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The authors correlated the computed tomographic (CT) findings and intracranial pressure (ICP) in 150 consecutive comatose head injury patients to determine if noninvasive CT can help identify the patients not requiring ICP monitoring. The study reveals that a majority of patients (55%) with hemorrhagic lesions shown by CT suffer from intracranial hypertension and require ICP monitoring for proper management. Of the patients with normal initial CT, 98% had normal ICP during the first 24 hr. Of the patients with normal CT, 15% developed intracranial hypertension later, irrespective of the initial Glasgow coma scale score or age. More than half of the patients who developed intracranial hypertension subsequently had normal ICP through the first 48 hr. The study indicates that ICP monitoring need not routinely be performed on admission on severe head injury patients with a normal CT. However, repeat CT at 24–48 hr before ICP monitoring in patients with initially normal CT may be valuable, particularly if their clinical status deteriorates.

Computed tomography (CT) is valuable for the detection of structural damage to the brain resulting from head injury [1–3]. It also obviates angiography and other invasive diagnostic procedures for the evaluation of most acute traumatic cerebral lesions that require immediate surgical intervention [4]. But its role in guiding nonsurgical management is less clear. Intracranial pressure monitoring is believed to be an important measure in the management and determination of prognosis of patients with severe head injury, especially in the immediate post-traumatic period. Several investigators have commented that continuous intra-cranial pressure (ICP) monitoring is essential for appropriate therapy [5–10]. However, intracranial pressure monitoring itself is associated with a certain morbidity [11, 12]. Since the structural damage detectable on CT and the pressure response of the brain to such damage are interdependent, we believe it is logical to evaluate the correlation between the two. Except for a few brief reports [13, 14], such a correlation has not been well documented.

As part of a major prospective study of patients with severe head injury who were managed by a standardized protocol and who underwent multiple sequential studies including computed tomography and ICP monitoring during the first 3 days or more, we correlated CT findings and intracranial pressure. This communication reports the results of that study and discusses the role of CT in identifying the patients who may not need continuous ICP monitoring.

Subjects and Methods

This series consists of 172 consecutive patients who were admitted to the Medical College of Virginia within 12 hr of severe head injury. The minimum criterion for entry into the study was that the patient was unable to utter coherent words or obey simple commands. The neurologic deficit ranged from this level to abnormal posturing and absent pupillary light reflexes and oculovestibular responses. Patients with cerebral death, alcohol or drug overdoses, and gunshot wounds were excluded.

TABLE 1: **CT/Intracranial Pressure Correlation in 137 Patients**

	CT (n = 137*)	Intracranial Pressure†, no. (%)			
		≤20 mm Hg	21–40 mm Hg	≥41 mm Hg	≥41 mm Hg and Uncontrollable ICP
Normal	47	39 (83)	7 (15)	1 (2)	0/1
Hematoma:					
Extracerebral	30	13 (43)	12 (40)	5 (17)	5/5
Intracerebral	39	17 (44)	13 (33)	9 (23)	6/9
Intra- and extracerebral					
hematoma	21	7 (33)	7 (33)	7 (33)	5/7

* Thirteen patients with lucent areas alone are excluded (see Results).
† Highest ICP recorded during the monitoring period.

Twelve patients did not have admission CT for one reason or another (equipment malfunction, or the patient was taken to surgery immediately) and ICP monitoring was not done in another 10 patients. This leaves 150 consecutive patients who had both the admission CT and ICP monitoring. They form the subject group of this report.

The age range was 1–72 years with 44 patients under 20 years, 60 patients between 20 and 39 years, 35 patients between 40 and 59 years and 11 over 60 (mean, 30.6 years; median, 25 years). CT was performed immediately on admission and then on days 3–5, day 14, and at 3 months and 1 year in surviving patients. Computed tomography was performed using the EMI Mark I Scanner with 160 × 160 matrix during the first 20 months and subsequently using a Delta 25 Scanner (Ohio Nuclear, Solon, Ohio) with a 256 × 256 matrix.

Intracranial pressure monitoring was delayed until the patient was stabilized by establishment of proper ventilation with intubation and adequate hemodynamics. After stabilization, ICP monitoring was begun using an intraventricular catheter or Richmond subarachnoid screw [15] and continued for at least 3 days or longer if necessary. The peak intracranial pressure reading during a 24 hr period was taken into account in each patient. ICP was considered normal if it was 20 mm Hg or less. Intracranial hypertension was considered to be present if the pressure was persistently above 20 mm Hg and was treated with hyperventilation and cerebrospinal fluid (CSF) drainage. Patients also underwent sequential neurologic examinations, electroencephalography, and multimodality evoked potentials on designated days.

