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Head Injury: Early Results of Comparing CT and High-Field MR

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The sensitivity and specificity of CT and high-field MR (1.5 T) were compared in an evaluation of 30 patients with head injuries (eight acute, 15 subacute, and seven chronic). By using T1- and T2-weighted images, it was possible to detect various stages of hemorrhages and their separation from edema. In the acute category, both CT and MR showed acute hemorrhagic lesions, but only MR demonstrated coexisting chronic hematomas or small hypothalamic or brainstem infarctions. MR was far superior to CT in the detection and characterization of subacute injuries, including shearing injuries, hemorrhagic and nonhemorrhagic contusions, and subdural hematomas. In chronic injury, atrophy was demonstrated by both techniques, but only MR showed parenchymal abnormalities and old hemorrhages. Its ease in monitoring patients and its greater speed make CT the procedure of choice for the evaluation of acute cases. CT also provides information that is useful in deciding between surgery and medical management. However, the more precise anatomic depiction of MR and its sensitivity to parenchymal abnormalities make MR the key to correct prognosis in patients with subacute or chronic injury.

In order for MR to replace or supplement CT in the evaluation of head injury, it will have to provide either new or better diagnostic information. MR at present requires significantly longer scan times than CT, and motion during these long scans can detract from image quality. Life-support and monitoring systems that perform well with CT function poorly with MR. The purpose of this study is to ascertain whether the information provided by high-field MR justifies the longer scan times and greater difficulty of patient management. To answer this question, we evaluated 30 head injury patients studied with 1.5-T MR and compared those findings with the CT findings when the trauma was acute, subacute, or chronic. The comparison was carried out for the three stages of trauma defined because of the now well-recognized changes in MR signal intensity of blood with time [1-4]. While the results in this series are preliminary, the data support the idea that MR contributes valuable information during the acute, subacute, and chronic stages after cerebral injury.

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Materials and Methods

MR studies were performed on a 1.5-T GE Signa proton imaging system. T1-weighted images were obtained using a repetition time (TR) of 400 to 800 msec and an echo delay time (TE) of 20 to 25 msec. T2-weighted images used a TR of 2000-2500 msec and a TE of 30-80 msec. Two echoes were routinely obtained. Matrix size was 128 × 256, resulting in a pixel size of 1 mm × 0.5 mm. Slice thickness was either 3 or 5 mm. All images were done multislice using one average. Examination was initiated in the sagittal plane with subsequent T2 studies done in the axial and coronal planes. Coronal and/or axial T1-weighted images were also obtained, as indicated, primarily for evaluation of hematomas. A set of T1-weighted images took 2.5-3.3 min, while a set of T2-weighted images took 8.5-10.5 min.

CT studies were done on a GE 9800, GE 8800, or Philips 310 scanner. The CT studies

were done without contrast enhancement in the axial plane with slice thickness of 9–10 mm. MR studies, CT, clinical presentation, course, and outcome were reviewed.

Results

During a 1-year period, 1543 patients were examined for CNS diseases. Thirty patients with head injuries had 38 MR studies. There were 26 males and four females, ranging in age from 3 to 60 years; 60% were 29 years old or younger. Eight patients were initially examined acutely (within 72 hr of injury), 15 patients were examined subacutely (5 to 20 days after injury), and seven patients were examined chronically (1 month to 3 years after injury). Eight patients had follow-up examinations 1 to 3 months after the first study. All patients had CT scans without contrast enhancement.

Acute Head Injury

Seven of the eight patients had severe injuries and were stuporous to comatose at presentation (Table 1). Three patients underwent craniotomy after CT examination and before MR. All three had evacuation of acute subdural hematomas; one also underwent removal of an accompanying intracerebral hematoma and another removal of an epidural hematoma. Six of the eight patients showed evidence of hemorrhagic contusions, usually multiple, which appeared as areas of markedly decreased signal intensity on T2-weighted images owing to the presence of deoxyhemoglobin surrounded by a high signal intensity area caused by edema (Fig. 1). These areas appeared only as a region of moderate hypointensity on T1-weighted images (Fig. 1B). Edema and hemorrhage could not be separated on the T1-weighted images. In addition, four patients showed acute subdural hematomas, one with an additional chronic subdural hematoma, one with an intraventricular hemorrhage (Figs. 1D and 1E), one with a subarachnoid hemorrhage, and one with a posterior cerebral arterial territory infarct with hypothalamic and upper brainstem infarction (Fig. 2).

CT showed all the acute hemorrhagic contusions, the subdural hematomas, and the intraventricular bleed, but did not show a large isodense infratemporal-occipital chronic subdural hematoma in one patient or an infarction of the upper brainstem and hypothalamus in another (Fig. 2A).

TABLE 1: Acute Head Injury^a

Finding ^b	CT	MR
Hemorrhagic contusions	6	6
Nonhemorrhagic contusion	0	1
Subarachnoid hemorrhage	1	1
Intraventricular hemorrhage	1	1
Acute subdural hematoma	4	4
Chronic subdural hematoma	0	1
Brainstem hypothalamic infarction	0	1
Posterior cerebral infarction	1	1

^a Eight patients were examined 1–3 days after injury.

