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Cranial computed tomography (CT) scans of 1,600 head trauma patients, 67 of which demonstrated evidence of brainstem injury, were reviewed. CT diagnosis of brainstem injury was based on direct and indirect evidence. Direct signs, which include focal hemorrhage, significant intraparenchymal contrast enhancement, hemorrhagic contusion, and edema of the brainstem, appear as areas of high density, mixed density, and low density on the CT scan. Indirect signs are obliteration of the pontine, cerebellopontine angle, and perimesencephalic cisterns. Mortality and morbidity rates after brainstem injury are 2–3 times greater than for head trauma with descending transtentorial herniation, but without brainstem injury.

The clinical manifestations and pathologic findings of brainstem injury have been well described. Head trauma carries a much graver prognosis when brainstem involvement is present [1–10]. Since severe head trauma is often characterized by injury to several sites, both intra- and extraaxial, there may be no clear-cut clinical evidence of a specific brainstem lesion. The most significant lesion may not be suspected until the patient fails to exhibit normal signs of recovery or it may be an unexpected autopsy finding [1–5, 11–16].

Because of its ability to demonstrate the nature, extent, sites, and multiplicity of brain injury, computed tomography (CT) is now the primary diagnostic method for head trauma. Although there have been numerous reports on the CT findings of most types of intracranial injury, the findings in brainstem injury have not been well described [17, 18]. We reviewed our experience in CT diagnosis of brainstem injury and conclude that the diagnosis and prognosis can often be appreciated at the time of the initial evaluation.

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Materials and Methods

During a 2 year period more than 1,600 patients with head trauma were examined in our hospital with an EMI CT-1010 head scanner. In most patients a noncontrast scan was followed immediately by a contrast-enhanced scan with infusion of 300 ml of 30% meglumine diatrizoate (Reno–M–DIP). Brainstem injury was diagnosed prospectively and retrospectively in 67 cases, and CT findings were correlated with clinical manifestations and pathologic findings. A supplementary group of 100 patients having head trauma with downward transtentorial herniation (based on clinical and CT findings) but no CT evidence of brainstem injury were also studied. Of the 67 brainstem cases with secondary injury (resulting from downward transtentorial herniation or posterior fossa hematoma), 21 were autopsy proved. Of the 67 who showed primary brainstem injury (a direct injury without significant additional trauma), 11 were proved at autopsy.

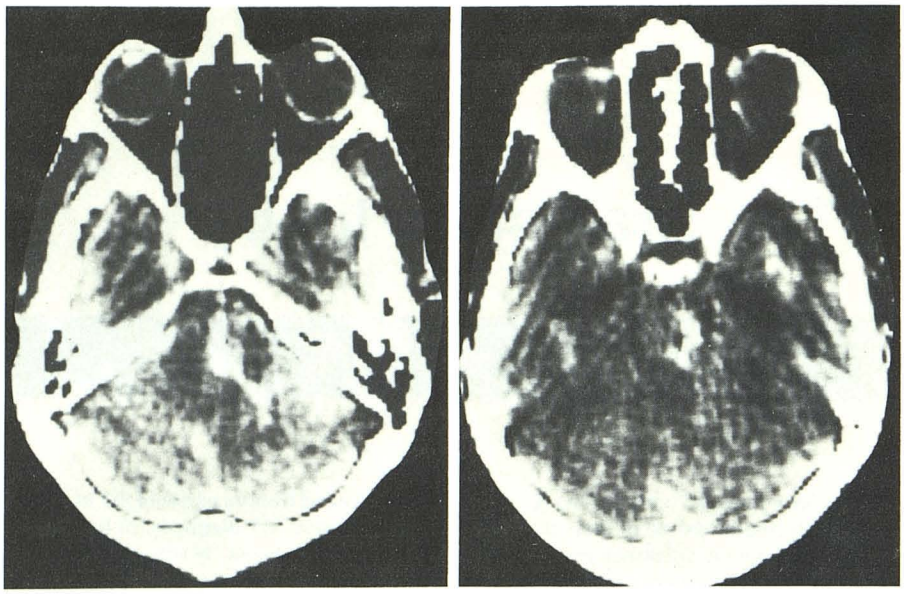


Fig. 1.—Case 1. **A**, Hemorrhage at pons and vermis. Right cerebellopontine angle cistern visible. **B**, Hemorrhage at upper pons; perimesencephalic cisterns visible (primary brainstem injury with intact surrounding cisterns).

