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MR imaging of cerebral ischemia: findings in the first 24 hours.

W T Yuh, M R Crain, D J Loes, G M Greene, T J Ryals and Y Sato

MR Imaging of Cerebral Ischemia:
Findings in the First 24 Hours

William T. C. Yuh[1]
Martin R. Crain[1]
Daniel J. Loes[1]
George M. Greene[2]
Tony J. Ryals[1]
Yutaka Sato[1]

MR changes of cerebral ischemia have been shown to occur as early as 1–2 hr after vessel occlusion in experimental models of stroke. However, the MR findings in the early stages of ischemic stroke in the clinical population have not been well established. We studied 41 lesions in 39 patients in whom MR was performed within the first 24 hr after onset of ischemic symptoms. Twenty-five lesions were studied with gadopentetate dimeglumine. Vascular flow-related abnormalities, including absence of normal flow void and presence of arterial enhancement, were the earliest MR findings, detected within minutes of onset. Morphologic changes (brain swelling) on T1-weighted images without signal changes on T2-weighted images could be detected within the first few hours. Signal changes were not usually found before 8 hr on T2-weighted images or before 16 hr on T1-weighted images. In contrast to the absence of parenchymal enhancement typically found in cortical infarctions in the first 24 hr, a few lesions (including transient occlusions, partial occlusions, and isolated watershed infarctions) exhibited early, exaggerated parenchymal enhancement.

We conclude that signal changes may not be reliable in detecting ischemic stroke within the first 8 hr after onset. Vascular abnormalities, when present, are the most reliable and earliest findings. Other MR findings of early ischemic stroke, including morphologic changes and early, exaggerated parenchymal enhancement, may also precede signal changes. Paramagnetic contrast administration often provides valuable information in the detection and evaluation of acute ischemia.

The diagnosis of acute stroke in its very early stages by clinical and radiologic methods can be difficult. An early diagnosis of ischemia might allow prompt initiation of treatment aimed at reducing morbidity and mortality and also enable the ongoing assessment of treatment. MR imaging has been shown to be more sensitive than CT in the detection of acute cerebral ischemia (either completed infarct or transient deficit) within the first 72 hr [1–8]. Abnormal MR findings with and without paramagnetic contrast enhancement have also been reported as early as 1–2 hr after onset of vessel occlusion in experimental models of stroke [9–12]. However, the early MR findings of acute stroke in the clinical population have not been well established. Because there are anatomic and physiologic differences between species, the temporal development of ischemic changes in the experimental animal model may differ from that of the human clinical population. The purposes of this study were to examine the earliest MR findings of clinical cerebral ischemia and their temporal progression using unenhanced and enhanced imaging. We also attempted to understand how these changes may reflect underlying pathophysiologic mechanisms in acute cerebral ischemia.

Materials and Methods

All patients who had an MR examination within 24 hr after the onset of ischemic symptoms between November 1988 and February 1990 were evaluated retrospectively. Chosen for

TABLE 1: Time of MR After Onset of Cerebral Ischemia and MR Findings at Each Time Interval in 39 Patients

Time After Onset (hr)	No. of Lesions	Absent Flow Void	Arterial Enhancement[a]	Morphologic Changes on T1-Weighted Images	Signal Changes on T1-Weighted Images	Signal Changes on T2-Weighted Images	Parenchymal Enhancement[a]
0–2	8	3	1/4	4	0	0	2/4
2–4	3	2	2/2	2	0	1	0/2
4–8	5[b]	3	4/5	4	2	4[b]	0/5
8–12	2	0	1/1	1	1	2	0/1
12–18	5	1	2/3	4	2	3	1/3
18–24	18	5	6/10	12	10	17	2/10
Total	41	14	16/25	27	15	27	5/25

[a] Expressed as number of lesions with abnormal enhancement per total number receiving gadopentetate dimeglumine at that time interval.
[b] Three of five lesions in this interval were not examined until 8 hr after onset.

inclusion in the final study group were all patients who had positive MR findings in a distribution that corresponded with an initial history and physical examination strongly suggestive of cerebral ischemia. In order to accurately determine the temporal appearance and progression of MR findings, asymptomatic patients (obviously without a time of onset) were excluded from the study group. Also excluded from consideration were patients with clinical signs or symptoms but without corresponding MR abnormalities, owing to the variability in degree of clinical suspicion of ischemia and the resultant lack of specificity of the diagnosis. Forty-one ischemic lesions in 39 patients were studied. Each of two patients had two separate clinical ischemic events that corresponded to separate anatomic lesions on MR images. The timing of onset (either the acute onset of ischemic symptoms in a previously asymptomatic patient or rapid progression of symptoms in a patient with intermittent ischemic symptoms) was determined from the best available clinical history. Patients whose MR examination was performed more than 24 hr after onset of symptoms or whose initial MR or CT examination revealed hemorrhagic infarct were also excluded.

Patients ranged in age from 22 to 91 years (mean, 57 years). Twenty-three were men and 16 were women. Seven patients were imaged after developing neurologic signs due to an iatrogenic cause, including cerebral angiography (three), cardiovascular surgery (two), percutaneous occlusion of the internal carotid artery for treatment of an aneurysm (one), and failed test occlusion of the internal carotid artery (one). Twenty-five lesions were studied with contrast material.

Scans were reviewed retrospectively by two radiologists without knowledge of the duration of ischemic symptoms. Lesions were categorized as cortical when the distribution involved the cortex predominantly and as noncortical when confined primarily to deep white or noncortical gray structures. Special attention was given to the absence of normal flow-void phenomenon and to the presence of any abnormal signal intensity changes, brain morphologic changes (e.g., swelling), and abnormal arterial or parenchymal contrast enhancement. Abnormal arterial enhancement was defined as asymmetric enhancement of arterial structures on short TR spin-echo pulse sequences.