^b In some patients, there was more than one finding.

Subacute Injury

The patients in this stage fell into the moderate-to-severe head injury category and were confused, stuporous, or comatose. Four patients had surgery before MR, two for elevation of depressed fractures associated with underlying contusions, one for evacuation of an acute epidural hematoma, and one for evacuation of an acute intracerebral hematoma (Table 2). One patient had surgery after the MR examination for removal of a subacute (isodense on CT, high signal on T1- and T2-weighted images) subdural hematoma (Fig. 3). Subdural hematomas were found in five patients. Hemorrhagic contusions were found in 13 patients and nonhemorrhagic contusions in three patients. On T1-weighted images, contusional hemorrhages and related hematomas showed peripheral high-signal change early on, and more central high-signal change over time (Fig. 4) at sites of methemoglobin formation [3]. On T2-weighted images, signal change to high intensity was slightly delayed behind T1-weighted images, corresponding to a breakdown in the cell membrane surrounding methemoglobin. Intracellular methemoglobin has low signal intensity while extracellular methemoglobin has high signal intensity [3]. Shearing injuries were found in six patients. All the patients with shearing injury showed zones of high signal abnormality (T2-weighted image) in the cerebral white matter (corpus callosum = 4, basal ganglia = 1, corticomedullary juncture zones = 1) (Figs. 4C, 4D, and 5). Two patients with shearing injuries had focal hemorrhages at the site of injury. One patient had posterior cerebral artery territory infarction with involvement of the thalamus and hypothalamus. CT scans in these patients had not revealed the shearing injuries or subdural hematomas. Thirty-seven percent of the contusions seen on MR (including all nonhemorrhagic contusions) were not identified on CT.

Chronic Injuries

These patients had survived moderate-to-severe head injuries (Table 3). Two patients had had surgery, one for decompression of a frontal hemorrhagic contusion and one for removal of acute subdural and epidural hematomas. Five patients showed focal atrophy at a site consistent with old frontal or temporal contusions. Two patients showed generalized atrophy, one patient had evidence of diffuse gray- and white-matter damage, and another had evidence of multiple areas of old intracerebral hemorrhage (Fig. 6), including the brainstem. CT scans in these patients showed either focal or generalized loss of brain parenchyma.

Discussion

CT is the mainstay in the diagnostic armamentarium for evaluation of traumatic head injury. This is because of its rapid scan times and its ability to demonstrate, on the basis of density differences, the presence, location, and extent of intracerebral and extracerebral hemorrhage and hematomas, bony fractures, and compressed brain parenchyma, ventri-

