

Kyo Noguchi, MD • Toshihide Ogawa, MD • Hikaru Seto, MD • Atsushi Inugami, MD  
 Hiromu Hadeishi, MD • Hideaki Fujita, MD • Jun Hatazawa, MD • Eku Shimosegawa, MD  
 Toshio Okudera, MD • Kazuo Uemura, MD

## Subacute and Chronic Subarachnoid Hemorrhage: Diagnosis with Fluid-attenuated Inversion-Recovery MR Imaging<sup>1</sup>

**PURPOSE:** To evaluate fluid-attenuated inversion-recovery (FLAIR) magnetic resonance (MR) imaging in the detection of subacute and chronic subarachnoid hemorrhage.

**MATERIALS AND METHODS:** The authors performed 19 FLAIR MR imaging examinations at 0.5 T in 14 adult patients with subarachnoid hemorrhage 3–45 days after the ictus and 22 FLAIR examinations in 22 adult control subjects. The detection of subacute and chronic subarachnoid hemorrhage on FLAIR images was compared with the detection on conventional spin-echo MR and computed tomographic (CT) images.

**RESULTS:** In the detection of subacute subarachnoid hemorrhage, FLAIR (100% detection) was significantly superior to T1-weighted imaging (36% detection,  $P < .01$ ), T2-weighted imaging (0% detection,  $P < .02$ ), and CT (45% detection,  $P < .02$  [Fisher exact test]). Although FLAIR imaging (63% detection) was superior in chronic subarachnoid hemorrhage detection, there were no statistically significant differences between modalities. FLAIR imaging demonstrated all subarachnoid hemorrhage areas as high-signal-intensity areas within 18 days and up to a maximum of 45 days after the ictus. In a blind comparison, no FLAIR images acquired in control subjects were confused with those acquired in patients.

**CONCLUSION:** FLAIR diagnostic images are superior to conventional MR or CT images in patients with subacute subarachnoid hemorrhage.

**M**AGNETIC resonance (MR) imaging shows high sensitivity and accuracy in the diagnostic evaluation of a wide variety of intracranial diseases. Conventional MR imaging has been demonstrated to be superior to computed tomography (CT) for the diagnosis of subarachnoid hemorrhage in the subacute and chronic stages (1–6). It is generally accepted, however, that acute subarachnoid hemorrhage is difficult to detect with conventional MR imaging (1,7–9); thus, MR is not necessarily useful for the diagnosis of subarachnoid hemorrhage in all stages.

CT plays an important role in the diagnosis of acute subarachnoid hemorrhage and has generally replaced lumbar puncture for this purpose (10). The appearance on CT scans, however, is almost normal several days or weeks after the bleeding event has occurred; thus, at this stage, subarachnoid hemorrhage is difficult to diagnose with CT (10–12).

The fluid-attenuated inversion-recovery (FLAIR; Picker International, Cleveland, Ohio) sequence and its variations nulls the signal from cerebrospinal fluid (CSF) and produces heavy T2 weighting as a consequence of its long echo time (TE) (13–18). Recently, it was reported that acute subarachnoid hemorrhage within 3 days

after the ictus could be clearly demonstrated as an area of high signal intensity on FLAIR images and that FLAIR is comparable with CT in the detection of acute subarachnoid hemorrhage (19,20). The present study was performed to evaluate the detection of subarachnoid hemorrhage with FLAIR in comparison with detection with CT or conventional MR imaging in the diagnostic imaging of subacute or chronic subarachnoid hemorrhage.

