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Intraventricular Hemorrhage in Blunt Head Trauma: An Analysis of 43 Cases

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ABSTRACT

BEFORE THE ADVENT of computed tomography, intraventricular hemorrhage (IVH) from any source was thought rare and invariably fatal. Although intraventricular blood is readily identifiable with computed tomography, there has been little systematic study of its significance in blunt head trauma. Forty-three patients with traumatic IVH were prospectively identified in 1 year at Harborview Medical Center (University of Washington). Most were victims of motor vehicle accidents and suffered severe head injuries. IVH occurred alone in two patients; superficial contusions and subarachnoid hemorrhage were the most common associated finding. Blood was present in only one or both lateral ventricles in 25 patients; only the 3rd or 4th ventricles in 4 and all ventricles in 14 instances. There were 3 intracerebral hematomas and 14 basal ganglion hemorrhages. All of the former and half of the latter communicated with the adjacent lateral ventricle. Extra-axial hematomas appeared more common when only the lateral ventricles were involved, whereas corpus callosum or brain-stem hemorrhage appeared more likely when all the ventricles were involved. Acute hydrocephalus was rare, and ventricular drainage was needed in only four cases. Intracranial pressure (ICP) was elevated (> 15 mm Hg) in 46% of patients. The amount of IVH was related inversely with the Glasgow Coma Scale, but not with increased ICP. The presence of IVH indicated a poor outcome, with only half of the patients being independent at a 6-month follow-up. Poor outcome was associated with increasing age, low admission Glascow Coma Scale, the presence of space occupying lesions if only the lateral ventricles were involved, and hemorrhage in all four ventricles. IVH after blunt head trauma is uncommon and reflects the severity of the head injury. The pattern of associated lesions suggests that the extent of IVH may be related to the direction of force applied to the head.

Historically, the occurrence of intraventricular hemorrhage (IVH) from any source was thought rare and viewed as a poor prognostic indicator (5,18,26,27,30). Since the advent of computed tomography (CT), intraventricular blood can be readily diagnosed and can be found in 1.5 to 3% of all patients with nonmissile head trauma and in nearly 10% of patients with severe head injury (4,7,8,10,23,28,31,38). Despite this, there has been little systematic study of this phenomenon in relation to blunt head trauma, so its pathogenesis remains speculative. Previous reports suggest that traumatic IVH may result from the spread of an adjacent intracerebral hematoma (16,23,31). Others have suggested it may be secondary to hypoxia (3) or coagulopathy (31). In addition, traumatic IVH has been reported in patients with radiological evidence of diffuse axonal injury (36,37), suggesting that different pathogenic mechanisms may lead to IVH development. Outcome is often poor; however, it is unclear if IVH affects the clinical course directly or is a result of hydrocephalus and intracranial hypertension (3,4).

The present clinicoradiological study was undertaken for the following reasons: 1) to establish the clinical characteristics of IVH in nonpenetrating head injury; 2) to identify associated pathological and radiological findings and their influence on the pathogenesis of IVH; 3) to determine the effect on hydrocephalus and intracranial pressure; and 4) to ascertain the outcome.

PATIENTS AND METHODS

We analyzed all patients with nonmissile head trauma who were evaluated at Harborview Medical Center (HMC) during 1 year (1989). Forty-three patients were prospectively identified with the diagnosis of traumatic IVH. Patients with IVH from other causes were excluded. The diagnosis of IVH was made by computed tomography (CT) of the head.

Patients with head injury treated at HMC are initially evaluated and aggressively resuscitated by paramedics in the field. An admission Glasgow Coma Score (GCS) was determined based on physical examination findings as described by Teasdale and Jennett (33). Intubated patients were assigned one point for the GCS verbal category. Additional clinical data collected at admission included the following: 1) mechanism of injury; 2) presence of other injuries; 3) arterial blood gas analysis (hypoxia defined as PaO_2 < 60 mm Hg); and 4) coagulation screen including prothrombin and partial thromboplastin time, platelet count, fibrinogen, and fibrinogen degradation products.

After initial evaluation and resuscitation in the emergency room, all patients underwent a head CT scan (GE 9800, General Electric Medical Systems, Milwaukee, WI; 10 mm axial interval; 360 × 360 matrix). Head CT scans were reviewed for the presence of the following: 1) IVH; 2) skull fracture; 3) hemorrhage (hyperdense lesions < 2 cm diameter and no mass effect); 4) intracerebral hematomas (hyperdense lesions > 2 cm diameter with mass effect); and 5) extra-axial hematomas (acute extradural or subdural hematomas).

