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Prospective Comparative Study of Intermediate-Field MR and CT in the Evaluation of Closed Head Trauma

Lindell R. Gentry^{1,2}
 John C. Godersky³
 Brad Thompson¹
 Val D. Dunn^{1,4}

Forty patients with closed head trauma were evaluated prospectively with CT and intermediate-field-strength MR imaging to compare the diagnostic efficacies of the two techniques. Traumatic lesions were detected in 38 patients. The severity of injury, as determined by the Glasgow Coma Scale, ranged from 3 to 14. The sensitivities of CT and MR were calculated for all subgroups of lesions: (1) hemorrhagic and nonhemorrhagic intraaxial lesions (diffuse axonal injury, cortical contusion, subcortical gray-matter injury, primary brainstem injury); (2) extraaxial hematomas (subdural, epidural); and (3) diffuse hemorrhage (subarachnoid, intraventricular).

CT and MR (T1- and T2-weighted) studies were both highly and comparably sensitive in the detection of hemorrhagic intraaxial lesions. MR scans, however, were much more sensitive in detecting nonhemorrhagic lesions. Cortical contusions and diffuse axonal injury constituted 91.9% of all intraaxial lesions. The sensitivities of the imaging techniques for this combined group of lesions were (1) nonhemorrhagic lesions (CT = 17.7%, T1-weighted MR = 67.6%, T2-weighted MR = 93.3%); (2) hemorrhagic lesions (CT = 89.8%, T1-weighted MR = 87.1%, T2-weighted MR = 92.5%). MR was also significantly better in detecting brainstem lesions (CT = 9.1%, T1-weighted MR = 81.8%, T2-weighted MR = 72.7%). The sensitivities of the diagnostic studies in the detection of extraaxial hematomas were CT = 73.2%, T1-weighted MR = 97.6%, T2-weighted MR = 90.5%). Intraventricular hemorrhage was consistently seen with all three imaging studies, but subarachnoid hemorrhage was detected much more frequently with CT.

In summary, MR has clear advantages over CT in evaluating closed head trauma. Although its sensitivity in detecting hemorrhagic lesions is similar to that of CT, it is much better than CT in detecting nonhemorrhagic lesions, which are more prevalent. MR is more useful than CT in classifying primary and secondary forms of injury and directing treatment. CT's one advantage over MR is its ability to more rapidly assess unstable patients who may need surgery.

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¹ Department of Radiology, University of Iowa Hospitals and Clinics, Iowa City, IA 52242.

² Present address: Department of Radiology, University of Wisconsin Hospital and Clinics, 600 Highland Dr., Madison, WI 53792. Address reprint requests to L. R. Gentry.

³ Department of Surgery, Division of Neurosurgery, University of Iowa Hospital and Clinics, Iowa City, IA 52242.

⁴ Present address: Department of Radiology, American Fork Hospital, American Fork, UT 84003.

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During the past decade, CT has contributed significantly to the management of patients with closed head trauma [1-9]. CT has improved the survival and reduced the morbidity of patients with closed head trauma, primarily through the early recognition and treatment of extracerebral hemorrhage [1, 2, 5-9]. CT has been less beneficial, however, in the recognition and management of other types of cerebral injury [3-5, 9-14]. In the acute phase, CT findings often do not correlate with neurologic impairment or the level of consciousness as measured by the admission Glasgow Coma Scale (GCS) [9-15]. Although certain CT findings are associated with a poor outcome [3, 16-22], the extent of posttraumatic recovery of neurologic and cognitive function is generally not reliably predicted from the pattern of injury on the initial CT scan [5, 9-13]. The lack of correlation of CT findings with the acute and chronic sequelae of closed head trauma is due, in part, to the insensitivity of CT to many types of cerebral injuries [3, 9-11, 19]. Although MR imaging has been shown to be more sensitive than CT in detecting many diseases involving the CNS, it has not been extensively used for the evaluation of acute closed head trauma. A few reports deal only with subacute or chronic posttraumatic extracerebral hematomas [23-26]. A number of investigators have

studied the MR appearance of hemorrhagic lesions, but many of these lesions were not posttraumatic in nature [27–33]. A few series of patients with closed head trauma have also been studied by MR [34–39]. But one of these series was very small [35], and others included patients in the subacute/chronic phase of injury [37–39]. A large prospective comparative study of CT and intermediate-field-strength MR has not been reported. In this study, we prospectively evaluated 40 patients with acute closed head trauma to determine (1) the feasibility of MR for evaluating these patients and (2) the relative sensitivities of CT and MR for detecting various types of cerebral injuries.

Subjects and Methods

A prospective comparative study of the usefulness of CT and MR for the evaluation of 40 patients with acute closed head trauma was initiated after approval of the protocol by our hospital's institutional review board. Inclusion in the study required a thorough neurologic evaluation and determination of the level of consciousness (GCS) [40] by an experienced neurosurgeon or neurologist within 6 hr of admission to the hospital. Patients who experienced transient loss of consciousness without focal neurologic deficits and who had complete recovery of consciousness within the first 12 hr were excluded from the study. Also excluded were patients who died shortly after hospitalization, those with contraindications to MR scanning, and those with multisystem trauma who were medically unstable. The age of the patients ranged from 1 month to 82 years (mean, 26.6 years). Eight female and 32 male patients constituted the study group. The severity of injury, as measured by the admission GCS, ranged from 3 to 14 (mean, 9.8).

The MR scans were obtained as soon as possible after injury but only after the patients were considered stable. The interval from injury to MR evaluation was 2–19 days (mean, 7.6; median, 5–6). The intervals between CT and MR studies for the study population are summarized in Table 1. Those patients with the most severe injuries were usually evaluated after the intracranial pressure had stabilized. Twelve patients were examined with MR while being mechanically ventilated with a fluidic ventilator, as recently described by Dunn et al. [41]. This was accomplished by modification of a Monaghan 225 SIMV Volume Ventilator* to eliminate ferromagnetic material from the supporting stand.

Nonenhanced CT scans were obtained in all patients within the first 24 hr after trauma by using a fourth-generation scanner.† A slice thickness of 8 mm was used in all instances. MR was performed with a 0.5-T cryogenic system.‡ Thirty-nine patients were examined with both T1- and T2-weighted pulse sequences while one patient was studied only with a T2-weighted sequence. Scans were obtained with a contiguous interleaved multislice technique with a slice thickness of 10 mm. The T2-weighted scans were obtained with a spin-echo sequence with a repetition time (TR) of 2300–2900 msec and echo time (TE) of 80–120 msec. T1-weighted scans were obtained in 37 patients with an inversion-recovery sequence with a TR of 2000–2300 msec and inversion time (TI) of 500–600 msec and in 12 patients with a spin-echo sequence (TR = 400–1000 msec, TE = 25–40 msec). Two or more imaging planes were used in 34 of 40 patients. Images were obtained in the axial plane in 39 patients, while the coronal and sagittal planes were used in 31 and 5 patients, respectively.

* Monaghan Medical Corp., Plattsburg, NY.

† Picker 600/1200 scanner (Picker International, Highland Heights, OH).

‡ Picker International, Highland Heights, OH.

TABLE 1: Time Interval Between Head Trauma and CT and MR Scans

No. of Days	No. of Patients		
	Injury–MR Scan	First CT–MR Scan	Closest CT–MR Scan ^a
0	0	1	4
1	0	6	9
2–5	21	20	21
6–8	11	8	4
9–12	4	4	1
13–19	4	1	1

^a The CT scan closest to the time of the MR scan.