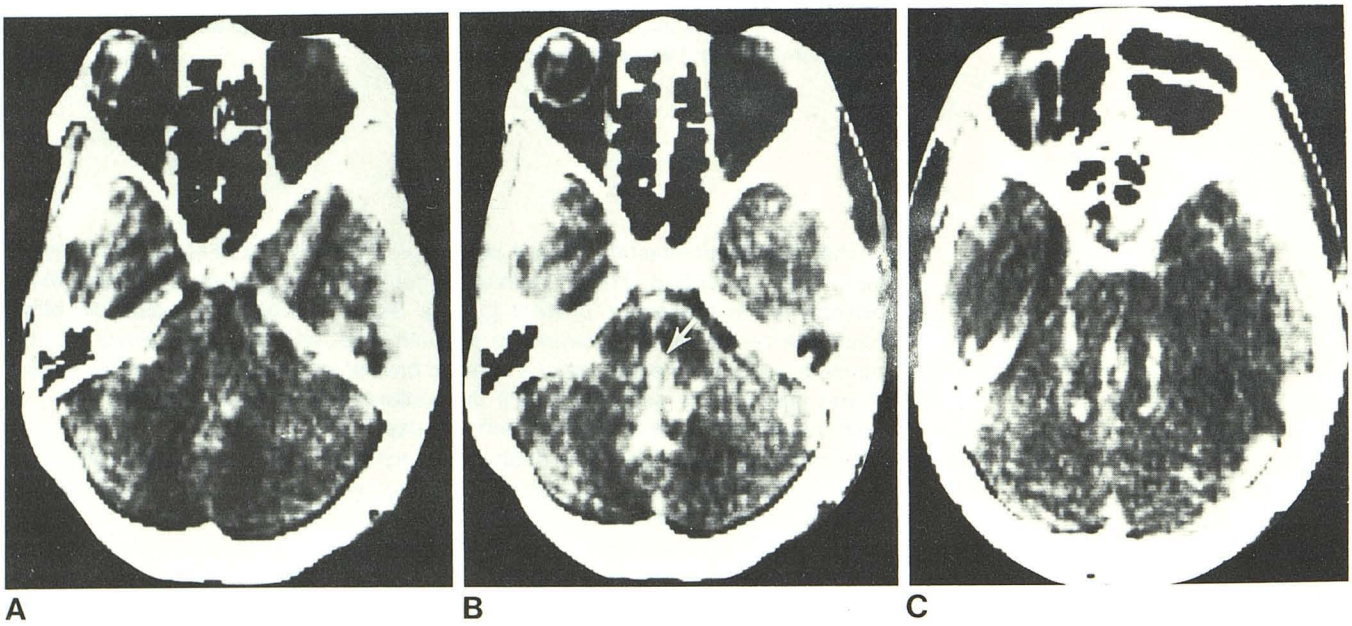


Fig. 2.—Case 2. **A**, Hemorrhagic density, central posterior fossa, possibly at vermis. Fourth ventricle not seen. **B**, Enhanced scan. Enhancing contusion at brainstem (*arrow*). **C**, Superior slice of enhanced scan. Obliteration of cisternal spaces.