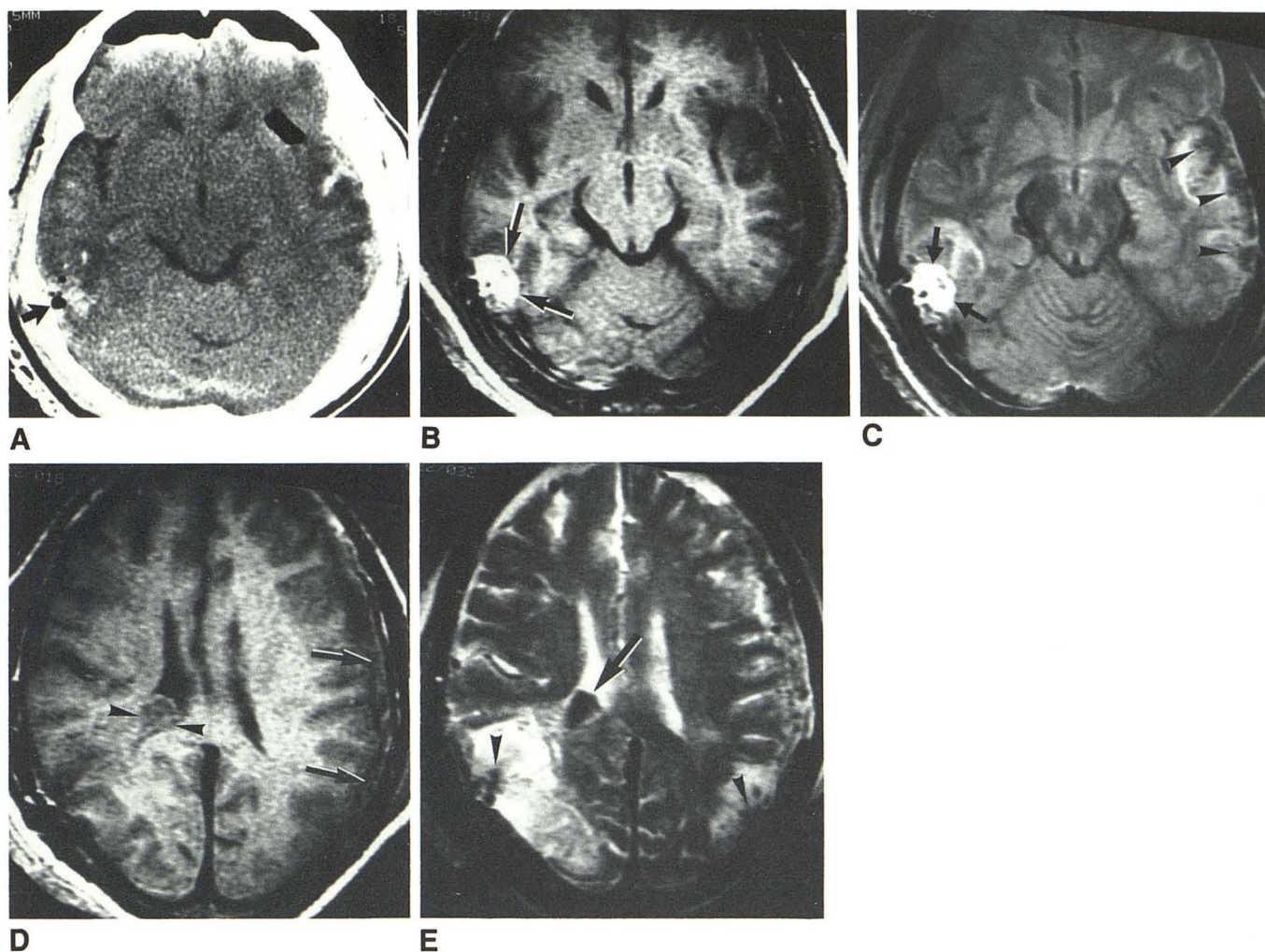


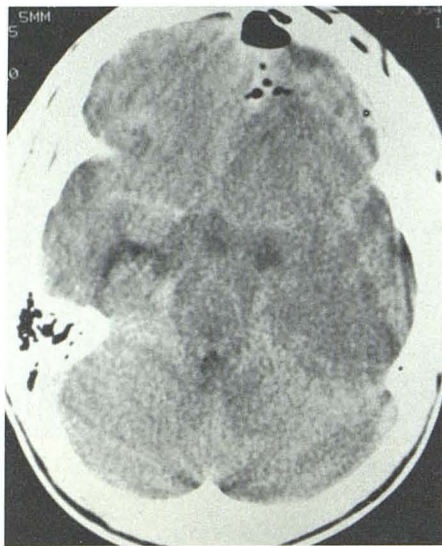
Fig. 1.—Acute brain injury in a 55-year-old man who was hit by a bus and rendered comatose. **A**, CT examination within 24 hr of injury, after evacuation of bilateral acute subdural hematomas, shows air over frontal lobes within subdural space, at craniotomy site (*arrow*), and at site of intratemporal hemorrhagic contusion. Left temporal lobe shows hemorrhagic contusion, edema, and residual subdural hematoma. **B**, Axial T1-weighted image (TR = 600 msec, TE = 25 msec). Examination performed approximately 1 hr after CT in **A** shows high intensity signal (methemoglobin) (*arrows*) at site of partially evacuated hemorrhagic contusion. Hemorrhagic contusion of surface of left temporal lobe is hypointense relative to nonhemorrhagic cortex. **C**, Axial T2-weighted image (done minutes after T1-weighted image) at same plane of section as in **B** (TR = 2500 msec, TE = 35 msec) shows high signal of extracellular methemoglobin (*arrows*) at same site as T-1 weighted image. Less intense high signal is present bilaterally in white matter underlying sites of hemorrhagic contusion secondary to edema. Marked hypointensity is seen from deoxyhemoglobin of hemorrhagically contused cortex (*arrowheads*) in left temporal lobe. **D**, Higher axial T1-

weighted image (TR = 600 msec, TE = 25 msec) shows deoxyhemoglobin of intraventricular hematoma in right lateral ventricle as area of hypointensity relative to white matter, but more intense than CSF. Subdural hematoma on left is identifiable (*arrows*) as band of hypointensity distinguishable from more marked signal void of inner table of skull. **E**, Axial T2-weighted image (TR = 2500 msec, TE = 80 msec) shows high signal abnormalities of ischemia and edema involving both parietal occipital areas and underlying hypointense cortical areas of hemorrhagic contusion (*arrowheads*). Markedly hypointense clot (*arrow*) is present in right lateral ventricle, in part outlined by hyperintense CSF. Residual left acute subdural hematoma is seen as area of variable hypointensity. In this instance, areas of hyperintensity between acute subdural hematoma and inner table of skull are thought to represent serum extruded from clot retraction of subdural hematoma. Similar high intensity within subdural space is seen overlying upper portion of left and right frontal lobes. CSF within sulci is hyperintense.