All examinations were performed on either a 0.5-T (Picker International, Highland Heights, OH) or a 1.5-T (General Electric, Milwaukee, WI) superconductive scanner. At least one T1-weighted, 350–700/20–26 (TR/TE), and one T2-weighted (2000–2500/80–100) spin-echo pulse sequence was obtained with 3–10 mm slice thickness. Multiecho pulse sequences were included in all T2-weighted examinations performed at 1.5 T. There was a 10–50% slice gap in the examinations performed at the higher field strength. At least two orthogonal planes were imaged. Extra pulse sequences and planes of imaging were added depending on the individual case. Any patient receiving contrast material was imaged immediately after an IV injection of 0.1 mmol/kg of gadopentetate dimeglumine (Magnevist, Berlex Laboratories, Inc., Wayne, NJ). Postcontrast T1-weighted imaging

TABLE 2: MR Findings in Acute Cerebral Ischemia

Mechanism	MR Finding	Possible Causes	Estimated Time[a] (hr)
Flow kinetics	Absent flow	Slow flow; occlusion	Early
	Arterial enhancement	Accentuation of flow derangement	Early
Biophysiologic	T1 morphologic change	Cytotoxic edema (free water)	2–4
	T2 signal change	BBB breakdown; vasogenic edema; macromolecular binding	8
	T1 signal change	BBB breakdown; vasogenic edema; macromolecular binding	16–24
Combination	Delayed parenchymal enhancement[b]	Impaired delivery of significant contrast agent	>24[c]
	Early exaggerated enhancement[d]	Intact delivery of contrast agent; BBB leakage; focal hyperemia	2–4[d]

Note.—BBB = blood-brain barrier.
[a] Time at which findings generally could first be detected by available MR examinations; this does not necessarily imply the exact time of onset.
[b] Typical finding in completed cortical infarctions.
[c] Usually not detected before 5–7 days.
[d] Found in a few cases with transient or partial occlusions and in watershed infarctions.

(without gradient moment nulling) was performed with pulse parameters and imaging planes identical with those of the precontrast T1-weighted sequences.

Results

The temporal distribution of MR examinations after the onset of symptoms and the individual MR findings in each time interval are summarized in Table 1. MR was obtained within the first 4 hr in 11 lesions. Twenty-five lesions were cortical and 16 were noncortical. Table 2 summarizes the early MR findings of ischemia and the estimated time at which they could be detected.

Abnormal Vascular Findings

The earliest MR findings detected in acute cerebral ischemia were vascular abnormalities in major intracranial vessels, including the absence of normal flow-void phenomenon and/

or the presence of arterial enhancement (Fig. 1A). The earliest findings were seen in a patient who had a cardiac arrest during the MR examination. Absence of normal flow void was seen within 8 min of the last documented normal flow void on an earlier pulse sequence (Fig. 2). Most (22 of 41) of the lesions imaged in the first 24 hr were associated with vascular abnormalities (Fig. 1A); eight showed both absence of flow void and arterial enhancement, eight showed only arterial enhancement, and the remaining six lesions showing absence of flow void were not studied with contrast material. Vascular abnormalities were detected in three of eight lesions evaluated in the first 2 hr and in more than half of the total lesions evaluated by 4 hr (six of 11) and by 6 hr (seven of 13).

An absence of normal flow void in a major intracranial artery was detected in association with 14 lesions. Eight lesions were in internal carotid/middle cerebral artery distributions (Fig. 3) and six were in the vertebrobasilar system (Fig. 4).

Arterial enhancement was found in association with 16 of the 25 lesions studied with contrast MR within the first 24 hr (Fig. 5). Thirteen of the 16 were in a cortical distribution.

Arterial enhancement was seen in one of four lesions evaluated in the first 2 hr, in two lesions evaluated between 2 and 4 hr, and in one of two lesions evaluated between 4 and 6 hr. No arterial enhancement was seen within the first 24 hr in any case of isolated supratentorial noncortical infarction.