### MATERIALS AND METHODS

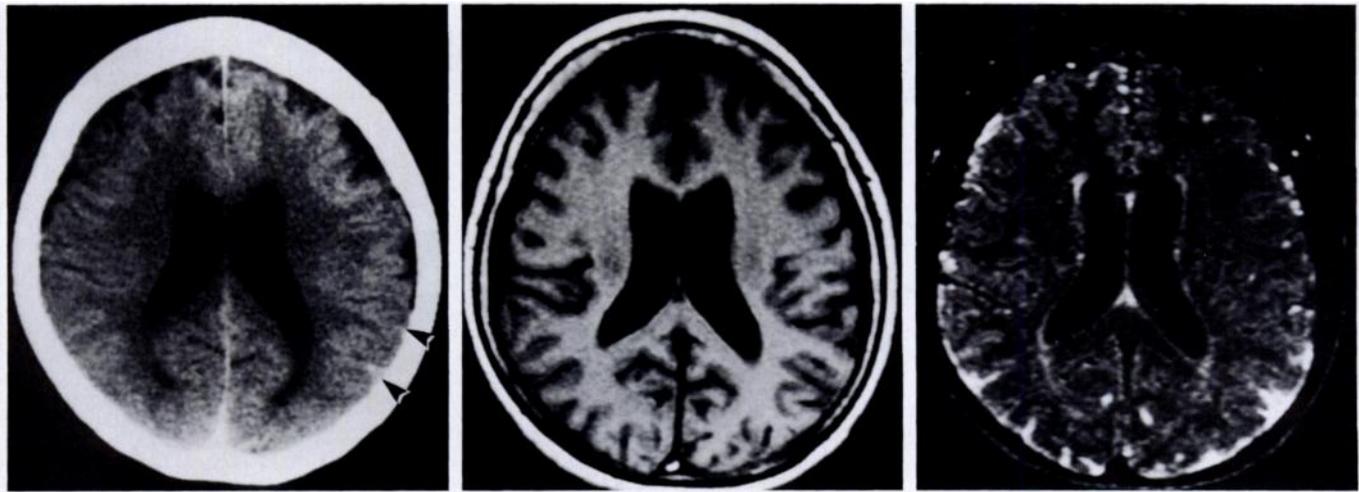
The study group consisted of 14 consecutive patients with subacute or chronic subarachnoid hemorrhage caused by a ruptured aneurysm (10 patients), a ruptured arteriovenous malformation (one patient), arterial dissection (two patients), or moyamoya disease (one patient). There were three men and 11 women aged 33–70 years (mean age, 58.7 years). Subarachnoid hemorrhage was confirmed on the basis of findings of the initial CT examination performed at the time of hospitalization as an area of high attenuation in the subarachnoid space in nine patients (including four patients at the acute stage, ie, within 3 days after the ictus) and on the basis of the lumbar puncture results for the CSF in the other five patients, in whom high-attenuation subarachnoid hemorrhage was not detected on the CT scans. Four-vessel cerebral angiography was performed in all patients for evalua-

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**Abbreviations:** CSF = cerebrospinal fluid, FLAIR = fluid-attenuated inversion recovery, RBC = red blood cell, SE = spin echo, TE = echo time, TI = inversion time, TR = repetition time.

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<sup>1</sup> From the Department of Radiology, Toyama Medical and Pharmaceutical University, 2630 Sugitani, Toyama City, 930-01 Toyama, Japan (K.N., H.S.); and the Departments of Radiology and Nuclear Medicine (T. Ogawa, A.I., H.F., J.H., E.S., T. Okudera, K.U.) and Surgical Neurology (H.H.), Research Institute of Brain and Blood Vessels, Akita, Japan. Received July 8, 1996; revision requested August 28; revision received October 15; accepted October 21. Address reprint requests to K.N.



**Figure 1.** Subarachnoid hemorrhage due to a ruptured aneurysm of the left internal carotid artery in a 65-year-old woman. CT scan and MR images were obtained 4 days after the ictus. (a) CT scan shows a high-attenuation change in the left parietal cerebral sulci (arrowheads). However, it is difficult to diagnose subarachnoid hemorrhage on the basis of this CT scan. (b) T1-weighted MR image (500/35) shows no area of high signal intensity caused by subarachnoid hemorrhage. (c) FLAIR MR image (5,000/120/2,000) clearly shows subarachnoid hemorrhage as diffuse high-signal-intensity areas in the bilateral cerebral sulci.

tion of underlying causative diseases. No patients underwent surgery to treat these diseases before the MR examinations. A total of 19 MR examinations were performed in the 14 patients 3–45 days after the ictus. One patient underwent three MR examinations; three patients underwent two MR examinations. Informed consent was obtained from either the patients directly or their relatives.

MR images were obtained by using a 0.5-T superconducting unit (SMT-50X; Shimadzu, Kyoto, Japan). The 19 FLAIR examinations were performed with 5,000/120/2,000 (repetition time [TR] msec/TE msec/inversion time [TI] msec) with a 180 × 256 matrix, one signal acquired, and an 8-mm section thickness. The overall imaging time with matrix reduction (60%) was about 9 minutes. No cardiac gating or flow compensation was used. We performed 19 spin-echo (SE) T1-weighted examinations (500/35) and nine SE T2-weighted examinations (3,000/90) with a 192 × 256 matrix and an 8-mm section thickness. Seventeen CT examinations in 13 patients were performed without contrast material enhancement and with a 5-mm or 10-mm section thickness by using a 9800 scanner (GE Medical Systems, Milwaukee, Wis). Four patients underwent two CT examinations each. The interval between the linked CT and MR examinations was less than 12 hours.