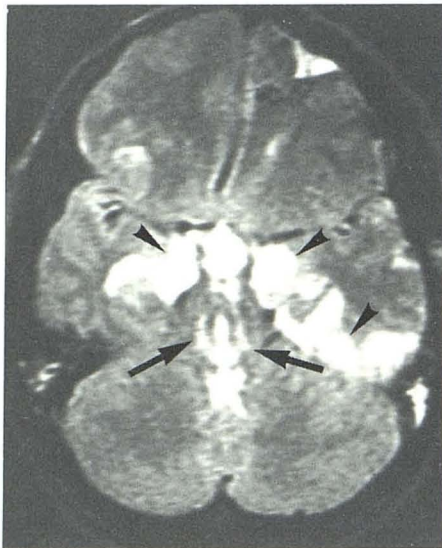
cles, and subarachnoid space. The significance of CT findings is understood and their clinical importance recognized [5]. The limitations of CT are apparent in evaluation of the comatose patient and the patient with focal neurologic deficit whose CT examination appears normal [6, 7]. Superficial contusions of the cortex are frequently obscured on CT by volume averaging with contiguous bone. Small hypothalamic and brainstem infarcts are not resolved by CT, and shearing injuries of the cerebral white matter, unless hemorrhagic,

usually are not identifiable [8]. Thus, there is a role for a new imaging technique that is both sensitive and reasonably specific in identifying those lesions that are poorly seen (or not seen at all) by CT. If MR can both identify and quantify these latter lesions while at the same time demonstrate the lesions already well shown by CT, then it has the potential to become the critical imaging technique in the evaluation of acute head injury.

Blood in acute intraparenchymal hematomas, in the state



A



B



C



D

Fig. 2.—Infarction of hypothalamus, midbrain, and vascular territory of posterior cerebral artery secondary to cerebral herniation caused by frontal intracerebral hematoma. Patient is a 23-year-old man who was involved in a motor vehicle accident and rendered comatose. First CT examination done within hours of injury revealed intracerebral hematoma. Surgical evacuation was undertaken, and CT and MR studies shown were performed 48 hr after trauma. **A**, CT scan shows air at operative site in left frontal lobe region, compression of chiasmatic cistern, and loss of ambiens cisterns. Subarachnoid hemorrhage is present in basilar cisterns. **B**, Axial T2-weighted image (TR = 2500 msec, TE = 35 msec) study done several hours after CT in **A** shows high signal intensity in midbrain (arrows) and medial temporal lobes (arrowheads) consistent with infarction. **C**, CT at a higher level shows area of hypodensity involving left occipital region, midline shift to right, and obliteration of atrial region of left lateral ventricle. Findings are consistent with infarction in distribution of left posterior cerebral artery. Subarachnoid hemorrhage is present in frontal interhemispheric fissure. **D**, Axial T2-weighted image (TR = 2500 msec, TE = 35 msec) shows area of high signal intensity in distribution of left posterior cerebral artery, consistent with infarction and midline shift to right.

TABLE 2: Subacute Head Injury^a

Finding ^b	CT	MR
Hemorrhagic contusion	10	13
Nonhemorrhagic contusion	0	3
Shearing injury	0	6
Subdural hematoma	0	5
Posterior cerebral infarction	1	1
Hypothalamic infarction	0	1

^a Fifteen patients were examined 5–20 days after injury.

^b In some patients, there was more than one finding.

of deoxyhemoglobin, examined at high field strength (1.5 T) appears as an area of slight hypointensity on T1-weighted images and as an area of marked hypointensity on T2-weighted images (Fig. 1) [3]. After oxidation to methemoglo-

bin, the hematoma becomes high in signal intensity on T1-weighted images and eventually on T2-weighted images (Figs. 1, 3, 4, and 7B). This change occurs from the periphery centrally, and, in the nonoperated patient, was first seen 5 day after injury (Fig. 4B). At this stage, T2-weighted images still distinguish edema from hematoma because the intracellular methemoglobin remains low in signal intensity (Fig. 4C). However, as the cell membranes surrounding the methemoglobin lyse, the signal intensity changes, becoming higher [3]. In this case, a combination of T2- and T1-weighted images, done in the same plane, is needed in order to discriminate between edema and hematoma. Contusions and hematomas that are surgically exposed and that undergo oxidation at the time of craniotomy undergo rapid signal change, and high signal with both T1- and T2-weighted images may be seen on the immediate postoperative MR (Figs. 1B and

