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Clinical Analysis of 34 Diffuse Axonal Injured (DAI) Patients Below GCS 8

Chong Oon Park and Young Soo Ha

A consecutive series of 34 severe head-injured patients (DAI) were studied prospectively. Patients were categorized according to a new, simple classification system comprised of four lesion types according to the compression or obliteration of the ventricles or cisterns. Five patients belonged to type II and 19 patients to type IV. Each type was further subdivided into two GCS score ranges (5 to 8 and below 5). The distribution of the posttraumatic infarction was mainly in the frontal and temporal lobes (60% of all cases). Our data demonstrated that the ICP was significantly lower at a 30° head elevation than at 0° (18.6±7.21 mmHg vs 23.0±10.60 mmHg. t=4.22 P<0.001), but head position did not statistically affect CPP (69.4±19.86 mmHg vs 68.2±19.87 mmHg. t=−0.54, P<0.59). The effect of intensive therapy on ICP, CPP and $AVDO_2$ was studied in all cases, employing steroids and diuretics in a modified intensive care scale. In cases where barbiturates were employed, there were statistically significant changes in ICP and $AVDO_2$ (P < 0.001), but CPP was not affected (P<0.59). Surviving patients were analyzed by using the GOS and the neurological grading score (NGS, Nihon University) of the persistent vegetative state. Our data suggests that head elevation of 30° and barbiturate therapy are more effective on ICP and $AVDO_2$, and NGS more exact than GOS in vegetative patients.

Key Words: DAI, ICP, CPP, $AVDO_2$, intensive care scale, classification by CT

The incidence of mortality and morbidity from severe head injury remains surprisingly high in spite of the current aggressive strategies of diagnosis and treatment based on the control of intracranial pressure (Becker *et al.* 1977; Bouma *et al.* 1991).

This suggests that increased intracranial pressure (ICP), in many cases, reflects irreversible end results of biochemical and cellular events leading to neuronal death, rather than being the cause itself of such damage. Increased ICP or the combination of inadequate cerebral perfusion pressure (CPP) and arteriovenous oxygen difference ($AVDO_2$) are common causes of secondary brain damage in head injury patients (Rossanda *et al.* 1973). This idea suggests that the success of new therapeutic efforts may depend upon intervention in the pathophysiological mechanisms that occur early after injury.

Whether reduced cerebral blood flow (CBF) is sufficient to meet the metabolic demand of the injured brain depends on the control of elevated cerebral metabolism with barbiturates, hyperventilation, and induced hypertension. Reduced CBF does not necessarily produce ischemia in these patients. Determination of $AVDO_2$ is necessary for the proper evaluation of brain metabolism and important in determining the method of controlling the metabolism following head injury (Jaggi *et al.* 1990; Bouma *et al.* 1991). Mannitol and barbiturates are used widely in the management of severe head injury for reducing ICP and brain metabolism (Johnston *et al.* 1970; Overgaard *et al.* 1973; Marshall *et al.* 1978; Mendelow *et al.* 1985).

The aim of this study is to document the effects of: non-barbiturate and barbiturate treatment on ICP, CPP, and $AVDO_2$; and head position on cerebral parameters in patients with diffuse axonal inju-

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Department of Neurosurgery, In Ha Hospital, College of Medicine, In Ha University, Sungnam, Korea
Address reprint reguest to Dr. C O Park, Department of Neurosurgery, In Ha Hospital, College of Medicne, In Ha University, Sungnam, Korea

ry (DAI) below a Glasgow Coma Scale (GCS) score of 8. In addition, this study reports on ischemic brain areas common in DAI patients.

CLINICAL MATERIAL AND METHOD

Patient population and clinical mangement

Thirty-four patients with DAI (below a GCS score of 8), following only nonsurgical resuscitation, were admitted into the study between january and December 1991. Ten patients were less than 20 years old (29.4%) and 24 were 20 to 69 years old (70.6 %); most were between the ages of 20 and 40 (50 %)(Fig. 1). Eighteen cases were caused by traffic accidents (52.9%) and 15 cases by falls (44.1%)(Table 1). All patients were treated according to a protocol that emphasized early intubation, respiratory support, and prevention of secondary injury to the brain.

Only patients with non-surgical lesions were admitted into the study. Diffuse brain lesions were defined as head injuries accompanied by coma for longer than 24 hours, where CT scanning did not indicate mass lesions.