Representative Case Reports

Case 1

A 38-year-old male intoxicated driver involved in an automobile accident was brought to the emergency room. He was unresponsive and exhibited decerebrate posturing. The corneal and oculocephalic reflexes were absent. Retinal hemorrhage and papilledema were noted on the right. After a respiratory arrest, he was maintained on a respirator. CT demonstrated several traumatic hemorrhages of the cerebrum, cerebellum, and brainstem (fig. 1). He died shortly after admission.

Case 2

A 44-year-old man with head trauma was comatose when treated by paramedics. On arrival at the hospital, his pupils were asymmetrical (5 mm on the left and 6 mm on the right) and sluggishly reactive to light. Corneal and occulocephalic reflexes were absent. CT demonstrated small bilateral subdural hematomas (fig. 2). There was a small hemorrhagic density in the center of the posterior fossa, and the pontine and perimesencephalic cisterns were not visible. Since CT findings were incompatible with the clinical manifestations, a contrast-enhanced study was recommended. After infusion of contrast material, enhancing contusions in the pons, midbrain,

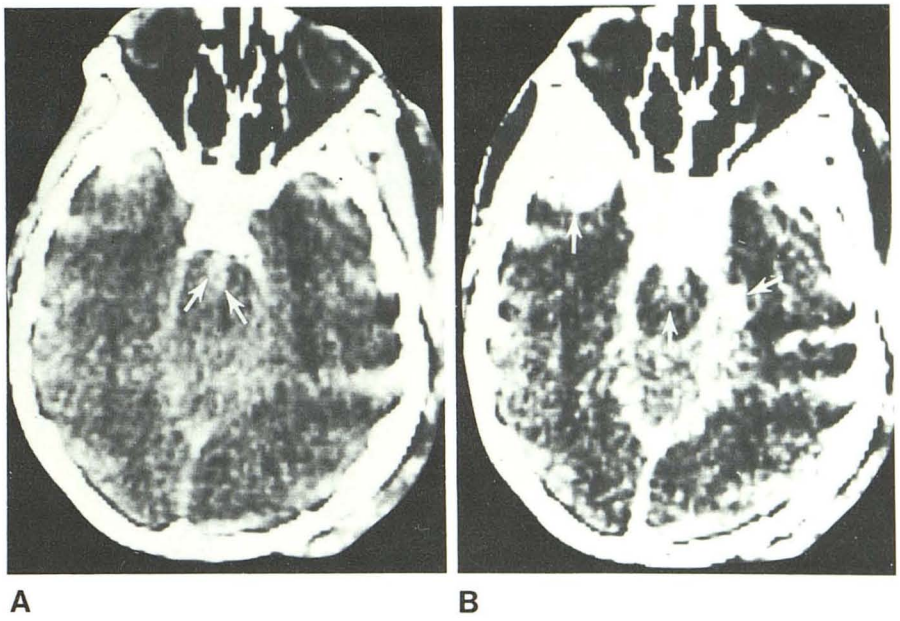


Fig. 3.—Case 3. **A**, Noncontrast scan. Small hemorrhage in region of midbrain (*arrows*). Obliteration of perimesencephalic and prepontine cisterns. **B**, Contrast-enhanced scan. Enhanced contusions in right uncus, pons, and left temporal tip (*arrows*).