The location of each hemorrhage was recorded as follows: 1) superficial (cortical surface contusion); 2) deep (white matter exclusive of corpus callosum); 3) basal ganglia; 4) corpus callosum; or 5) brain stem. The size of the IVH was scored using a numerical system (Table 1). All CT scans were obtained within 1 hour of admission. Twenty-eight were obtained within 2 hours of injury. No scan was obtained later than 6 hours after injury. In surgically treated patients, CT scans were repeated after craniotomy. In survivors, CT scans were obtained at 24 and 48 hours, 1 week and 1 month after injury, respectively. All patients obtained a second CT scan (24 hours), but only 29 underwent a 1-month follow-up scan.

Score 1 = Trace of blood
Score 2 = Less than half a single ventricle filled with blood
Score 3 = More than half a single ventricle filled with blood
Score 4 = Entire ventricle filled and expanded with blood
Note: Each ventricle is scored separately and a total score calculated. Minimum score = 1; Maximum score = 16.

Table 1. Numerical Scoring System for Grading Severity of Intraventricular Hemorrhage

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Intracranial pressure (ICP) monitors (subarachnoid screws) were placed in 39 of the 43 patients. The mean maximum ICP was identified for the duration of monitoring. Raised ICP was defined as greater than 15 mm Hg and further subdivided into categories of responsive and nonresponsive to treatment. Treatment included hyperventilation, sedation and/or muscular paralysis (pancuronium bromide; Pavulon, Organon Pharmaceuticals, West Orange, NJ), osmotherapy with mannitol or furosemide, ventricular drainage and/or surgery.

All surviving patients were followed, and outcome was assessed at least 6 months after injury. For statistical comparison, outcome was rated as “favorable” in patients capable of an independent existence. This includes a Glasgow Outcome Score of “good” or “moderate disability” (14). “Poor” outcome was assigned to those patients who died or were not capable of an independent life.

Data are expressed as the mean ± the standard deviation of the mean. Stastical comparison between groups was assessed with Student's unpaired *t*-test. In the case of small observed numbers in any classification category, Fischer's exact test was used. Statistical significance is assumed for comparisons in which $P < 0.05$. In the case of multiple associations, Bonferronian inequality was used to protect against the inflation of chance.

RESULTS

Clinical characteristics of patient population

Traumatic IVH was identified in 43 of 1526 (2.8%) patients who received CT scans for head injury during 1 year at HMC. The results are summarized in Tables 2 and 3. There were 33 male and 10 female patients, ranging in age between 7 and 83 years (mean, 34.9 ± 19.1 years). Most patients were victims of motor vehicle accidents; the remainder suffered either a fall or blunt assault. Twenty-one suffered multiple injuries, including significant chest, abdominal, or orthopedic injuries. Neither the mechanism of injury nor the presence of other injuries affected the extent of IVH. The admission GCS ranged between 3 and 14 and was related inversely with the amount of IVH ($r = 0.379$, $P < 0.01$). Hypoxia was identified in six patients and coagulopathy in seven. Neither influenced the extent of IVH.