Five patients who received contrast material in our series also underwent cerebral angiography within 24 hr of the MR examination. Of the three in whom arterial enhancement was detected, two demonstrated slow antegrade flow in the middle cerebral artery at angiography (one case of nonoccluding intraluminal clot and one case of moyamoya disease). The third showed proximal occlusion of the middle cerebral artery with delayed retrograde filling of distal middle cerebral branches via leptomeningeal collaterals. The two other patients who had angiography within 24 hr of the MR examination included one person with a failed test occlusion of the internal carotid artery before MR and one person whose aneurysm was successfully treated by balloon occlusion of the internal carotid. In both cases, MR failed to demonstrate arterial enhancement.
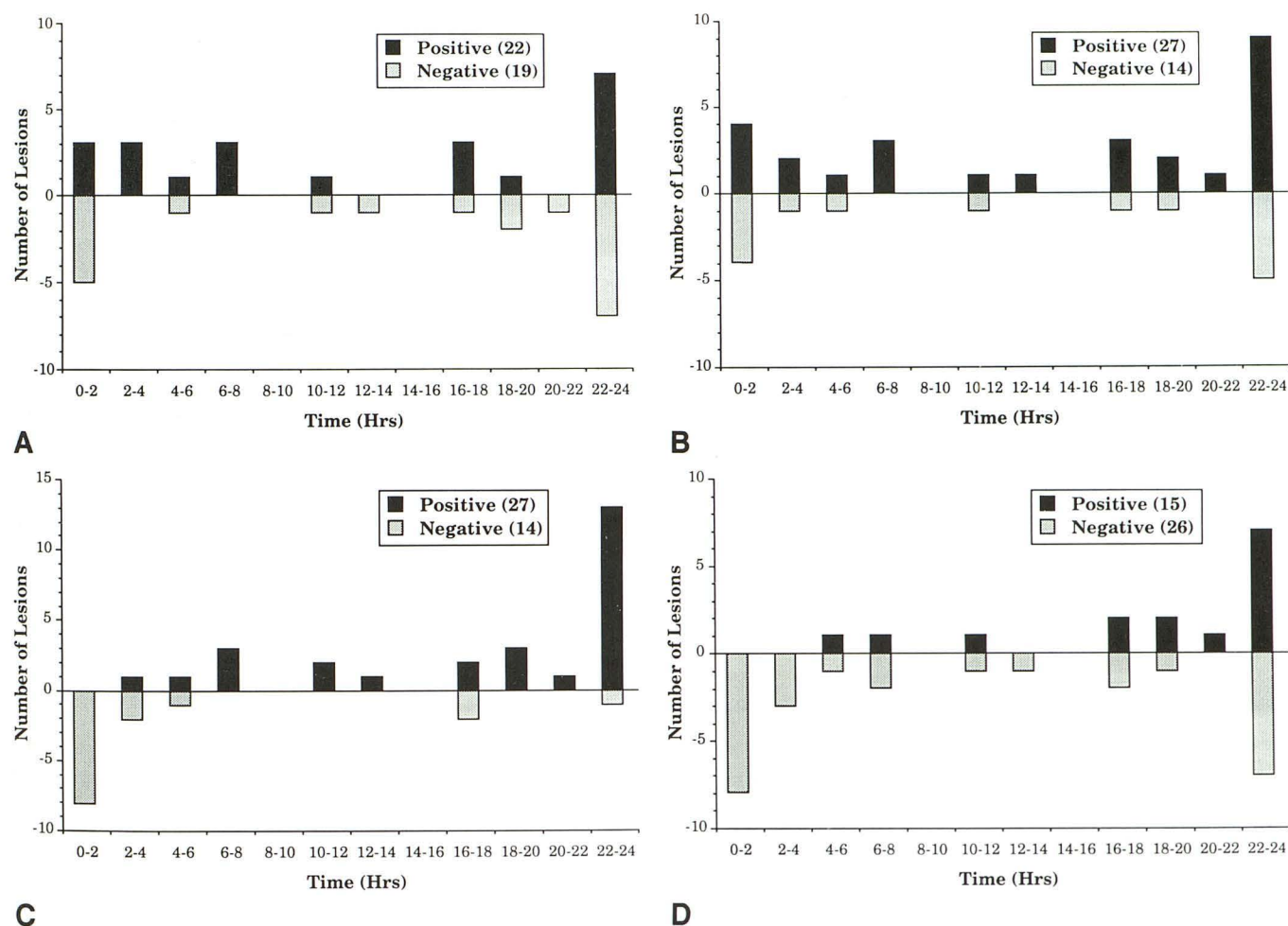

Fig. 1.—Temporal distribution of MR findings within first 24 hr after onset of ischemic symptoms.
A, Positive vascular findings in first 24 hr. Vascular findings include absence of flow void and/or presence of arterial enhancement.
B, T1 morphology in first 24 hr. Morphologic change is defined as evidence of brain swelling demonstrated by T1-weighted images.
C, T2 signal changes in first 24 hr. T2 signal change refers to abnormally high signal intensity of brain parenchyma.
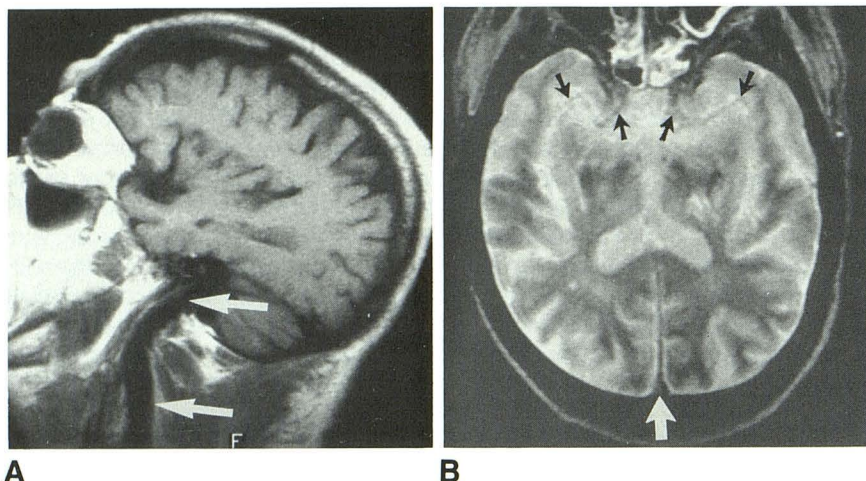D, T1 signal changes in first 24 hr. T1 signal change refers to abnormally low signal intensity of brain parenchyma.

A

B

Fig. 2.—71-year-old man who had a cardiac arrest during MR examination.

A, Initial parasagittal T1-weighted image (400/20) shows flow-void phenomenon within jugular vein (*arrows*).