TABLE 2: Classification and Frequency of Primary Traumatic Brain Lesions in 40 Patients

Type: Location	No. of Patients ^a	Total No. of Lesions
Intraaxial lesions:		
Diffuse axonal	34	149
Cortical contusion	31	135
Subcortical gray matter	8	14
Primary brainstem	10	11
Total	38	309
Extraaxial hematoma:		
Subdural	26	39
Epidural	3	3
Total	27	42
Diffuse hemorrhage:		
Subarachnoid	20	20
Intraventricular	11	11
Total	25	31

^a Some of the 40 patients had more than one type of lesion.

The MR and CT scans were analyzed independently, and all visualized abnormalities were classified [42]. An attempt was made to differentiate between injuries resulting from the initial traumatic force (primary) and those arising from delayed posttraumatic (secondary) causes such as diffuse brain swelling, brain displacement and herniation, delayed hemorrhage, cerebral infarction, and diffuse hypoxic injury [42]. In many instances it was difficult to separate primary and secondary damage. For purposes of statistical analysis, however, all focal abnormalities that were not obviously secondary in nature were classified as primary lesions (Table 2). Primary intracranial lesions were classified as (1) intraaxial lesions; (2) extraaxial hematomas (subdural, epidural); and (3) diffuse hemorrhage (intraventricular, subarachnoid). Intraaxial lesions were subclassified as diffuse axonal injury (white-matter "shear" injury), cortical contusion, subcortical gray-matter injury, or primary brainstem injury. All intraaxial lesions were further separated into hemorrhagic and nonhemorrhagic subsets.

We used the following criteria to classify a lesion as hemorrhagic on MR. On T1-weighted images we required that the shortening of T1 by the paramagnetic properties of methemoglobin be of sufficient degree that the hematoma was at least partially hyperintense relative to white matter (Fig. 1). On T2-weighted images we required the

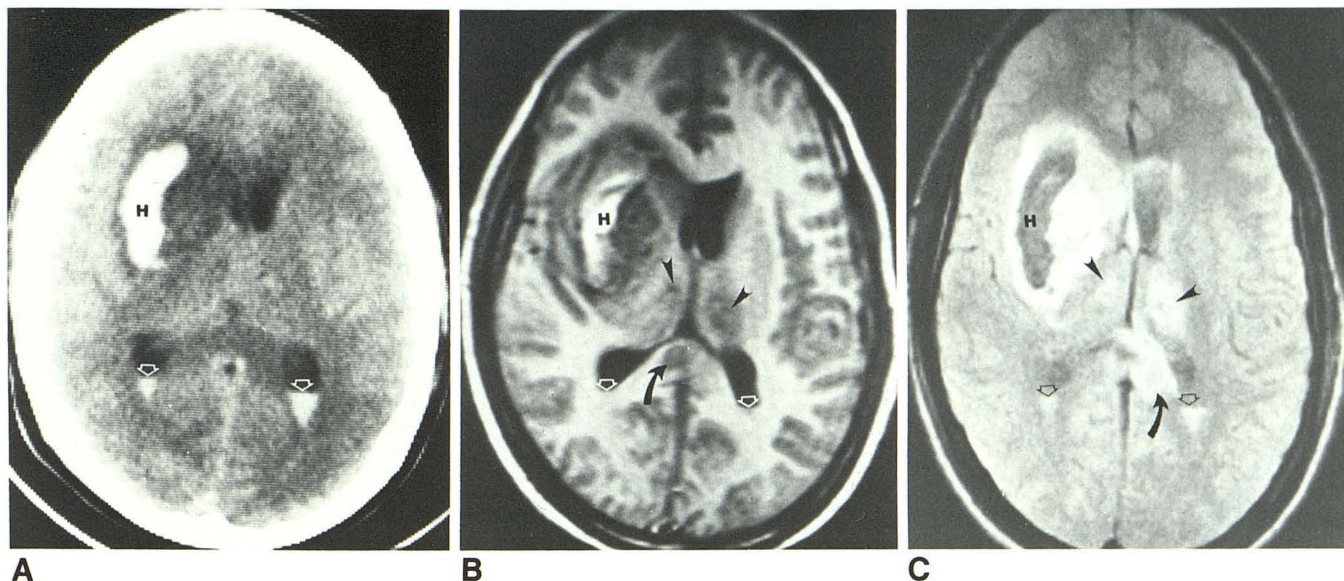


Fig. 1.—19-year-old man 2 days after head trauma.

A, Axial CT scan shows large basal ganglia intracerebral hematoma (H) with surrounding edema as well as intraventricular hemorrhage (arrows). B, and C, T1-weighted inversion-recovery (TR = 2100 msec, TI = 600 msec) (B) and T2-weighted spin-echo (TR = 2300 msec, TE = 80 msec) (C) scans confirm hematoma and intraventricular hemorrhage (open arrows). Large nonhemorrhagic diffuse axonal lesion is in splenium of corpus callosum (curved arrows), and bilateral subcortical gray matter lesions (arrowheads) are seen. Note.—Acute hematoma is hyperintense relative to brain parenchyma on T1-weighted image and hypointense on T2-weighted image. Intraventricular hemorrhage is hyperintense relative to CSF on T1- and T2-weighted scans and isointense relative to white matter on T1-weighted scan.

presence of central hypointensity within the hematoma. As recently described by Gomori et al. [30], central hypointensity in acute hematomas appears to be secondary to preferential T2 proton relaxation enhancement caused by methemoglobin or deoxyhemoglobin within intact RBCs (Figs. 1 and 2).

After initial individual analysis, the CT and MR scans were compared and the total number of lesions in each category determined. Since autopsy confirmation of the lesions was only possible in one patient, we required that a lesion be seen on more than one study (CT, T1-weighted MR, T2-weighted MR) or imaging plane (axial, sagittal, coronal) to be regarded as a true traumatic lesion. Equivocal abnormalities seen on only one study or imaging plane were omitted from further analysis. The sensitivities of CT and MR were determined from the tabulated data. Separate calculations were made for each MR pulse sequence, imaging plane, and category of intraaxial and extraaxial lesion. The influence of hemorrhage on lesion detectability was also assessed.

Results

Intraaxial Lesions

The most common type of traumatic lesion identified in this series was diffuse axonal injury [21, 42–46]. These lesions have also been called shear lesions [3, 45] or diffuse damage to the white matter of the immediate-impact type [10, 13, 46]. These lesions typically were less than 1 cm in greatest dimension, spared the adjacent cortical surface of the brain, and were located entirely within white matter or at the gray-/white-matter interface. The vast majority (81.2%) of these lesions were nonhemorrhagic (Table 3). In the nonhemorrhagic subset there was a marked difference in the sensitivi-

ties of the three imaging studies. T2-weighted MR was most sensitive, detecting 92.4% of lesions. CT was very insensitive, detecting only 19% of lesions. Diffuse axonal lesions were typically seen by CT only if larger than 1.5 cm or located in the corona radiata or internal capsule. Small nonhemorrhagic lesions in other locations were rarely seen by CT (Figs. 2 and 3). T1-weighted MR was also more sensitive (72.3%) than CT (19.0%) but less sensitive than T2-weighted MR. For the hemorrhagic subset, the sensitivities of all three imaging methods were quite high and very similar (T2-weighted MR, 91.3%; T1-weighted MR, 86.2%; CT, 85.7%) (Table 3).

The second most common type of traumatic lesion seen in our series was that involving the cortical surface of the brain (cortical contusion) [13, 43]. These lesions were usually much larger than diffuse axonal lesions, often being 2–4 cm in greatest dimension (Figs. 4–6). By definition, they were primarily localized to the cortical surface of the brain with relative sparing of the underlying white matter. There was a great tendency for confluence of the lesions and associated hemorrhage. Hemorrhage was detected in 51.8% of cortical contusions in this series (Table 3). All three imaging methods were very sensitive in the detection of hemorrhagic cortical contusions (T2-weighted MR, 92.4%; T1-weighted MR, 87.5%; CT, 91.4%). The true size of the lesion (hemorrhagic and nonhemorrhagic components), however, was usually underestimated by both CT and T1-weighted MR (Figs. 4–6).

