Original Article

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Role of Invasive ICP Monitoring in Patients with Traumatic Brain Injury: An Experience of 98 Cases

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Abstract: The outcome of patients with head injury depends upon several factors, and most important among them is the raised intracranial pressure. ICP monitoring using subdural Richmond bolt system was used in 98 cases of TBI. The age range was 9-75 years; there were 78 adults and 20 children. This included 52 cases of severe HI and 46 cases with moderate HI.The GCS range was 4-10(mean: 5.6). Mortality was 64% in severe HI (34/52) and 9.5% in moderate HI patients (4/42). ICP ranged from 6-28mm Hg in moderate HI (mean 11.6 mm) and 8-42mm Hg in severe HI (mean 19.6 mmHg). Of 98 cases of HI, 41(41.8%) patients were operated (26 had moderate HI and 15 cases had severe HI. Seventy-three (74.5%) cases had single/multiple contusions in frontal/temporal regions while 25/73(25.5%) patients had diffuse axonal injury (10 cases) and thin subdural hematoma (15 cases). Fifty-seven patients were managed conservatively with decongestants, elective ventilation and continuous ICP monitoring for 3 days. Of these, 16(43%) had moderate HI and 41(71%) cases had severe HI. In the absence of intracranial hypertension (ICP <10 mm Hg), good recovery (Glasgow outcome scale: 4-5) was noted in 8/57(14%) cases in conservative group and in 4/41(9.7%)cases in operative group. In patients with ICP in 11-20 mm range, good recovery was noted in 6/41(14.6%) patients in operative group and in 9/57(15.8%) patients managed conservatively. When the ICP was >20 mm Hg, 10/41(24.3%) operated patients expired while only 6/57(10.5%) patients in conservative group expired. ICP malfunction after day 1 was noted in 27 cases ((27.5%) while ICP site infection/CSF leak was noted in 8 cases (8.2%). Outcome was related to severity of head injury and intracranial hypertension. Outcome was better in patients of moderate HI after surgery than in severe HI patients. Mortality was higher in patients operated in the presence of intracranial hypertension than in those managed conservatively. ICP monitoring was associated with a high blockage (27.5%) and infection rate (8.2%). ICP monitoring improved overall outcome in moderate head injury patients.

Keywords: intracranial pressure monitoring, head injury, outcome, Glasgow outcome scale, and Richmond bolt

INTRODUCTION

Intracranial pressure (ICP) is a reflection of the relationship between alterations in craniospinal volume and the ability of the craniospinal axis to accommodate added volume[1]. The outcome of patients with head injury depends upon several factors, and most important among them is the raised intracranial pressure. An increasing body of evidence supports the concept that intracranial pressure (ICP) slow B waves represent the autoregulatory response of spontaneous fluctuations of cerebral perfusion pressure (Figure1-3). A relationship between cerebral autoregulation and clinical outcome in patients with traumatic brain injury has been established[2]. Moreover, raised ICP is the most common cause of death, in patients with head injury. Uncontrolled intracranial hypertension produces secondary damage by reducing cerebral blood flow. However, surprisingly routine ICP monitoring in head injury still continues to be a subject of controversy[3-8]. Lundberg[8] was the first person to introduce ICP monitoring. Today, ICP monitoring has gained a place in neurosurgical ICU management and become an integral part of ICU care. ICP monitoring is extensively studied in head injury patients in last three decades[3, 5,6,9-11] either to establish its practical value for therapy or as a prognostic factor[6, 7,11-12]. Several studies linked high level of ICP with high mortality and morbidity. Persistent raised ICP has a direct effect on the brain tissue inspite of cerebral perfusion pressure being normal. ICP monitoring is routinely carried out in severe head injury patients, patients with good coma scale, CT showing multiple small hematomas not meriting surgery, patients with good coma scale with single large intracranial hematoma diagnosed on CT scan, patients with diffuse

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brain swelling those who need aggressive management and for postoperative ICP monitoring following evacuation of hematoma. Till 1996, there were 146 articles published on ICP monitoring in head injury, 41% publications on head injury and ICP monitoring and of these 27 articles have dealt with indications for ICP monitoring. The correlation between high ICP and poor outcomes has been well reported by many authors[4, 8,10,11-15.] Narayan et al[6] reported 53-63% raised ICP in severe closed head injury patients with abnormal CT scan while patients with normal CT scan had 13% incidence of raised ICP. The risk of raised ICP in moderate head injury is 10-20% especially more so in the presence of mass lesion.