B, Axial T2-weighted image (2000/100) obtained 8 min later shows absence of flow-void phenomenon within middle cerebral and distal internal carotid arteries (*small black arrows*) and in superior sagittal sinus (*large white arrow*). Note old stroke in left thalamic region.
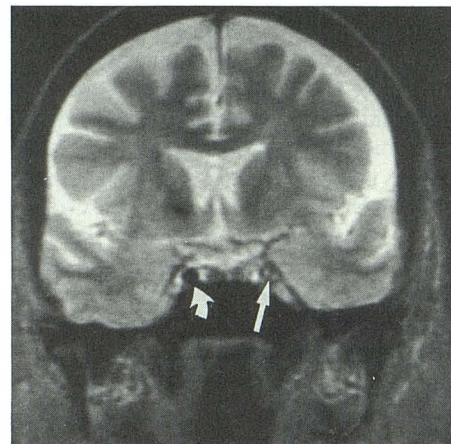
Fig. 3.—63-year-old man with acute onset of diminished vision in left eye. Coronal T2-weighted image (2000/100) obtained 18 hr after onset shows absence of flow-void phenomenon in left internal carotid artery (*straight arrow*). Note normal right internal carotid artery (*curved arrow*).
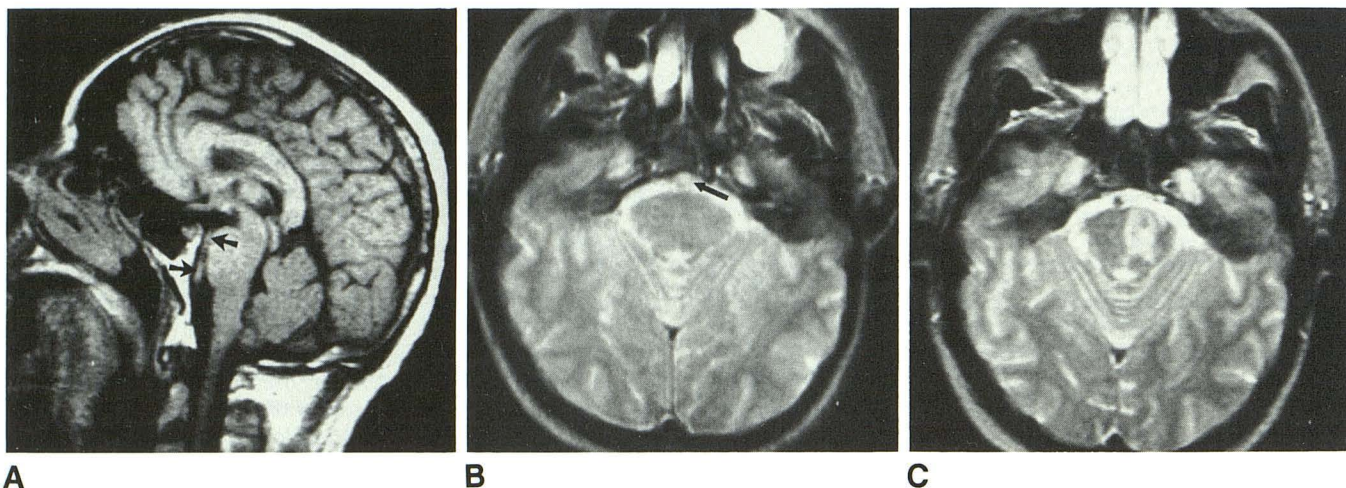

A

B

C

Fig. 4.—50-year-old man with symptoms of brainstem ischemia.

A, Parasagittal T1-weighted image (350/26) obtained 4 hr after onset shows linear signal isointense with brain in prepontine region (*arrows*) along course of basilar artery, suggesting intraluminal clot.

B, Axial T2-weighted image (2000/100) shows absence of flow void in basilar artery (*arrow*). No apparent T2 signal abnormality is detected within the pons at this time.

C, Repeat axial T2-weighted image (2000/100) obtained 48 hr later shows the interval development of ischemic changes in the pons.

Morphologic Changes

Morphologic changes caused by tissue swelling were found in most (27 of 41) of the lesions studied in the first 24 hr after the onset of symptoms (Fig. 1B). Morphologic changes were detected in at least half of the lesions imaged by 2 hr (four of eight), between 2 and 4 hr (two of three), and between 4 and 6 hr (one of two). Swelling was detected either by gross enlargement of structures or more often by distortion of normal adjacent structures. These morphologic changes were best seen on T1-weighted scans. Rapid development of massive parenchymal swelling over the course of 1 hr was documented in two patients. This occurred before the appearance of signal changes on T1- or T2-weighted images (Fig. 6).

Morphologic changes occurred frequently in cortical lesions (20 of 25), where swelling was best demonstrated by obliteration of cortical sulci. Morphologic changes in isolated noncortical lesions, including those in the deep white matter, noncortical gray matter, and brainstem, were detected less often (only seven of 16) and were also not as obvious. When noncortical swelling was detected, this was usually manifested by distortion of the ventricular system or by brainstem swelling.