T2-weighted MR was significantly more sensitive (95.0%) in the detection of nonhemorrhagic cortical contusions than was T1-weighted MR (58.3%) or CT (15.4%) (Figs. 4–6). In some instances a lesion could be indirectly suspected on CT

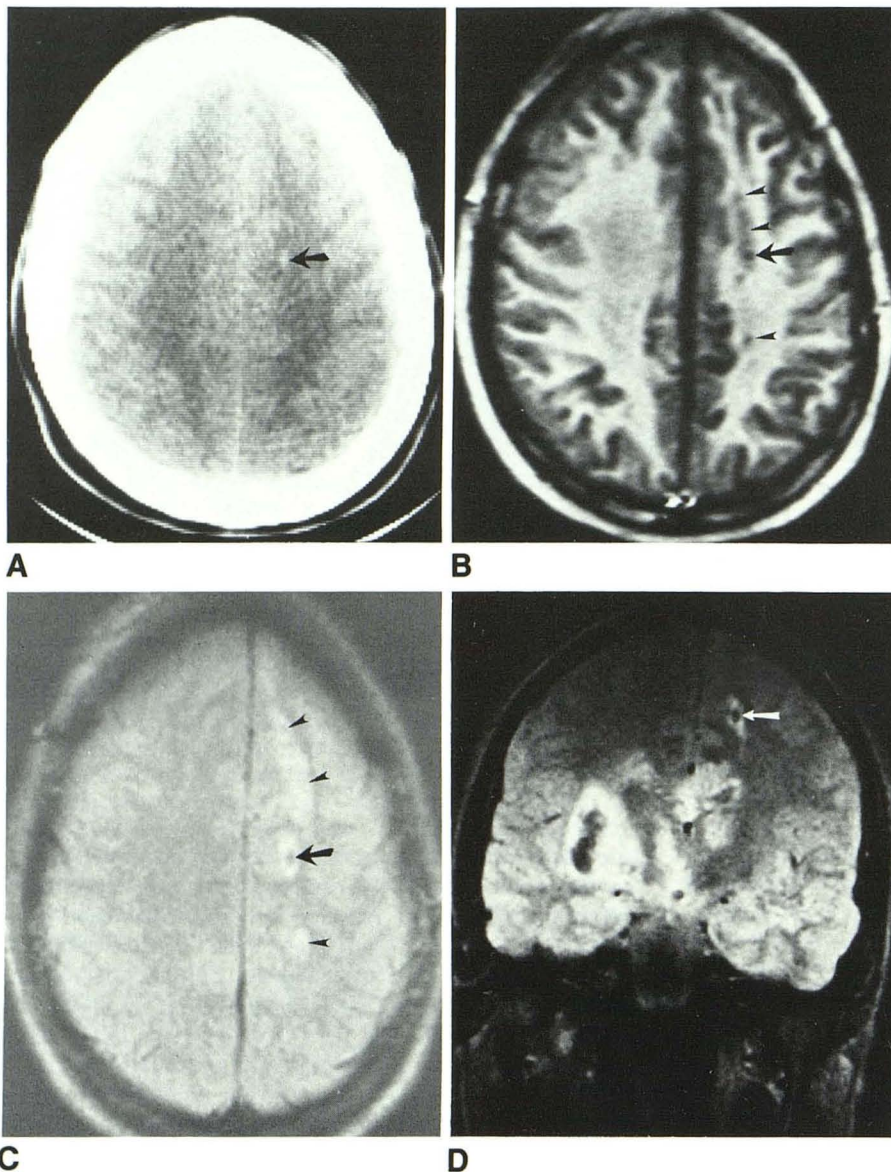


Fig. 2.—19-year-old man.

A, Axial CT scan 1 day after head trauma shows very subtle 0.5-cm hemorrhagic lesion (arrow) consistent with diffuse axonal injury in deep white matter.

B–D, Axial T1-weighted inversion-recovery MR image (TR = 2100 msec, TI = 600 msec) (B), axial T2-weighted spin-echo MR image (TR = 2300 msec, TE = 80 msec) (C), and coronal T2-weighted spin-echo MR image (TR = 2300 msec, TE = 80 msec) (D) 2 days after head trauma confirm hemorrhagic diffuse axonal lesion (arrows) as well as multiple other nonhemorrhagic diffuse axonal lesions (arrowheads).

The presence of acute hemorrhage is indicated on T2-weighted images (C and D) by central hypointensity (arrows). Note that the small hemorrhage is not hyperintense 2 days after trauma on T1-weighted image, as methemoglobin has not yet formed (D). Also present are multiple other hemorrhagic and nonhemorrhagic lesions.

scans by local mass effect (ventricular or sulcal effacement), but the lesion itself was often not apparent. This was because the contrast between the normal and injured cortex was typically very low on CT scans. The relative insensitivity of T1-weighted MR was from very low contrast differences between normal cortex; surrounding CSF; and edematous, contused cortex (Fig. 5). The T1-weighted spin-echo images were slightly more sensitive than inversion-recovery images in lesion detection, although the latter were superior for anatomic delineation.

Only 14 lesions were confined entirely to the subcortical gray matter. These involved the thalamus and lentiform nucleus (Fig. 1) and were similarly distributed between the hemorrhagic and nonhemorrhagic subsets (Table 4). The pattern of sensitivities for this small but clinically important [13, 44] number of lesions was similar to that for diffuse

axonal injury and cortical contusion. Hemorrhagic lesions were detected accurately by all three examinations, while nonhemorrhagic lesions were poorly visualized with CT.

Eleven brainstem lesions, thought to be primary lesions, were identified in 10 patients. All but one of the lesions were initially detected with MR. The lesion that was not seen initially by MR was identified retrospectively. T1- and T2-weighted images were similarly sensitive, detecting nine and eight lesions, respectively (Table 4). CT identified only one (hemorrhagic) brainstem lesion.

Extraaxial Hematomas

Thirty-nine subdural hematomas were identified in 26 patients. Thirty were supratentorial and located along the convexity, eight were interhemispheric or along the tentorium,

TABLE 3: Sensitivities of CT and MR in the Detection of Cortical Contusions and Diffuse Axonal Injury

Lesion: Study	True Positive/Total = % Sensitivity		
	Diffuse Axonal Injury (<i>n</i> = 149)	Cortical Contusion (<i>n</i> = 135)	Total (<i>n</i> = 284)
Nonhemorrhagic	<i>n</i> = 121	<i>n</i> = 65	<i>n</i> = 186
CT	23/121 = 19	10/65 = 15.4	33/186 = 17.7
T1-weighted MR:			
Axial	53/68 = 77.9	21/38 = 55.3	75/106 = 69.8
Coronal	33/51 = 64.7	14/22 = 63.6	47/73 = 64.4
Both views	86/119 = 72.3	35/60 = 58.3	121/179 = 67.6
T2-weighted MR:			
Axial	114/121 = 94.2	62/65 = 95.4	176/186 = 94.6
Coronal	43/49 = 87.8	33/35 = 94.3	76/84 = 90.5
Both views	157/170 = 92.4	95/100 = 95	252/270 = 93.3
Hemorrhagic:	<i>n</i> = 28	<i>n</i> = 70	<i>n</i> = 98
CT	24/28 = 85.7	64/70 = 91.4	88/98 = 89.8
T1-weighted MR:			
Axial	18/22 = 81.8	39/43 = 90.7	57/65 = 87.7
Coronal	7/7 = 100	24/29 = 82.8	31/36 = 86.1
Both views	25/29 = 86.2	63/72 = 87.5	88/101 = 87.1
T2-weighted MR:			
Axial	26/28 = 92.9	65/70 = 92.9	91/98 = 92.9
Coronal	16/18 = 88.9	32/35 = 91.4	48/53 = 90.6
Both views	42/46 = 91.3	97/105 = 92.4	139/151 = 92.5

Note.—Some totals are greater than the number of lesions because more than one scan plane was used for some T1- and T2-weighted pulse sequences.