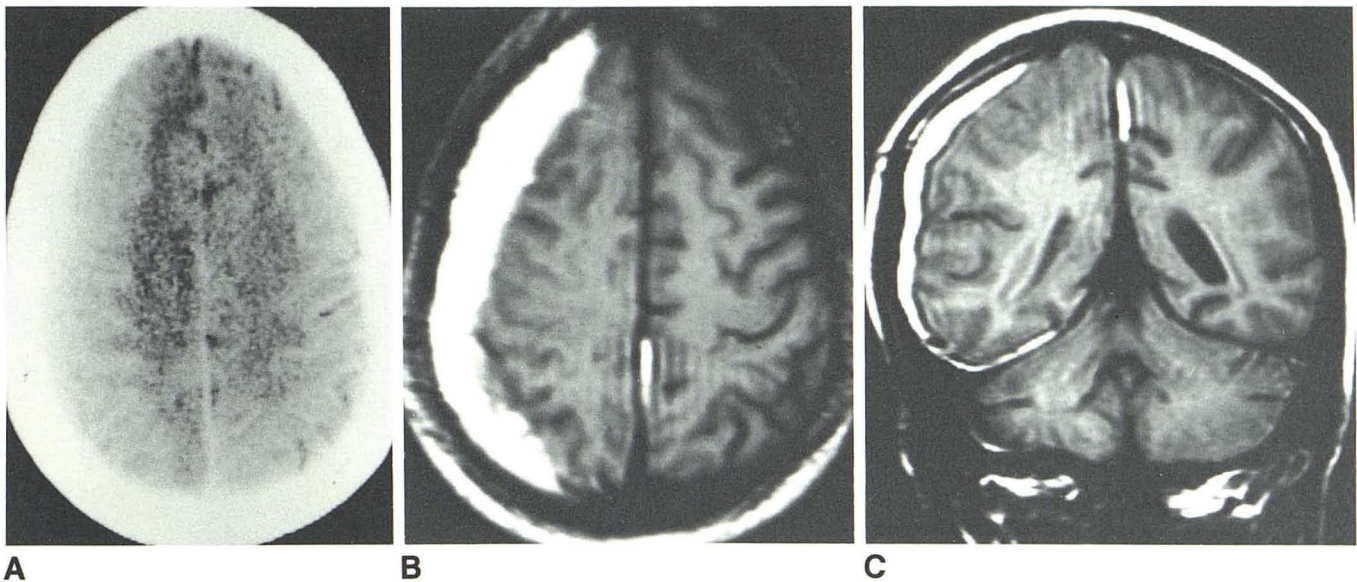


Fig. 3.—Subacute subdural hematoma in a 34-year-old man who was studied by CT and MR 12 days after a motor vehicle accident that resulted in an acute subdural hematoma, which was surgically removed. **A**, Axial CT was interpreted as showing no definite abnormality. In retrospect, cortical gray matter does not reach inner table of skull on right. **B**, Axial T1-weighted image

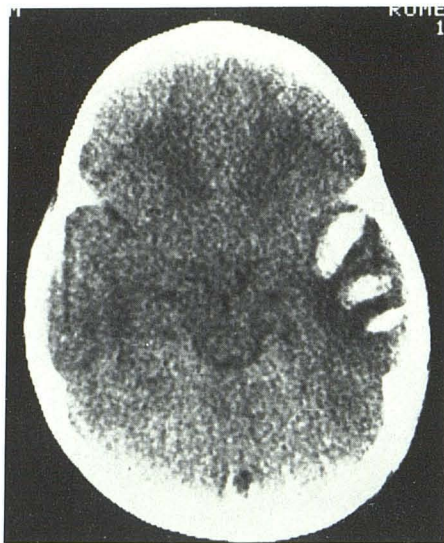
(TR = 800 msec, TE = 20 msec) shows high signal intensity from a large subacute subdural hematoma that is displacing cortex medially. **C**, Coronal T1-weighted image (TR = 800 msec, TE = 20 msec) shows subdural hematoma to cover lateral convexity, extending infratemporal with a small interhemispheric component. Atrium of right lateral ventricle is smaller than left.

1C). With breakdown of the hematoma, macrophagic activity results in removal of blood products. The hemosiderin in these products produce a marked hypointensity on T2-weighted images on the periphery of the hematoma (Fig. 6) [3].

In this series, the eight patients with acute head injury all had initial CT examinations and the MR was done after any emergency surgical procedure or medical therapy that was indicated. At the time the MR study was done, its success was equal to that of CT in identifying the hemorrhagic contusions and focal hematomas within the brain, ventricles, and subdural space. Only one patient was examined who had evidence of significant subarachnoid hemorrhage, and in this instance, both the CT (day 1) and MR examination (day 3) were positive. This series does not represent sufficient experience to permit us to draw conclusions regarding the ability of MR to demonstrate subarachnoid hemorrhage.

In the acute patients, the multiplanar MR examination, using coronal and sagittal planes in addition to the axial, gave much better localization and assessment of the extent of contusions and hematomas. The acute cortical hemorrhagic contusion is identified not only because of the hypointensity of the blood, but also because of the hyperintensity on the T2-weighted image of the surrounding edema and the interspersed necrotic brain tissue. In the acute injury patient, a difference in signal intensity between the normal and edematous cortex from the hypointense calvarium serves to identify the extracerebral hematoma. On the T2-weighted image, the hypointensity of the acute subdural hematoma may appear similar to the signal void of the nonmobile protons in the inner table of the calvarium. In this instance, the T1-weighted image is apt to show a

higher signal intensity from the acute subdural hematoma than from the cortical bone, identifying the presence and location of the extracerebral hematoma [Fig. 1D]. However, the extrusion of serum from the acute subdural hematoma (because of clot retraction) can produce a thin rim of high-intensity signal (T2-weighted image) on the inner and/or outer margins of the subdural hematoma (Fig. 1E) that also helps to identify the extracerebral hematoma. With CT, in the smaller acute subdural hematomas, there may be relatively little contrast between the hematoma and the calvarium. With MR, using both T1- and T2-weighted images, we have found better contrast overall between brain, hematoma, and calvarium. Although we did not examine an acute epidural hematoma in this series, we would expect on the T2-weighted image that the hypointensity of the deoxyhemoglobin in the acute epidural hematoma would not be easily separable from the lack of signal in both the displaced dura and in the calvarium, and that the MR diagnosis would depend more on the T1-weighted image difference in signal intensity between dura, clot, and calvarium as well as on the configuration of the extracerebral hematoma. Air, regardless of its location, produces a signal void, and, as such, may not be separable from the hypointensity of the deoxyhemoglobin on T2-weighted images. However, on T1-weighted images, the signal intensity of air is less than that of deoxyhemoglobin, thus allowing separation. The signal void associated with rapidly flowing blood in surface arteries will not be easily separated from the hypointensity of an acute subdural hematoma on T2-weighted images. Once the signal intensity of the hematoma has become higher, the displaced vessels are easily