It is of note that morphologic changes on T1-weighted images could be detected in all lesions associated with arterial
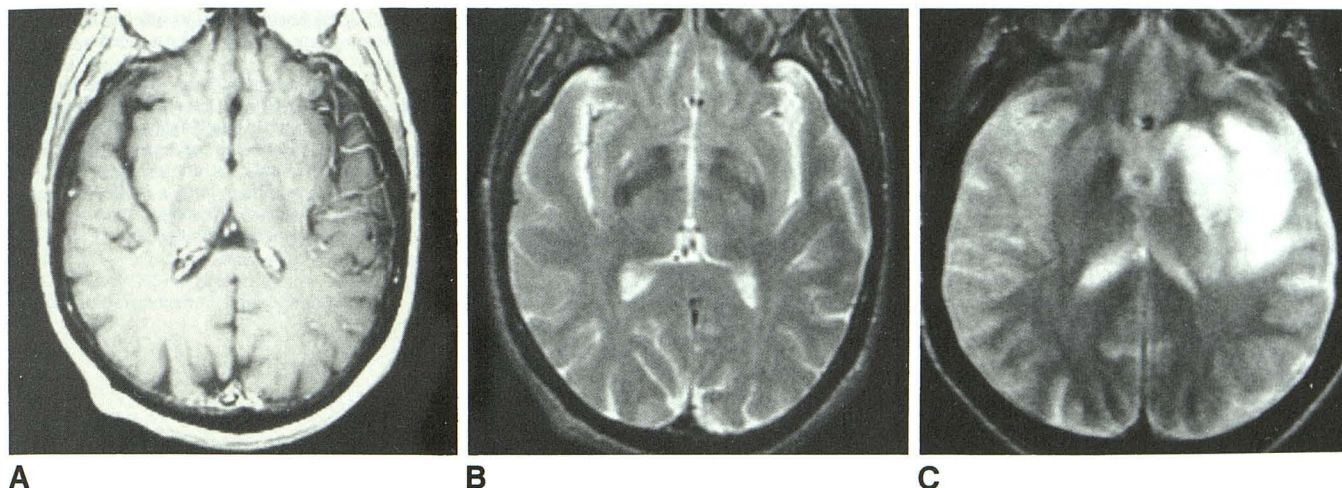
A   **B**   **C**

Fig. 5.—62-year-old man with acute left hemispheric stroke symptoms.

A, Axial T1-weighted contrast-enhanced image (700/20) obtained 3 hr after onset shows abnormal arterial enhancement in distribution of left middle cerebral artery. No abnormal parenchymal enhancement is noted.

B, Axial T2-weighted image (2200/90) at corresponding level shows no parenchymal T2 signal changes at this time.

C, Axial T2-weighted image (2000/100) obtained 24 hr later shows extensive T2 signal changes consistent with ischemia of the left middle cerebral artery.

enhancement. However, vascular enhancement was frequently more obvious.

Signal Changes

Signal changes on T1- and T2-weighted images were usually not detected within the first few hours of symptoms. Most (11 of 13) of the lesions evaluated before 8 hr after onset showed no signal abnormality on T2-weighted images, whereas most (25 of 28) of the lesions evaluated from 8 to 24 hr demonstrated such abnormalities (Fig. 1C). Therefore, signal changes on T2-weighted images were not usually present until 8 hr after onset of cerebral ischemia. Although one lesion did show signal abnormality on T2-weighted images obtained at 4 hr, the exact time of onset may have been more than 4 hr, because the patient was first noted to have symptoms of stroke when he awoke.

Evaluation of the scans for signal abnormalities included a review of both long TR/short TE (proton density) and long TR/long TE images. Although small cortical lesions were frequently depicted better by proton density images and deep noncortical lesions were more easily seen on long TR/long TE images, all lesions seen on long TR images could be seen on short and long TE images.

As compared with T2-weighted images, signal changes on T1-weighted images were even less sensitive in the detection of early cerebral ischemia (Fig. 1D). Only three of 19 lesions evaluated before 16 hr and only 12 of 22 evaluated between 16 and 24 hr showed signal abnormalities on the T1-weighted images.

Parenchymal Enhancement Patterns

In contrast to the typical gyriform enhancement of cortical infarction seen in the subacute to chronic stages of stroke,

enhancement of brain parenchyma was not found in 20 of 25 patients receiving contrast material in this series. However, five of the 25 lesions did show parenchymal enhancement within the first 24 hr; four of these showed a pattern of exaggerated enhancement exceeding the area of signal change on the T2-weighted images. One patient developed acute ischemic symptoms after 2 min of transient balloon occlusion of the ipsilateral internal carotid artery. MR obtained at 2 hr showed a diffuse exaggerated parenchymal enhancement (Fig. 7). An MR study in another patient showed faint enhancement without signal abnormality on T2-weighted images in a watershed distribution after occlusion of the internal carotid artery for treatment of an aneurysm. An enhanced MR examination in a third patient, who presented with acute onset of diminished vision due to occlusion of the proximal internal carotid artery, showed abnormal parenchymal enhancement in the watershed zone of the contralateral hemisphere without signal abnormality on T2-weighted images or clinical sequelae. An MR study in the fourth patient, who had moyamoya disease, showed a lesion with early, exaggerated enhancement in the posterior watershed zone.