Most studies recommend ICP monitoring for 48-72 hrs, as the intracranial hypertension is maximum between 48-72 hours following traumatic recording not only helps in guiding therapy, but also helps in predicting the outcome.

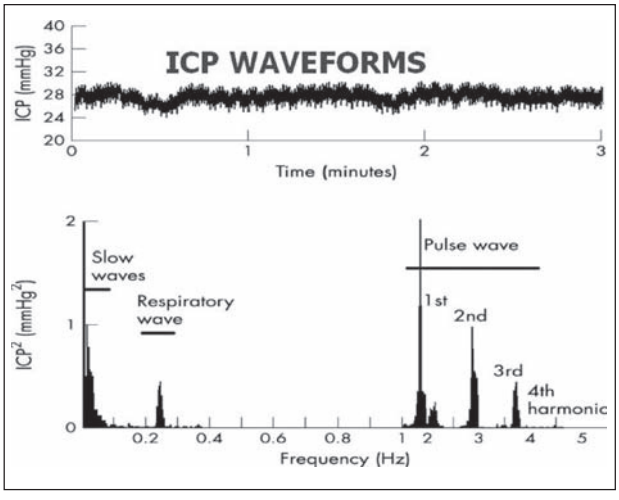

Fig 1: ICP recording showing pulse, respiratory and 'slow waves' overlapped in time domain.

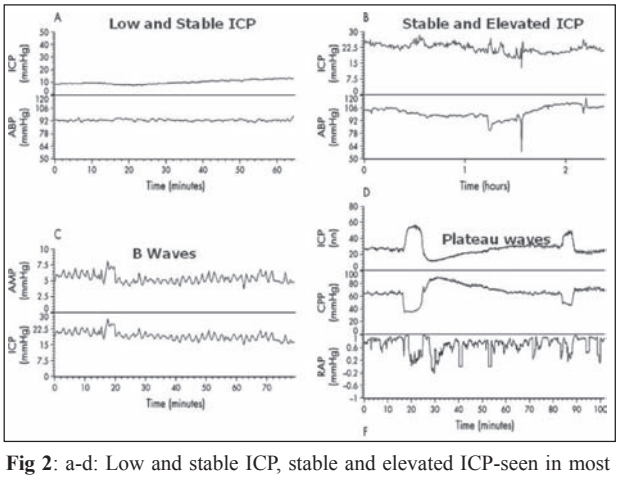

Fig 2: a-d: Low and stable ICP, stable and elevated ICP-seen in most of the time in head injury patients

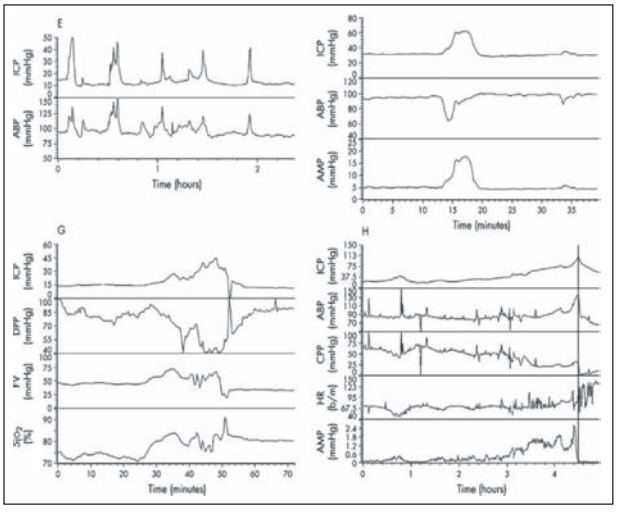

Fig 3 (top right, top left, bottom right, bottom left image): High spiky waves of ICP caused by sudden increases in ABP, increase in ICP caused by temporary decrease in ABP, increase in ICP of 'hyperemic nature'.

