SUMMARY We used three-dimensional proton NMR images to study ischemic infarction in the territory of the vertebral-basilar posterior cerebral circulation. The study includes sixteen cases, eight of which are presented in detail. In seven cases, the infarctions were secondary to demonstrable large artery occlusive disease — vertebral, basilar, or posterior cerebral. In nine cases, the infarctions were secondary to what was presumably small vessel disease. In fifteen of the sixteen cases, NMR imaging could locate the infarct, inversion recovery and spin-echo pulse sequences being more sensitive than the saturation recovery pulse sequence. This efficiency rests on the high sensitivity of ischemic infarction to changes in $T_1$ and $T_2$ relaxation time, highlighted in the inversion recovery and spin-echo images, respectively. The additional advantages of the three-dimensional approach, and the lack of bone artifact, make NMR imaging superior to CT scanning in identifying areas of infarction in the territory of posterior cerebral circulation.

CASES OF ISCHEMIC STROKE in the brain stem and cerebellum often present a stunning array of clinical symptoms and signs. When they do, the areas of encephalomalacia can usually be located adequately. But less dramatic cases can pose difficulties. Then an imaging technique that could confirm the presence of an ischemic insult would be very helpful. Unfortunately, current imaging techniques do not locate with precision ischemic infarction in the vertebral basilar posterior cerebral circulation. Therefore, symptoms of ischemic infarction cannot be ascribed confidently to either of their probable causes — large vessel, vertebral-basilar posterior cerebral atherothrombotic disease or small vessel, basilar branch atherothrombotic or lipohyalinotic disease. This imprecision has become a serious constraint, because newly developed therapies for ischemic stroke in this vascular territory depend on precise in vivo documentation of the extent and location of the infarct.

Modern CT scanning can locate an infarct in the pons and cerebellum, sometimes; but it fails too often to document the precise extent of an infarct within these areas, because of interference from bone artifacts, computer ripple ("overshoot") artifacts, partial volume effects, or an inept choice of the two-dimensional plane. Only pathologic examination has been able to delineate the area of an infarct precisely enough to allow a correlation between specific areas and clinical syndromes. Another weakness of CT scanning is that it does not follow satisfactorily the early phases of an ischemic insult. However, proton $T_1$ and $T_2$ relaxation times change markedly during the early stage of ischemic infarction, and this can be utilized in generating diagnostically useful proton NMR images. Recent developments permit proton NMR imaging to pinpoint small areas of encephalomalacia in the mesencephalon, pons, and cerebellum, especially when utilizing three-dimensional techniques that produce thin sections and pulse sequences that accentuate change in $T_1$ and $T_2$ relaxation times. We report our initial results in proton NMR imaging ischemic infarction in the territory of the vertebral-basilar posterior cerebral circulation. We include 16 cases, seven with occlusion of large arteries (basilar, vertebral, posterior cerebral) and nine with disease presumably of small penetrating arteries (table 1). In each case but one, findings aided greatly in the clinicoanatomic correlation. We present eight of the 16 cases in detail and describe their images because they illustrate specific NMR imaging points.

Methods

NMR imaging was performed using a prototype system based on a water-cooled, 4-coil electromagnet operating at a field strength of 1.4 KiloGauss and generating proton images at 6.25 MHz. True, three-dimensional data were collected from the entire volume of the head for subsequent filtered backprojection of
data and reconstruction of 1 or 3mm thick planes selected at any angle desired. In these studies, three radiofrequency (RF) pulse sequences were used: (1) Inversion recovery (IR), (b) Saturation recovery (SR), and (c) Carr-Purcell-Meiboom-Gill (CPMG) spin echo (SE). (a) IR images were obtained in all patients using an IR T1, sequence. This sequence, the details of which have been previously reported,3-5 generate two NMR signals (S1 and S2) for each gradient angle. Reconstruction of the S1 signal generates an image that approximates proton density; regions of long T1, such as CSF have reduced signal intensity and thus do not reflect the true proton density. Reconstruction of the S2 signal generates a T2 weighted inversion-recovery image; the T value between 180° and 90° pulse was set at 400 ms. A 180° refocusing echo, applied 16 ms after the 90° ‘read’ pulse, is utilized to generate both nuclear signals (S1 and S2), therefore, a small T2 component is also present in these images. (b) Volumetric SR images were obtained in four patients. The SR acquisition utilizes a 90°, τ, 90° type sequence with an interpulse delay (repetition rate) of 230 ms. This sequence also employs a 180° refocusing pulse. The signal intensity of the SR images depends on proton density, T1, and a small T2 component. The T2 discrimination of SR images is less apparent than for IR images. (c) SE images were acquired in eight patients using a Carr-Purcell-Meiboom-Gill Sequence. This acquisition uses a 90°, τ, 180°, 2τ, 180°, . . . sequence with a τ value of 30.7 msc.; four 180° pulses are employed resulting in four spin-echo images at 61, 123, 184 and 246 msec. Further details on the principles of NMR imaging and on our techniques may be found elsewhere.3-3