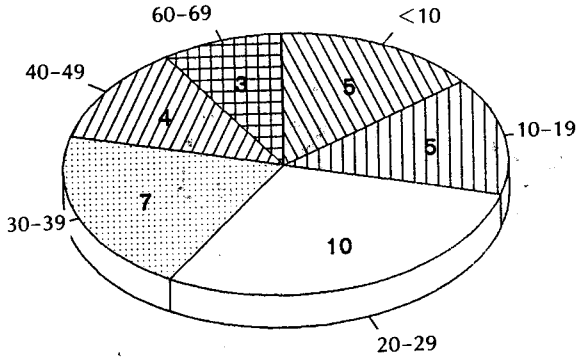

Fig. 1. *Age distribution.*

Table 1. Nature of injury

Nature	Number	Sex distribution (M : F)
Motor Vehicle		
Struck by Automobile	16	13 : 3
Accident in Automobile	2	1 : 1
Fall	15	14 : 1
Hit by some objects	1	1 : 0
Total	34	29.5

Patients with DAI below a GCS score of 8 were then categorized into four lesion types according to the compression or obliteration of the ventricles or cisterns as verified by CT scan. According to this classification, five patients (15%) showed diffuse injury with no visible pathology (DAI Type 1), five patients showed DAI Type II, five Type III, and 19 showed DAI Type IV (Table 2, Fig. 2).

Diagnostic CT scans were obtained upon admission and at regular intervals thereafter. A few cases required resuscitation to restore arterial pressure. ICP was monitored in all patients, using a parenchymal monitor (Camino Co.).

Arterial blood pressure (ABP) was monitored with an intra-arterial catheter and recorded as mean arterial blood pressure (MABP = 1/3 systolic ABP + 2/3 diastolic ABP). ICP and BP were assessed at 1-hour intervals. Each 24-hour physiological recording "day" was subdivided into six 4-hour intervals Neurological examination, including GCS and motor scoring, was performed twice daily for the first three days and once daily thereafter. Two consecutive sets of physiological measurements were performed. The initial set was taken at a zero degree head elevation; the second set at a 30° elevation, four hours later.

On arrival, six study patients had GCS scores of 3 to 4, while 28 patients had scores of 5 to 8. Abnormal pupil size was demonstrated by 17 patients having GCS scores of 5 to 8 (61%), and by six patients with scores below 5 (100%). Hypotension (BP $<$90 mmHg) appeared in six patients (Table 3). Initial gas studies indicated that PaO_2 was above 60 mmHg in 28 patients, $PaCO_2$ was above 45 mmHg in 32 patients, and $AVDO_2$ was initially above normal in 32 of the 34 patients in the study (94%). ICP was above 20 mmHg in 15 cases (Table 4).

In order to determine venous O_2 content accurately, the internal jugular vein was punctured at the posterior one third site on the baseline of the medial and lateral head of sternocleidomastoid-muscle-clavicle triangle. A Vygon subclavian catheter was guided to the jugular bulb, while arterial blood was drawn from a radial intra-arterial catheter. $AVDO_2$ determination was made at six hour intervals during the first days following injury. Patients were initially ventilated to maintain a PaO_2 of 100 mmHg or above and a $PaCO_2$ of 30 to 35 mmHg.

Differences were computed by subtracting the value obtained when the patient was lying flat from the value obtained when the head was elevated to

Table 2. Diagnosis categories of types of abnormalities visualization of CT scanning

Category	Definition	No fo pts.
DAI type I	No specific abnormal findings of ventricles or cisterns	
DAI type II	Only abnormality of ventricle	
DAI type III	Abnormality of ventricles and perimesencephalic cisterns	
DAI type IV	All abnormality of ventricles and cisterns	