Patient Number	Sex/ Age (yr)[a]	Mechanism of Injury[b]	Admission Glasgow Coma Scale	Maximum ICP[c]	IVH Score[d]	Description IVH[e]	Extraxial or ICH[f]	Diffuse Axonal Injury[g]	Ventricular Drainage[h]	Glasgow Outcome Score[i]
1	M/44	MVA	3	10	13	RLLV,3V,4V		+	+	Died
2	M/14	MVA	4	18	2	LRLV				Mod disability
3	M/81	Fall	10	8	2	LRLV	RICH		+	Died
4	M/34	MVA	10	7	2	LRLV				Good
5	F/27	MVA	11	5	2	LRLV				Mod disability
6	M/53	Fall	14	11	2	LRLV	RSDH		+	Mod disability
7	M/36	MVA	5	22	4	LRLV,3V,4V		+		Died
8	M/21	MVA	6	25	2	LRLV				Vegetative
9	M/19	Fall	5	27	1	LLV				Vegetative
10	M/27	MVA	14	12	4	LRLV,3V,4V	RSDH			Severe disability
11	M/16	MVA	9	13	1	LLV				Mod disability
12	M/83	Fall	4	10	1	LLV	RSDH			Died
13	M/58	MVA	4	19	4	RLV,3V,4V	LSDH	+		Died
14	M/16	MVA	7	8	2	RLLV	REDH			Mod disability
15	M/28	Fall	3	35	12	RLLV,3V,4V		+	+	Died
16	M/8	MVA	10		2	RLLV				Mod disability
17	M/17	Fall	13		1	3V	LSDH			Good
18	M/24	MVA	7	20	2	RLLV				Mod disability
19	F/74	Ped	3	11	4	RLLV,3V,4V		+		Died
20	M/7	Fall	3	21	6	RLLV,3V,4V		+		Died
21	F/43	MVA	13		1	RLV				Good
22	M/47	Ped	4		12	RLLV,3V,4V				Died
23	F/68	Fall	3	20	2	RLLV	LICH			Severe disability
24	M/17	MVA	10	10	4	RLLV,3V,4V		+		Mod disability
25	F/30	Fall	4	25	6	RLLV,3V,V4V		+		Died
26	M/35	Assault	12	19	2	RLLV				Good
27	M/23	MVA	7	8	2	RLLV				Good
28	M/46	Fall	3	21	3	4V	LSDH			Died
29	M/53	Assault	4	7	2	RLLV	LSDH			Died
30	M/27	MVA	5	14	1	RLV	LSDH			Vegetative
31	M/47	MVA	10	18	2	RLLV	RSDH			Mod disability
32	F/21	MVA	5	24	1	LLV		+		Died
33	M/27	MVA	3	40	5	RLLV,3V,4V				Died
34	M/46	Assault	7	14	8	RLLV,3V,4V	RICH			Good
35	F/20	MVA	6	34	4	3V,4V				Mod disability
36	M/29	MVA	3	17	2	RLLV	RSDH		VPS	Severe disability
37	F/31	Fall	14	11	2	RLLV				Good
38	F/11	MVA	6	7	2	RLLV				Mod disability
39	M/54	MVA	3	14	2	3V	LSDH			Died
40	M/27	MVA	4	12	2	LLV		+		Mod disability
41	M/36	Logging	10	13	2	LRLV	LSDH			Good
42	M/56	MVA	5	10	6	RLLV,3V,4V		+		Severe disability
43	F/22	MVA	4	25	7	LRLV,3V,4V		+		Vegetative

[a] M, male; F, female.
[b] MVA, motor vehicle accident; PED, pedestrian struck by car and assault—blunt assault.
[c] Maximum intracranial pressure in mm Hg.
[d] Numerical score for grading severity of intraventricular hemorrhage (Table I).
[e] R, right; L, left; LV, lateral ventricle; 3V, third ventricle; 4V, fourth ventricle.
[f] ICH, intracerebral hematoma; SDH, subdural hematoma; EDH, extradural hematoma.
[g] As depicted on CT by brain stem or corpus callosum hemorrhages. +, present.
[h] VPS, ventricular peritoneal shunt.
[i] As described by Jennett and Bond (14). Mod, moderate.

Table 2. Clinical Characteristics: Computed Tomography and Outcome in Patients with Intraventricular Hemorrhage after Blunt Head Injury

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	Type of Intraventricular Hemorrhage		
	Lateral Ventricles Only	3rd or 4th Ventricles Only	All Ventricles
Number	25	4	14
Mean age (yrs)	33.9 ± 20.4	34.3 ± 18.5	36.4 ± 18.7
Sex: male/female	19/6	3/1	11/3
Mean admission GCS[a]	7.7 ± 3.5	6.3 ± 4.7	5.1 ± 3.2
Mean IVH Score[b]	1.8 ± 4	2.5 ± 1.3	6.8 ± 3.3
Mean maximum ICP (mm Hg)[c]	14 ± 6.4	23 ± 10.2	20 ± 9.2
Associated radiological abnormalities			
Extra-axial hematomas	9	3	2
Intracerebral hematomas	2		1
Corpus callosum or brain stem hemorrhage	2		10
Outcome[d]			
Favorable	16	2	2
Poor	9	2	12

[a] GCS, Glasgow Coma Scale (33).
[b] Mean IVH score, Mean intraventricular hemorrhage numerical score (Table 1).
[c] ICP, intracranial pressure.
[d] Outcome assessed at least 6 months after injury. Outcome was rated as favorable in patients capable of an independent existence. Poor outcome was assigned to those patients who died or were not capable of independent life.