Discussion

The detection of acute cerebral ischemia by MR may be dependent on many pathophysiologic factors (Table 2) that are often coexistent. Alteration of normal blood flow is a flow-kinetics phenomenon that should be detected immediately, as was seen in one patient in this series with cardiac arrest. The remaining factors are biophysiologic phenomena that may take time to be appreciated by MR. Sensitivity in detecting ischemia may be increased through the addition of paramagnetic contrast agents, most likely due to either accentuation of the underlying flow derangement or accumulation of abnormal amounts of contrast agent in the ischemic tissue.
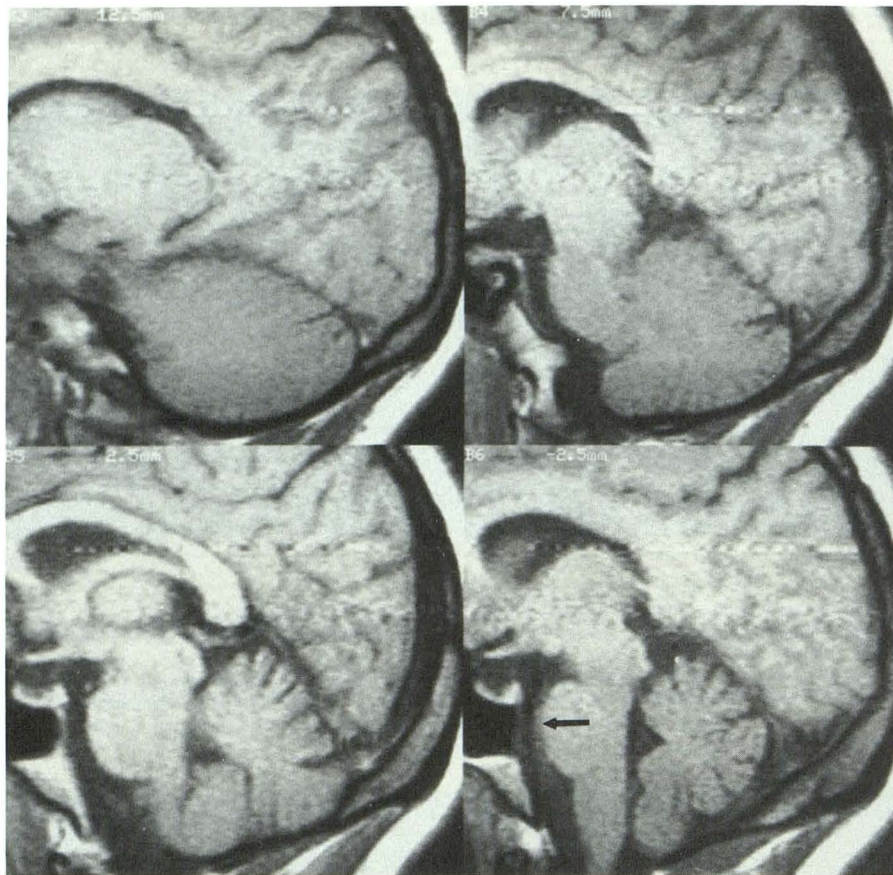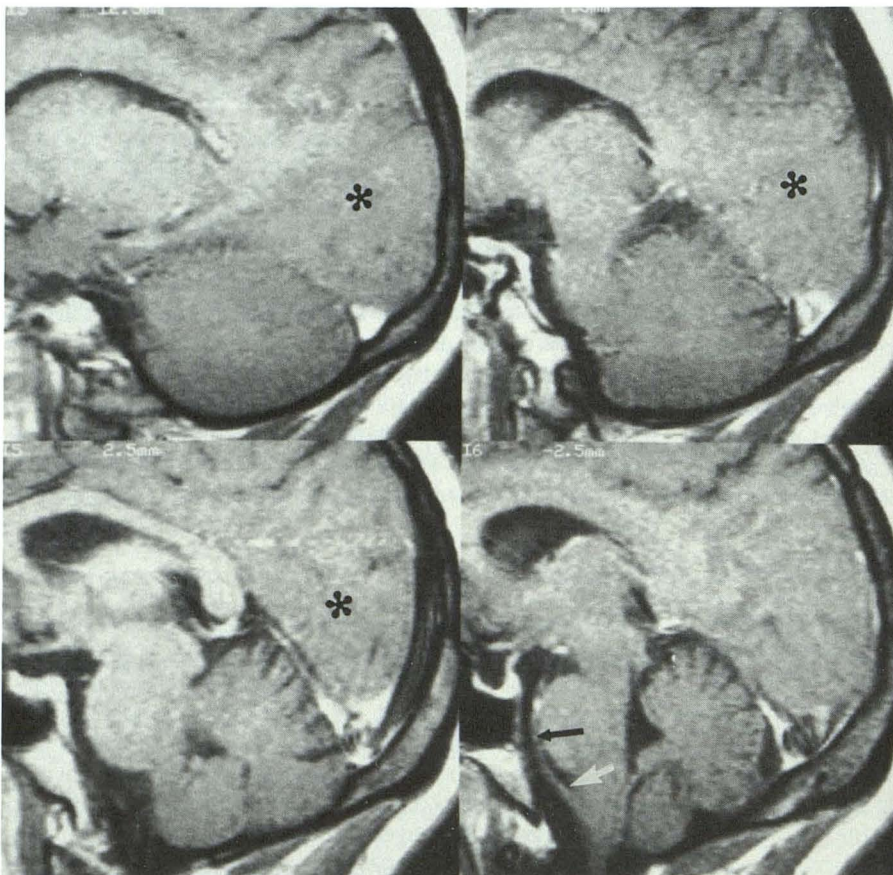
Fig. 6.—35-year-old woman with stroke symptoms rapidly progressing from cerebellar dysfunction to unresponsiveness.

A, Precontrast parasagittal T1-weighted images (450/20) obtained 2½ hr after onset show signal isointense with brain in prepontine cistern (*arrow*).

B, Corresponding postcontrast parasagittal images obtained 40 min after *A* show development of massive brain swelling in occipital lobes (*asterisk*) and cerebellum, and progression of a blood clot in the basilar artery (*arrows*).

*C*, Axial T2-weighted image (2000/100) obtained before contrast administration shows no parenchymal signal abnormality.
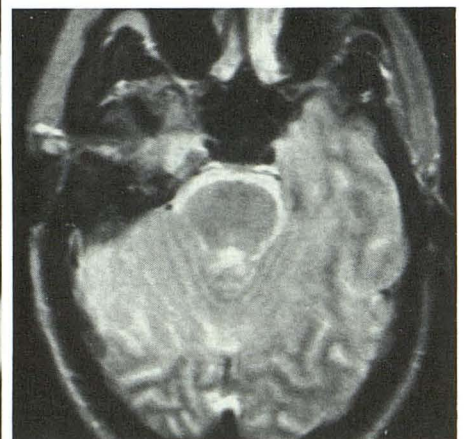
Fig. 7.—67-year-old man who underwent preoperative balloon test occlusion of left internal carotid artery and developed acute ischemic symptoms after 2 min of carotid occlusion. After balloon deflation, preservation of carotid flow was demonstrated angiographically.