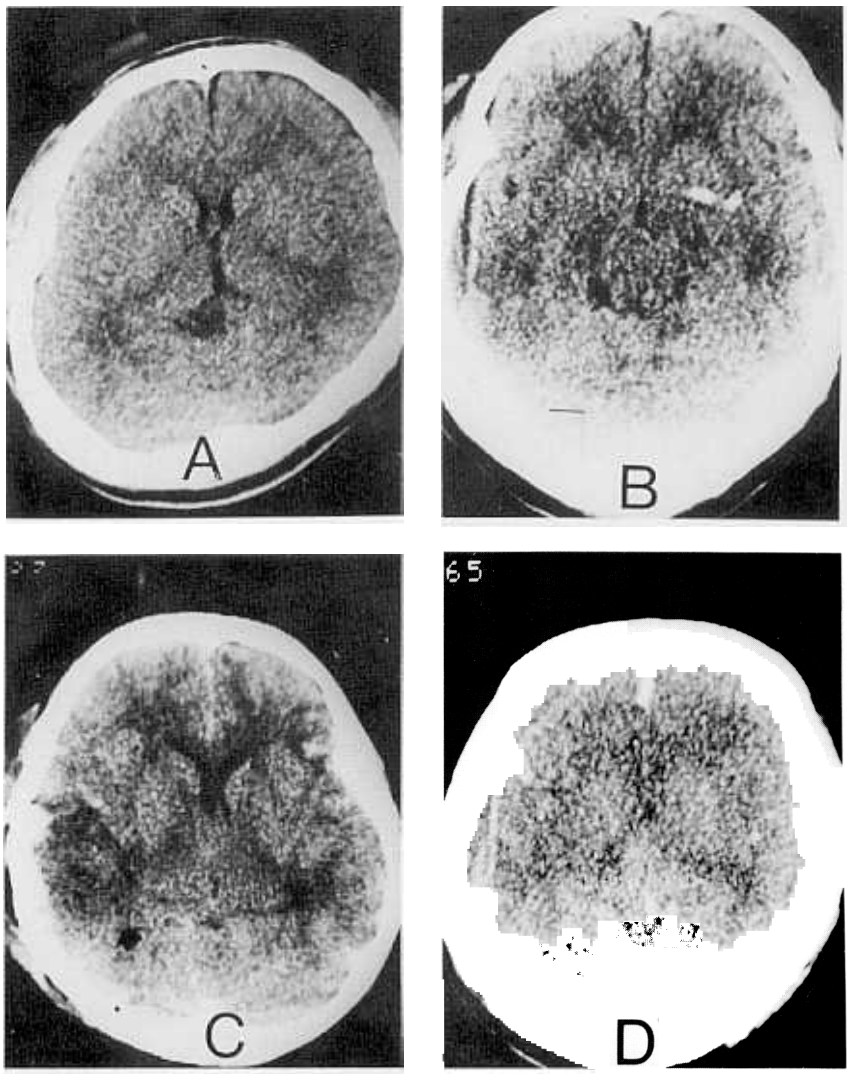

Fig. 2. A: Type I: no specific abnormality of ventricle or cisterns
B: Type II: only abnormality of ventricles
C: Type III: compression of ventricles and mesencephalic cistern
Posttraumatic infarction was shown on the figure C (↑)

Table 3. Clinical signs at ER

GCS	Pupil size		Systolic BP		Total
	Normal	Abnormal	≥90 mmHg	90 mmHg>	
8-5	11	17	26	2	28
<5	0	6	2	4	6
Total	11	23	28	6	34

Table 4. Gas study at ER

GCS	PaO_2		$PaCO_2$		$AVDO_2$		ICP		Total
	≥60 mmHg	60 mmHg>	≥45 mmHg	45 mmHg>	≥4-5 mmHg	4-5 mmHg>	≥20 mmHg	20 mmHg>	
8-5	26	2	27	1	26	2	10	18	28
>5	2	4	5	1	6	0	5	1	6
Total	28	6	32	2	32	2	15	19	34

Table 5. Therapy intensity level scale

Therapy	Score
Barbiturates	15*
Mannitol	
>1 gm/kg/hr	6
≤1 gm/kg/hr	3
Ventricular drainage	
>4 times/hr	2
≤4 times/hr	1
Hyperventilation	
intensive (PaCO<30 mmHg)	2
moderate (PaCO≥30 mmHg)	1
Steroid	1
Diuretics	1
Paralysis	1
Sedation	1

*Maximum score is 15. Without barbiturates, the score is the sum of the other components.

30°. These differences were evaluated using the paired t-test and 2-way ANOVA test.

Therapy intensity level concept

The new therapy index developed by investigators at the Medical College of Virginia provides greater resolution by grading ICP treatment according to a 15-point scale (Maset *et al.* 1978) (A score of 12 corresponds to maximum conventional therapy). We modified this scale by adding 2 points: one point for the use of steroids and one for the use of diuretics (Table 5). Considering all combinations, a score of 14 corresponds to maximum conventional therapy in the modified scale used in this study. Any patient given barbiturates at any dosage (18 in this study) was given the maximum scale score of 15. Therapy intensity levels were assessed at 6-hours intervals.