A total of 16 patients with ischemic strokes in the vertebral-basilar posterior cerebral circulation were scanned and are included in the study (table 1). This includes ten males and six females between the ages of 40 and 75. Each of the 16 patients with ischemic strokes in the territory of the vertebral-basilar circula-

<table>
<thead>
<tr>
<th>Case presented in detail</th>
<th>Location of infarct by NMR</th>
<th>Site of vascular disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>left lateral occipital lobe cortex and borderzone between the posterior and middle cerebral artery territories</td>
<td>occluded left posterior cerebral artery 2 mm above the origin with the basilar with reconstituted flow distally</td>
</tr>
<tr>
<td>No</td>
<td>right and left basis pontis and pontine tegmentum</td>
<td>severe midbasilar stenosis (selective cerebral angiography)</td>
</tr>
<tr>
<td>No</td>
<td>right medial occipital lobe left median basis pontis</td>
<td>retrograde flow down the basilar artery basilar origin not seen (digital subtraction angiogram)</td>
</tr>
<tr>
<td>case 1</td>
<td>left medial occipital lobe and left midbrain cerebral peduncle</td>
<td>left posterior cerebral artery at its origin from the basilar (selective cerebral angiography)</td>
</tr>
<tr>
<td>case 2</td>
<td>left inferior cerebellar hemisphere</td>
<td>occlusion of the vertebral basilar junction with retrograde flow down the basilar artery (digital subtraction angiogram)</td>
</tr>
<tr>
<td>case 3</td>
<td>left medial occipital lobe left lateral ponto medullary</td>
<td>occluded right and atretic left vertebral artery, retrograde flow down the basilar artery (digital subtraction angiogram)</td>
</tr>
<tr>
<td>case 4</td>
<td>left lateral pontine tegmentum right basis pontis</td>
<td>bivertebral occlusion, retrograde flow down the basilar artery (selective cerebral angiography)</td>
</tr>
<tr>
<td>no</td>
<td>left pontine tegmentum</td>
<td>presumed occlusion of a basilar penetrating branch artery resulting in a residual peripheral, left facial weakness</td>
</tr>
<tr>
<td>no</td>
<td>left basis pontis</td>
<td>presumed occlusion of a basilar penetrating branch artery resulting in a right pure motor hemiparesis</td>
</tr>
<tr>
<td>no</td>
<td>presumed subthalamic paramedian infarction NOT identified</td>
<td>presumed occlusion of the medial thalamic no perforating artery resulting in a supranuclear paralysis of upgaze with upbeatng nystagmus and absence of convergence</td>
</tr>
<tr>
<td>no</td>
<td>left basis pontis</td>
<td>presumed occlusion of a basilar penetrating branch artery resulting in a right pure motor ataxic hemiparesis</td>
</tr>
<tr>
<td>no</td>
<td>right paramedian midbrain tegmentum and peduncle</td>
<td>presumed occlusion of a basilar tip paramedian penetrating artery resulting in a right third nerve palsy and left hemiparesis</td>
</tr>
<tr>
<td>case 5</td>
<td>right basis pontis</td>
<td>presumed occlusion of a basilar penetrating branch artery resulting in a left motor hemiparesis</td>
</tr>
<tr>
<td>case 6</td>
<td>left basis pontis</td>
<td>presumed occlusion of a basilar penetrating branch artery resulting in a right pure motor hemiparesis</td>
</tr>
<tr>
<td>case 7</td>
<td>right paramedian midbrain</td>
<td>presumed occlusion of a basilar tip paramedian penetrating artery resulting in a right third nerve palsy and ataxic left arm</td>
</tr>
<tr>
<td>case 8</td>
<td>left middle cerebellar peduncle and lateral pontine tegmentum</td>
<td>presumed occlusion of a basilar penetrating artery resulting in ataxia of the right arm and leg</td>
</tr>
</tbody>
</table>
tion have documentation of the pathophysiological nature of their stroke supported by transfemoral angiography (seven), digital subtraction angiography (six), or clinically (three). For purposes of comparison, all patients underwent cerebral CT scanning (GE 8800 or EMI 1010), but it was not possible to have the two studies made on the same day. The period between the CT and NMR scan varied from 0–10 days. In four patients the CT scan was done within the first 24 hours of the stroke, and would therefore not be expected to show the stroke. It was not possible to obtain the NMR scan within the first 24 hours of the stroke in any of the patients.