All patients were managed by a standardized protocol with emphasis on early diagnosis and evacuation of intracranial mass lesions, artificial ventilation, control of ICP, and aggressive medical therapy [5, 6]. The outcome was graded at the end of 3 months or 1 year according to the recovery scale proposed by Jennett and Bond [16], and was considered to be good if the patient had little or some residual disability but suffered no impairment of daily activities. The outcome was considered to be poor if there was severe disability or worse.

Results

The initial CT findings in 137 patients are summarized in table 1. CT was normal in 47 patients. Thirty patients had extraaxial hematomas, 39 had intraaxial contusions or hemorrhages, and an additional 21 had both intra- and extraaxial hemorrhagic lesions. The intracranial pressure recorded in various categories of CT lesions is also shown in table 1 and the clinical outcome of the patients with various lesions on CT is summarized in table 2.

TABLE 2: **CT Findings and Outcome**

Initial CT Findings	No. Patients	Outcome*, no. (%)	
		Good	Poor
Normal	47	38 (81)	9 (19)
Hematoma:			
Extracerebral	30	14 (47)	16 (53)
Intracerebral	39	17 (43)	22 (57)
Intra- and extracerebral			
hematoma	21	7 (33)	14 (67)
Total	137	76	61

* Good: good to moderate; poor: severe disability to death.

Thirteen patients showed areas of decreased density on the admission CT without evidence of mass effect or associated hemorrhagic lesions and were considered to have "edema." In view of the well recognized relationship between posttraumatic edema and intracranial hypertension [5], it is necessary to clarify the CT findings in patients reported to have edema. The diagnosis of edema can be made without difficulty if low density zones surround an area of contusion or hematoma, but lucent areas not associated with hemorrhagic lesions should be viewed with caution. They may or may not represent posttraumatic edema.

It is not possible to differentiate the edema secondary to ischemic infarction and "true" posttraumatic edema on convential CT. The former does not have the same connotation as the latter for the management or outcome. Furthermore, ischemic infarction was seen in 90% of the autopsied head injury victims reported by Graham et al. [17]. The confusion regarding the diagnosis of edema was also pointed out in one recent report on a large series of head injury patients [2]. Of the patients who had CT performed within the first 24 hr after injury, 72% were diagnosed to have contusion compared to 83% who were considered as having edema on CT performed subsequently. This report shows how the resolving contusions may simulate edema. For these reasons, we have excluded the 13 patients with only lucent areas on CT from this discussion and will limit our correlation between CT findings and ICP only to the patients with unequivocally normal initial CT and those with hemorrhagic lesions.

It is beyond the scope of this paper to discuss the pathophysiology of posttraumatic intracranial hypertension.

However, it is well known that several factors play a role. The primary structural brain damage resulting in a mass lesion leads to intracranial hypertension, but the secondary insults like edema and subsequent compression of normal brain may lead to further brain dysfunction and elevation of intracranial pressure [5].

A high correlation is noted between the CT findings and intracranial pressure in this study. Of the 47 patients with normal initial CT 39 (83%) had intracranial pressure of 20 mm Hg or less. In seven patients, the pressure was 21–40 mm Hg during the monitoring period and none of them had uncontrollable intracranial hypertension. Only one had ICP over 41 mm Hg.

Patients with hemorrhagic lesions, whether extraaxial, intraaxial, or combined, had a higher incidence of elevated intracranial pressure and more often it was of moderate to severe intensity. Of the 30 patients with extraaxial hematomas, 12 (40%) had intracranial hypertension of 21–40 mm Hg and five (17%) had severe intracranial hypertension. The ICP was uncontrollable in all of the latter group.

Of the 39 patients with intracerebral hemorrhagic lesions, 13 (33%) had moderately increased ICP and nine (23%) had severe intracranial hypertension. Two thirds (6/9) of the latter group had uncontrollable hypertension.

Of the 21 patients with combined intra- and extraaxial hematomas 14 (67%) had elevated ICP. Seven (33%) had ICP over 41 mm Hg and five (71%) of these seven had uncontrollable ICP. In summary, patients with hemorrhagic lesions tend to have a higher incidence of elevated ICP with more of them having severe intracranial hypertension. Thus, it is clear from the results that patients with hemorrhagic lesions require immediate ICP monitoring so that appropriate treatment may be initiated.