Patient treatment in the study followed non-barbiturate or barbiturate courses depending on the following criteria: (1) ICP greater than 20 mmHg was treated by: hyperventilation ($PaCO_2$ 25 to 30 mmHg), sedation (morphine), paralysis (pancuronium), and administration of mannitol and phenytoin for seizure. A bolus of 20% mannitol (0.5 to 1.0 gm /kg) was given intravenously over 10 to 15 minutes. (2) Barbiturate coma was induced in patients only if ICP was refractory to the above treatment and the initial ICP was above 30 mmHg.

RESULTS

The effects of head elevation on ICP are illustrated in Fig. 3. The mean values (±standard deviation) for ICP were significantly more affected at 30° head elevation than at 0°: 18.6±7.21 mmHg vs 23.0± 10.60 mmHg ($t=4.22$, $p<0.001$). The mean values

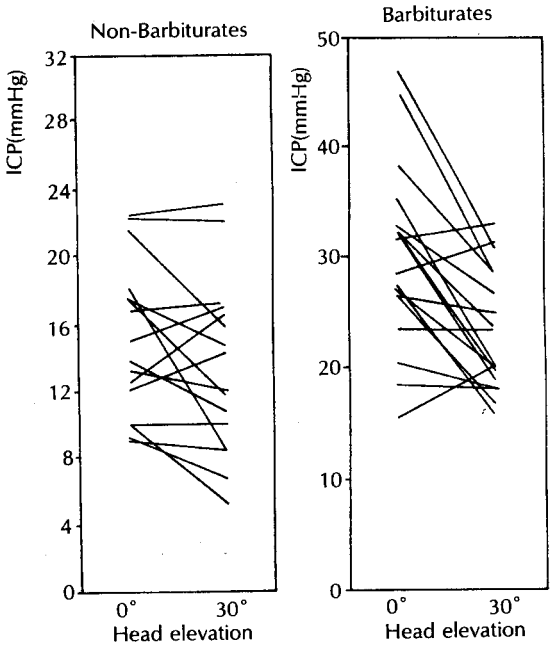

Fig. 3. Relation between ICP & Head elevation (30°).

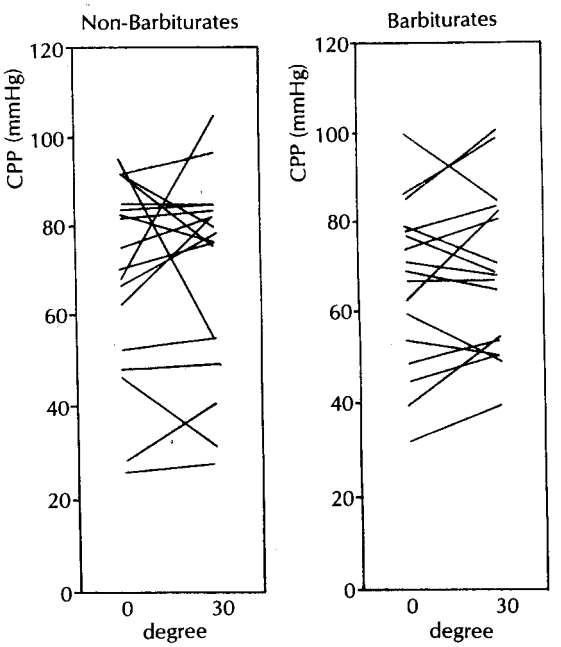

Fig. 5. Relation between CPP & head elevation (30°).

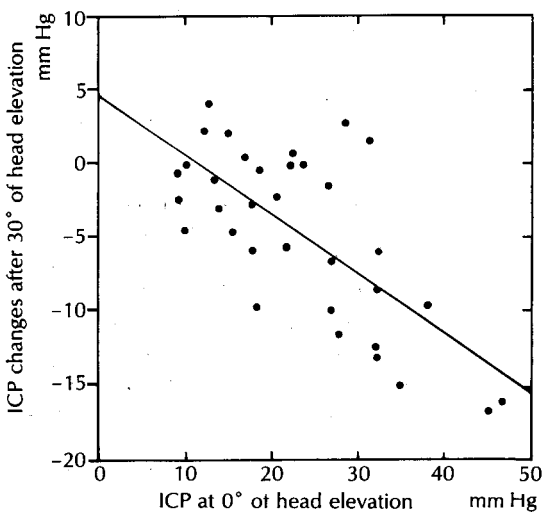

Fig. 4. Correlation between the level of intracranial pressure (ICP) at 0° head elevation and the amount of change in ICP after head elevation to 30° (r = -0.4226).