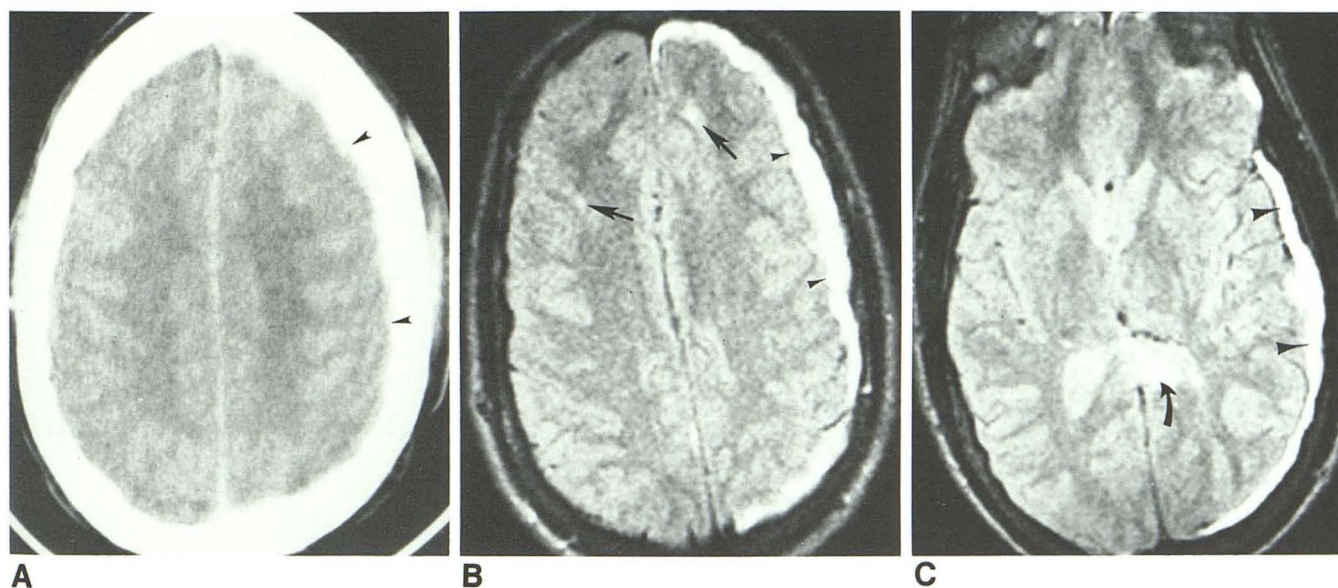


Fig. 3.—18-year-old man.

A, Axial CT scan 1 day after cranial trauma appears normal except for very subtle subdural hematoma (arrowheads).

B and C, T2-weighted spin-echo MR images (TR = 2300 msec, TE = 80 msec) 9 days after trauma. Subdural hematoma (arrowheads) is more easily appreciated on MR. Also present are focal nonhemorrhagic diffuse axonal lesions at gray-white-matter interface (straight arrows) of frontal lobe and in splenium of corpus callosum (curved arrow), not apparent on CT.

and one was infratentorial. T1-weighted MR identified all but one (97.4%) 2-mm interhemispheric lesion that was isointense relative to adjacent brain parenchyma. T2-weighted MR identified 89.7% of the subdural lesions, missing four isointense

lesions that were less than 3 mm thick. All lesions missed by T2-weighted MR, however, were hyperintense on T1-weighted MR and easily visible.

Only 27 (71.1%) of 38 subdural hematomas were detected

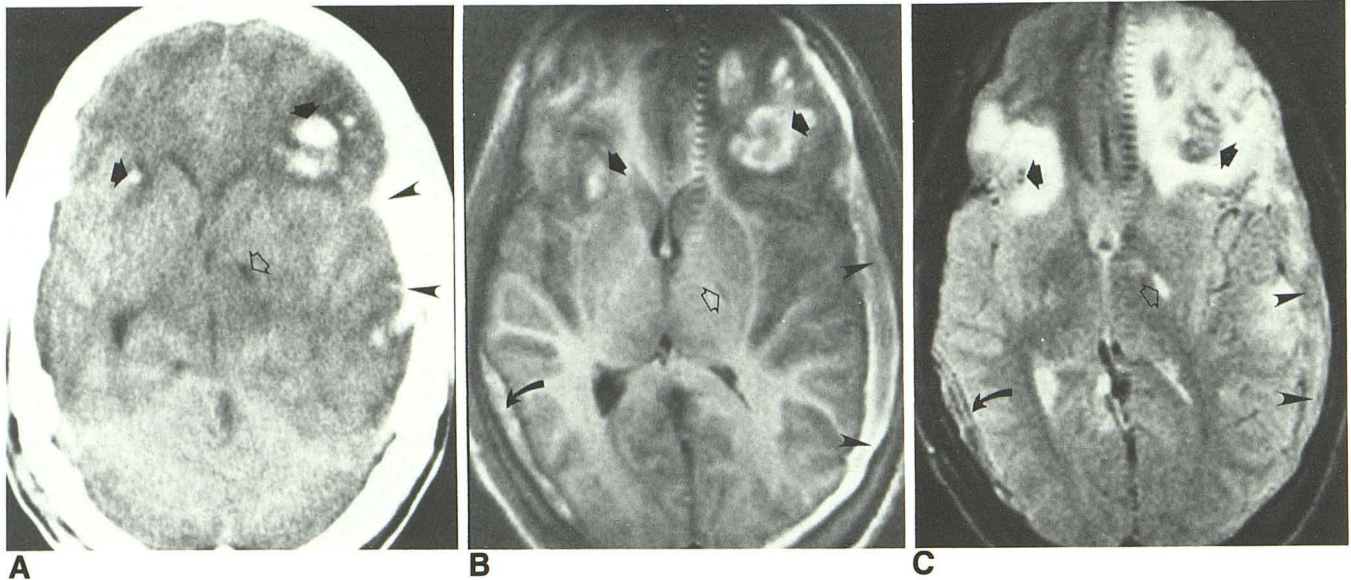


Fig. 4.—17-year-old man.

A, Axial CT scan 1 day after cranial trauma reveals left subdural hematoma (arrowheads), bilateral hemorrhagic cortical contusions of inferior aspects of frontal lobes (solid arrows), and nonhemorrhagic diffuse axonal lesion of internal capsule (open arrow).

B and C, T1-weighted inversion-recovery MR image (TR = 2100 msec, TI = 600 msec) (B) and T2-weighted spin-echo MR image (TR = 2300 msec, TE = 80 msec) (C) 5 days after trauma confirm left subdural (arrowheads) and small right subdural (curved arrows) hematomas. Bifrontal hemorrhagic cortical contusions (straight solid arrows) and nonhemorrhagic diffuse axonal internal capsule lesion (open arrows) are also seen. Extent of contusions is seen more clearly on T2-weighted scan. Also note hyperintensity of acute subdural hematoma on T1-weighted image but relative isointensity on T2-weighted image.