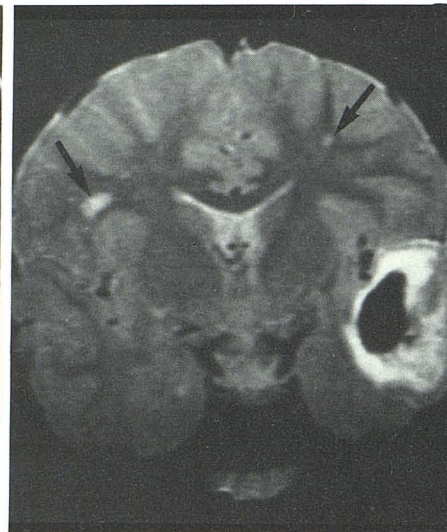


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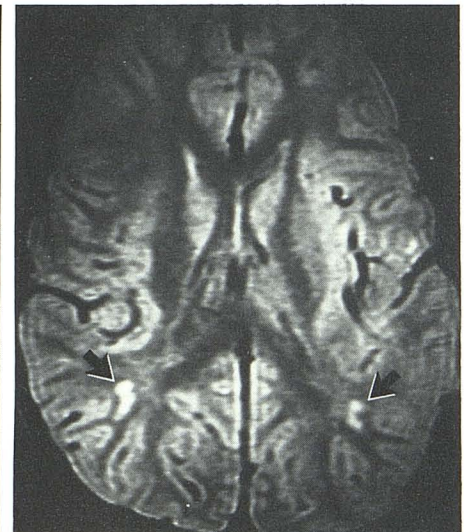
Fig. 4.—Subacute examination of intratemporal hematomas and shearing injury in a 5-year-old boy who was thrown from a motor vehicle during a high-speed collision. CT examination on day of injury; MR study 5 days after injury. **A**, Axial CT shows three intratemporal hematomas surrounded by some edema. No other abnormalities were identified on either this CT examination or on follow-up examinations. **B**, Sagittal T1-weighted image (TR = 800 msec, TE = 20 msec) shows peripheral high signal intensity of methemoglobin at site of one intracerebral hematoma within temporal lobe. Temporal lobe is enlarged. **C**, Coronal T2-weighted image (TR = 2000 msec, TE = 40 msec) shows marked hypointensity centrally within temporal intracerebral hematoma. Peripheral high signal intensity corresponds to edema. Intracellular methemoglobin on periphery is not of high signal intensity. Note two scattered areas of high signal intensity near corticomedullary junctures (arrows) secondary to shearing injury. **D**, Axial T2-weighted image (TR = 2000 msec, TE = 40 msec). Same bilateral parietal high signal areas at corticomedullary junctures (arrows) are seen.



B



C



D

identified by their signal void.

With the conversion of deoxyhemoglobin to methemoglobin, the signal intensity becomes high on T1-weighted images [1]. This high signal intensity on T1-weighted images provides very precise gyral localization of hemorrhagic contusions and graphically shows the location and extent of the extracerebral hematomas (Fig. 3). Again, relative to CT, the multiplanar capabilities of MR make a more precise anatomic depiction possible.

However impressive the subacute demonstration of focal hemorrhages and hematomas may be, it is the demonstration of nonhemorrhagic parenchymal changes that is the real province of MR. These are areas of edema that occur at sites of brain injury as a result of shearing injuries and necrosis, nonhemorrhagic contusions, and infarction (Figs. 2 and 5). In this series, the majority of patients showing the nonhemor-

TABLE 3: Chronic Head Injury^a

Finding ^b	CT	MR
Shearing injury	0	1
Focal contusional encephalomalacia	5	5
General atrophy	2	2
Gray-white matter damage	1	1
Old intracerebral hemorrhage	0	1

^a Seven patients were examined 1 month to 3 years after injury.

^b In some patients, there was more than one finding.

rhagic consequences of trauma fell into the head injury group that was studied subacutely. One of the eight acute head injury patients also demonstrated such findings—infarction of the upper brainstem and hypothalamus—not seen on CT (Fig. 2A). MR demonstration of infarctive changes and non-

