# MR Imaging of Intracerebral Blood: Diversity in the Temporal Pattern at 0.5 and 1.0 T 

Amer Zyed ${ }^{1}$
L. Anne Hayman ${ }^{2}$
R. Nick Bryan ${ }^{3}$

Received March 7, 1989; revision requested May 1, 1989; final revision received November 26, 1990; accepted November 26, 1990.

This work was supported in part by grant NS 19056-06.
${ }^{1}$ Department of Radiology, Al-Azhar University, Cairo, Egypt.
${ }^{2}$ Department of Radiology, Baylor College of Medicine, 1 Baylor Plaza, Houston, TX 77030.
${ }^{3}$ Department of Radiology and Radiological Science, The Johns Hopkins University School of Medicine, The Johns Hopkins Hospital, 600 N. Wolfe St., Baltimore, MD 21205. Address reprint requests to R. N. Bryan.
0195-6108/91/1203-0469
© American Society of Neuroradiology


#### Abstract

MR scans were obtained at 0.5 and 1.0 T in 40 patients with 46 intracerebral hematomas categorized as hyperacute ( $0-2$ days), acute (3-7 days), subacute ( $8-14$ days), and chronic ( 15 days to 6 years). In a retrospective review, the signal intensity of the lesions was compared with that of normal white matter of the brain on spin-density, T1-, and T2-weighted spin-echo and T1-weighted gradient-echo sequences. The classic appearance and evolution of hematomas described in the literature at 1.5 T were not found in a significant number of the cases reviewed. In the hyperacute group, only five of eight hematomas had signal intensities that were hypointense relative to brain on T2-weighted images. Two of eight hyperacute hematomas were hyperintense relative to brain on the T1-weighted spin-echo images. However, T1-weighted gradient-echo images reliably demonstrated a hypointense signal in some portion of the hematoma in 45 of 46 cases.

We conclude that while there is no constant temporal pattern on spin-echo or gradientecho sequences, there are signal-intensity changes suggestive of hemorrhage in nearly all hematomas imaged at 0.5 and 1.0 T . Although the inconsistency may be frustrating from a diagnostic standpoint, this variability may reveal important individual differences in hematomas and the brain that surrounds them, and thus be clinically significant. Before these data can be mechanistically analyzed, the reason for contrast on MR scans of hematoma must be better understood.


AJNR 12:469-474, May/June 1991; AJR 157: July 1991

MR of intracranial hematomas should be clinically useful because of the sensitivity of MR to many physical and chemical changes that accompany hemorrhage. Current concepts of the appearance of intracerebral hemorrhage are based on empirical clinical observations, theoretical explanations predicated on prior MR data, and very limited in vitro analysis of human blood clot and components. Currently, the most popular mechanistic explanation for the appearance of intracerebral hemorrhage is that of Gomori et al. [1], which emphasizes the importance of the paramagnetic hemoglobin phenomenon at 1.5 T . However, this hypothesis, which depends on magnetic field strength, does not explain certain obvious clinical observations, such as low-signal lesions on T2-weighted spin-echo (SE) images obtained with lower field strengths [2, 3], nonspecific bright T2-weighted SE images of very early ( $<48 \mathrm{hr}$ ) hematomas at any field strength, and the occasional high signal intensity on T1-weighted SE images of very early hemorrhage [3]. In this report, we review the clinical appearance of intracerebral hemorrhage at field strengths of 0.5 and 1.0 T . In a companion article [4], we propose a theory to explain the MR patterns of cerebral hemorrhage at these field strengths by focusing on factors that influence T1 and T2 relaxation and thereby determine MR image contrast in clinical hematomas examined by SE and gradient-echo (GE) images at field strengths below 1.5 T .


Fig. 1.- $A$ and $B$, Signal intensities of the center $(A)$ and $\operatorname{rim}(B)$ of hematomas vs age of hemorrhage on spin-density-weighted SE images obtained at 0.5 and 1 T .


Fig. 2.- $A$ and $B$, Signal intensities of the center $(A)$ and rim $(B)$ of hematomas vs age of hemorrhage on T2-weighted SE images obtained at 0.5 and 1 T .