Results (table 1)
All 16 patients were hypertensive. Seven of the 16 patients had large vessel disease. Five of the patients in the large vessel group had basilar insufficiency with retrograde flow due to proximal basilar occlusion (one), proximal to mid basilar stenosis (two), bivertebral occlusive disease (two). The two remaining patients in the large vessel group had proximal posterior cerebral artery occlusion, one at its origin from the basilar artery and one more distally. Nine patients had presumed small vessel disease, thrombotic (atherothrombotic or lipohyalinotic), or embolic, which on digital subtraction angiography or conventional angiography showed patent large arteries (six) or clinically had no evidence of basilar insufficiency (three).

In 15 of the 16 cases the location of the infarct could be detected by NMR scanning, particularly utilizing inversion recovery and spin-echo pulse sequences that accentuate change in T1 and T2 relaxation times, respectively. The one case in which the infarction was not demonstrated was that of a 56 year old man with amaus but blurring due to presumed movement artifact. Images obtained using the CPMG sequence highlighted areas of infarction. Three of the eight patients had saturation recovery studies, while five of the eight patients underwent CPMG spin-echo imaging sequence that generates four echo images; the latter pulse sequence was added to our capabilities more recently. Images obtained using the CPMG sequence have a lower spatial resolution than IR or SR images, however, the T2 weighting of the spin-echo images, particularly in later echoes (numbers 3 and 4), reliably highlights areas of infarction as prolonged T2 relaxation times.

Case Histories

Group 1
Large vessel atherothrombotic disease of the vertebral basilar system.

Case 1
This 47 year old hypertensive man suddenly became dizzy and staggered. He slept for six hours and awoke with nausea and vomiting, trouble speaking and persistent staggering on walking. The following day he developed numbness and weakness of the right face, arm and leg. His speech difficulty persisted; initially, a few words would come out, but then none. Forty-eight hours later the right face, arm and leg had become completely paralyzed, and he was transferred to the Massachusetts General Hospital.

Upon his arrival four days after the onset of his symptoms, speech was hesitant. He would make a few well-formed words and then lapse into literal and verbal paraphasic errors; complex words were correctly repeated from dictation but he did not read headlines. There was a right homonymous hemianopia. He easily showed two fingers and waved goodbye to command with his left hand. There was a complete right hemiplegia. He easily identified touch over the left arm, but not on the right. Vertebral angiography showed an occlusion of the left posterior cerebral artery at its origin from the basilar artery. A CT showed a left occipital and medial temporal lobe area of decreased intensity consistent with infarction, corresponding to the entire territory of the left posterior cerebral artery. Inversion recovery NMR study, obtained two weeks after the onset of symptoms, showed a similar area of decreased signal intensity (prolonged T1 relaxation time) in the left temporal occipital lobe (fig. 1a). In addition, the inversion recovery NMR image showed an area of decreased image intensity (infarction) in the left cerebral peduncle which accounted for his right hemiplegia (fig. 1b).