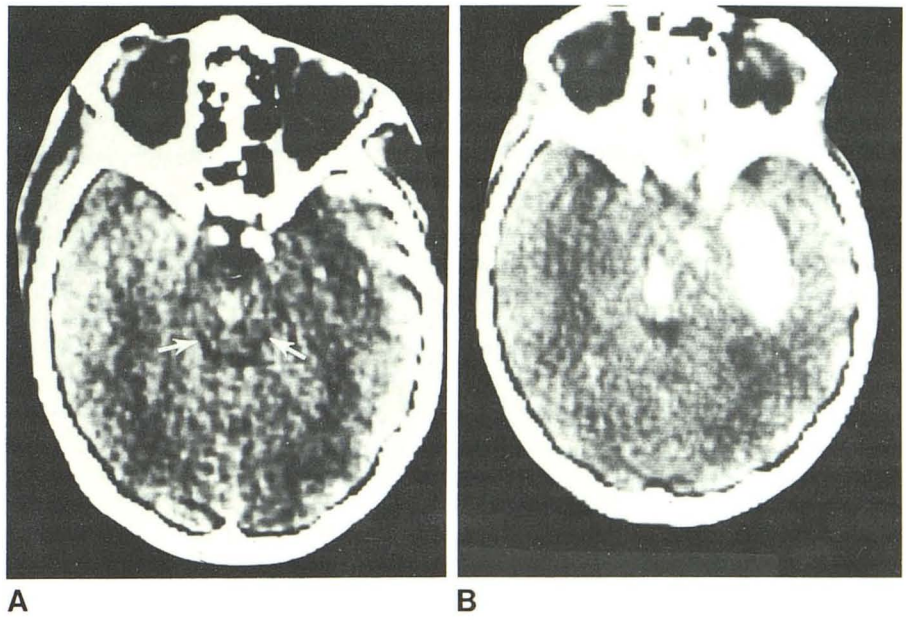


Fig. 4.—Case 4. **A**, Hemorrhage in central part of brainstem. Prepontine and mesencephalic cisterns intact (*arrows*). **B**, 1 day later. Increased hemorrhage in brainstem. Obliteration of mesencephalic and prepontine cisterns. Hemorrhage in right temporal lobe.

and vermis became apparent. The patient progressively deteriorated over the next 48 hr and died. CT findings of bilateral subdural hematomas and brainstem and vermian contusion were confirmed at autopsy.

Case 3

A 21-year-old male motorcycle-accident victim was found at admission to have fixed, dilated pupils with no corneal and oculocephalic reflexes. A right hemotympanum was noted. The noncontrast CT scan showed no supratentorial lesion or midline shift (fig. 3). A small area of hemorrhagic density was present in the midportion of the pons. The pontine, cerebellopontine angle, and perimesencephalic cisterns were obliterated. Contrast-enhanced scans demonstrated enhancing contusions in the pons and the left temporal tip. The patient died 2 days after admission. CT findings were confirmed at autopsy.

Case 4

A 24-year-old woman was admitted after severe head trauma sustained in an automobile accident. She was comatose, unresponsive to pain, and decerebrate. Her pupils were fixed and dilated and her gaze disconjugate. The occulocephalic reflex was present, but

the corneal reflex was absent. Her Glasgow coma score was 5. A CT scan was negative with the exception of a small hemorrhagic density in the midportion of the upper pons (fig. 4). The pontine, cerebellopontine angle, and perimesencephalic cisterns were intact. The patient's clinical status remained unchanged for 24 hr, then deteriorated. Repeat CT disclosed an apparent increase in the size of the brainstem hemorrhage, as well as a hemorrhage within the right basal ganglia and obliteration of the same cisterns. The patient died 2 weeks later. CT findings were confirmed at autopsy.

Case 5

A 7-year-old boy was struck by an automobile. He became comatose, unresponsive to pain, and had no spontaneous respiration. His pupils were fixed and dilated. Corneal and occulocephalic reflexes were absent. CT showed no supratentorial lesion, but there was a small area of hemorrhagic contusion of the brainstem, with swelling and obliteration of the pontine, cerebellopontine angle, and perimesencephalic cisterns (fig. 5). The patient died several hours later. Brainstem contusion was found at autopsy.

Case 6

A 44-year-old woman was brought to the emergency room in a coma. She had a respiratory arrest and was placed on a respirator. Her pupils were fixed and dilated, and she responded to pain with decorticate posturing. The corneal and occulocephalic reflexes were absent. CT demonstrated a subdural hematoma with midline shift and transtentorial herniation (fig. 6). The perimesencephalic, pontine, and cerebellopontine angle cisterns were obliterated and the brainstem appeared isodense. The patient died 2 days later. Subdural hematoma and transtentorial herniation were confirmed at autopsy. Hemorrhage was noted in the midbrain.