Table 3. Clinical, Radiological, and Outcome Characteristics of Different Types of Traumatic Intraventricular Hemorrhage

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Radiographic findings

IVH was the only abnormality disclosed by CT in two patients. Hemorrhage involved only the lateral ventricles in 25 patients; only the 3rd or 4th ventricle in 4, and involved all ventricles in 14 instances. The size of the IVH (by

numerical score) ranged between 1 and 13 (mean, 3.4 ± 3.0). A skull fracture was identified in 16 patients. Subarachnoid hemorrhage and superficial contusions were frequent findings (Fig. 1). Deep hemorrhage was present on a third of the admission CT scans, but its presence did not appear to impact the degree of intraventricular blood. Intracerebral hematomas with associated mass effect were identified on three occasions; each communicated with the adjacent ventricle. Extra-axial hematomas (extradural or subdural) usually occurred when IVH involved the lateral ventricles only. In contrast, hemorrhage involving the corpus callosum or brain stem was more likely to be associated with hemorrhage involving all four ventricles ($P < 0.001$) (Fig. 2). A single cerebellar hemorrhage associated with blood in both lateral ventricles only was identified. Basal ganglion hemorrhages were present in 14 patients. In half of these, the ventricular wall appeared intact between the lesion and the intraventricular blood. Five patients developed delayed traumatic intracerebral hematomas on follow-up CT scans. IVH did not appear to increase but was gradually reabsorbed (in survivors) in all by 1-month follow-up and most of the smaller hemorrhages by 1 week.

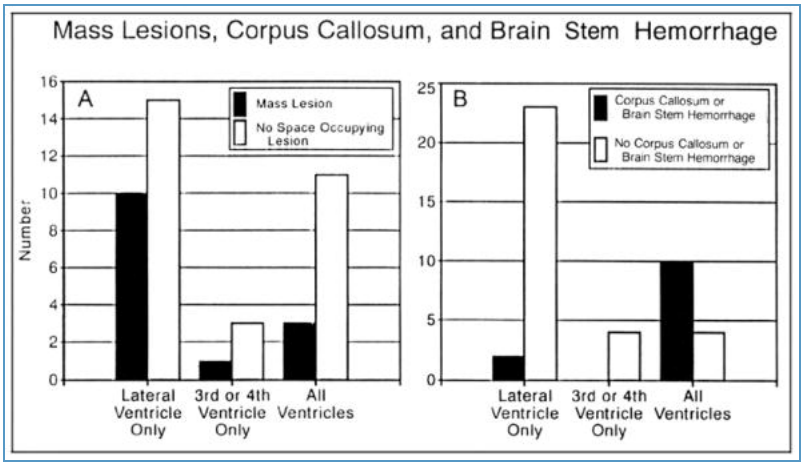

Figure 2. Bar graph depicting the relationship between the type of intraventricular hemorrhage and the presence of mass lesions (intracerebral or extra-axial hematomas) or the presence of corpus callosum or brain stem hemorrhage. The latter were more common if blood was present in all ventricles ($P < 0.001$).

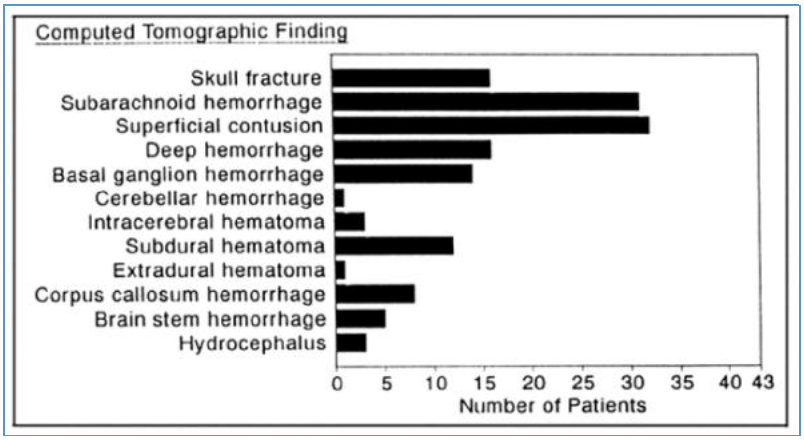

Figure 1. Bar graph depicting computed tomographic findings associated with traumatic intraventricular hemorrhage. Hemorrhage refers to hyperdense lesions < 2 cm in diameter. Hematoma refers to hyperdense lesions > 2 cm in diameter with associated mass effect.