Case 2
This 73 year old, hypertensive, diabetic woman suddenly became dizzy and began to vomit. She was unsteady when she walked. Examination on her arrival at the hospital showed nystagmus on right and left lateral gaze, anisocoric left pupil, and slight ptosis of the left eye. Although there was slight left facial droop, teeth show was equal. Dysarthria was present, yet her tongue protruded in the midline and she could swallow.
Cerebellar ataxia was noted in the left arm and leg and she fell over when seated unaided. Intravenous heparin was begun. Over the following two hours her nystagmus and her right lateral gaze paresis disappeared. She was less unsteady on sitting and the ataxia of the left arm and leg had diminished. Left ptosis persisted. A CT scan obtained 12 hours after the onset of illness was negative. Inversion recovery NMR study, obtained 36 hours after the onset of illness, showed an area of decreased signal intensity in the left cerebellar hemisphere corresponding to ischemic infarction in the distribution of the left posterior inferior cerebellar artery (fig. 2a). The same area had an increased signal intensity in the last two echo images of the CPMG study indicating a prolonged $T_2$ relaxation time (fig. 2c–f). The spin density image (fig. 2b) showed no abnormality in the area of infarction seen on the inversion recovery and spin-echo scans. Forty-eight hours after the onset of illness, she became stuporous and hyponatremic. Because of the rather large area of infarction seen on the inversion recovery NMR scan, cerebellar swelling was suspected as the cause of the clinical deterioration and was documented by a follow-up CT scan. She responded to mannitol and fluid restriction. Digital subtraction angiography showed retrograde flow down the basilar artery; small, atretic vertebral arteries could be seen at their origin, but the vertebral-basilar junction could not be identified. Her neurologic examination improved to normal over the next two weeks.

The left cerebellar infarction seen on the inversion recovery and spin-echo NMR scans at 36 hours was of crucial importance as it alerted her physicians to the possibility of cerebellar swelling. Although she had some elements of left lateral medullary infarction on physical examination (left eye ptosis), the lateral medulla could not be seen clearly to allow a diagnosis to be made or excluded.

**Case 3**

This 68 year old, hypertensive man with arteriosclerotic cardiovascular disease was neurologically well until five months prior to admission, when suddenly he became dizzy, unsteady and had difficulty using his right arm and hand to open the door of his car. Symptoms cleared later in the day. Two months prior to his current admission, while seated at the dinner table, he suddenly saw a kaleidoscope of lights in the right visual field; after a few seconds to a minute, the light had disappeared and he became blind in the right visual field. He got up and was unsteady on his
feet and his speech was slurred. During the week of hospitalization, both gait and speech improved greatly, but his hemianopia persisted. Four days after his discharge from the hospital, dizziness and unsteady gait suddenly recurred and he lost hearing in his left ear. Over the next month and a half his hearing loss and hemianopia persisted, but his gait and dysarthria again improved.

On admission he walked unaided on a wide-base but could not tandem walk. There was cerebellar ataxia of the right arm and leg but no dysarthria. He had a right homonymous hemianopsia, and hearing loss in the left ear had persisted.

CT scan and inversion recovery NMR image both showed an abnormality in the left medial occipital area consistent with infarction in the territory of the left posterior cerebral artery (fig. 3c & d). The saturation recovery image also showed a lesion in the same area. In addition, there was a decreased area of image intensity in the region of the left lateral ponto-medullary junction, corresponding to an area of infarction in the territory of the anterior inferior cerebellar artery (figure 3b–arrow). The CT scan also showed a similar area of infarction (figure 3a). The spin-echo pulse sequences, particularly in the third and fourth echo images, identified the infarctions as areas of increased image intensity corresponding to prolongation of the $T_1$ relaxation time in the left medial occipital lobe (fig. 4) and left lateral ponto-medullary junction (fig. 5).

Digital subtraction angiogram showed retrograde flow down the basilar artery and demonstrated an occluded right vertebral artery and an atretic left vertebral artery. This case, therefore, represents basilar artery insufficiency with infarction of the territory of both the left posterior cerebral artery and the left anterior inferior cerebellar artery. The patient was placed on warfarin and was stable over the subsequent year.

**Case 4**

This 75 year old physician with hypertensive arteriosclerotic cardiovascular disease suddenly developed a burning feeling in the left cheek which resolved within an hour. The next morning he developed horizontal diplopia while watching television; an hour later, he became nauseated and vomited. On arrival at the hospital he was noted to have a left gaze paresis, full gaze to the right, a rightward-beating counter-clockwise rotatory nystagmus, a slight left facial droop with decreased forehead wrinkle and a decreased left corneal reflex; there was no ataxia of the arm or leg, and he could walk well. One week later there was still slight left facial weakness and limitation of left lateral gaze.