A, Postcontrast coronal T1-weighted image (583/20) obtained 2 hr after onset shows diffuse parenchymal enhancement of left cerebral hemisphere (*arrows*). Parenchymal morphologic changes (swelling) are noted without evidence of discrete arterial enhancement.

B, Axial T2-weighted image (2000/100) shows no parenchymal signal changes.

A   B
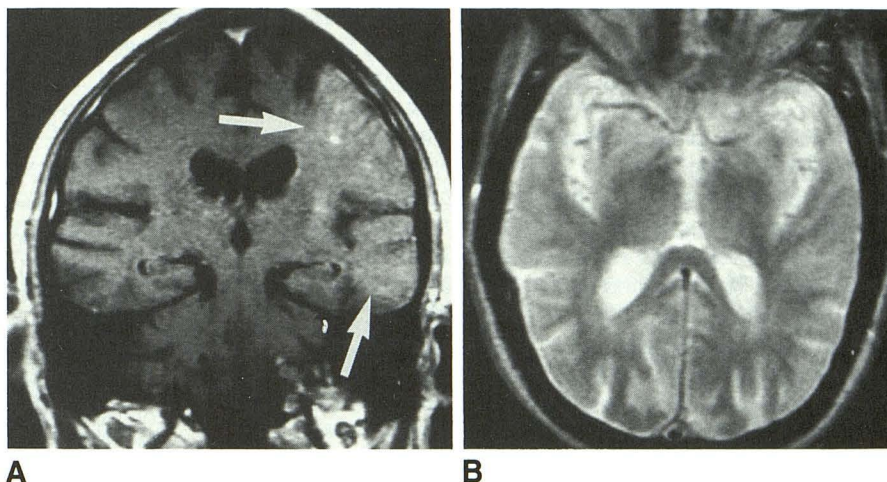
Altered Flow Kinetics

The ability of unenhanced MR imaging to detect absence of flow in large cerebral arteries early in complete thrombotic occlusion has been reported previously [13–15]. Normal arterial structures are generally devoid of intraluminal MR signal due to rapid and/or turbulent flow, the so-called flow-void phenomenon. Normal flow-void phenomenon is seen better on T2-weighted images because of increased susceptibility to motion on long TE sequences and better contrast between the dark vessel and surrounding bright CSF. All lesions with detectable absence of flow void in this series (approximately one third) were demonstrated better with T2 weighting than with T1 weighting.

We have shown that abnormal vascular enhancement by contrast material further improved the ability of MR to detect early cerebral ischemia by accentuating the underlying flow alteration. This could be detected not only in large vessels in both anterior and posterior circulation lesions but also in smaller vessels in distal cortical distributions. However, we were not able to detect enhancement of the terminal arteries supplying isolated deep noncortical lesions, most likely because of their small size. Absence of normal flow void was seen better on T2-weighted images, whereas arterial enhancement was seen better on the enhanced T1-weighted images with respect to the surrounding dark CSF. The contrast agent, therefore, improved the detection of flow derangement in distal cortical branches, which went undetected on the unenhanced images.

Arterial enhancement in stroke has been reported recently [16] and has been attributed to sluggish arterial flow near the infarct. We have encountered cases with arterial enhancement that had angiographic evidence of slow antegrade or retrograde (collateral) flow in the enhancing arteries, while other cases with angiographically proved complete proximal occlusion showed no arterial enhancement in the distal arterial tree when excellent collateral flow was present. Therefore, we also believe that slow flow is the likely mechanism for enhancement of the arterial tree in many of these lesions [17].

In the evaluation of flow alteration in the proximal internal carotid and middle cerebral arteries, we prefer the axial imaging plane because the entire course of these vessels is demonstrated best with the fewest images without significant interference from flow-related enhancement. Optimal evaluation of basilar artery occlusions frequently requires both sagittal T1-weighted imaging and axial T2-weighted imaging [13].

Biophysiologic Phenomena

The other pathophysiologic factors that may result in abnormal MR findings in cerebral ischemia are biophysiologic phenomena (Table 2). The ability of MR to detect parenchymal abnormalities in ischemic stroke caused by these biophysiologic mechanisms depends mostly on anatomic distortion seen best on T1-weighted imaging and/or signal changes seen best on T2-weighted imaging.

T2-weighted images have proved to be sensitive in detecting CNS lesions, such as tumor, on the basis of the abnormal accumulation of fluid that develops over a relatively long period of time. However, the ability of T2-weighted imaging to detect early ischemia appears to be limited. Signal abnormalities on T2-weighted images in this series were not sensitive in detecting ischemia in the first 8 hr after onset and often lagged behind detectable physical flow alterations, abnormal arterial contrast enhancement, and morphologic changes on T1-weighted images. The cause for the delayed development of the signal change on T2-weighted images is unknown. It is probably related to the relatively delayed onset of extracellular swelling or vasogenic edema, which is thought to be caused by the breakdown of the blood-brain barrier (BBB) [10, 18–22]. Experimental work has shown this breakdown does not occur to a significant extent until approximately 6 hr after onset and is then associated with leakage of water as well as proteins and macromolecules into the extracellular and intracellular spaces. The time course of signal change on T2-weighted images found in our series is in accordance with the temporal development of vasogenic

edema. Significant signal change on T2-weighted images, before the development of vasogenic edema, may be limited by relatively insufficient changes in water content (3%) [19], macromolecular shifts, and hydration binding of free water to macromolecules during the first few hours of ischemia (cytotoxic edema phase).