MATERIALS AND METHODS

In this study, 98 cases of traumatic brain injury admitted to our centre between 2001-2004 and subjected to intracranial pressure monitoring using Richmond bolt subdurally were evaluated (Figure 4). The age range was 9-75 yrs (78 adults and 20 children; 80 males and 18 females). There were 52 cases of severe head injury (GCSd"8) and 46 cases with moderate head injury with (GCS: 9-12) [GCS range: 4-10, mean GCS was 5.6]. Forty-one patients (41.8%) were subsequently operated (26 patients had moderate head injury and 15 had severe head injury) and 57 patients (58.2%) were conservatively managed with decongestants/ ventilatory support and continuous ICP monitoring using subdural Richmond bolt system for 3 days. Twenty of these had moderate head injury and 37 had severe head injuries. Of the patients who were managed conservatively, 24/

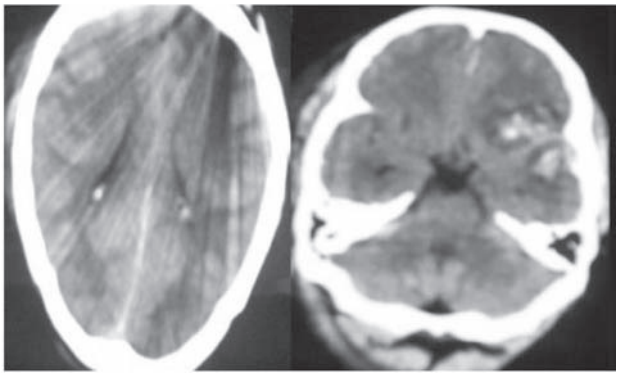

Fig 4: Clinical picture of a case of a severe head injury with ICP monitoring being shown on the right side of the image.

37(64.8%) of severe HI while 2/20(10%)of moderate HI expired. Of the operated group, 10/15(66.6%) of severe HI expired, while only 2/26(7.7%) of moderate HI in operated group expired. Thirty-eight (38.7%)patients expired, of these 34/52(65.4%) had severe HI and 4/42(8.7%) had moderate HI.

Radiological assessment

Contusions were noted in 73/98 patients (74.4%), of these single contusions were seen in 28/73(38%) while the remaining 45 cases (62%) had multiple contusions on initial CT scanning. Frontal contusions were noted in 40/73(54.8%) patients, of these 20 cases had single contusions while the remaining 20 cases had multiple contusions. Temporal contusions were noted in 33/73(45.2%) cases, of these 5/33(15.1%) patients had single contusions while 25/33(84.9%) had bilateral temporal contusions. CT scan was carried out within 1-2 hours on arrival to the hospital (within 2-24 hours of injury, mean 5.8 hours post injury in these patients). Diffuse axonal injury was noted in 10 cases (10.2%), amongst these 4 cases had DAI grade 3 and 7 cases had DAI grade 2 (Marshall grading). Fifteen cases had thin subdural hematoma/traumatic subarachnoid hemorrhages and were subjected to elective ICP monitoring intiallly. Repeat CT scanning was done in all patients 12-24 hours after the initial CT scan or earlier in case of neurological deterioration. (Figure 5-6)

Richmond screw ICP bolt was put subdurally under local anesthesia when a decision for conservative management and to electively monitor/ventilate the patients were taken. After head shaving and cleaning, Richmond bolt was put subdurally after making a twist drill burr hole at a point 1.5cm anterior and 3 cm lateral to the midline. Dura was incised by giving a cruciate incision and edges were coagulated and Richmond bolt was put subdurally and

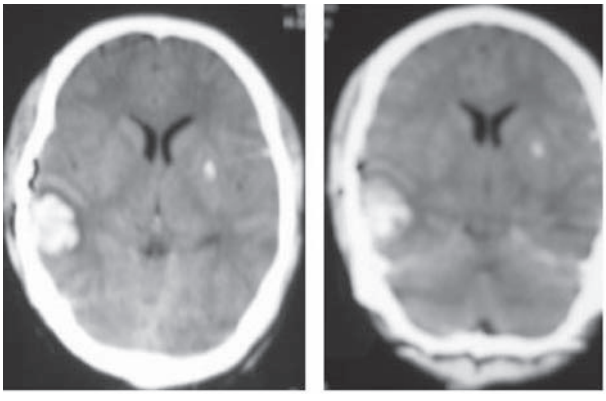

Fig 5: NCCT head of a patient with left temporal contusion with ICP monitoring. Patient subsequently required operation in view of intractable intracranial hypertension