Surgical procedures and ICP measurements

Twelve craniotomies were performed to evacuate either an extra-axial or intracerebral hematoma. A total of 39 patients had intracranial pressure monitors inserted. Mean maximum ICP ranged between 5 and 40 mm Hg (mean, 16.5 ± 8.4 mm Hg). Twenty-one patients had normal ICP (<15 mm Hg); 12 had elevated ICP that responded to treatment; and 6 developed intractable intracranial hypertension. Although increased intracranial pressure and admission GCS showed some relationship ($r = 0, 398$, $P < 0.02$), the amount of intraventricular blood had little impact on ICP.

Hydrocephalus was rare (7%), and only four ventriculostomies were inserted. One patient required ventricular peritoneal shunting.

Outcome

Outcome in this group of 43 patients with IVH was poor: 15 died, 4 remained vegetative, 4 were severely disabled, 12 moderately disabled, and 8 had a good outcome 6 months after their respective injuries. Only 20 (47%) of the patients were capable of independent life.

Both increasing age and decreasing admission GCS ($P < 0.001$) adversely affected outcome. Twenty-nine patients had admission GCS of less than or equal to 8; 21 (72%) had a poor outcome; and 8 (28%) were independent at follow-up. In contrast, among those with a GCS greater than 8, 12 (86%) were independent, and 2 (14%) had a poor outcome at a 6-month follow-up.

Outcome varied depending on the type and size of hemorrhage and the occurrence of other abnormalities (Table 4). The presence of hemorrhage in all four ventricles worsened outcome ($P < 0.01$); 14% of patients with blood in each ventricle were independent at a 6-month follow-up, compared with 64% of patients with hemorrhage in the lateral ventricles only. A space-occupying lesion tended to worsen outcome in this latter group. There was a trend toward a worse outcome with increased ICP.

	Glasgow Coma Scale > 8		Glasgow Coma Scale < 8		
	Outcome[a] Favorable	Outcome Poor	Outcome Favorable	Outcome Poor	Total
Lateral ventricle only	9	0	4	2	15
All ventricles/or 3rd or 4th ventricle	0	0	3	5	8
Lateral ventricle and space occupying lesion	3	2	0	5	10
All ventricles and corpus callosum or brain stem hemorrhage	0	0	1	9	10
Total	12	2	8	21	43

[a] Outcome assessed at least 6 months after injury. Outcome was rated as favorable in patients capable of an independent existence. Poor outcome was assigned to those patients who died or were not capable of independent life.

Table 4. Outcome by Ventricular Hemorrhage and Glasgow Coma Scale

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DISCUSSION

In the present report, we studied 43 patients with IVH after blunt head injury. Although strong predictors are evident, moderate associations may be missed because of this small sample size. Patients were usually victims of motor vehicle accidents and suffered severe head injury (GCS < 8). The admission GCS was related inversely with the extent of IVH. Superficial contusions and subarachnoid hemorrhage were frequent findings. Extra-axial hemorrhages were usually seen when only the lateral ventricles were involved, whereas hemorrhage involving all ventricles was commonly seen in association with corpus callosum or brain-stem lesions. Although hydrocephalus was rare, raised intracranial pressure occurred in nearly half the patients. Outcome was poor; less than half the patients were independent at a 6-month follow-up.

Pathogenesis

The pathogenesis of traumatic IVH is unclear except where there is the spread of blood from adjacent parenchymal lesions (16,23,31). Frontal, temporal, and basal ganglia hematomas with extension into the adjacent ventricles are reported to coexist in about one-third of patients with IVH (4,8,38). This is more likely after an impact to the head in the sagittal direction (8). However, secondary spread is not evident in over half the cases of traumatic IVH, and so alternate hypotheses have been advanced, including hypoxia (3) and coagulopathy (31).