However, the occasional development of intracranial hypertension in some patients with normal initial CT needs further analysis to determine the need for immediate ICP monitoring. Related factors like initial Glasgow Coma Scale (GCS) score, secondary complications, and ultimate clinical outcome are considered in the following section.

Normal CT, Elevated ICP, and Outcome

Of the 47 patients with initially normal CT, eight (17%) had elevated ICP during the monitoring period. The average age of these eight patients was 37 years, higher than the average of 25 years in the group but not statistically significant ($\chi^2 = 2.12$). Six of the eight patients had a good outcome. Their average age was 35 years and the initial GCS score was 6 or less in three (table 3). (GCS range is 3–15; persons with low scores are poorer neurologically (coma is deeper); persons with high scores are better. People with scores of 4 or less have poor neurologic functions and outcome compared with persons with a score of 8 or more, who have a good outcome.) The ages of the two patients who had a poor outcome were 39 and 48. The admission coma scores were 5 and 11 (table 3) and were not significantly different from those of the six patients with good outcome ($\chi^2 = 0.67$).

One patient developed bilateral diffuse cerebral edema as

TABLE 3: **Clinical Course and Outcome in Eight Patients with Normal CT and Increased ICP**

Outcome/ Case No. (age, gender)	GCS	Complications
Good:		
1 (48, M)	7	Wound infection, bleed
2 (52, F)	9	IADH
3 (42, M)	6	None
4 (24, F)	5	Gastrointestinal bleed, diabetes mellitus
5 (24, F)	7	None
6 (24, M)	4	Pulmonary infection, meningitis, ventriculitis
Poor:		
7 (39, M)	11	Pulmonary infection and edema, meningitis, ventriculitis, gastrointestinal bleed, renal failure, IADH, delayed hematoma
8 (48, F)	5	Pulmonary infection, IADH, cerebral edema on 3d day CT, cardiac arrest

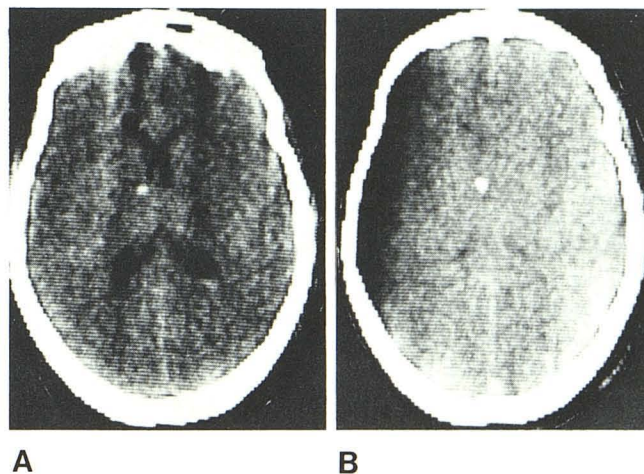Note.—IADH = inappropriate antidiuretic hormone response.

A   **B**

Fig. 1.—**A**, On admission. Normal, except for intraventricular catheter. **B**, Third day. Nonvisualization of ventricles due to diffuse edema. ICP remained normal through first 3 days and was elevated on fourth day.

demonstrated by nonvisualization of the lateral ventricles on the third day CT (fig. 1). She went on to develop intracranial hypertension on the fourth day and was noted to have uncal herniation at autopsy. The other patient had delayed traumatic intracerebral hematoma shown on the third day CT. He developed meningitis and ventriculitis and succumbed to Gram-negative septicemia. His ICP was persistently elevated on the third day. Similar secondary complications were less common in six of the patients with elevated ICP but who had a good outcome. Only one of the six in this group had the combination of pulmonary dysfunction, meningitis, and ventriculitis. Half (3/6) of this group had a poor coma score, 6 or less.