Case 7

A 21-year-old man suffered head trauma in a motorcycle accident. Examination on admission revealed fixed, dilated pupils. He had no spontaneous respirations, and was intubated. He was areflexic and flaccid. CT showed massive hemorrhage into the midline cerebellar structures and obliteration of the pontine, cerebellopontine angle, and perimesencephalic cisterns (fig. 7). The brainstem appeared hypodense. He died several hours later. Autopsy showed petechial hemorrhage in the pons, in addition to the cerebellar hemorrhage. (Cf. fig. 9.)

Case 8

A 31-year-old man was transferred to our emergency room from another hospital where he was reported to be alert and oriented after head trauma. Examination in our hospital revealed his pupils to be fixed and dilated, and he responded to painful stimuli with decerebrate posturing, more vigorous on the right. He had very shallow spontaneous respirations. The corneal and occulocephalic reflexes were absent. The patient was intubated and placed on a respirator. CT demonstrated a huge right epidural hematoma and transtentorial herniation (fig. 8). The right perimesencephalic, pontine, and cerebellopontine angle cisterns were obliterated. The brainstem area was isodense. He was taken immediately to surgery and the hematoma was evacuated. Postoperatively, his condition was unchanged, and he died 9 days later. Minimal residual extradural hematoma, multiple foci of midbrain, and pontine hemorrhage were found at autopsy. Evidence of transtentorial herniation was also noted.

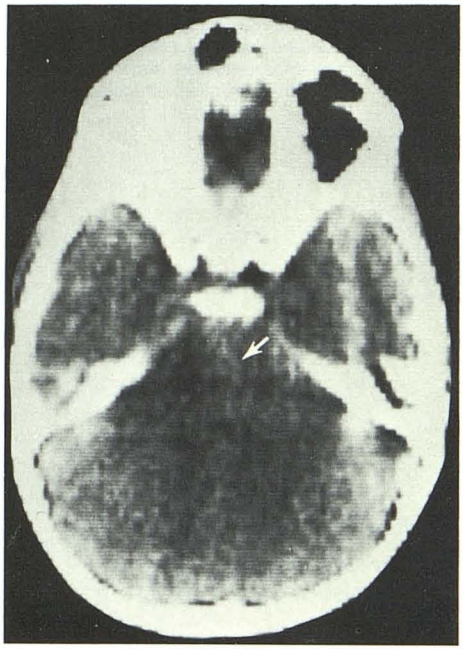


Fig. 5.—Case 5. Minimal hemorrhagic contusion, central pons (*arrow*). Swelling of pons, with complete obliteration of pontine, right cerebellopontine angle, and perimesencephalic cisterns. Small fourth ventricle. Only partial visualization of left cerebellopontine angle cistern.

Results and Discussion

Brainstem injuries occur often in cases of fatal cranial trauma. Such injuries may be primary (occurring at the time of the original trauma) or secondary (the result of mechanical stress within the brainstem produced by downward transtentorial or direct compression) [17, 18]. Since a primary brainstem injury may be associated with widespread injury to the rest of the brain, and since transtentorial herniation may produce secondary brainstem injury very soon after the initial insult, it is difficult to distinguish between them with certainty [1–6, 8, 9, 11, 15, 16, 19–21]. However, differentiation between primary and secondary injuries may be useful since only some cases have secondary brainstem injury after severe transtentorial herniation [22–25]. Also, some cases of autopsy-proven brainstem injury have no evidence of transtentorial herniation. Our analysis revealed a dismal prognosis in patients with CT evidence of both transtentorial herniation and brainstem injury, but a better prognosis with transtentorial herniation alone.

The 67 patients in our study included 19 patients with primary brainstem injury and 48 with secondary injury. They were 1½–73 years old with average age of 32.