Hypoxia has been found in neonates with IVH (22,35) and is common after head injury (6,24). Although Christie et al. (3) have suggested that hypoxia may be implicated in the pathogenesis of traumatic IVH, we did not find a relationship between hypoxia and IVH in our series. Thromboplastin is commonly released in severe head injury and may cause a coagulopathy. Piek et al. (31) suggested that this may lead to secondary IVH. Coagulopathy did not appear to influence the extent or later development of IVH in our series, nor the findings of Fujitsu et al. (8). The presence of an unsuspected choroid plexus arteriovenous malformation has been reported in traumatic IVH (11,22). These, however, are rare. Ten of our patients received contrast scans, all of which disclosed nothing abnormal, as did those in other studies (8). Unterharnscheidt (34) hypothesized that midline traumatic lesions (primarily hemorrhages in the ventricles or subependymal area) result from the development of negative pressure in the veins of this region. Impact along the sagittal diameter of the skull deforms it by increasing the minor axis with resultant ventricular dilatation. This is thought to cause a negative pressure and rupture of the subependymal veins leading to IVH. Fujitsu et al. (8) speculated that a similar mechanism led to the development of IVH secondary to basal ganglia hematoma.

In addition, we suggest that some types of IVH, particularly those involving all ventricles, may be related to diffuse axonal injury (DAI), which is more commonly caused by angular acceleration or movement in the coronal plane (9). DAI complicates about 3% of all head injuries and has characteristic pathological findings, including hemorrhage in the corpus callosum and dorsolateral brain stem (1). These are commonly visible on CT scan after severe head injury (36,37). Cordobes et al. (4) have noted these CT findings in 50% of patients with traumatic IVH; similarly, Wilberger et al. (36) and Zimmerman et al. (37) have found that half the cases with CT evidence of DAI have intraventricular blood. In our patients, lesions in the corpus callosum or brain stem were more likely when hemorrhage involved all four ventricles. In those same patients, intracranial hematomas were uncommon. At autopsy, Adams et al. (1) found a much lower incidence of intracranial hematomas in patients with DAI. Five patients in our series with hemorrhage in all ventricles and hemorrhagic lesions in the corpus callosum and brain stem underwent autopsy. Microscopically, all showed axonal retraction balls or microglial clusters–the histological characteristics of DAI (1,32).

It is rare for IVH to be found in isolation (4,8,16,28,29,38); subarachnoid hemorrhage and small superficial contusions being commonly associated. The centripetal theory of Ommaya and Gennarelli (29) suggests that progressively deeper lesions are found after greater impact and therefore worse head injury (17,29). IVH is commonly found in association with lesions in the deep white matter (36). In our series, although these lesions were commonly present, they did not appear to affect the extent of intraventricular blood. Blood in all four ventricles, however, was more commonly associated with callosal or brain-stem hemorrhage, which, in models of head injury, appear related to lateral acceleration (9). Conversely, subdural hematomas in this model are more likely to occur with sagittal acceleration. In our patients, subdural hematoma was usually associated with lateral ventricular blood only. It is thus possible that the type of IVH (lateral ventricular or panventricular) is determined by the direction of acceleration applied to the head.

Intracranial pressure/ventricular drainage

Intracranial pressure has not been systematically studied in relation to traumatic IVH. Case reports suggest that it may be raised (> 15 mm Hg) in nearly half the patients (3,27,28), similar to our results. A third of these patients develop intractable intracranial hypertension. This probably reflects the severity of the head injury rather than acute hydrocephalus, which appears rare in posttraumatic IVH. The amount of IVH, particularly when all four ventricles were involved, did not appear to impact intracranial pressure. This is in keeping with the postulate that blood in all four ventricles is a radiological manifestation of DAI, as raised ICP is uncommon in these patients (1). With time, the blood is quickly resorbed, and later ventricular enlargement is more often secondary to parenchymal atrophy rather than hydrocephalus (18). External ventricular drainage or ventriculoperitoneal shunts are not often needed (4,18), and although they may help control ICP, they do not necessarily improve outcome as in our series. In contrast to aneurysmal IVH where hydrocephalus may worsen outcome (26), it does not appear to be a factor in traumatic IVH.