In the 39 patients with normal CT and ICP, seven had secondary complications and poor outcome (table 4). One patient succumbed to septicemia and Gram-negative shock and two died of liver laceration. Two others suffered from

TABLE 4: **Clinical Course in Seven Patients with Normal CT and ICP but Poor Outcome**

Case No. (age, gender)	GCS	Complications
1 (28, M)	4	Anoxia, pulmonary infection delayed hematoma
2 (55, F)	4	Septicemia
3 (25, F)	6	Meningitis, ventriculitis, gastrointestinal bleed, pulmonary infection, IADH
4 (57, F)	6	Pulmonary infection
5 (27, M)	7	Pulmonary infection, gastrointestinal bleed
6 (62, M)	5	Hyperthermia, meningitis, septicemia
7 (22, M)	3	Hyperthermia, pulmonary infection, cardiac and respiratory arrest

Note.—IADH = inappropriate antidiuretic hormone response.

TABLE 5: **Patients with Normal CT and No ICP Monitoring, Good Outcome**

Case No. (age, gender)	GCS	Complications
1 (14, M)	8	Multiple fractures and retroperitoneal hematoma
2 (43, M)	10	Tracheostomy
3 (34, M)	7	None
4 (25, M)	6	Rib fracture, pneumothorax
5 (16, F)	9	None
6 (18, F)	9	Anoxia
7 (25, M)	NA	Multiple fractures, CSF otorrhea, meningitis
8 (17, F)	7	Anoxia, pulmonary infection
9 (6, M)	9	Pelvic fracture, pneumothorax, pulmonary edema, gastrointestinal bleed, IADH, diabetes mellitus
10 (19, M)	7	None

Note.—NA = not available. IADH = inappropriate antidiuretic hormone response.

meningitis and ventriculitis in addition to other complications. One of these two developed multifocal edema on the third day CT. The remaining two had severe pulmonary dysfunction, one of whom had *Klebsiella* pneumonia. The other had delayed hematoma on the third day CT.

The secondary complications were far less common in the 32 patients with normal CT and ICP who had a good outcome. Only three patients in that group had severe pulmonary complications and only one had septcemia. Thus, secondary complications seem to be significant ($p < 0.0001$) in the outcome of the patients with normal CT whether or not they have elevated ICP.

The average age of the 32 patients with normal CT and ICP and a good outcome was 22 years, compared to 39.5 years for the patients with poor outcome. The good outcome in the younger age group is not surprising and has been discussed by Bruce et al. [18]. The coma score in the seven patients with poor outcome was 6 or less in six as compared to a similar score in five of the 32 patients with normal CT and ICP and good outcome. Thus the coma score alone remains a significant factor in predicting the outcome ($p < 0.01$).

Discussion

The few reports correlating CT and ICP are somewhat contradictory in their conclusions [13, 14]. Sadhu et al. [13] observed that no patient with a normal initial CT had intracranial hypertension. Another report concluded that no reliable relation exists between the morphologic pictures as shown on CT and ICP, and warned about the reliability of the correlation between the ICP level and CT findings [14].

Our results suggest that some clinically useful assumptions can be made from the CT findings. They indicate that a majority of patients—55% or more—with CT-demonstrated hemorrhagic lesions do have intracranial hypertension in the immediate posttraumatic period and need ICP monitoring for proper management.

However, 83% of the patients with normal initial CT did not have intracranial hypertension. More important is the fact that 98% of these patients had normal ICP during the first 24 hr after trauma and 91% did not have an increase in ICP until the third day or later.

The complication rate associated with ICP monitoring is estimated to be 2.1%–10.0% [11, 12, 19]. In our study, four (9%) of the 47 patients with normal CT had ventriculitis and/or meningitis. Although three of the 47 had either a basal skull fracture or a compound fracture of the calvarium, the four patients who developed ventriculitis and/or meningitis had an intact calvarium on admission. Only one of 10 patients with normal CT who did not have ICP monitoring during the study had meningitis (table 5). Meningitis in this patient was attributed to his basal skull fracture and CSF otorrhea.

Because of the high degree of correlation between normal CT and ICP, and because complications attributable to ICP monitoring developed in 9% of these patients, we believe ICP monitoring need not be routinely performed on admission in patients with normal CT. More than half of the patients who developed intracranial hypertension did not do so until the third day or later, and two of them had new hemorrhagic lesions on the third day CT. Another patient had bilateral diffuse edema. A recent report points out that two-thirds of the severe head injury patients who deteriorate neurologically after 48 hr have new lesions on CT [20]. If neurologic status does not improve or shows deterioration, CT should be repeated to detect new lesions [21–23] that may cause intracranial hypertension requiring ICP monitoring. Likewise, with a satisfactory clinical course, both CT and intracranial pressure monitoring can be safely deferred.

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