We evaluated the FLAIR findings in the patients with subarachnoid hemorrhage in comparison with the CT and conventional MR imaging findings. The presence of subarachnoid hemorrhage was evaluated visually: The features assessed were high-attenuation areas in the subarachnoid space on the CT scans, high-signal-intensity areas in the subarachnoid space on FLAIR and T1-weighted images, and high- or low-signal-intensity areas in the subarachnoid space on T2-weighted images.

Subarachnoid Hemorrhage	High-Signal-Intensity Areas Depicted			
	FLAIR MR Imaging	T1-weighted MR Imaging	T2-weighted MR Imaging	CT
Subacute (4–14 d)	11 of 11 (100)*	4 of 11 (36)	0 of 2 (0)	5 of 11 (45)
Chronic (> 14 d)	5 of 8 (62)	4 of 8 (50)	3 of 7 (43)	1 of 6 (17)
Total	16 of 19 (84)	8 of 19 (42)	3 of 9 (33)	6 of 17 (35)

Note.—Numbers in parentheses are percentages.  
\* In the detection of subacute subarachnoid hemorrhage, FLAIR imaging was significantly superior to T1-weighted imaging ( $P < .01$ ), T2-weighted imaging ( $P < .02$ ), and CT ( $P < .02$ ).

We performed a blind study to verify the accuracy of the comparative evaluation. Images from 15 FLAIR examinations performed in the patients with subarachnoid hemorrhage 3–17 days after the ictus and from 22 FLAIR examinations performed in a sex- and age-matched control group (eight men, 14 women; aged 19–72 years; mean age, 46.3 years) were evaluated in the blind study. The control subjects had undergone FLAIR imaging as part of evaluation for transient ischemic attack, vertigo, or fever; the findings were almost normal except for a few small infarctions. The two neuroradiologists (T. Ogawa, A.I.) were requested to independently determine, without access to any clinical information, whether each of the images from the 37 FLAIR examinations demonstrated subarachnoid hemorrhage.

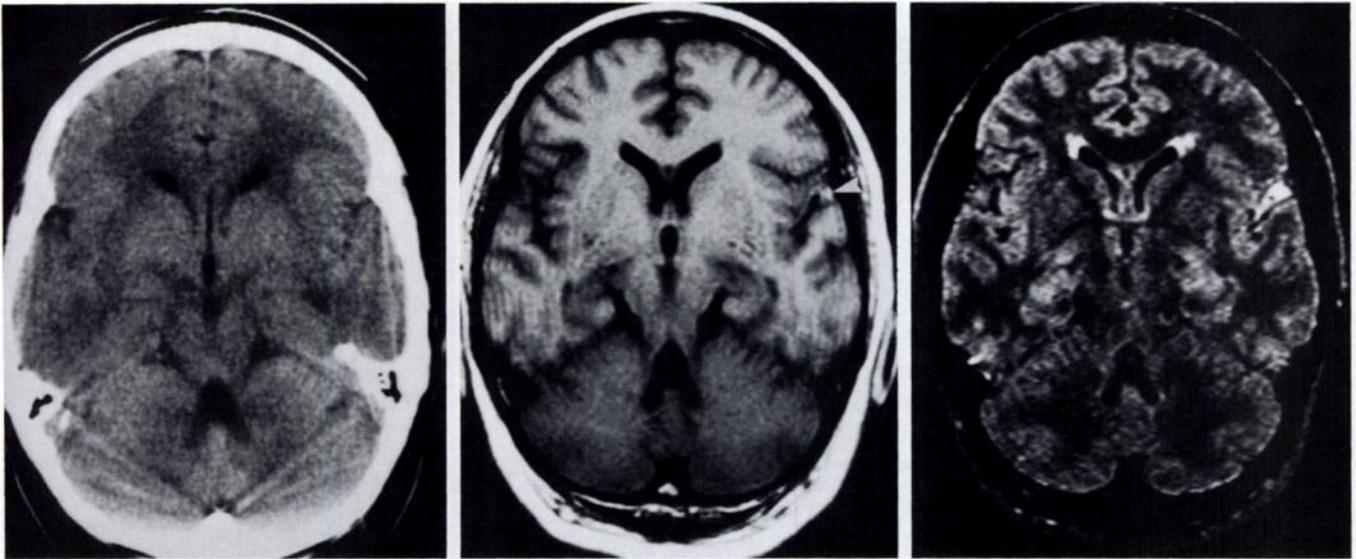
We remeasured, by using previously acquired images, the T1 and T2 relaxation times in the gray matter, the normal CSF, and the subacute subarachnoid hemorrhage 4 days after the ictus in one patient to calculate these times for the superconducting unit used in this study. The two regions measured in the subacute subarachnoid hemorrhage were the dense portion of subarachnoid hemorrhage