Vertebral angiography demonstrated an occlusion of the right vertebral artery at the junction with the basilar and an occluded left vertebral artery as it entered the skull; the basilar artery was not opacified. CT scan (fig. 6a) obtained the day after the onset of symptoms, was normal. The inversion recovery NMR study (fig. 6b–e) obtained one week after the onset of symptoms showed an area of decreased image intensity in the left lateral pontine tegmentum and right basis pontis. The sagittal (fig. 6b–arrow) and serial horizontal sections...
FIGURE 5. Case 3. CPMG spin-echo images at same level as in Figure 3a & b. The left AICA territory infarction is well seen. The third and fourth echo images show the infarct to have the longest $T_2$ relaxation time value; i.e., apparent brightness and later echoes (c & d-arrows).

Group 2
Small vessel or branch occlusive disease (atherothrombotic or lipohyalinotic).

Case 5
In June of 1982 this 71 year old man noted the sudden onset of right arm weakness and difficulty speaking in sentences. CT scan showed an infarct in the white matter adjacent to the left frontal horn of the lateral ventricle and a smaller lacunar infarct in the right corona radiata. A tightly stenotic atherothrombotic lesion narrowing to lumen diameter of less than 2mm was demonstrated angiographically at the origin of the left internal carotid artery and the patient underwent endarterectomy. Over the next month his arm weakness and speech improved to almost normal. He then suddenly developed difficulty walking and incoordination of the left limbs. Upon awakening the following morning, he noted that he was unable to move his left limbs and he cried uncontrollably in conversation. On examination there was dysarthria and a pure motor left hemiplegia involving face, arm and leg; there was no left hemineglect or gaze paresis. Digital subtraction angiography demonstrated normal vertebral, basilar and carotid arteries. CT scan showed an abnormality in the same place, but the diagnosis of lacunar infarction was uncertain because of the presence of bone artifact.

Case 6
Two months prior to the NMR imaging study, a 42 year old hypertensive, diabetic architect awoke with weakness of the right arm and leg which worsened by the afternoon; in addition, he complained of slurred speech. On examination at the time of the NMR study he had improved to the point that he could walk with a circumduction gait and had moderate arm and leg weakness and a slight right facial droop; his speech was fluent without dysarthria, and he easily repeated complex sentences from dictation.

Saturation recovery and inversion recovery NMR studies (fig. 9a & b) demonstrated an abnormal reduction of image intensity (prolonged $T_1$, consistent with a lacunar infarction in the left basis pontis corresponding to the territory of a paramedian branch of the basilar artery. CT scan showed an abnormality in the same place, but the diagnosis of lacunar infarction was uncertain because of the presence of bone artifact.

Case 7
A 72 year old hypertensive woman presented with a one-and-a-half day history of falling to the left fol-
followed by the sudden onset of diplopia and ptosis of the right eye. On examination there was mild dysmetria of the left arm and leg, as well as a right third nerve palsy with almost complete ptosis. Strength was equal in the arms and legs, but finger-thumb tap was slower on the left. A CT scan done 2 days after the onset of symptoms was normal. Vertebral angiography showed mild atheromatous changes at the proximal basilar artery and a normal distal basilar-posterior cerebral artery junction. NMR inversion recovery study, obtained four days after the onset of stroke, showed an area of diminished image intensity (prolonged T₁) in the right paramedian midbrain tegmentum (fig. 10a–arrow). The four spin echoes of the CPMG pulse sequence (fig. 10b–e) showed the same lesion as bright in the third (d) and fourth (e) echo images, representing an area of prolonged T₂ relaxation time. It is suspected that the patient had a thrombotic or embolic occlusion of a midbrain penetrating branch of the right posterior cerebral artery proximal to the posterior communicating artery, which led to ischemic infarction in the area of altered image intensity on both the inversion recovery and spin-echo NMR images.

Case 8

A 63 year old hypertensive man awoke from his sleep to go to the bathroom and noted that he was unsteady in using his right arm and leg. The following morning, although improved, he was still unsteady and came to the hospital. His voice was dysarthric and he
FIGURE 10. Case 7. The IR (A) image four days after onset of stroke shows an area of diminished image intensity, suggesting prolonged $T_1$ values, in the right paramedian midbrain (arrows). CPMG spin-echo images at the same level (b-e) show bright signal intensity in the affected area not only in early echoes (b,c) suggestive of increased proton density, but also in later echoes (d,e,arrows), compatible with prolonged $T_2$.

had cerebellar ataxia of his right arm and leg; extraocular movements were full, without nystagmus, and there was no facial droop. Vertebral angiography was normal. CT scan, obtained the day of admission, was negative, but the pons and cerebellum were obscured by artifact (fig. 11a). Inversion recovery NMR study, obtained eight days after the onset of symptoms, was compatible with a left middle cerebellar peduncle pontine tegmentum lacunar infarction (fig. 11b–arrow). The area of infarction is depicted as an area of decreased image intensity corresponding to an area of prolonged $T_2$ relaxation time. The saturation recovery (fig. 11c) and spin-density images (figure 11d) did not show the lesion. A repeat CT scan done the same day as the NMR also showed the lesion but less clearly (fig. 12).