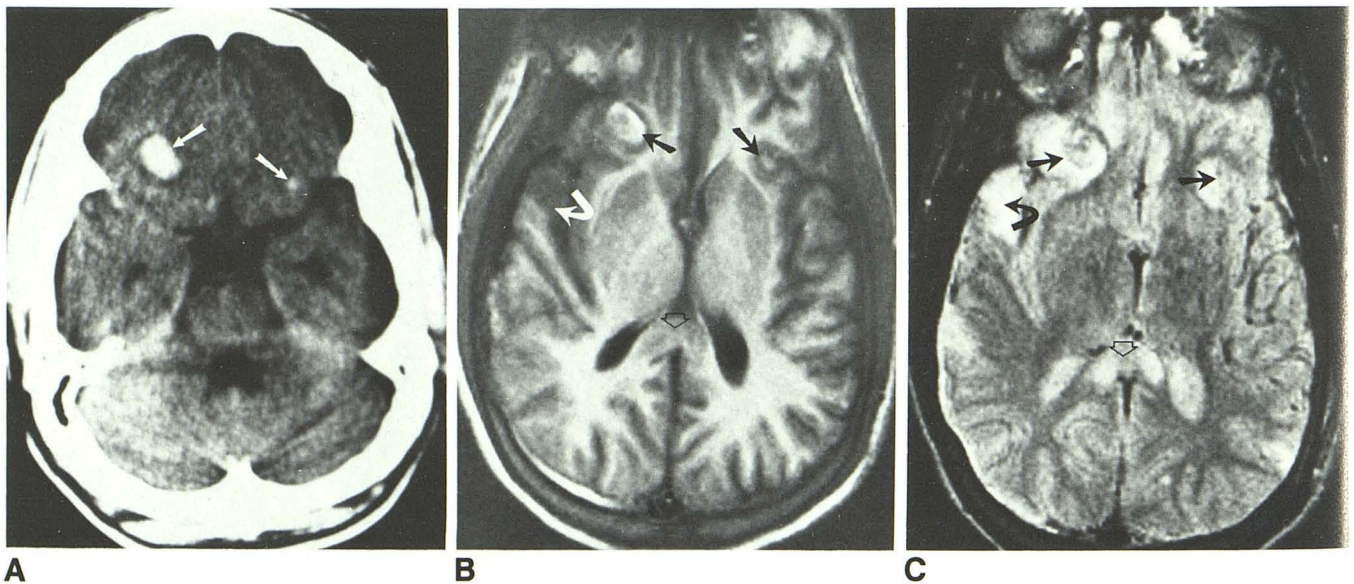


Fig. 5.—25-year-old man.

A, Axial CT scan 1 day after cranial trauma reveals bifrontal hemorrhagic contusions (arrows).

B and C, T1-weighted inversion-recovery MR image (TR = 2100 msec, TI = 600 msec) (B) and T2-weighted spin-echo MR image (TR = 2300 msec, TE = 80 msec) (C) 5 days after trauma confirm bifrontal hemorrhagic contusions (straight solid arrows). There is also a nonhemorrhagic contusion of temporal lobe (curved arrows) and nonhemorrhagic diffuse axonal lesion in corpus callosum (open arrows). On T1-weighted image, note low level of contrast between edematous and normal temporal lobe cortex (curved arrow).

on the initial CT scan. All of the 11 subdural hematomas that were missed by CT were considered clinically insignificant. Only one of 11 missed subdural hematomas was more than 5 mm thick; most were 1–3 mm. Five of these 11 subdural

hematomas were visualized retrospectively when CT scans were compared with MR scans. One large hematoma (14 mm) had obviously developed between the initial CT and MR examinations and was excluded from statistical consideration.

Fig. 6.—18-year-old man.

A and B, Axial CT scans 1 day after trauma reveal acute epidural hematoma (arrows) and several small frontotemporal petechial hemorrhagic contusions (arrowheads). Follow-up CT scan 2 days later (not shown) was unchanged.

C, Coronal T1-weighted inversion-recovery MR image (TR = 2100 msec, TI = 600 msec) 6 days after trauma reveals epidural hematoma (curved arrow) similar in intensity to adjacent parenchyma. It also shows adjacent temporal lobe hemorrhagic contusion (straight arrow).

D and E, Axial T2-weighted MR images (TR = 2300 msec, TE = 80 msec) also show acute epidural hematoma (curved arrows), which is iso-/hypointense relative to brain. Displaced dura (straight solid arrow) is seen between hematoma and adjacent brain. T2-weighted scans more clearly show extent of nonhemorrhagic zones of injury around small frontotemporal petechial hemorrhagic contusions (arrowheads). Multiple nonhemorrhagic left frontotemporal contusions are also seen (open arrows).

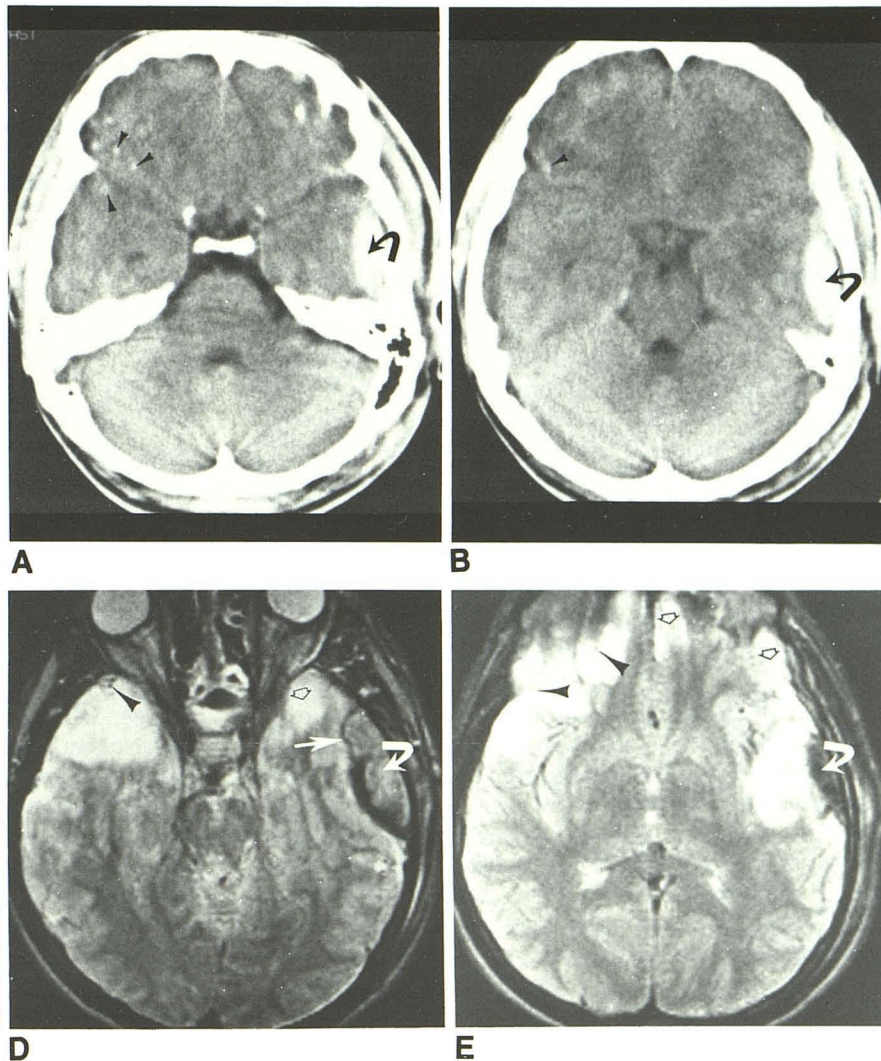


TABLE 4: Relative Sensitivities of CT and MR in the Detection of Primary Brainstem and Subcortical Gray-Matter Lesions

Type of Lesion	No.	True Positive/Total = % Sensitivity		
		CT	T1-Weighted MR	T2-Weighted MR
Primary brainstem	11	1/11 = 9.1	9/11 = 81.8	8/11 = 72.7
Subcortical gray	14	8/14 = 57.1	10/14 = 71.4	14/14 = 100.0

All three epidural hematomas identified in this series were larger than 5 mm in thickness and were readily seen by CT and T1- and T2-weighted MR (Fig. 6).