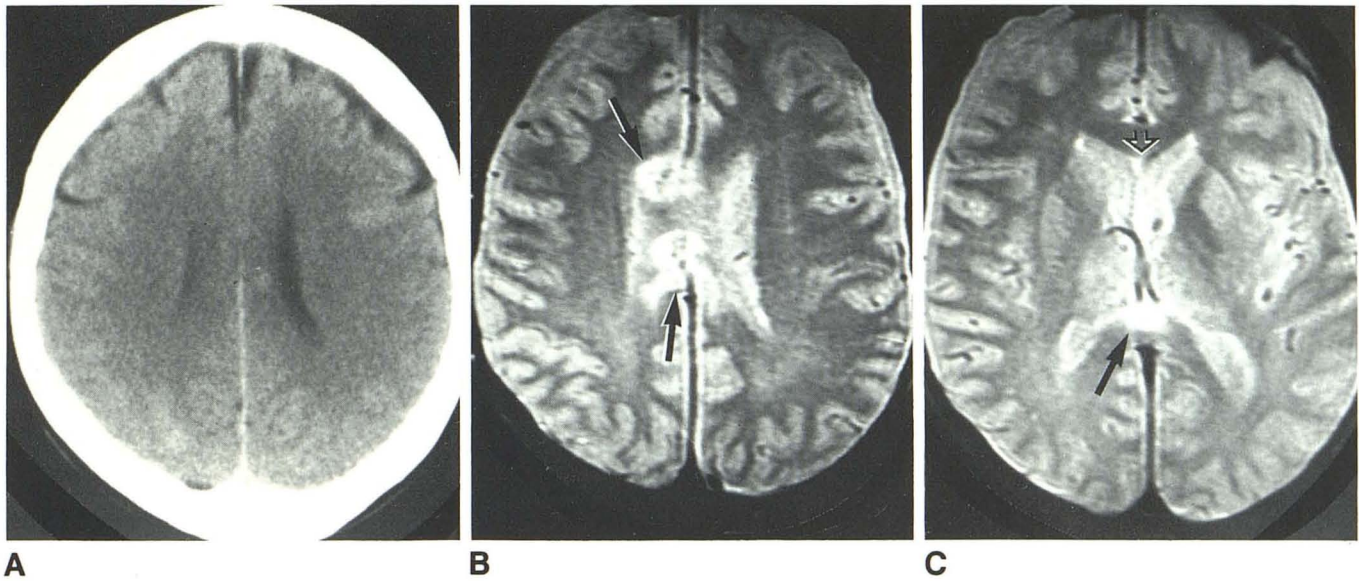


Fig. 5.—Shearing injury corpus callosum in a 60-year-old woman rendered comatose by a motor vehicle accident. **A**, CT scan on day of accident shows no apparent abnormality at level of corpus callosum. **B**, Axial T2-weighted image (TR = 2500 msec, TE = 40 msec), 6 days after accident, shows high

signal abnormality involving corpus callosum (arrows). **C**, Lower axial section T2-weighted image (TR = 2500 msec, TE = 40 msec) shows high signal abnormality in splenium of corpus callosum (arrow) and in septum pellucidum (open arrowhead).

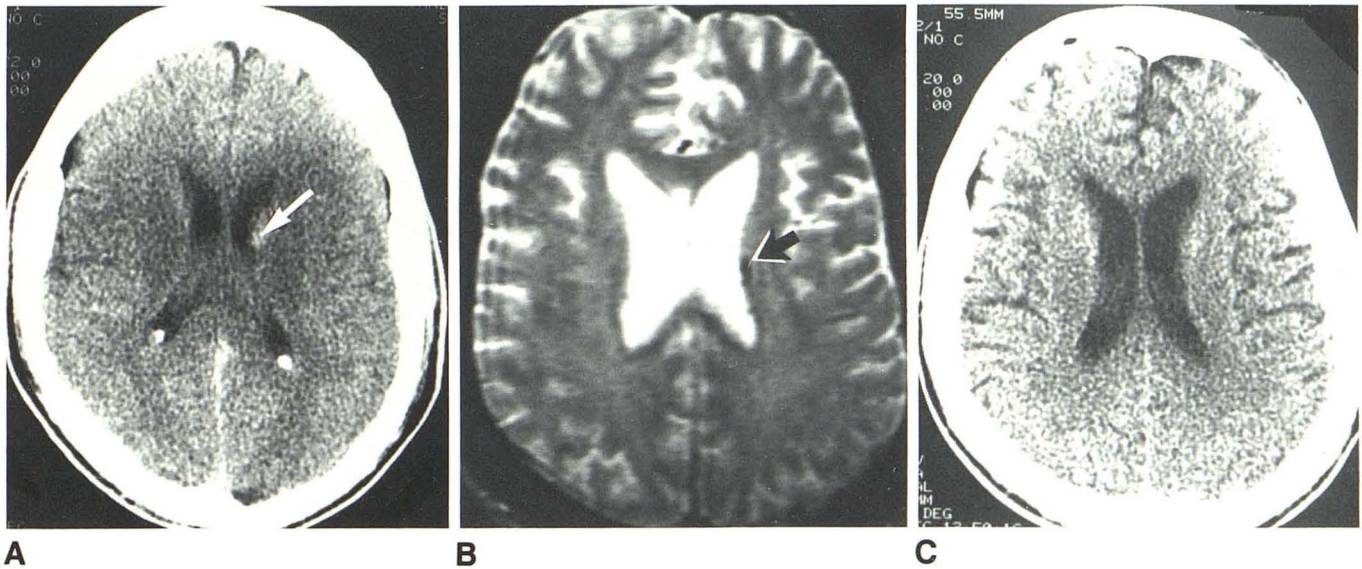


Fig. 6.—Hemosiderin deposition at site of old injury. Patient is a 38-year old man who suffered shearing injury with hemorrhages in brainstem and white matter of centrum semiovale after a motor vehicle accident. CT examination at time of injury, and at 6 months; MR examination at 6 months. **A**, Axial CT section shows hemorrhage (arrow) in white matter contiguous to body of left