for ICP were, especially, more affected by head elevation in the patients treated with barbiturate treatment (23.2±5.36 mmHg at 30° vs 30.00±8.17 mmHg at 0°: t=4.18, p<0.001) than in those without (13.3±5.12 mmHg at 30° vs 15.1±4.58 mmHg at 0°: t=1.41, p<0.046). Correlationship analysis revealed a significant relationship between ICP level at 0° head elevation and the amount of change in ICP after 30° head elevation (r=-0.4226). The higher the ICP in patients at 0°, the greater the reduction in ICP at a head elevation of 30° (Fig. 4). The effect of barbiturates on ICP, disregarding head position, revealed a significant relationship. The mean values for ICP were significantly lower in barbiturate treatment cases than in non-barbiturate treatment cases. Statistical analysis was also done by 2-way ANOVA test. By 2-way ANOVA test, there is no correlation between head elevation and barbiturate treatment.

The effects of head elevation on CPP are illustrated in Fig. 5. The mean values for CPP were slightly higher at 30° head elevation than at 0° head elevation, but not significantly so (69.4±19.86 mmHg vs 68.2±19.87 mmHg; t=-0.54, p<0.59). There was also no significant relationship between head elevation and barbiturate/non-barbiturate therapy (Barb.; 70.5±21.85 mmHg vs 69.5±21.34 mmHg; t=-0.24, p<0.810, Non-barb.; 68.2±17.99 mmHg vs 66.7±18.63 mmHg, t=-0.67, p<0.514)(Fig. 5). Mean values for CPP were unaffected by barbitu-

Table 6. Relation between Posttraumatic infarction & DAI Type

Location Classification	F.	F.-T.	T.-P.	F.-T.-O.	Brain stem	Total
Type I						
Type II	1					1
Type III	1	2	1			4
Type IV	4	4	2	4	1	15

F.: Frontal, F.-T.: Frontotemporal, T.-P.: Temporoparietal, F.-T.-O.: Frontotemporooccipital

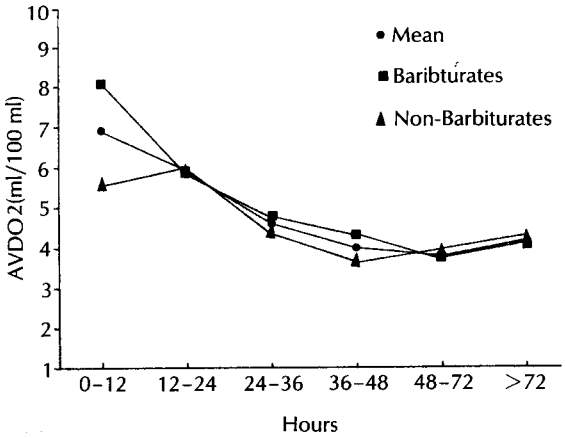

Fig. 6. *Time course of mean $AVDO_2$.*

Table 7. Neurological grading of the persistent vegetative state

Score	Neurobehaviour
1.	Alive with spontaneous respiration
2.	Withdrawal response to pain
3.	Spontaneous eye opening and closing
4.	Spontaneous movement of extremities
5.	Pursuit by eye movement
6.	Emotional expression
7.	Oral intake
8.	Producing sounds
9.	Obeying orders
10.	Verbal response

rate treatment (0° hed elevation: 69.5±21.35 mmHg (barbiturates) vs 66.7±18.63 mmHg (non-barbiturates), t=0.42, p<0.678. 30° head elevation: 70.4±21.85 mmHg (barbiturates) vs 67.9±19.27 mmHg (nonbarbitura-tes). t=0.37, p<0.713). With the 2 way-ANOVA test, there is no significant relationship (p=0.580 at barb. treated group, p=0.836 at head elevation)

$AVDO_2$ values were initially above normal in 94% of the patients (Table 4), The highest $AVDO_2$ values early after injury were typically found in patients with low GCS and motor scores. In non-barbiturate cases, $AVDO_2$ levels increased slightly in the first 24 hours (5.58±0.68 ml/100 ml initially vs 5.98±1.10 ml/100 ml in 12 hours vs 4.36±0.91 ml/100 ml in 24 hours). In barbiturate cases, $AVDO_2$ fell the fastest during the first 24 hours (8.13±1.00 ml/100 ml initially vs 5.84±1.13 ml/100 ml in 12 hours vs 4.89±0.53 ml/100 ml in 24 hours), while mean values progressively decreased to normal levels after 24 hours (Fig. 6).