Only 12 of the 67 patients demonstrated significant brainstem hemorrhage on CT scans. Six brainstems were felt to be purely edematous, as demonstrated by uniform low density on CT. Mixed densities were seen in 11 brainstems; six of these had surrounding edema. Uniformly isodense brainstems were seen in 38 of the 67 patients. Of these isodense lesions, 11 demonstrated contrast enhancement.

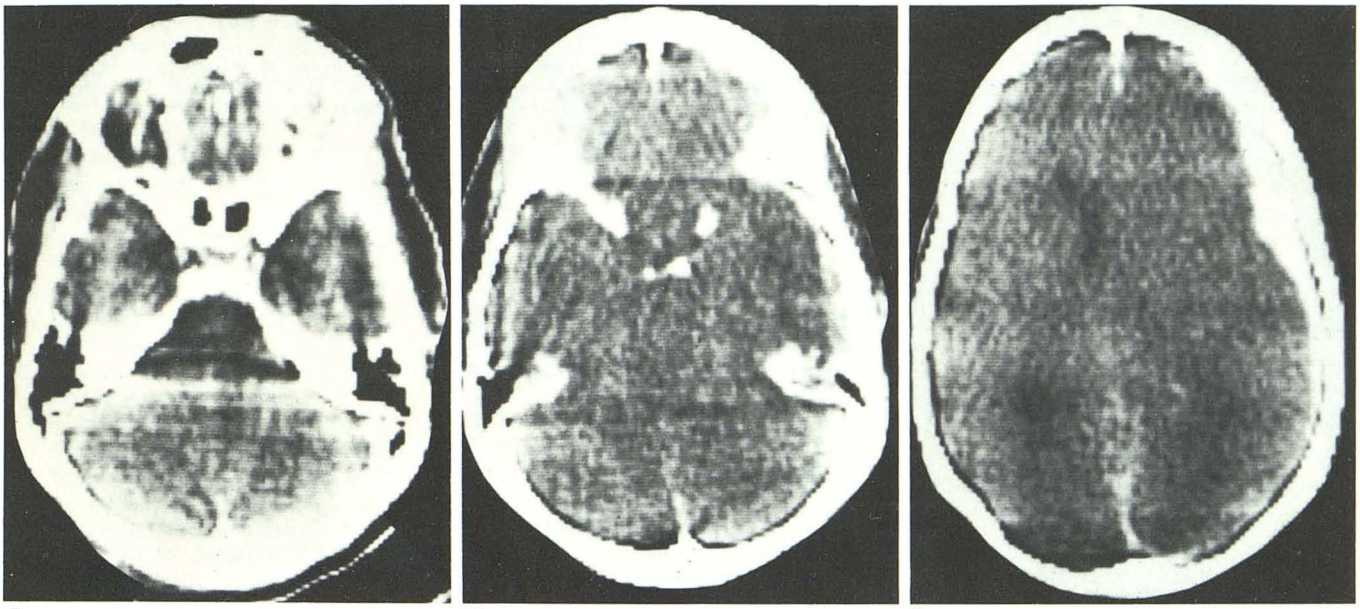


Fig. 6.—Case 6. **A**, Partial obliteration of prepontine cistern and fourth ventricle. **B**, Isodense brainstem, but obliteration of prepontine and perimesencephalic cisterns. Neither temporal horn dilated. **C**, Subdural hematoma along left anterior and middle cranial fossa, with displacement of midline structures toward left side.