(dense subacute subarachnoid hemorrhage) and the portion diluted by CSF (diluted subacute subarachnoid hemorrhage). Those regions measured in the subacute subarachnoid hemorrhage were determined as follows: Dense subacute subarachnoid hemorrhage was determined to be areas of high signal intensity or high attenuation demonstrated on all T1-weighted images, proton-density-weighted (3,000/35) images, and CT scans; diluted subacute subarachnoid hemorrhage was determined to be areas of slightly increased signal intensity demonstrated on only proton-density-weighted images.

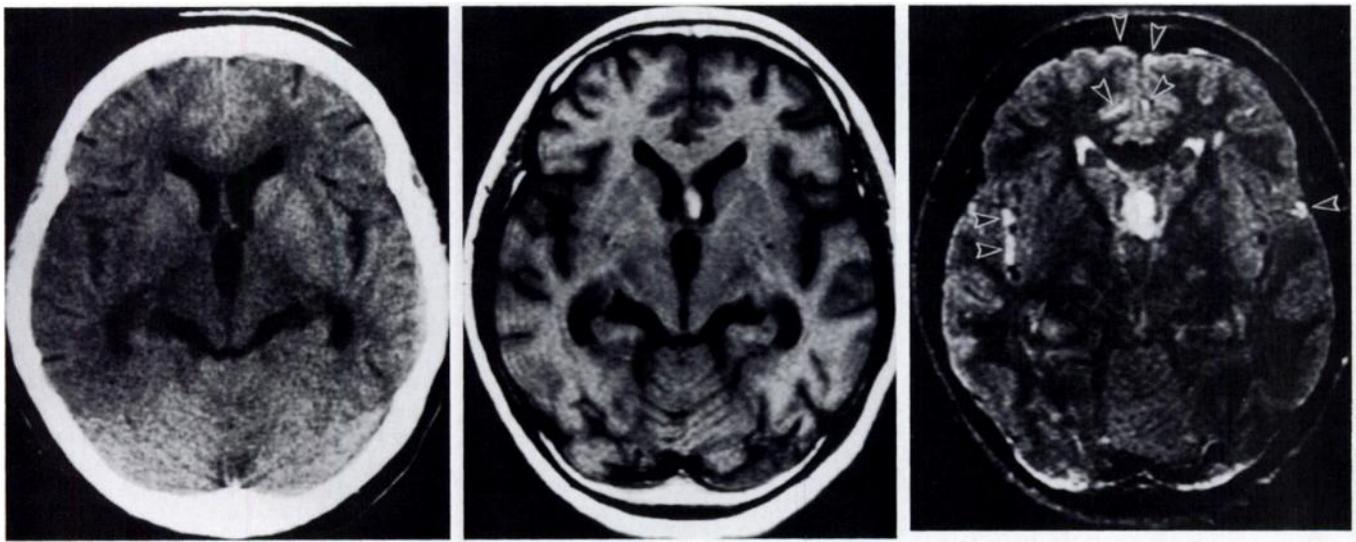
Statistical analysis of the detection of each phase of subarachnoid hemorrhage was conducted with the Fisher exact test. A  $P$  value of less than .05 was considered to indicate a statistically significant difference.

## RESULTS

The MR and CT findings of subacute and chronic subarachnoid hemorrhage are summarized in the Table. Subacute or chronic subarachnoid



**Figure 2.** Subarachnoid hemorrhage due to a ruptured aneurysm of the left middle cerebral artery in a 58-year-old woman. CT scan and MR images were obtained 7 days after the ictus. (a) CT scan shows no areas of high attenuation caused by subarachnoid hemorrhage but only obscuration of the left sylvian fissure. (b) T1-weighted MR image (500/35) shows subarachnoid hemorrhage as an area of slightly high signal intensity in the left sylvian fissure (arrowhead). (c) FLAIR MR image (5,000/120/2,000) clearly shows subarachnoid hemorrhage as an area of high signal intensity in the left sylvian fissure.