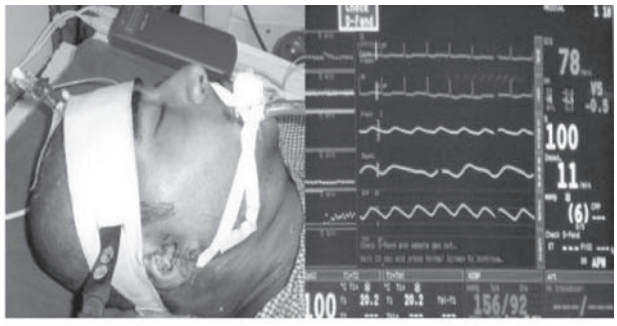

Fig 6: NCCT of a patient with temporal contusion, ICP remained low and patient made good recovery

connected to a monitoring set and ICP was subsequently monitored. ICP monitoring was done for 0-2 days in 60 patients in the present series (in 27 of these ICP catheter got blocked after initially functioning for 12-24 hrs), 3-4 days in 26 patients and >4 days in 2 patients. All patients had elective ventilatory care and received decongestants (intravenous mannitol and frusemide) and underwent CT scanning in the event of clinical deterioration/persistent intracranial hypertension (>20 mm Hg). There was no technical failure in any patients and good waveforms and opening pressure readings were obtained in all patients. Intracranial pressure ranged from 6-28 mm Hg in moderate head injury patients and 8-42 mm Hg in severe head injury patients. Intracranial pressure was <20 mm Hg in 35/52 (65.4%) severe head injury patients and in 38/42(90.4%) of moderate head injury patients and this difference was found to be significant in the two groups. Intracranial pressure of >20 was noted in 17/52(34.4%) cases of severe head injury and in 8/42(9.5%) cases of moderate head injury and this difference was significant. Higher intracranial pressure was noted in severe head patients while majority of moderate head injury patients were having ICP <20 mm Hg. However, worth noting is the fact that 26/42(62%) moderate head injury patients were operated while only 15/52(28.9%) severe head cases were operated. ICP monitoring in mod HI therefore helped in early decision making in mod HI patients and improved outcome in this group. ICP site infection and CSF leak was seen in 8 (8.2%)patients. (Table 1)

ICP and CT correlation

Intracranial hypertension (>20 mmHg) was noted in 14/73(19.7%) patients with contusions (single frontal contusion in 3, bifrontal contusion in 6 and bilateral/multiple temporal contusions in 5 cases) and in 11/25(44%) patients without any contusion on initial or repeat CT scanning. In the presence of mass effect/cisternal effacement, intracranial hypertension was noted in 22/76(28.9%) cases. Fifty patients (50/56: 65.8%) in the

presence of cisternal effacement on initial CT had ICP in the range of 11-20 mm Hg while 4/76(5.3%) had normal ICP values even in the presence of mass effect/cisternal effacement (catheter got blocked in all these patients in 6-24 hrs period and had to be removed subsequently, all these cases were operated subsequently for expanding hematoma in frontal lobe 3 patients and in temporal lobe in 1 patient). Eight of 10 cases of diffuse axonal injury had intracranial hypertension and required electively ventilatory support for over 3 days. Two patients with difuse axonal injury and 8 cases with thin subdural hematoma had ICP in 11-20 mm range and were managed conservatively. Good recovery was noted in only one case with normal initial ICP recording, remaining 24 cases with diffuse axonal /subdural hematomas had poor recovery (GOS 1-3). (Figure 7)

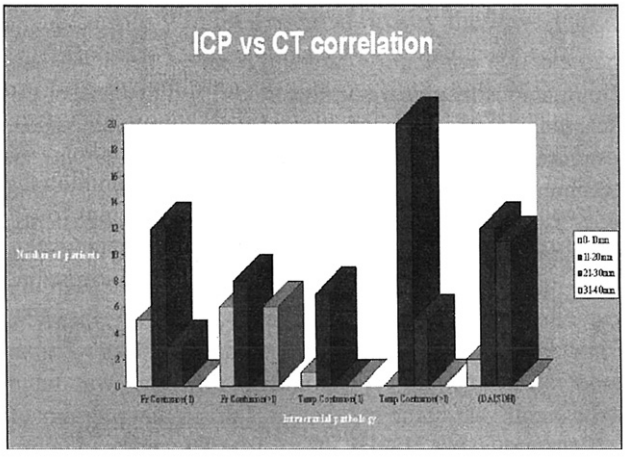

Fig 7: Bar diagram showing the correlation of contusions with degree of intracranial hypertension.