Diffuse Hemorrhage

In 20 patients there was enough subarachnoid blood after trauma to be recognizable on CT. In seven instances only a small amount of blood was present adjacent to a hemorrhagic cortical contusion. The blood was diffuse in 10 cases, localized to the sylvian fissure in two instances, and confined to the interpeduncular cistern in one patient. T2-weighted MR detected only those three focal clots within the sylvian and

interpeduncular cisterns. We did not identify any subarachnoid clots with T1-weighted MR scans in this series.

Eleven of 40 patients had evidence of intraventricular hemorrhage. The majority of the patients had only a trace of blood and it was limited to the occipital horns, although in one case blood was also present in the third ventricle. Intraventricular hemorrhage was seen with T2-weighted MR in all 11 instances, with CT in nine cases, and with T1-weighted MR in five of seven patients whose scans included the region in which the blood was localized. In four patients, coronal T1-weighted scans were not obtained through the occipital horns. Intraventricular hemorrhage was always visualized on T2-weighted spin-echo scans (TR = 2300 msec, TE = 80 msec)

as an area of hyperintensity within the dependent portion of the occipital horns (Fig. 1). On T1-weighted inversion-recovery scans (TR = 2100 msec, TI = 600 msec), the blood was always hyperintense relative to CSF and, depending on its age, either iso- or hyperintense relative to white matter.

Discussion

The best diagnostic imaging test for the evaluation of traumatic brain injury should possess a number of characteristics. It should be highly sensitive and specific, provide useful anatomic information for guiding therapeutic intervention, allow classification of the type of injury, offer accurate prognostic information, and be easily and safely performed in severely injured patients. In the last decade CT revolutionized the care of patients with closed head trauma because it represented a significant improvement over previous diagnostic methods [1–9]. Despite its significant contribution to the management of closed head trauma, CT suffers from a number of diagnostic limitations. Numerous authors have noted a marked discrepancy between the clinical severity of injury and the CT findings in the acute setting [9–15, 34]. CT findings often correlate poorly with focal neurologic deficits, as well as with more global indicators of cerebral injury such as intracranial pressure and level of consciousness. Autopsy and histopathologic studies confirm the relative insensitivity of CT to many forms of neural injury [13, 16, 43, 44, 46]. The primary deficiency of CT is its insensitivity to small nonhemorrhagic lesions (Table 3). These lesions are difficult to discern because the difference in X-ray attenuation between normal and injured brain is small, resulting in a low level of contrast between these tissues (Figs. 1, 2, and 6). Since many nonhemorrhagic lesions are not detected, therapeutic decisions must be made without complete delineation of the extent of injury. Previously, CT has been used mainly to diagnose “surgical” lesions. Choosing proper “medical” therapy and judging the prognosis of recovery has been primarily based on the clinical manifestations of injury (GCS, intracranial pressure, neurologic examination) [34, 37].

Early experience with MR suggested that it might be more sensitive than CT in detecting some types of traumatic lesions [23–39]. Our prospective study has confirmed that MR is more sensitive than CT for detecting all types of traumatic lesions, with the exception of subarachnoid hemorrhage. Although the difference in sensitivity was minimal for the hemorrhagic lesions, there was a great discrepancy for nonhemorrhagic intraaxial lesions. An increased sensitivity might be expected with MR, since differentiation of normal and injured tissues with this technique depends on several imaging parameters. Lesion contrast is influenced by relative differences in the T1 and T2 relaxation times of the tissues as well as the proton density [23–25, 29–35]. The superior sensitivity of MR for nonhemorrhagic lesions is most likely from its greater ability to detect local alterations of water in injured tissues. Neural injury, like most pathologic processes, is invariably accompanied by changes in the local water environment [43]. Intracellular edema may accompany trauma-induced alterations of cellular metabolism from hypoxia or hypoxemia. Extracellular edema may be produced by disruption of the blood-brain barrier or as a result of direct injury to the axons (axoplasmic leakage) [43].

MR was also superior in the detection of extraaxial hematomas, although the difference was less striking. The only

lesions typically missed by CT were very small, clinically silent subdural hematomas usually less than 3 mm in thickness. These small, high-density lesions were very hard to distinguish from adjacent bone on CT scans obtained at standard window settings (Figs. 3 and 4). They were easily seen, however, with either T1- or T2-weighted MR scans as high-intensity lesions adjacent to the signal void of cortical bone. Since both the CT and MR scans were analyzed without the benefit of optimizing the image contrast on the monitor, this factor does not bias against CT. Rather, the wider latitude of contrast differences between the hematoma and adjacent structures was responsible for MR's greater sensitivity. The sensitivities of CT and MR were similar for intraventricular hemorrhage. We concur with others, however, that CT has a significant advantage over MR in the detection of acute/subacute subarachnoid blood [32, 37]. Visualization of subarachnoid hemorrhage with MR was difficult in the acute phase unless it was a large, localized clot.

There are a number of methodologic observations concerning this study that should be emphasized when comparing the relative sensitivities of MR and CT. Owing to medical and logistic reasons, the CT and MR scans could not usually be obtained on the same day. CT was usually performed first, so that therapeutic decisions could be made on the basis of a proven diagnostic method. MR was performed only after the patient was considered stable, usually resulting in a delay between CT and MR of zero to 10 days. For this reason it is possible that some traumatic lesions became more apparent over time, thus improving the sensitivity of MR. Although this factor may have been operative, it cannot be the sole explanation for the vast differences in the sensitivities of the two methods. To test the importance of this factor, we also compared the sensitivities of initial or follow-up MR or CT scans obtained within 1 day of each other (Table 5). The sensitivities in the 13 patients in this subgroup were (1) nonhemorrhagic parenchymal lesions (CT, 26.8%; T1-weighted MR, 61.1%; T2-weighted MR, 100%) and (2) hemorrhagic parenchymal lesions (CT, 84.8%; T1-weighted MR, 80.6%; T2-weighted MR, 90.9%). Although a few more nonhemorrhagic lesions were detected on the follow-up CT scans, more small hemorrhagic lesions were missed.

Because the MR scans were usually obtained after the CT scans, the possibility also exists that the MR examinations were “tailored,” thus improving the MR sensitivity. This is not apparent to us from looking at our data, since a “routine” MR examination consisted of both a T1- and T2-weighted study, usually in at least two planes. In no instance was the slice thickness for the MR scans smaller than that for CT. The CT scans generally were not available to the MR examiner before the MR study.