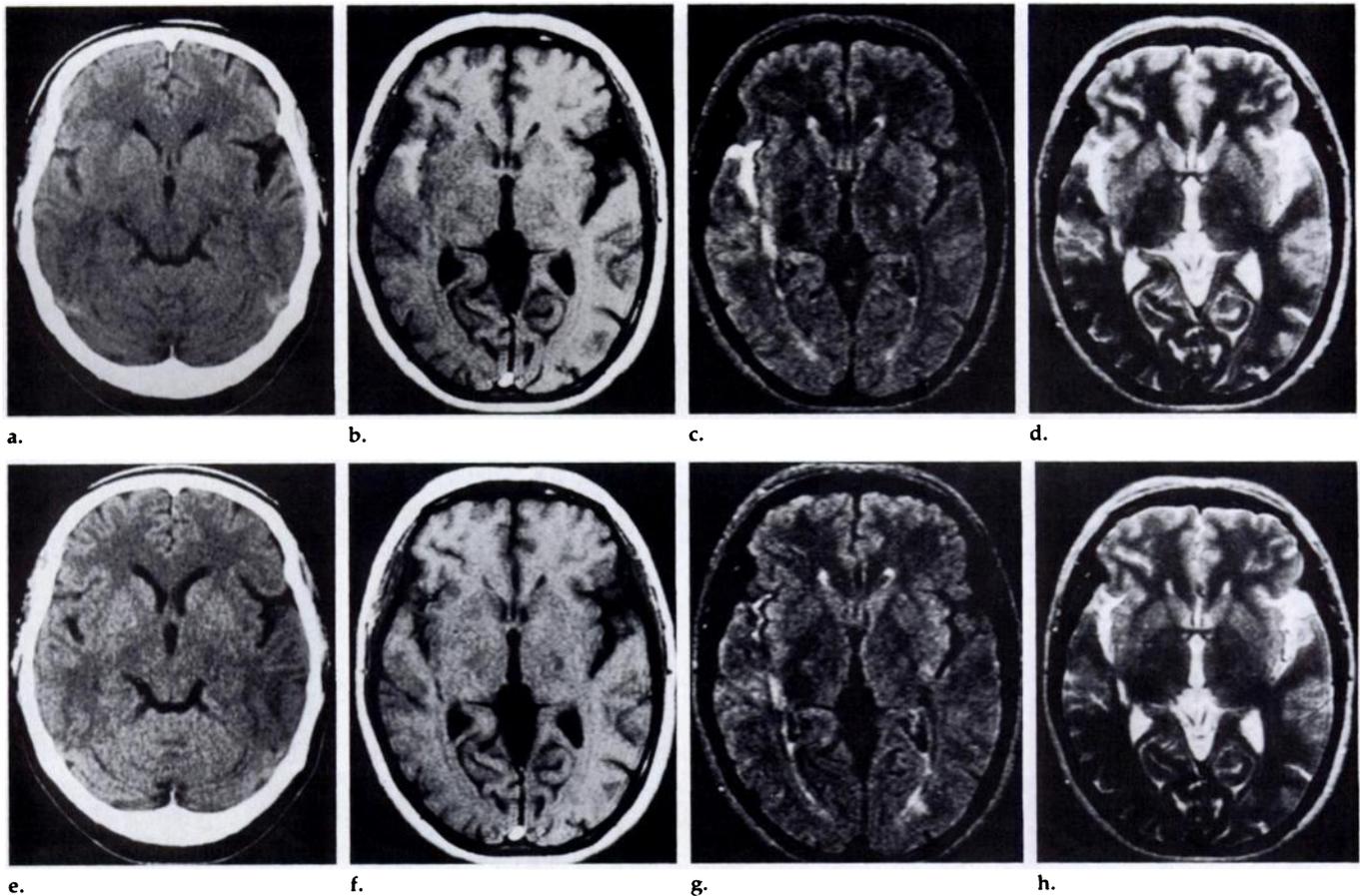
**Figure 3.** Subarachnoid hemorrhage due to a ruptured aneurysm of the anterior communicating artery in a 59-year-old woman. CT scan and MR images were obtained 15 days after the ictus. (a) CT scan shows dilatation of the third ventricle and both lateral ventricles and no areas of high attenuation caused by subarachnoid hemorrhage. (b) T1-weighted MR image (500/35) shows a hematoma as an area of high signal intensity in the septum pellucidum. (c) FLAIR MR image (5,000/120/2,000) clearly shows subarachnoid hemorrhage as areas of high signal intensity in the frontal subarachnoid space and bilateral sylvian fissures (arrowheads; especially in the right sylvian fissure) in addition to a hematoma in the septum pellucidum.

hemorrhage was demonstrated as an area of high signal intensity compared with the surrounding brain parenchyma and CSF on FLAIR, T1-weighted, and T2-weighted images. No subarachnoid hemorrhage had low signal intensity compared with CSF on T2-weighted images. FLAIR images demonstrated small areas of subarachnoid hemorrhage in the sylvian fissure and in the cerebral sulci that could not be detected on con-

ventional MR images and CT scans (Figs 1–4).