Post-traumatic infarction developed in 20 patients, significantly, in 15 of 19 patients involved in type IV (79%). The frontal and temporal lobes were the most infarcted areas (60% of all cases). According to our new head injury classification, based on computerized tomography, no cerebral infarction occured in type I, 1 in Type II, 4 in Type III, and 15 (79%) in Type IV (Table 6).

Fifty-six percent of the study patients died during their hospital stay: thirteen of those patients treated with barbiturates (72%), and six of those treated with non-barbiturates (38%) (Table 9). At the time of discharge, 24% of the study patients were vegetative, 14% were severely disabled, and 6% showed a good outcome using the GOS scale. The neurological grading score (NGS of Nihon University, Table 7) was used only to evaluate the vegetative state. In this study we correlated the relationship between GOS and NGS. Our study found GOS IV (8 patients) equated to an NGS score of 3 to 6, while GOS III (5 patients) correlated to an NGS score of 7-10 points (Table 8).

Table 8. Relation between GOS & NGS

GOS \ NGS	3	4	5	6	7	8	9	10	Total
IV	1	2	3	2					8
III					1	1	2	1	5
Total	1	2	3	2	1	1	2	1	13

Table 9. Mortality rate between non-barbiturates & barbiturates

	<1 wk	<2 wks	<3 wks	Mortality rate
Barbiturates (18)	10	2	1	72%
Non-Barbiturates (16)	5	0	1	38%
Total (34)	15	2	2	56%

DISCUSSION

The management of patients with severe head injury continues to present neurosurgeons with a major challenge: mortality and morbidity are appallingly high, compared with other intracranial pathologies. Detailed neurological and psychosocial follow-up studies reveal that many apparent recoveries are in fact tragedies. The outcome of serious illness concerns not only the patient and their family, but therapeutic teams and the whole community. Modern intensive medical therapy has assured that some critically brain-damaged patients can survive for long periods in prolonged coma. Among these cases, 30-40% fall into a persistent vegetative state, a "wakefulness without awareness", and the prognosis is almost universally poor. Available treatments for such patients raise numerous social, economic, and judicial questions (Pagni, 1973; Tsubokawa *et al.* 1990).

Current intensive care of DAI patients has focused on controlling ICP. This study monitored ICP, CPP, $AVDO_2$, and the effects of head elevation in severe head injury cases treated by conventional intensive therapy without surgery. (barbiturates, mannitolization, steroid and diuretics, hyperventila-tion, etc.).

Brain enlargement may have resulted from edema, an increase in cerebral blood volume, or both. Although reports of diffuse brain bulk enlargements after head injury have increased with the widespread use of CT scanning (Tsai *et al.* 1978; Zimmerman *et al.* 1978; Bruce *et al.* 1991), the cause is still not completely understood. Even terms and definitions are not clearly established. For example: diffuse brain swelling (Obrist *et al.* 1979), generalized brain edema (Crone *et al.* 1987), and malignant brain edema (Bruce *et al.* 1981).

The most common neuropathological pattern includes diffuse microscopic damage to innumerable axons throughout the brain as well as focal lesions in the corpus callosum and the dorsolateral quadrants of the rostral brainstem (Lidenberg *et al.* 1970; Gennarelli *et al.* 1982). They preferred the descriptive term, diffuse axonal injury (DAI). The group reported that lower grade DAI injuries consisted of axonal abnormalities mainly restricted to the parasagittal white matter of the cerebral hemispheres. Higher grade injuries were characterized by focal lesions in the corpus callosum or superior cerebellar peduncle. They also felt that mass lesions and secondary complications such as hypoxia, ischemia, and other metabolic factors accentuate the neurological effects of DAI. Axonal retraction balls, microglial clusters, and long tract degeneration were microscopically demonstrated. These descriptions required pathologic findings based on CT scans and clinical findings. Lidenberg *et al.* (1970) classified focal lesions as space-occupying and diffuse brain lesions as non space-occupying. The diffuse category was further subdivided by coma duration (less than or greater than 24 hours) and occurrence of