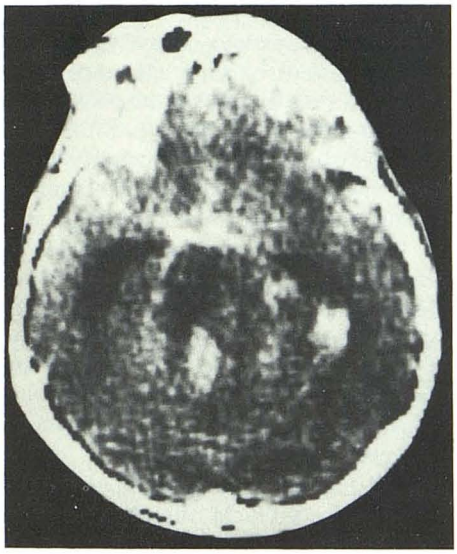


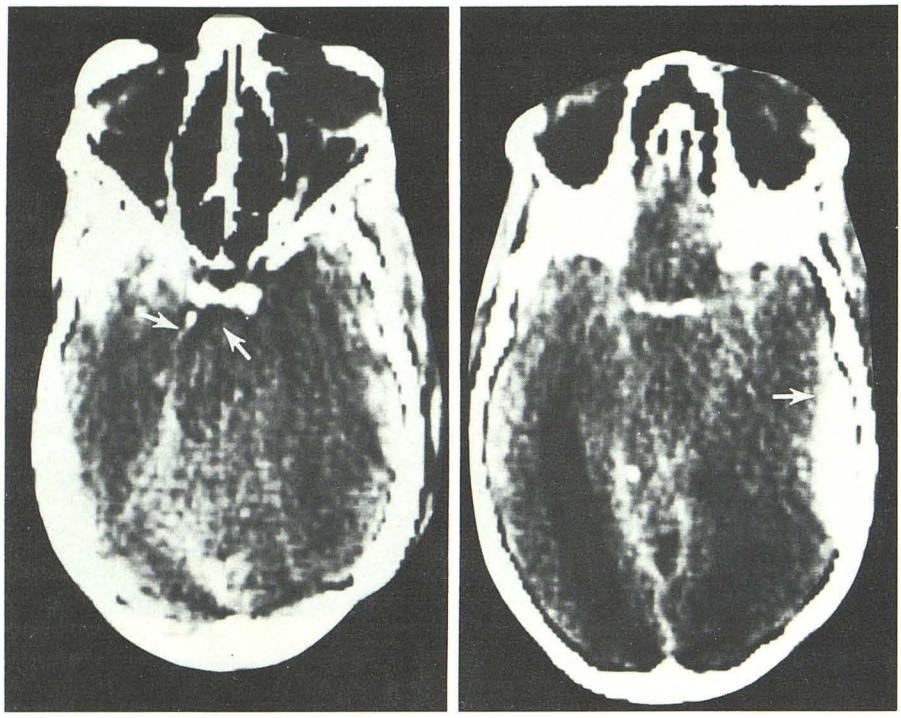
Fig. 7.—Case 7. Noncontrast scan. Multiple hemorrhage at vermis and right hippocampal region and subarachnoid hemorrhage. Deformation and foreshortening of edematous pons (low density). Temporal horns dilated.

Correlation of CT and autopsy findings indicates that injury to the brainstem may include hemorrhage, contusion, and/or edema. Hemorrhage may be unifocal or multifocal, and the extent of hemorrhage ranges from petechial to massive bleeds involving much of the brainstem cross section. These brainstem lesions may occur alone or in association with other cranial injuries. CT manifestations of brainstem injury are similar to those of cerebral injury. They may be hyperdense, isodense, or hypodense depending on the degree of hemorrhage and edema [18].

The CT diagnosis of primary brainstem injury requires demonstration of direct evidence, such as hemorrhage, edema, or contusion, or indirect evidence, such as complete obliteration of the pontine, cerebellopontine angle, and perimesencephalic cisterns. We believe the indirect findings constitute reliable signs of brainstem injury in head trauma patients, and can be seen in either primary or secondary brainstem injuries. Similar findings have been noted in brainstem neoplasms, but these are easily differentiated by clinical history. CT signs for downward transtentorial herniation have been previously described as contralateral temporal-horn dilatation, obliteration of the suprasellar cistern, and widening of the ipsilateral cerebellopontine angle cistern [22–24, 26].