Glasgow coma scale and intracranial hypertension

Intracranial hypertension was noted in 17/52 (32.7%)cases of severe head injury and in 8/46(17.3%) cases of moderate head injury. Decerebration was noted in 15/17 patients when ICP was >10 mm Hg.There was no correlation between the intracranial pressure and the initial motor response of the patients(Figure 8).

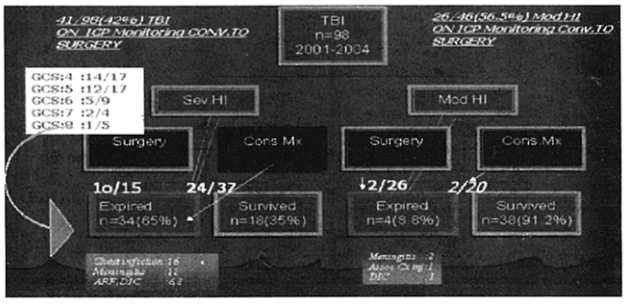

Fig 8: Flow chart of the patients studied for ICP monitoring.

Glasgow out come scale and intracranial hypertension

In the presence of intracranial hypertension, 16 patients expired (10 were operated and 6 were managed conservatively amongst these) while 8 had good recovery, 22 patients expired even when ICP was less than <20 mm Hg (9 of these were operated and 13 were managed conservatively). In the absence of intracranial hypertension, 22 patients expired (9 in surgical group and 13 in conservative group). There was no correlation of initial intracranial pressure (initial peak high ICP) recording with final neurological outcome. Intractable intracranial hypertension (ICP >20 mm Hg for more than 30 hour continuous ICP recording) was noted in 20 cases and of these 12 expired while no patient expired in the absence of intractable intracranial hypertension. In the absence of intracranial hypertension, good outcome (GOS of 4 or 5) was noted in 25/98(25.5%) cases while only 8/98(8.2%) had good outcome in the presence of intracranial hypertension and this difference was significant. (Figure9-12)

ICP RANGE(mmHG)	Moderate head injury Number(%)	Severe head injury Number(%)	Decerebrating Number(%)
0-10	9(20%)	5(9.6%)	2(11.5)
11-20	29(63%) >15mm:14(30.4%)	30(58%) >15mm:26(50%)	13(77)
21-30	4(8.5%)	15(29%)	2(11.5)
>30	4(8.5%)	2(3.4%)	0

Fig 9: Table showing correlation of degree of intracranial hypertension with severity of head injury.

Outcome in Surgical/Conservative group and ICP				
ICP (mmHg)	SURGICAL GROUP		CONSERVATIVE GROUP	
	GOS2-3	GOS4-5	GOS2-3	GOS4-5
0-10	0	4/41(9.7%)	0	8/57(14%)
11-20	5	6(14.6%)	19	9(15.8%)
21-30	2	4(9.7%)	1	0
>30	0	3(7.3%)	0	1(2.2)

Fig 10: Table showing Glasgow outcome scale correlation with ICP monitoring in surgical and conservative groups

GOS correlation with Peak ICP and Intractable ICP

Outcome Glasgow outcome scale	Peak ICP >20mmHg Number(%) N=27/98	Peak ICP <20mmHg Number(%) N=71/98	With Intractable ICP N=20	Without Intractable ICP N=7
GOS(4-5)Good outcome	8/98(8.2)	25/98((25.5)	1/20(5%)	5/7(71%)
GOS(2-3)Poor outcome	3/98(3.1)	24/98(24.5)	7/20(35%)	2/7(29%)
GOS(1) Dead	16/98(16.3) 10 surgical gp 6 conserv.gp	22/98(22.4) 9 surgical gp 13 conserv gp.	12/20(60%)	0

Fig 11: Table showing Glasgow outcome scale correlation with peak ICP and intractable ICP

GOS correlation with age and CPP

Outcome GLASGOW OUTCOME SCALE	Age>18yrs Number(%)	Age<18 yrs Number(%)	Lowest recorded CPP >60 mm Hg Number(%)	Lowest recorded CPP <60 mm Hg Number(%)
GOS(4-5)Good outcome	17/78(21.8)	16/20(80)	23/36(63.9)	10/62(16.1)
GOS(2-3)Poor outcome	25/78(32)	2/20(10)	5/36(13.8)	22/62(35.5)
GOS(1) Dead	36/78(46.2)	2/20(10)	8(22.2)	30(48.4)

Fig 12: Table showing Glasgow outcome scale correlation with age and cerebral perfusion pressure.

