Outcome 3 months					Total	
			Dead	Vegetative	Severe disability	Moderate disability	Good recovery	Lost to follow-up	
ICP > 25 four category	0 hours	Count	24		4	11	19	23	81
		% within ICP > 25 four category	29.60%		4.90%	13.60%	23.50%	28.40%	100%
	1 to 10 hours	Count	11	2	8	20	7	5	53
		% within ICP > 25 four category	20.80%	3.80%	15.10%	37.70%	13.20%	9.40%	100%
	11 to 50 hours	Count	23		5	6	3	9	46
		% within ICP > 25 four category	50%		10.90%	13.00%	6.50%	19.60%	100%
	More than 50 hours	Count	17	2	1	1	2		23
		% within ICP > 25 four category	73.90%	8.70%	4.30%	4.30%	8.70%		100%
	Total	Count	75	4	18	38	31	37	203
		% within ICP > 25 four category	36.90%	2%	8.90%	18.70%	15.30%	18.20%	100%
	Total patients 203								

Table 3: CPP group

			Dead	Vegetative	Severe disability
CPP group	CPP < 60 for < 14 hours	Count	22	2	10
		% within CPP group	30.10%	2.70%	13.70%
	CPP < 60 for 14 or > hours	Count	29	1	7
		% within CPP group	40.80%	1.40%	9.90%
Total	Count	51	3	17	
	% within CPP group	35.40%	2.10%	11.80%	
Total patients 203					

Table 4: ICP > 25

			3 month outcome—2 category		
			Dead	Alive	Total
ICP > 25 two category	0 hours	Count	24	34	58
		% within ICP > 25 two category	41.40%	58.60%	100.00%
	1 or more hours	Count	51	57	108
		% within ICP > 25 two category	47.20%	52.80%	100.00%
Total	Count	75	91	166	
	% within ICP > 25 two category	45.20%	54.80%	100.00%	
Chi-square tests					
Pearson Chi-square value 0.520					

Table 5: CPP < 60

			<i>Dead</i>	<i>Alive</i>	<i>Total</i>
CPP group	CPP < 60 for < 14 hours	Count	22	39	61
		% within CPP group	36.10%	63.90%	100.00%
	CPP < 60 for 14 or > hours	Count	29	33	62
		% within CPP group	46.80%	53.20%	100.00%
Total		Count	51	72	123
		% within CPP group	41.50%	58.50%	100.00%
Chi-square tests					
Pearson Chi-square value 1.453					

DISCUSSION

The pathophysiology of traumatic brain injury remains controversial. In an attempt to improve outcomes many techniques have been attempted. One approach is based on physiologic concept called the vasodilatory cascade. According to this hypothesis, a reduction in CPP—either a decrease in arterial blood pressure, an increase in ICP, or both—stimulates the cerebral vessels to dilate in an attempt to maintain CBF.⁵⁻⁹ This is the normal pressure autoregulatory response to a decrease in CPP. The increase in cerebral blood volume that accompanies the vasodilation further reduces CPP by increasing ICP. This cycle may lead to ever reducing CPP.⁹⁻¹¹ An increase in arterial blood pressure under this circumstance has been observed to break the cycle and reduce ICP. A detailed description of this approach is given in a recent report of a clinical. In this series of 158 patients admitted with glasgow coma scale score less than 7, mortality was only 29%, and 59% achieved a good recovery or moderate disability at 6 months.² There was believed to be sufficient value in this practice that it was included in the 1996 Head Injury Guidelines. There has been wide acceptance of this approach. In fact our centers protocol has been driven by this thesis.

Another recent approach, called the Lund therapy, emphasizes reduction in microvascular pressures to minimize edema formation in the brain. The goals of this approach are to preserve a normal colloid osmotic pressure (infusion of albumin and erythrocytes), to reduce capillary hydrostatic pressures by reducing systemic blood pressures (metoprolol and clonidine), and to reduce cerebral blood volume by vasoconstricting precapillary resistance vessels.¹⁶

Another more recent approach has been to treat traumatic brain injury as compartment syndrome. This has led to liberal use of ventriculostomy, as well as early decompressive craniotomy to control ICP. Fluid and pressure agents as adjuncts to maintain MAP to appropriate levels and ICP by decompression of either fluid or restrictive space.¹³⁻¹⁵

The approach in this study emphasized the use of fluids as well as pressure agents (neosynephrine) to maximize CPP. Management of ICP was limited to medical therapy including mannitol, sedation and phenobarbital coma. Frequently this approach when ICP is greater than 25 mm Hg leads to MAP of 90 to 100 mm Hg in order to maintain an CPP of 60. MAP in these ranges may be counter productive. Although our data does not demonstrate any deleterious effects there was no improvement either.¹²

There are several shortcomings in his study. This study is a retrospective study and inherently is biased. Despite protocol driven care driving MAP to artificially high values may have been counter productive and exacerbated penumbral areas of injury. ICP is part of the CPP calculation making ICP an contributing factor in analysis of effects of CPP. Finally, although we saw trends indicating ICP is a better predictor of mortality than CPP.

Despite these shortcomings, the data presented in this study would support the view that severe TBI produces a compartment syndrome. Efforts to drive CPP in order to break the cycle of vasoconstriction are not supported by our data. We would recommend all endeavors to decrease ICP including ventriculostomy and decompressive craniotomy should be utilized while maintaining normal MAP values.

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