Discussion

This study presents a group of patients whose ischemic infarctions in the vertebral-basilar posterior cerebrovascular circulation were studied by proton NMR imaging. We did not design a sensitivity/specificity study correlating CT with NMR for three reasons: 1) the timing of the CT scans and NMR scans was not uniform, 2) the cases were not taken randomly or sequentially, and 3) new NMR imagers offer better resolution, now. Nonetheless, these 16 cases prove that proton NMR imaging techniques can locate the infarcted areas in the territory of the vertebral basilar circulation. The infarct was found in 15 of the 16 cases (table 1). In the exception, an upper midbrain subthalamic infarct, motion artifact resulted in a blurred image. We did not study lateral medullary infarcts because our prototype scanner does not give adequate resolution in this area.

In the present study using our prototype NMR imaging system, data may be collected over the total volume of the head. This 3-D methodology allowed us to reconstruct any plane through the head and provides good images of the pons, cerebellum and midbrain, as well as the supratentorial structures supplied by the posterior cerebral arteries and their proximal penetrating branches. This technique, therefore, allows us to visualize the lesion in three dimensions and to select the plane that best shows the full extent of the lesion, a factor which is so important for clinicopathologic diagnoses. Furthermore, 3-D techniques allow us to select planes which match those of CT scans or necropsy brain sections closely. Thus, in 15 of the 16 patients
NMR IMAGING OF VERTEBRAL-BASILAR STROKE/Kistler et al

with suspected ischemic infarction in the posterior cerebral circulation, the infarct was located precisely. The exception was a midbrain subthalamic infarct which we could not locate because motion artifact obscured the area. In spite of our three-dimensional methodology, an image of the medulla often lacks enough resolution to visualize an infarction of that structure. Thus, in one patient with transient symptoms referable to the lateral medulla, the NMR study was not helpful.

The eight cases we present in detail made specific points about proton NMR imaging in this arterial territory. The first four cases presented in detail showed that ischemic infarctions resulting from large vessel occlusive disease in the posterior cerebral circulation (either posterior cerebral artery, vertebral artery, or basilar artery), can be identified. Cases 3 and 4 document, in vivo, that in vertebral basilar ischemic disease the infarcted area can either be small and discreet (Case 4) or occupy widely separate locations in the arterial territory (Case 3). The second four cases presented in detail illustrate that the topography of lacunar infarctions resulting from small penetrating or branch vessel disease can be identified in the posterior fossa.

While the selection of these cases does not allow comparative studies of the timing of the onset or evolution of NMR alterations versus CT scan changes, probably NMR will detect ischemic infarction sooner after onset of symptoms than CT scan does. From our experimental stroke studies, changes in proton $T_1$ and $T_2$ relaxation times, as measured by NMR spectroscopy, were detected within 30 minutes of arterial occlusion. Similar findings were subsequently reported by Mano et al using $T_1$ and $T_2$ weighted imaging. This would suggest that NMR scanning should be able to detect ischemic infarction also in humans within the first minutes to hours after the onset of symptoms. Although there has been no systematic study documenting the acuteness of the onset of NMR changes in human ischemic infarction, changes have been easily detected on inversion recovery images less than 24 hours after the onset of symptoms.