decerebration (flaccid or not). Other authors have classified DAI, by CT findings, as follows: diffuse injury with no visible pathology (Type 1), diffuse injury with cisterns present and <5 mm shift (Type II), diffuse injury with swelling and cisterns compressed or absent (Type III), diffuse injury with a shift >5 mm (Type IV)(Marmarou *et al.* 1991; Marshall *et al.* 1991). Our group felt that midline shifting was not useful since ventricular or cisternal compression findings alone would adequately indicate the patient's condition and prognosis in DAI cases with GCS below 8. Subsequently, we simplified the Marmarou's classification by only considering ventricular and/or cisternal compression.

Overgaard *et al.* (1973; 1981) reported that ischemic change occurred at the highest rate within the first few hours of injury. They also confirmed that, from a hemodynamic perspective, the cortical areas most susceptible to damage from low-flow states (the frontoparietal arterial boundary zone) are the same in head injury patients as in patients with various systemic derangements. Gentry *et al.* (1988) reported that magnetic resonance imaging (MRI) was superior to CT scanning in the detection of traumatic head lesions such as DAI. While T_2 weighted images were most useful in lesion detection, T_1 weighted images proved most useful for anatomic location and classification of DAI patients, The group found that the most commonly involved locations were the frontal and temporal lobes, followed by the corpus callosum. The vast majority of the callosal lesions were in the splenium (71.9%), while the majority of primary brainstem lesions were localized in the dorsal and lateral aspects of the rostral brainstem. The shape of the lesions was usually ovoid to elliptical with the long axis parallel to the direction of the fiber bundles. Our study found the distribution of the cerebral infarction to be mainly in the frontal and temporal lobes (60%). In ten cases using MRI, five cases showed infarction in the corpus callosum (1 case in the anterior 1/3 of corpus callosum and 4 cases in the posterior 1/3 of corpus callosum), and three cases in the rostal brainstem.

We modified the Medical College of Virginia's 15-point ICP intensive therapy index by adding two factors, while maintaining a 15 point maximum. Accounting for the use of diuretics and steroids, valued at one point each, provided greater resolution. Considering all combinations, a score of 14 corresponded to maximum conventional therapy. Administration of barbiturates at any dosage resulted in a mandatory maximum score of 15. At their time of admission, six patients had levels of PaO_2 below 60 mmHg, 32 had $PaCO_2$ levels of 45 mmHg or above, 32 had $AVDO_2$ levels of 4-5 ml/100 ml or above, and 15 had ICP levels of 20 mmHg or above. Our treatment initially tried to maintain a PaO_2 level of 100 mmHg or above and a $PaCO_2$ level of 30-35 mmHg. Barbiturate coma was induced (in 18 cases) only if ICP was refractory to the above treatment.

ICP and CPP are common causes of secondary brain damage in head injury patients. Some studies suggest that head elevation may lower ICP and CPP (Kenning *et al.* 1981; Rosner *et al.* 1986, 1990; Feldman *et al.* 1992).

Durward *et al.* (1983) studied the effect of head elevation at 0°, 15°, 30°, and 60° on ICP and CPP in patients with intracranial hypertension. They concluded that 15° and 30° significantly reduced ICP while maintaining CPP and cardiac output. However, further elevation of the head to 60° caused an increase in ICP and a significant decrease in CPP and cardiac output. Kenning *et al.* (1981) reported that elevations of 45° or 90° significantly reduced ICP. More recently, Rosner *et al.* (1986; 1990) emphasized the importance of adequate CPP with physiological and clinical evidence suggesting that CPP is maximum when patients are in the horizontal position, even though ICP is usually higher. Davenport *et al.* (1990) found inconsistent evidence of head elevation on ICP but a significant decrease in MABP and CPP at 40° and 60°. Feldman *et al.* (1992) however reported that ICP was significantly reduced at 30° head elevation, but CPP did not show any statistically significant difference between 0° and 30° elevations. Our data suggest that for barbiturate and non-barbiturated cases, head elevation is effective in reducing ICP. In barbiturate treatment cases, head elevation is more effective in reducing ICP. This is apparently due to: 1) hydrostatic displacement of the CSF from the cranial cavity to the spinal subarachnoid space, and 2) increased venous outflow from the brain. Barbiturate treatment disregarding the change of head position had a statistically significant effect in reducing ICP. Head position also had some effect on CPP, but didn't produce a statistically significant difference between head elevation of 30° and 0°.