DISCUSSION

Intracranial pressure monitoring is reported to improve mortality from head injury. In 1977, Jennett et al[17] reported nearly 50% mortality in severe head injury. Subsequently other authors[3, 5,10-11] reported lower mortality with aggressive treatment. Aggressive management meant ICU care, ventilation and ICP monitoring. Saul and Ducker[18] treated two groups of severe head injury with mannitol and CSF drainage. In the patients in whom ICP was between 20-25mm Hg had 46% mortality as compared to 28% mortality in patients in whom ICP was 15mm Hg. In the current series, severe head injury was seen in 53% patients and mortality amongst these was 65%. Intracranial hypertension was noted in 17 severe head injury patients, 13/17(76%) cases expired. Normal intracranial pressure was observed in 35 cases and of these 21 patients expired (60%). Mahapatra et al[12] noted raised ICP in 65% of cases, overall 52% patients with raised ICP had good outcome and only 15% had good outcome in their series. In the current series, when ICP was >20 mm Hg, 16 patients expired while 8 had good recovery. Twenty two patients expired even when ICP was less than <20 mm Hg. There was no correlation of initial intracranial pressure (initial peak high ICP) recording with final neurological outcome. Intractable intracranial hypertension was noted in 20 cases and of these 12 expired while no patient expired in the absence of intractable intracranial hypertension. In the absence of intracranial hypertension, good outcome (GOS of 4 or 5) was noted in 25.5% cases while only 8.2% had good outcome in the presence of intracranial hypertension and this difference was significant. The determination of cerebral perfusion pressure is regarded as vital in monitoring patients with severe traumatic brain injury. Besides indicating the status of cerebral blood flow, it also reveals the status of intracranial pressure. The abnormal or suboptimal level of CPP is commonly correlated with high values of ICP and therefore with poor patient outcomes. Isa R et al studied outcome of severe traumatic c brain injury and compared three monitoring approaches: ICP alone, CPP and CBF and conservative methods during two different observation periods. The authors noted that only time between injury and arrival was statistically significant. There was a statistically significant difference in the proportions of good outcomes between the multimodality group compared with the group of patients that underwent a single intracranial based monitoring method and the group that received no monitoring based on a disability rating scale after a follow up of 12 months[19].

In Mahapatra et al[12] series, 65% of their patients had raised ICP. In the current series, intracranial hypertension was noted in 25/98(25.5%) patients only while majority of the patients had ICP in the range of 11-20 mm Hg (59/98:60.2%) in the current series. Mahapatra et al[12] reported normal ICP in 26% of their patients in the presence of mass lesion and 67% normal ICP when CT scan did not reveal mass lesion. In the current series, normal ICP in the presence of mass lesion was noted in 55/98((56.1%) patients while 10/98(9.8%) had normal CT and normal ICP recordings. The presence or absence of mass lesion did not correlated with mass lesion in the present series. Over all 52% patients with raised ICP had poor outcome and only 15% had a good outcome. Poor outcome in the presence of raised ICT was noted in 17/98(17.3%) patients and 8/98(8.2%) had good outcome even in the presence of intracranial hypertension in the present series. Contusions were noted in 74.5% cases. Single frontal contusion was seen in 20 cases, single temporal contusion in 8 cases. Multiple frontal contusions were noted in 20 cases while multiple temporal contusions were observed in 25 cases. Thin subdural hematoma/traumatic subarachnoid haemorrhage was noted in 15.3% cases.

Diffuse axonal injuries were seen in 10.2% cases, ICP more than 20 mm Hg was seen in 80% of these cases. No significant difference in the incidence of single/multiple and frontal/temporal contusions with the degree of intracranial hypertension was noted.

ICP monitoring for 0-2 days was done in 60 patients

(62%)(seventeen of whom had ICP malfunction after initially working for 12-24 hrs), for 3-4 days in 26 patients (26.5%) and for more than 4 days in 12 patients (11.5%). Our policy is to monitor ICP for 2-3 days and if patient improves or remains unchanged neurologically and/or CT scanning shows no increase in hematoma or new lesions. ICP range was 6-28 mmHg (Mean: 11.6) in moderate HI and 8-42 mm Hg (Mean: 19.6) in severe HI patients. ICP related problems included malfunction/poor or no waveforms in 17 patients (17.3%): infection of ICP port site/CSF leak was noted in 8 cases (8.2%). Narayan et al[4] noted infection in 6.3% cases and hemorrhage in 1.4% patients subjected to intracranial pressure monitoring. Mahapatra et al noted infections in 6% of their cases[10].