In the present series, the NMR images were obtained between 36 hours and two months after the onset of symptoms. In each case the area of infarction was identified as an area of decreased image intensity on the inversion recovery scan. In inversion recovery images, areas of variously decreased image intensity may signify either decreased proton density or various prolongations of the $T_1$ relaxation time. Presumably, in ischemic infarction the areas of decreased intensity seen on images made utilizing this pulse sequence are due to prolongation of the $T_1$ relaxation time. In five of the eight patients, $T_2$-weighted images were obtained with the CPMG spin-echo pulse sequence in the same NMR imaging session as the $T_1$-weighted studies. In each case, areas of infarction appeared as increased image intensity persisting in later echoes using the CPMG sequence, signifying a prolongation of $T_1$ relaxation times. An example of prolonged $T_2$ in cerebral infarction is that of Case 2, where NMR studies were performed 36 hours after the onset of symptoms; the "spin density" image (fig. 2b) did not show the infarction seen in the IR (fig. 2a) or CPMG studies (fig. 2c–d). Thus, alterations in $T_1$ and $T_2$ relaxation times were the major factors in showing the abnormality on the IR and CPMG studies in this case. Currently, our "spin density" images do not reflect true proton density and carry some $T_1$ information. In the future it may be possible to produce "true" proton density images and therefore quantitate the amount of edema associated with infarction. At the time of the NMR study the patient did not have any symptoms of cerebellar swelling, but such symptoms did develop eight hours later.

Since rigidly bound protons in bone produce very prolonged $T_1$ and very short $T_2$ relaxation times, they generate little or no signal in the NMR pulse sequence used and therefore bone is not positively visualized on NMR images. As a result, there is no bone artifact in the images, a feature of considerable advantage over CT scanning in the investigation of posterior fossa strokes. In Cases 3, 6 and 8, artifact present on the CT scan limited its diagnostic certainty. While the question of abnormality was debated on the CT scans, the lesions were nevertheless positively identified on the NMR studies.

NMR imaging techniques thus appear to hold great promise in the study of posterior fossa strokes and in elucidating their clinicopathological correlates. In addition, because the topography of the lesion can be identified by NMR imaging, this technique promises to be of help in differentiating large from small vessel isch-
Anticoagulant-Related Hemorrhage in Acute Cerebral Embolism

ROBERT W. SHIELDS, JR., M.D.,* ROBERT LAURENO, M.D.,† TIM LACHMAN, M.D.,‡ AND MAURICE VICTOR, M.D.*

SUMMARY Five patients with nonseptic cerebral embolism of cardiac origin are reported in whom early anticoagulant therapy resulted in clinical deterioration or death from frank hemorrhage into the acute infarct. In each patient an initial CT scan excluded the presence of intracerebral hemorrhage and a second CT scan, after clinical deterioration had occurred, documented frank hemorrhage into the infarcted zone. All five patients had large infarctions in the right middle cerebral artery territory and three patients were mildly hypertensive. Four patients received heparin within 36 hours of their stroke and one was on warfarin anticoagulation therapy. Clinical deterioration occurred after intervals of several hours (2 cases), 5–6 days (2 cases) and 30 days (1 case). In only 2 patients was anticoagulant activity excessive at time of clinical deterioration. This report illustrates the danger of early anticoagulant therapy of acute nonseptic cerebral embolism, particularly in the setting of large infarction.

NONSEPTIC CEREBRAL EMBOLISM of cardiac origin is a common cause of stroke accounting for nearly one third of all cerebral infarctions.1 Despite this high incidence the correct management of this disorder remains uncertain. Although chronic anticoagulation therapy has proven to be effective in decreasing the incidence of recurrent arterial embolism of cardiac origin,2-4 its use in the setting of acute embolic cerebral infarction remains controversial.5-8 At issue is whether the potential benefit of anticoagulants in reducing the risk of early recurrent embolism outweighs their potential danger of inducing massive hemorrhage into the infarcted tissue. The concern that anticoagulation therapy may lead to frank hemorrhage in acute embolic cerebral infarcts has been based largely upon pathologic observations that such infarcts are often stippled with small petechial hemorrhages.9 There have been only a few well documented examples of intracerebral hematoma following the administration of anticoagulants to patients with acute cerebral embolism.10-14 The rarity of these reports has fostered the view that such an event is extremely uncommon and that early use of anticoagulants is uniformly safe.8 15-17 Our experience contradicts this view, and for this reason we are reporting five patients who developed catastrophic cerebral hemorrhage following anticoagulant therapy of nonseptic cerebral embolism.

Case Reports

The following five cases were extracted from a retrospective analysis of 51 consecutive patients with acute nonseptic cerebral embolism of cardiac origin, seen on the Neurology services of The Cleveland Metropolitan General Hospital, Cleveland, Ohio, The...
Vertebral-basilar posterior cerebral territory stroke--delineation by proton nuclear magnetic resonance imaging.


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