In the statistical analysis of the detection of subacute subarachnoid hemorrhage, FLAIR imaging (100% detection) was significantly superior to T1-weighted imaging (36% detection,  $P < .01$ ), T2-weighted imaging (0% detection,  $P < .02$ ), and CT (45% detection,  $P < .02$ ). Although FLAIR was superior in the detection of chronic subarachnoid hemorrhage, it

was not significantly superior. On FLAIR images, all areas of subarachnoid hemorrhage imaged within 18 days after the ictus were demonstrated as high-signal-intensity areas. On the other hand, at T1-weighted and T2-weighted MR imaging and CT within 18 days after the ictus, the subarachnoid hemorrhage was demonstrated as high-signal-intensity or high-attenuation areas at 53%, 40%, and 46% of the examinations, respec-



**Figure 4.** Subarachnoid hemorrhage due to moyamoya disease in a 46-year-old woman. CT scans and MR images were obtained (a–d) 18 days or (e–h) 45 days after the ictus. (a) CT scan shows obscuration of the right sylvian fissure. (b) T1-weighted (500/35) and (c) FLAIR (5,000/120/2,000) MR images clearly show subarachnoid hemorrhage as areas of high signal intensity in the right sylvian fissure. (d) T2-weighted MR image (3,000/90) shows a slightly high signal intensity change in the right sylvian fissure. However, it is difficult to observe the abnormal signal intensity change. No area of high attenuation or high signal intensity caused by subarachnoid hemorrhage is demonstrated on the (e) CT scan, (f) T1-weighted MR image (500/35), or (h) T2-weighted MR image (3,000/90). (g) Only the follow-up FLAIR MR image (5,000/120/2,000) clearly shows subarachnoid hemorrhage as an area of high signal intensity in the right sylvian fissure.

tively. On FLAIR images, high signal intensity of the subarachnoid hemorrhage was depicted up to a maximum of 45 days after the ictus (Fig 4).

In the blind comparison, whether subarachnoid hemorrhage was present was correctly identified on all the images by each of the two observers (T. Ogawa, A.I.). No FLAIR images acquired in control subjects were misidentified as those acquired in patients.

The T1 and T2 relaxation times were 937 msec and 91 msec, respectively, in the gray matter and were 3,522 msec and 241 msec, respectively, in the normal CSF. The T1 and T2 relaxation times in the dense subacute subarachnoid hemorrhage were 544 msec and 108 msec, respectively. The T1 relaxation time in the dense subacute subarachnoid hemorrhage was markedly shorter than that in the gray matter, and the T2 relaxation time was nearly equal to that in the gray matter. The T1 and T2 relaxation times in the diluted subacute sub-

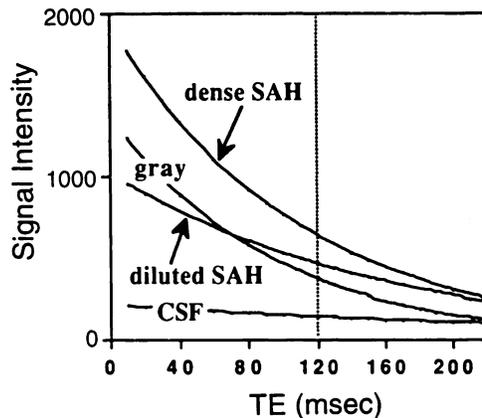
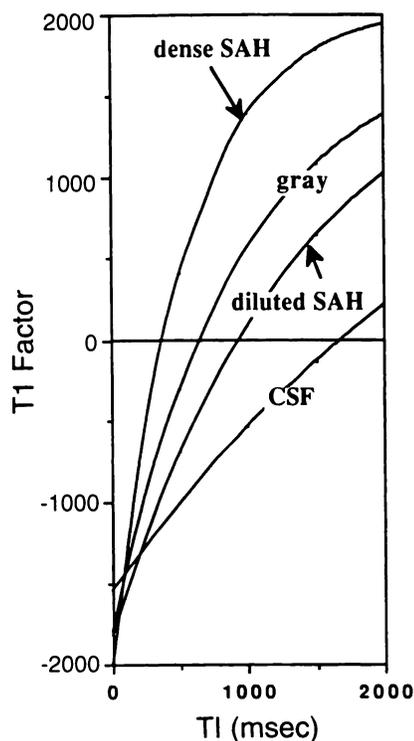
arachnoid hemorrhage were 1,404 msec and 149 msec, respectively. The T1 and T2 relaxation times were similar to those previously reported for acute subarachnoid hemorrhage (6).