Bouma *et al.* (1991) reported another factor in determining whether CBF is sufficient to meet the metabolic demands of an injured brain is that cerebral metabolism is often decreased after severe brain trauma. Consequently, reduced flow does not necessarily produce ischemia in these patients.

Therefore, determination of $AVDO_2$ is necessary to properly evaluate CBF, as this parameter indicates the link between flow and metabolism. Low $AVDO_2$ may or may not reflect metabolic (mitochondria) dysfunction, but high $AVDO_2$ always indicates the inadequacy of the CBF to support brain metabolism. In brain trauma cases, they reported that $AVDO_2$ was initially above normal, decreased rapidly in the first 24 hours, and levelled off at about 4.5 ml/100 ml. The highest $AVDO_2$ values early after injury and the steepest falls in the first 24 hours were typically found in the patients with low motor scores. In our study, for patients NOT undergoing barbiturate therapy, $AVDO_2$ values increased in the first 24 hours and then decreased to a level of about 4.3 ml/100 ml. In barbiturate treated patients, $AVDO_2$ values decreased rapidly in the first 24 hours after injury. Overall $AVDO_2$ values decreased progressively in the first two days.

Jennett and Bond (1975) started to document carefully and serially the clinical status of patients with severe head injury in a prolonged study of both in-hospital and outpatient follow-up cases. They developed a series of standardized descriptions of consciousness level, neurological signs, and outcome categories. A five-point scale was defined: death, persistent vegetative state, severe disability, moderate disability, and good recovery. We felt the GOS scale was a good classification but extended over too vast a range. We found there was a need to divide the persistent vegetative and moderate disability states more precisely to evaluate patient outcome more exactly. NGS, the 10 point scale developed at Nihon University (Tsubokawa *et al.* 1990) is well adapted to monitoring progress in chronic patients. In correlating the relationship between GOS and NGS, our study found GOS IV equated to an NGS score of 3 to 6, while GOS III correlated to an NGS score of 7 to 10 points.

Becker *et al.* (1977) reported in a study of 160 patients that 36% made a good recovery, 24% were moderately disabled but able to care for themselves, 8% were left severely disabled, 2% were in a vegetative state, and 30% of them died. Patients with intracranial mass lesions had a higher mortality (40%) than patients with diffuse brain injury (23%). The series of head injuries reported by Pazzaglia *et al.* (1975) consisted of 282 patients. Patients with mass lesions had a higher mortality (55 %) than those with diffuse injury (41%). Bruce *et al.* (1981) believed the presence of mass lesions did not influence outcome, since 11 of their 12 patients harboring mass lesions achieved good recovery or had only moderate disability. Our study involved only nonevacuated mass lesions. The total mortality rate was 56% (19 patients): 46.4% of patients with GCS scores between 8 and 5, and 100% of those under 5. All patients under a GCS of 5 had no reactive pupils to light, either bilaterally or unilaterally. Of those patients who died, 18 had Diffuse Injury Type IV and 1 patient had Type III. The mortality rate of the non-barbiturate cases was 38% and 78 % for the barbiturate cases.

Alberico *et al.* (1987) reported pediatric mortality was lower than adult mortality (24% vs. 45%). Additionally, Hendrick *et al.* (1964) reported a higher mortality in infants than in adolescents. Similarly, Teasdale *et al.* (1974; 1976) demonstrated an increasing mortality with age in patients over five years old. In general, good outcome decreases with age, excepting the very young (0 to 4 years of age), and mortality increases with age (Carlsson *et al.* 1968; Vollmer *et al.* 1971). This study did not analyze mortality rates since their were few pediatric cases.

CONCLUSION

In summary, this study indicates that disturbance of $AVDO_2$ or CPP occurring in the first few hours after severe brain trauma is one of the most important correlating factors to clinical status and outcome. Therefore, intensive therapy in the first few hours after severe head injury, involving barbiturate therapy, hyperventilation, and head elevation, is of great importance. The level of intensive care should be determined by the metabolic need of the brain. MRI, highly sensitive in detecting both hemorrhagic and nonhemorrhagic lesions, is now able to accurately identify, classify, and stage the extent of all types of traumatic lesions.

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