Signal changes on T1-weighted sequences were the most delayed change demonstrated, as expected, and were the least sensitive indicators in the early detection of stroke. This is mostly due to the limited sensitivity of the currently available, relatively T1-weighted pulse sequences in detecting abnormal water accumulation.

Morphologic changes in brain parenchyma demonstrated best on T1-weighted images were early indicators of acute cerebral ischemia and could be seen as early as 2 hr after onset. The occurrence of both abnormal arterial enhancement and morphologic changes (even without signal change) was strongly suggestive of ischemia early in its development. Although the cause of parenchymal morphologic swelling without concomitant signal change is unknown, we speculate that this is related predominantly to the formation of cytotoxic edema. Cytotoxic edema is considered to be the abnormal accumulation of intracellular water that develops in ischemic brain parenchyma as a consequence of decreased availability of ATP and the resultant dysfunction of the Na-K-ATP pump [10, 19, 20, 22, 23]. This occurs within minutes after the induction of experimental ischemia [19]. The excess intracellular fluid is thought to be primarily free water without proteins or macromolecules. The overall shift from the intravascular space at this early stage reportedly results in a modest 3% increase in the amount of parenchymal water. In addition to the relatively small shift in total water during this stage, this free water is not yet bound to macromolecular components. We speculate that this may explain the formation of detectable swelling on T1-weighted images without significant signal changes on T2-weighted images.

Evidence of massive brain swelling was seen to progress rapidly over the course of approximately 1 hr in two patients in this series. The radiologic detection of such dramatic brain swelling in the first few hours of ischemia has not been reported previously. Again, the cause for the shift of such a large amount of water without accompanying signal changes on T2-weighted images is unknown.

Mass effect was usually not detected in isolated noncortical infarcts in our series until signal changes on T2-weighted images were evident. This was probably due to the small size of many isolated noncortical infarcts and to the terminal arteriolar nature of these vascular zones, which lack significant collateral flow. The detection of mass effect is also frequently dependent on the distortion of normal surrounding structures, an indirect sign. The thin, delicate sulci interposed between cortical gyri, on the other hand, are easily distorted by a relatively small amount of cortical edema, and this distortion is easily appreciated by MR. The lack of sulci adjacent to deep brain structures limits the detectability of slight amounts of edema in these areas. In our study, the obliteration of sulci and effacement of gyri in cortical infarctions were usually detected better on the T1-weighted im-

ages. This was primarily due to the relatively better contrast between CSF and cortical gyri on the T1-weighted images.

Parenchymal Enhancement Patterns

Most infarcts in our study group did not demonstrate enhancement of brain parenchyma in the first 24 hr after onset of symptoms. This is in agreement with other series that have shown development of typical gyriform enhancement of cortical strokes only in the subacute and chronic (i.e., more than 1–2 weeks) phases [24–27]. The absence of parenchymal enhancement in the acute stage of ischemia is probably related to an inability to deliver sufficient amounts of contrast material to the ischemic area caused by the underlying vascular abnormality and a lack of significant collateral circulation. Those lesions that showed parenchymal enhancement within the first day were usually related to transient vessel occlusion, partial vessel occlusion (i.e., partially intact antegrade flow or partially intact collateral flow), or watershed zone infarct [17]. The mechanism for exaggerated and early enhancement of these types of ischemic lesions is unknown, but probably relates to the abnormal accumulation of contrast material in the presence of an intact supply of contrast material. Possible mechanisms for the local concentration of contrast material include BBB breakdown and/or local hyperemia. The explanation for involvement of a greater area by contrast enhancement than that by signal change on T2-weighted images in these cases is also unknown, but may be indicative of a lesser degree of tissue insult [17].

Limitations

Several limitations of the present study should be noted. The major purpose of this work was to investigate the early MR findings in acute stroke and the time course of their development. Our study population was limited to only those patients with acute clinical symptoms who had MR examinations and showed corresponding MR abnormalities. We have encountered at least three patients (excluded from the present study) with clear neurologic deficits who had no abnormalities on MR imaging. In addition, stroke was often only one of many possible diagnoses in the patients referred to us for MR studies. Clinically, many of these were considered unlikely to have had a stroke and others were lost to followup; therefore, we did not attempt to assess the sensitivity, specificity, or accuracy of MR findings early in acute cerebral ischemia. Another limitation stems from the nonuniform temporal distribution of MR examinations in the first 24 hr (Fig. 1). Lack of data points at several time intervals limits more precise determination of the time of onset of several of the MR abnormalities. A prospective and controlled study will be necessary to determine the reliability of MR in detecting early ischemic stroke.

Hemorrhagic lesions were excluded from the study because their mechanism probably differs from that of primary cerebral ischemia. Although T2-weighted images are less

sensitive in the detection of early brain ischemia, we do not suggest eliminating the T2-weighted sequence, as many other diseases may mimic the clinical signs of stroke. In addition, acute parenchymal blood may be detected on T2-weighted images in higher field scanners.

Finally, our results differ slightly from those reported in the imaging of experimental infarcts in other species [9–11, 24]. Whether this is attributable to a difference in vascular anatomy and/or physiology or to different mechanisms of occlusion (gradual or intermittent thrombosis vs abrupt surgical ligation) remains uncertain. Lack of precision in determining the time of occlusion may also contribute to these disparate results.

In conclusion, MR appears to be a useful method for diagnosing early acute cerebral ischemia. Early MR findings include the absence of normal flow void and the presence of arterial enhancement and morphologic changes that may precede detectable signal changes. These early MR signs represent additional evidence of early acute ischemia and further demonstrate the advantages of MR over CT in detecting early ischemic injury. Furthermore, the use of paramagnetic contrast agent is often valuable in the detection and evaluation of acute stroke in its very early stages.

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