## DISCUSSION

The depiction of subarachnoid hemorrhage at CT depends on the attenuation values of the blood in the CSF spaces, whereas that at MR imaging mainly depends on the difference in the T1 and T2 relaxation times between the subarachnoid hemorrhage and the CSF and brain parenchyma. Chakeres and Bryan (21) postulated that MR imaging may be superior to CT in the detection of subarachnoid hemorrhage on the basis of their *in vitro* data for CSF-blood mixtures, especially at lower CSF-to-blood mixture ratios. It was demonstrated in one previous study (20) that an area of acute subarachnoid hemorrhage diluted by CSF that is not well demonstrated as a high-attenuation area

at CT sometimes is shown as a high-signal-intensity area at FLAIR MR imaging and that such an area of acute subarachnoid hemorrhage is more readily identified with FLAIR imaging than with CT.

Figure 5 illustrates the T1 relaxation curves as a function of TI for a specific TR and the T2 relaxation curves as a function of TE for a specific TI and TR as obtained in a FLAIR sequence by using a theoretical equation of inversion-recovery signal intensity (22). The signal of normal CSF is reduced in this FLAIR sequence, whereas that of either the dense or diluted subacute subarachnoid hemorrhage is not reduced because of the shortening of the T1 relaxation time in these areas. FLAIR imaging more readily distinguishes subtle differences in the T1 relaxation times between bloody CSF and normal CSF than does conventional MR imaging. The dense subacute subarachnoid hemorrhage is hyperintense relative to the gray matter, because the T1 relaxation time is



**Figure 5.** T1 relaxation curves (left) as a function of TI for a specific TR (5,000 msec) and T2 relaxation curves (above) as a function of TE for a specific TI (2,000 msec) and TR (5,000 msec) in a FLAIR sequence; *gray* = gray matter, SAH = subarachnoid hemorrhage.

shorter than that in the gray matter while the T2 relaxation time is nearly equal to that in the gray matter. On the other hand, the T1 and T2 relaxation times in the diluted subacute subarachnoid hemorrhage were similar to those previously reported for acute subarachnoid hemorrhage (6). Because the T2 relaxation time is longer than that in the gray matter, diluted subarachnoid hemorrhage is hyperintense relative to the gray matter if a longer TE is selected in the FLAIR sequence.

The variable MR appearance of intracranial hemorrhage depends on the structure of hemoglobin and its various oxidation products. The characteristic marked T2 shortening due to deoxyhemoglobin is observed in acute intraparenchymal hemorrhage (23). However, subarachnoid hemorrhage differs from intraparenchymal hemorrhage in that it is mixed with CSF at high oxygen tension. Grossman and colleagues (24,25) presented in vitro data that suggested that the high oxygen tension of CSF imposed restrictions on the generation of paramagnetic deoxyhemoglobin in CSF blood; they proposed that CSF blood was not seen as an area of marked hypointensity because of the low hematocrit and the lack of formation of deoxyhemoglobin from diamagnetic oxyhemoglobin.

On the other hand, Hayman and colleagues (26) reported that the signal intensity on T2-weighted images

is dependent primarily on the state of hydration of the red blood cells (RBCs) and that RBC dehydration causes the T2 relaxation time to markedly decrease relative to that of the brain parenchyma, whereas RBC overhydration or lysis of RBCs causes a marked increase. Therefore, it is possible that the lack of marked hypointensity in the subarachnoid hemorrhage on T2-weighted images is caused by the low hematocrit and the overhydration or lysis of the RBCs, as opposed to their dehydration, owing to the bleeding into the CSF space and the restriction of the generation of paramagnetic deoxyhemoglobin that arises from the high oxygen tension of CSF.