CONCLUSIONS

Outcome related to severity of head injury. Mortality was higher in patients operated in presence of intracranial hypertension than in conservative group. Moderate head injury patients may have raised ICP.ICP monitoring especially useful in selecting patients for early surgery in moderate HI group. Outcome was better in patients of moderate HI after surgery than in severe HI patients. Mortality was higher in patients operated in the presence of intracranial hypertension than in those managed conservatively. ICP monitoring was associated with a high blockage (27.5%) and infection rate (8.2%). ICP monitoring improved overall outcome in moderate head injury patients.

REFERENCES

1. Giuseppe Citerio, Peter J D Andrews. Intracranial pressure: Part two: Clinical applications and technology. *Intensive Care Med* 2004; 30:1882-5.
2. Kostas N Fountas, Arturas Sitkauskas, Carlos H Feltes. Is non-invasive monitoring of intracranial pressure waveform analysis possible? Preliminary results of a comparative study of non-invasive vs. invasive intracranial slow wave waveform analysis monitoring in patients with traumatic brain injury. *Med Sci Monit* 2005; 112: CR 58-63.
3 Marshall LF, Smith RW, Shapiro HM. The outcome with aggressive treatment in severe head injury. Part I. The significance of intracranial pressure monitoring. *J Neurosurg* 1979; 80:20-25.
4. Johnston IH, Johnston JA, Jennett WB. Intracranial pressure following head injury. *Lancet* 1970; 2: 433-6.
5. Miller JD, Becker CD, Ward JD. Significance of intracranial hypertension in severe head injury. *J Neurosurg* 1977; 47:563-76.
6. Narayan RK, Kishore PR, Becker DP . Intracranial pressure to monitor or not to monitor? A review of our experience with severe head injury. *J Neurosurg* 1982; 56: 650-9.
7. Miller JD. ICP monitoring –current status and future direction. *Acta Neurochir* 1987; 85: 80-6.
8. Marmarou A, Anderson RL, Ward JD. Impact of ICP instability and hypotension on outcome in patients with severe head injury. *J Neurosurg* 1991; 75:559-66.
9. Lundberg N. Continous recording and control of ventricular fluid pressure in neurosurgical practice. *Acta Psychiatr Neurol Scand* (suppl) 1960; 36: 1-193.
10. Becker DP, Miller JD, Ward JD. The outcome from severe head injury with early diagnosis and intensive management. *J Neurosurg* 1977; 47:491-502.
11. Miller JD, Butterworth JF, Gudeman SK. Further experience in management of severe head injury. *J Neurosurg* 1981; 54:289-99.
12. Mahapatra AK, Bansal S. Role of intracranial pressure monitoring in head injury. A prospective study. *Neurology India* 1998; 46: 109-16.
13. Ostrup RC, Luerssen TG, Marshall LF, Zornow MH. Continous monitoring of intracranial pressure with a miniaturized fibreoptic device. *J Neurosurg* 1987; 67:206-9.
14. Jane JA, Luerssen Tg, Marmarou A , Foulkes MA. The outcome of severe closed head injury. *J Neurosurg* 1991; 75(suppl):528-36.
15. Marshall LF, Gautille T, Klauber MR. The outcome of severe closed head injury. *J Neurosurg* 1991; 75:528-36.
16. Eisenber HM, Gary HE Jr, Aldrich EF. Initial CT findings in 753 patients with severe closed injury. A report from the NIH Traumatic coma data bank. *J Neurosurg* 1990; 73:688-98.
17. Jennett B, Teasdale G, Galbraith S . Severe head injury in three countries. *J Neurol Neurosurg Psychiatr* 1977; 40:291-5.
18. Saul TG, Ducker TB. Effects of intracranial pressure monitoring and aggressive treatment on mortality in severe head injury. *J Neurosurg* 1982; 56:498-503.
19. Isa R, Wan Adnan WA, Ghazali G, et al. Outcome of severe traumatic brain injury: comparison of three monitoring approaches. *Neurosurg Focus 2003; 15(6): E1.*