lateral ventricle. **B**, Axial T2-weighted image (TR = 2000 msec, TE = 80 msec) shows area of marked signal hypointensity at site of hemosiderin deposition from previous hemorrhage (arrow). **C**, CT done at same time as MR shows enlargement of lateral ventricles, sylvian fissures, and sulci and no abnormality in region of old hemorrhage.

hemorrhagic shearing injuries are findings of prognostic significance [7, 8] since these lesions are either fatal or result in poor recovery.

Subacutely, on CT the high density of the hematoma fades [9]. On MR, the hyperintensity on T1- and T2-weighted images remains [2-4, 10, 11]. Thus, in one patient with an acute subdural hematoma, a subacute subdural hematoma in an

infratemporal occipital location was found on MR. This had not been seen on CT. A second isodense subacute subdural hematoma was missed on CT but showed clearly on MR (Fig. 3).

In the chronic phase after injury, focal or generalized atrophic changes are well seen with both CT and MR. Multiplanar MR displays the exact gyral or lobar involvement of

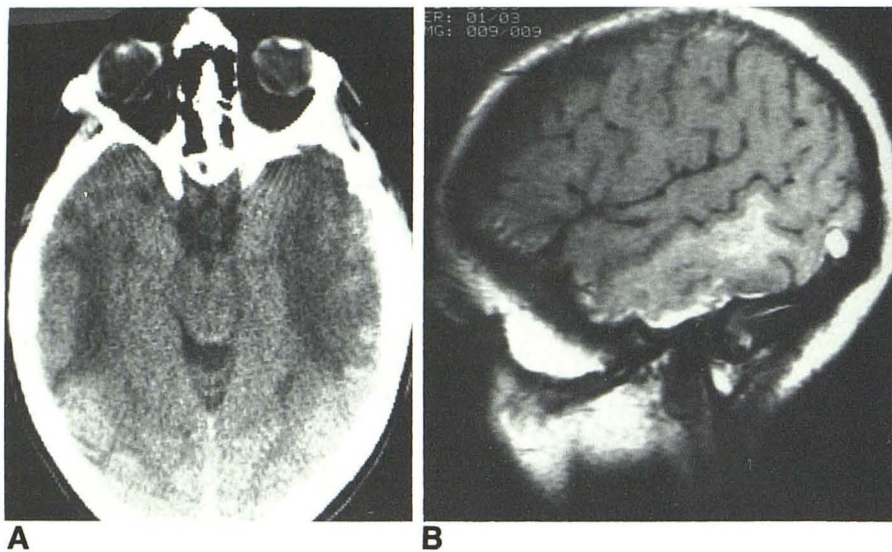


Fig. 7.—Hemorrhagic contusion of temporal lobes. Patient is a 30-year-old man who presented in a coma after being knocked off a bike by a car. CT and MR examinations 17 days after injury. **A**, CT shows bilateral temporal lobe mass effect consisting of edema within white matter and more superficial irregular areas of minimal hypodensity. Findings are consistent with bilateral temporal lobe hemorrhagic contusions. **B**, Sagittal T1-weighted image shows high signal from methemoglobin at site of hemorrhagically contused right middle and inferior temporal gyri.

the atrophic process better than CT, when focal. In this series the atrophic process was often the result of prior contusion. This anatomic information is invaluable in correlating the structural image with the functional abnormalities determined by psychological testing [12].

In the eight patients who had follow-up examinations, the additional benefits of MR were the demonstration of transverse sinus thrombosis in one, putaminal and uncus high-signal abnormalities in another, and progressive generalized atrophy in another, with abnormal high-signal changes in the white matter after a shearing injury.

Conclusions

Although the number of patients studied in each category is limited, certain preliminary conclusions appear to be warranted. The nonhemorrhagic consequences of trauma are much better displayed by MR than CT. Infarction, shearing injuries, and nonhemorrhagic contusions should be identifiable not only subacutely but acutely, and because they are significant consequences of trauma that are not well shown by CT, they should stimulate further efforts toward refinement of monitoring and life-support equipment for MR use with acute head injuries. CT remains the first diagnostic step in treating the emergency patient who needs a rapid examination to determine whether surgical evacuation of a hematoma or medical management is indicated. MR has the capacity to demonstrate the cause of unexplained coma and focal neurologic deficits as well as to provide more routine diagnostic information. Subacutely, MR has an edge over CT in that it can demonstrate with graphic and precise localization extra- and intracerebral hematomas, even when these are isodense on CT. In the chronic patient, while both CT and MR demonstrate local and generalized parenchymal loss, MR gives the type of anatomic information that is needed to make sense

of the patient's functional state [12]. In addition, old nonhemorrhagic and hemorrhagic areas can be demonstrated.

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