A stronger T2 shortening is observed in acute subarachnoid hemorrhage when massive bleeding has occurred (27–29), and the areas of marked T2 shortening due to heavily packed RBCs, which may be shielded from CSF and result in high hematocrit, increased deoxyhemoglobin formation, and RBC dehydration, may not be demonstrated as high-signal-intensity areas on the FLAIR images. However, the signal intensity in the subarachnoid hemorrhage may vary heterogeneously, depending on the mixture ratio of RBCs and CSF at each location. Although the areas of marked T2 shortening due to heavily packed RBCs may not be demonstrated as high-signal-intensity areas on the FLAIR images, the areas without marked T2 shortening due to the mixture of CSF that surrounds the area with heavily packed RBCs may be demonstrated as high-signal-intensity areas on the FLAIR images. In chronic subarachnoid hemorrhage,

however, the areas of marked T2 shortening due to the deposition of hemosiderin may not be demonstrated as high-signal-intensity areas on the FLAIR images.

The two primary mechanisms for T1 shortening in hemorrhage are bound-water effects and paramagnetic effects (28–30). Bradley and Schmidt (1), in an in vitro study, did not observe the formation of much methemoglobin until several days after the subarachnoid hemorrhage. Therefore, the T1 shortening of the acute subarachnoid hemorrhage relative to the CSF may reflect the increase in hydration-layer water due to the higher protein content of the bloody CSF (29). On the other hand, the T1 shortening of the dense subacute subarachnoid hemorrhage relative to the brain parenchyma may reflect the formation of much methemoglobin. The T1 shortening of the diluted subacute subarachnoid hemorrhage relative to the normal CSF may reflect the formation of a small quantity of methemoglobin or an increase in hydration-layer water due to the higher protein content of the bloody CSF.

In the results of our blind study, neither reviewer gave any false-positive or false-negative assessment. Artifacts due to CSF pulsation with fast FLAIR imaging have been described previously and are due to inflow and misregistration effects (16–18). In this study, however, a fast FLAIR sequence could not be performed because of the limitation of the MR imaging system, and the original FLAIR sequence with inversion recovery was performed. In this study, the CSF inflow artifacts in the basal cistern, such as those previously reported with the fast FLAIR sequences, were not seen on these original FLAIR images. Although artifacts from the vessels or the dural sinus sometimes were seen in the cerebropontine angle and medullary cistern on these original FLAIR images, these artifacts could be distinguished from bloody CSF by visually inspecting the image, by changing the phase encoding, or both.

In routine study, however, the fast FLAIR sequence is generally performed while the original FLAIR sequence is not. Therefore, it is important to describe the potential pitfalls in the diagnosis of subarachnoid hemorrhage with the fast FLAIR sequence. It has been reported (17,18) that a solution to this problem is incorporated into the fast FLAIR sequence: The width of the inverting section is increased (to use a nonselec-

tive inversion pulse). This approach helps minimize artifacts due to CSF pulsation but is not 100% effective (especially in the suprasellar cistern). A more effective device incorporated into the fast FLAIR sequence protocol must be developed to completely diminish artifacts due to CSF pulsation, which is a potential cause of false-positive diagnosis of subarachnoid hemorrhage.

In this study, FLAIR imaging was especially useful in demonstrating small areas of subarachnoid hemorrhage in the sylvian fissure and cerebral sulci that could not be detected at conventional MR imaging and CT. CSF inflow artifacts may not be seen in these regions. In the detection of small subarachnoid hemorrhage with FLAIR imaging, it is very useful to evaluate the signal intensity at the sylvian fissure and cerebral sulci.

The hyperintense CSF depicted on FLAIR images may also be seen in conditions other than subarachnoid hemorrhage (20). For example, severe purulent meningitis, granulomatous meningitis, arachnoiditis, meningeal metastasis or CSF disseminations caused by primary brain tumors, such as germinoma, ependymoma, medulloblastoma, or glioblastoma, may result in increased CSF signal intensity on FLAIR images. A ruptured dermoid or a deposition of oily contrast medium is also a potential simulator of subarachnoid hemorrhage on FLAIR images because of the short T1 of fat. These entities, however, should be distinguishable from subarachnoid hemorrhage on the basis of clinical information or with contrast material-enhanced MR imaging, together with the chemical shift artifact of fat in the case of a ruptured dermoid or the deposition of oily contrast medium.

In conclusion, all areas of subarachnoid hemorrhage could be demonstrated as high-signal-intensity areas on FLAIR images within 18 days and up to a maximum of 45 days after the ictus. The FLAIR sequence is useful in the evaluation of the signal intensity at the sylvian fissures and the cerebral sulci in the detection of small areas of subarachnoid hemorrhage diluted by CSF that are not demonstrated at con-

ventional MR imaging or CT. These results suggest that the FLAIR sequence provides superior diagnostic images in patients with subacute or acute subarachnoid hemorrhage. ■

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