It is also possible that the additional lesions seen with MR resulted from interval development of new (secondary) lesions. Previous authors have shown that, in up to 19% of patients whose clinical condition deteriorates after trauma, delayed hematomas will be evident [22]. In those patients in our series, however, who were restudied with CT for intervening neurologic deterioration, no delayed intraaxial hematomas were detected. It is apparent also that the discrepancy in sensitivity was in the nonhemorrhagic subset of lesions rather than the hemorrhagic lesions. It is possible that other intervening nonhemorrhagic secondary forms of injury (infarction, hypoxia, pressure necrosis, etc.) could explain the ad-

TABLE 5: Sensitivities of CT and MR in 13 Patients in Whom the Studies Were Performed 1 Day Apart

Type of Lesion	True Positive/Total = % Sensitivity		
	CT	T1-Weighted MR	T2-Weighted MR
Subdural/epidural	14/16 = 87.5	16/16 = 100.0	16/16 = 100.0
Parenchymal			
Nonhemorrhagic	11/41 = 26.8	22/36 = 61.1	41/41 = 100.0
Hemorrhagic	28/33 = 84.8	25/31 = 80.6	30/33 = 90.9
Total	39/74 = 52.7	47/67 = 70.1	71/74 = 95.9

Note.—Interval from injury to MR examination: mean = 6.6 days, range = 2–19 days.

ditional lesions seen by MR [43]. This seems unlikely in view of the data in Table 5 and because the clinical course of the patients was one of improvement rather than deterioration. Thus, it seems clear to us and in agreement with others [34, 37, 39] that MR is truly much more sensitive than CT in detecting traumatic lesions.

Early reports suggested that low- and intermediate-field-strength MR systems would be less sensitive for detecting acute hemorrhage [30]. The low signal intensity within acute hematomas on T2-weighted MR scans is thought to be from a heterogeneity of magnetic susceptibility caused by intracellular deoxyhemoglobin or methemoglobin. It is directly proportional to the square of the main magnetic field [30]. The difference in the magnetic susceptibility effects between a 0.5- and 1.5-T system should differ by approximately one magnitude. It is expected, therefore, that the hypointensity of acute hematomas at 0.5 T would be significantly less than at 1.5 T. We found this to be true, although the effect was still adequate enough at 0.5 T to detect acute hematomas as accurately as CT did in the first 4 days after trauma (Table 3). The intraparenchymal hematomas were always surrounded by peripheral halos of edema. This always aided in contrasting the central hypointensity of the hematomas with adjacent normal brain parenchyma. Although there are distinct theoretical advantages of higher magnetic fields for detection of acute hemorrhage, we believe that this is of lesser practical significance in the clinical imaging of patients. We believe that our data support the fact that acute hematomas can be accurately imaged in the first 4 days after trauma with an intermediate-field-strength system with strongly T2-weighted spin-echo pulse sequences (TR = 2300 msec, TE = 80–120 msec). Others have also reported visualization of central hypointensity in acute hematomas with intermediate-field-strength MR systems [32, 33, 47]. Recently, Edelman et al. [33] have also shown that it is possible to maximize the differences in magnetic susceptibility at intermediate and low field strengths. By using small radiofrequency pulse angles and gradient-recalled echoes, they have shown that acute hematomas can be detected easily at these field strengths.

The specificity of MR (and CT) in lesion characterization is difficult to determine from our study since surgical and pathologic confirmation of the lesions was not possible in the vast majority of patients. Two large intracerebral hematomas were surgically confirmed in two patients. Four extraaxial hematomas were surgically confirmed in four patients, and bilateral subdural hematomas were confirmed at autopsy in one patient. The other lesions in this series remain pathologically unsubstantiated but closely resemble the primary traumatic lesions described in many autopsy series in their location, size, distribution, and character [10, 13, 42–46]. The possibility should be considered that a few of the lesions that were

detected by MR and CT were preexisting nontraumatic lesions (demyelinating disease, subcortical arteriosclerotic encephalopathy, infarcts, etc.) It is doubtful, however, that preexisting disease could explain the vast majority of lesions seen in this series. The clinical histories did not reveal any patients with prior neurologic symptoms compatible with demyelinating disease or stroke. The mean age of the study population was only 26.6 years, and only 12.5% of patients were more than 40 years old, making it unlikely that vascular disease could account for any significant percentage of the lesions. Only one of the five patients older than 40 had a history of hypertension and vascular disease. Five subcortical lesions were found in this patient, and they were considered to be of indeterminate cause, compatible with either diffuse axonal injury or lacunar infarcts. These lesions were therefore excluded from statistical analysis. One of the five patients older than 40 had a normal MR scan, and in the three remaining patients the lesions were not suggestive of vascular disease.

MR also appears to be greatly superior to CT in its ability to precisely localize a lesion for purposes of classification. As Gennarelli [14] and others [20, 37, 43, 44, 46] have pointed out, it is important to accurately classify the various traumatic lesions since the prognosis, frequency of secondary complications, mechanism of injury, and therapeutic management may be quite different. MR is advantageous because direct multiplanar images allow a more precise localization of the lesion. Multiplanar images also provide a better three-dimensional view of the lesion, helping to separate primary from secondary forms of injury [42].

Our report indicates that MR is not only useful in patients with closed head trauma but that it is also quite feasible. Evaluation of respirator-dependent patients is more problematic with MR than with CT. A ventilatory system should be used that does not contain ferromagnetic materials, and close monitoring of patient's ventilation is required [41]. Monitoring of other vital systems is also more difficult than with CT scanning, but is also feasible [41]. Imaging mechanically ventilated patients was often advantageous, however, since agitated or combative patients could be pharmacologically paralyzed to eliminate motion artifacts.

Our study agrees with those of others who suggest that MR is valuable for detecting and characterizing traumatic lesions [23–37]. Most of these reports, however, were retrospective studies of patients with minimal intracranial injuries, subdural hematomas without intraparenchymal lesions, or studies of patients with a mixture of acute and chronic head injuries. Our study constitutes the first large prospective study comparing CT with intermediate-field-strength MR for detecting traumatic lesions in patients with moderate to severe acute closed head trauma. When using a 0.5-T system, our

conclusions are similar to those of Zimmerman et al. [34] in their retrospective study with a 1.5-T system. We agree that MR is significantly more sensitive than CT in the detection of acute and subacute nonhemorrhagic parenchymal lesions, small acute subdural lesions, subacute subdural lesions, subacute hemorrhagic lesions, brainstem injuries, and infarcts.

In summary, MR has significant advantages over CT in evaluating patients with closed head trauma. MR is similar in sensitivity to CT for detecting hemorrhagic lesions but vastly superior to CT in detecting the much more prevalent nonhemorrhagic lesions. MR is also more useful than CT for characterizing and classifying primary and secondary forms of injury [42]. More accurate detection and delineation of traumatic lesions with MR should permit more accurate prediction of neurologic and cognitive recovery and assist in optimizing "medical" forms of treatment. Because a more rapid assessment of "surgical" lesions can be made with CT, it is still of prime importance for the initial evaluation of unstable patients with head injuries.