Outcome

Before the CT era, IVH was thought usually fatal. It still carries a poor prognosis, particularly if there are other associated abnormalities (3,4,8,27,28,31,38). Younger patients fare better, which is well described in head injury reports (13,20). Similarly, the admission GCS is a strong predictor of outcome (8,20,21). Nontraumatic IVH carries an equally poor prognosis (2,5,18,21,26) and appears related to the volume of ventricular blood, particularly if it spreads through all the ventricles or involves the 3rd or 4th ventricles (5,12,30). Our results, like those of Christie et al. (3), suggest this, although Cordobes et al. (4) did not find that the extent of ventricular hemorrhage correlated with outcome. When blood involves the lateral ventricles only, a better outcome is expected unless there is an associated extra-axial or intracerebral hematoma. Although basal ganglia hemorrhage usually carries a poor prognosis (8,15,19,31,38), patients with this and only lateral ventricular blood often had a favorable outcome in our experience. Not un-expectedly, the presence of corpus callosum or brain-stem lesions is a grave sign (4,8), which probably reflects the extent of DAI, which generally carries a poor prognosis (1,36,38). As in other types of head injury, raised ICP worsened outcome (25).

In summary, IVH is uncommon after blunt head injury. The extent of hemorrhage may reflect both the direction of force applied to the head and other associated abnormality. Intracranial hypertension occurs in nearly half the patients but is not related to hydrocephalus, which is uncommon. Outcome remains poor, particularly if the hemorrhage involves all four ventricles.

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COMMENT

Computed tomographic (CT) scanning of head injuries enabled the recognition of intraventricular hemorrhage in survivors of head injury, not merely in those fatal injuries subject to post mortem. Intraventricular hemorrhage is nevertheless an uncommon finding, and the authors' review extends our knowledge of its occurrence, the various forms it may take, and its associations. The distinction drawn between hemorrhages that involve the lateral ventricles and are associated with extra axial clots and more widespread hemorrhage in association with CT features of diffuse axonal injury is novel and intriguing. In diffuse shearing injury, in addition to lesions in the corpus callosum, ventricular hemorrhage may be secondary to tears of the interventricular septum, often also ruptured (1). Other sources include tears of the choroid plexus that also occur in the spectrum of central injury (2). It seems likely that the underlying association with diffuse axonal injury explains the associations of intraventricular hemorrhage with a low (poor outcome) initial Glasgow Coma Score (which is not a cardinal number and, hence, is not appropriate to summarize, as in Table 3, as a mean including decimal points followed by either SD or SEM) and with a poor outcome. None of the patients whose initial coma score was over 8 showed hemorrhage affecting the 3rd or 4th ventricle; the only poor outcomes in this group were associated with an intracranial space occupying lesion. Patients whose initial coma score was under 8 and whose CT showed 3rd or 4th ventricular hemorrhage were worse (poor in 14 of 18 cases) than those patients with only a lateral ventricle involved (2 of 4). In nine of the group with 3rd or 4th ventricular hemorrhage, CT showed additional abnormalities commonly associated with diffuse axonal injury. Larger series will be needed to establish to what extent intraventricular bleeding has an independent association with outcome.

The association with diffuse axonal injury may also be the reason that drainage of any associated acute hydrocephalus rarely benefits the patient. This information is likely to be helpful to surgeons faced with the practical problem of making management decisions on patients with severe head injuries.

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KEY WORDS: Computed tomography; Head injury; Intraventricular hemorrhage; Ventricle

IMAGE GALLERY

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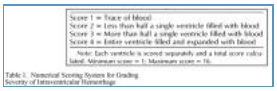

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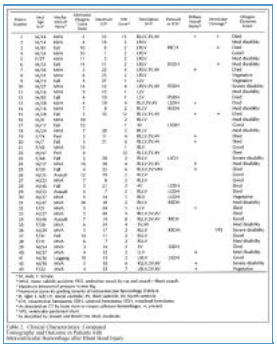

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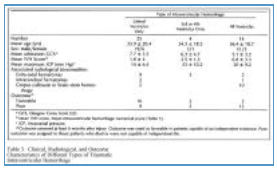

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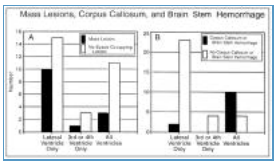

Figure 2

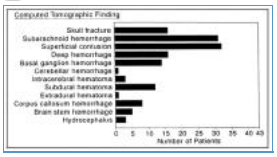

Figure 1

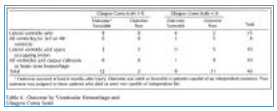

Table 4

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