Recognition of the type of brainstem injury may have some value in the management of severe head trauma. Among 48 patients with secondary brainstem injury, the mortality rate was 67% (32/48), and the rate of minimal recovery with resultant vegetative state was 29% (14/48). The other 4% (2/48) survived with moderate but significant deficits. Of our supplementary group of 100 patients with transtentorial herniation alone, the mortality rate was only 27%, and the rate of minimal recovery was 12%. The other 61% had mild to moderate deficits. In addition, of the 27 deaths, four were the result of intercurrent infection after a period of good recovery, not the direct result of the head injury. Thus, the survival rate in transtentorial herniation alone was at least 73%.

The mortality rate for secondary brainstem injury was 2–3 times greater than for patients with transtentorial herniation alone. Of the 19 patients with primary brainstem injuries, five survived, but in a permanent vegetative state. The mortality rate was 73.7% (14/19), compared with 67% in the secondary brainstem injury group. The rate of minimal



A B

Fig. 8.—Case 8. **A**, Partial visualization of left cerebellopontine angle cistern, but obliteration of prepontine and right-sided cerebellopontine angle and perimesencephalic cisterns. Contusion, right temporal lobe. **B**, Epidural hematoma (*arrow*). Obliterated perimesencephalic cisterns.

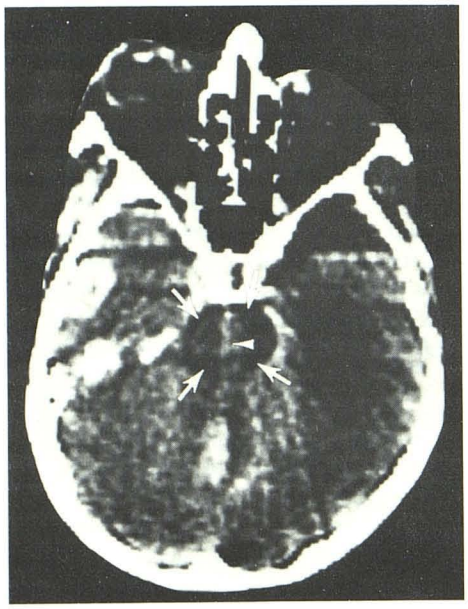


Fig. 9.—Hematoma at vermis and deformed brainstem, but prepontine, perimesencephalic, and cerebellopontine angle cisterns intact (*arrows*); artifact at pons (*arrowhead*). Patient survived with moderate deficit.

recovery is very similar to that of secondary injury. During the survival period, follow-up scans often showed low attenuation in the region of the brainstem, with widening of the surrounding cisterns. This was most likely due to evolution of the hemorrhage and to posttraumatic atrophy as the result of contusion [20, 21, 26].

Among those patients with complete obliteration of the pontine, cerebellopontine angle, and perimesencephalic cisterns or with enhanced contusion and partial obliteration of the cisterns, about one-third had autopsy proof of midbrain and/or pontine hemorrhage. In those patients without complete obliteration of the cisterns or enhanced contusion who received autopsies, no brainstem hemorrhage was identified. Therefore, we feel that complete obliteration of the cisterns or enhanced contusion is a valuable CT sign of brainstem injury in head trauma patients. The level of brainstem injury may be correlated with the parts of the cisterns that are obliterated. The perimesencephalic cisterns are obliterated with injury at the level of the midbrain, and pontine lesions may result in obliteration of the pontine and cerebellopontine angle cisterns.

Although CT scanning has an excellent capability for evaluation of brainstem injury, artifacts often obstruct our efforts. In addition, obliteration of the cisterns may be due to small amounts of subarachnoid hemorrhage in the cisterns, which appear isodense against adjacent brain. In this situation, careful evaluation of the size of the fourth ventricle may be helpful. A small fourth ventricle may also indicate brainstem swelling (case 5). If there is still a question, a contrast-enhanced scan may provide further evidence of brainstem injury (cases 2 and 3).

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