REFERENCES

- Zimmerman RD, Danziger A. Extracerebral trauma. *Radiol Clin North Am* 1982;20:105-121
- Hryshko FG, Deeb ZL. Computed tomography in acute head injuries. *J Comput Tomogr* 1983;7:331-334
- Zimmerman RA, Bilaniuk LT, Gennarelli T. Computed tomography of shearing injuries of the cerebral white matter. *Radiology* 1978;127:393-396
- Zimmerman RA, Bilaniuk LT, Bruce D, Dolinskas C, Obrist W, Kuhl D. Computed tomography of pediatric head trauma: acute general cerebral swelling. *Radiology* 1978;126:403-408
- French BN, Dublin AB. The value of computerized tomography in the management of 1000 consecutive head injuries. *Surg Neurol* 1977;7:171-183
- Moseley IF, Zilkha E. The role of computerized axial tomography (EMI-scanning) in the diagnosis and management of craniocerebral trauma. *J Neurosurg* 1976;3:277-296
- Bricolo A, Pasut ML. Extradural hematoma toward zero mortality. A prospective study. *Neurosurgery* 1984;14:8-12
- Seelig JM, Becker DP, Miller JD, Greenberg RP, Ward JD, Choi SC. Traumatic acute subdural hematoma. Major mortality reduction in comatose patients treated within four hours. *N Engl J Med* 1981;304:1511-1518
- Lanksch W, Grumme T, Kazner E. Correlations between clinical symptoms and computerized tomography findings in closed head injuries. In: Frowein RS, Wilcke D, Karimi-Nejad A, Brock M, Klinger M, eds. *Advances in neurosurgery. Vol 5: Head injuries. Tumors of the cerebellar region*. Berlin: Springer-Verlag, 1978:27-29
- Snoek J, Jennett B, Adams JH, Graham DI, Doyle D. Computerized tomography after recent severe head injury in patients without acute intracranial hematoma. *J Neurol Neurosurg Psychiatry* 1979;42:215-225
- Perrone P, Porazzi D, Ricotta E, Secchi P, Rovetta P. Brain contusion in minor cranial trauma. *Ital J Neurol Sci* 1985;6:43-45
- Bruce DA, Schut L, Bruno LA, Wood J, Sutton LN. Outcome following severe head injuries in children. *J Neurosurg* 1978;48:679-688
- Adams JH, Graham DI, Scott G, Parker L, Doyle D. Brain damage in fatal non-missile head injury. *J Clin Pathol* 1980;33:1132-1145
- Gennarelli TA, Spielman GM, Langfitt TW, et al. Influence of the type of intracranial lesion on outcome from severe head injury. *J Neurosurg* 1982;56:26-32
- Bruce DA, Alavi A, Bilaniuk L, Dolinskas C, Obrist W, Uzzell B. Diffuse cerebral swelling following head injuries in children: the syndrome of "malignant brain edema." *J Neurosurg* 1981;54:170-178
- Cooper PR, Maravilla K, Kirkpatrick J, et al. Traumatically induced brainstem hemorrhage and the computerized tomographic scan: clinical, pathological and experimental observations. *Neurosurgery* 1979;4:115-124
- Sweet RC, Miller JD, Lipper M, Kishore PRS, Becker DP. Significance of bilateral abnormalities on the CT scan in patients with severe head injury. *Neurosurgery* 1978;3:16-21
- Tsai FY, Huprich JE, Gardner FC, Segar HD, Teal JS. Diagnostic and prognostic implications of computed tomography of head trauma. *J Comput Assist Tomogr* 1978;2:323-331
- Tsai FY, Teal JS, Quinn MF, et al. CT of brainstem injury. *AJNR* 1980;1:23-29, *AJR* 1980;134:717-723
- Lipper MH, Kishore PRS, Enas GG, Domingues da Silva AA, Choi SC, Becker DP. Computed tomography in the prediction of outcome in head injury. *AJNR* 1985;6:7-10, *AJR* 1985;144:483-486
- Lobato RD, Cordobes F, Rivas JJ, et al. Outcome from severe head injury related to the type of intracranial lesion: a computerized tomography study. *J Neurosurg* 1983;59:762-774
- Clifton GL, Grossman RG, Makela ME, Miner ME, Handel S, Sadhu V. Neurological course and correlated computerized tomography findings after severe closed head injury. *J Neurosurg* 1980;52:611-624
- Young IR, Bydder GM, Hall AS, et al. Extracerebral collections: recognition by NMR imaging. *AJNR* 1983;4:833-834
- Sipponen JT, Sepponen RE, Sivula A. Chronic subdural hematoma: demonstration by magnetic resonance. *Radiology* 1984;150:79-85
- Moon Jr KL, Brant-Zawadzki M, Pitts LH, Mills CM. Nuclear magnetic resonance imaging of CT-isodense subdural hematomas. *AJNR* 1984;5:319-322
- Levin HS, Handel SF, Goldman AM, Eisenberg HM, Guinto FC. Magnetic resonance imaging after "diffuse" nonmissile head injury. *Arch Neurol* 1985;42:963-968
- Zimmerman RA, Bilaniuk LT, Grossman RI, et al. Resistive NMR of intracranial hematomas. *Neuroradiology* 1985;27:16-20
- Sipponen JT, Sepponen RE, Tanttu JI, Sivula A. Intracranial hematomas studied by MR imaging at 0.17 and 0.02 T. *J Comput Assist Tomogr* 1985;9:698-704
- Sipponen JT, Sepponen RE, Sivula A. Nuclear magnetic resonance (NMR) imaging of intracerebral hemorrhage in the acute and resolving phases. *J Comput Assist Tomogr* 1983;7:954-959
- Gomori JM, Grossman RI, Goldberg HI, Zimmerman RA, Bilaniuk LT. Intracranial hematomas: imaging by high-field MR. *Radiology* 1985;157:87-93
- Dooms GC, Uske A, Brant-Zawadzki M, et al. Spin-echo MR imaging of intracranial hemorrhage. *Neuroradiology* 1986;28:132-138
- DeLaPaz RL, New PFJ, Buonanno FS, et al. NMR imaging of intracranial hemorrhage. *J Comput Assist Tomogr* 1984;8:599-607
- Edelman RR, Johnson K, Buxon R, Shoukimas G, et al. MR of hemorrhage: a new approach. *AJNR* 1986;7:751-756
- Zimmerman RA, Bilaniuk LT, Hackney DB, Goldberg HI, Grossman RI. Head injury: early results comparing CT and high-field MR. *ANJR* 1986;7:757-764, *AJR* 1986;147:1215-1222
- Langfitt TW, Obrist WD, Alavi A, et al. Computerized tomography, magnetic resonance imaging, and positron emission tomography in the study of brain trauma. Preliminary observations. *J Neurosurg* 1986;64:760-767
- Gandy SE, Snow RB, Zimmerman RD, Deck MDF. Cranial nuclear magnetic resonance imaging in head trauma. *Ann Neurol* 1984;16:254-257
- Snow RB, Zimmerman RD, Gandy SE, Deck MDF. Comparison of magnetic resonance imaging and computed tomography in the evaluation of head injury. *Neurosurgery* 1986;18:45-52
- Hans JS, Kaufman B, Alfidi RJ. Head trauma evaluated by magnetic resonance and computed tomography: a comparison. *Radiology* 1984;150:71-77
- Jenkins A, Teasdale G, Hadley MDM, MacPherson P, Rowan JO. Brain lesions detected by magnetic resonance in mild and severe head injuries. *Lancet* 1986;2(8504):445-446
- Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet* 1974;2:81-84
- Dunn VD, Ely-Coffman C, McGowan JE, Ehrhardt JC. Mechanical ventilation during magnetic resonance imaging. *Magnetic Resonance Imag* 1985;3:169-172
- Gentry LR, Godersky JC, Thompson B. MR imaging of head trauma: review of the distribution and radiopathologic features of traumatic lesions. *AJNR* 1988;9:101-110
- Adams JH, Gennarelli TA, Graham DI. Brain damage in non-missile head injury: observations in man and subhuman primates. In: Smith WT, Cavanagh JB, eds. *Recent advances in neuropathology*, vol 2. Edinburgh: Churchill Livingstone, 1982:165-190
- Adams JH, Graham DI, Murray LS, Scott G. Diffuse axonal injury due to non-missile head injury in humans: an analysis of 45 cases. *Ann Neurol* 1982;12:557-563
- Strich SJ. Shearing of nerve fibers as a cause of brain damage due to head injury: a pathological study of twenty cases. *Lancet* 1961;2:443-448
- Adams JH, Mitchell DE, Graham DI, Doyle D. Diffuse brain damage of immediate impact type: its relationship to primary brainstem damage in head injury. *Brain* 1977;100:489-502
- Zimmerman RD, Deck MDF. Intracranial hematomas imaged by high-field MR (letter). *Radiology* 1986;159:565