## Materials and Methods

All MR examinations performed from May 1986 to April 1988 were retrospectively reviewed for evidence of cerebral hemorrhage. This included MR studies in patients referred for known or suspected intracranial hemorrhage, as well as MR studies suggesting the presence of a hematoma in patients referred for other indications. From the initial 107 patients reviewed, 40 patients were chosen for inclusion because there was convincing evidence of cerebral hemorrhage and the time of hemorrhage was well documented. When hematomas were multiple (two cases), they were evaluated separately because they often had different MR appearances. Serial studies (four cases) were also evaluated individually. Thus, the total number of hematomas (46) exceeded the number of patients (40). Twelve hematomas were confirmed surgically and pathologically, 20 hematomas were diagnosed and staged by clinical findings and CT examinations, while eight hematomas were documented by clinical findings and MR characteristics only.

Thirty-six hematomas were scanned on a 1.0-T superconducting unit (Siemens, Iselin, NJ ) and 10 hematomas on a 0.5 -T superconducting unit (Siemens). The following pulse sequences were used in all patients: spin-density-weighted $\mathrm{SE},>3000 /<35$ (TR/TE); T2weighted $\mathrm{SE},>3000 />88$; T1-weighted $\mathrm{SE},<500 /<35$; and T1weighted GE, $<500 / 17 / 90^{\circ}$ (TR/TE/flip angle). Images were obtained
with $1-4$ excitations and a $256 \times 256$ acquisition matrix. Images were $5-10 \mathrm{~mm}$ thick.

Each image type was independently evaluated by two neuroradiologists as to the signal intensity (SI) relative to the white matter of the brain. Since the SI pattern of a hematoma was not uniform, the hematoma was roughly separated into two compartments: center and rim (which presumably represents the outer part of the hematoma). The hematomas were arbitrarily divided by age into four stages. Eight hematomas were hyperacute ( $0-2$ days old), 19 were acute ( $3-7$ days old), 10 were subacute ( $8-14$ days old), and nine were chronic (more than 14 days old).

## Results

Rapid, dynamic, and even reversible changes in intensity patterns were observed that varied greatly with age of hemorrhage and pulse sequence. The Sls of hematoma regions are graphed as a function of the age of hemorrhage in Figures 1-4.

The hyperacute stage (0-2 days) was characterized by variable appearances rather than a consistent pattern. Hematomas examined with double-echo scans (spin-densityand T2-weighted SE) were noted to be predominantly hyper-


Fig. 3.- $A$ and $B$, Signal intensities of the center $(A)$ and $\operatorname{rim}(B)$ of hematomas vs age of hemorrhage on T1-weighted SE images obtained at 0.5 and 1 T .


Fig. 4.- $A$ and $B$, Signal intensities of the center $(A)$ and $\operatorname{rim}(B)$ of hematomas vs age of hemorrhage on T1-weighted GE images obtained at 0.5 and 1 T .
intense peripherally with foci of varying degrees of hypointensity scattered within the hematoma, particularly on the second-echo image. In three of eight cases, hypointensity on T2-weighted SE images was not sufficient to allow a definite diagnosis of hemorrhage. This nondiagnostic appearance occurred at both 1.0 T (two cases) and 0.5 T (one case). On T1-weighted SE images, most lesions were slightly hypointense, but two had hyperintense regions. On T1-weighted GE images, hematomas typically had a central hypointensity with variable hyperintensity about the rim. However, only minimal evidence of hypointensity was seen on T1-weighted GE images in one case (Fig. 5).

The acute (3-7 days) hematomas were typified by low SI centrally on spin-density- and T2-weighted SE and T1weighted GE pulse sequences. The rim most frequently had increased SI on spin-density- and T2-weighted SE images but low SI on T1-weighted GE images.

The central portion of subacute lesions (8-14 days) most often had decreased SI on spin-density- and T2-weighted SE and T1-weighted GE scans. In all cases (10 of 10), there was increased SI on T1-weighted SE sequences, either centrally or at the rim.

Chronic lesions ( $>14$ days) also varied in appearance, but the most common pattern was of central increased SI on spin-density-, T2-, and T1-weighted SE and T1-weighted GE images with decreased signal in the rim on spin-density- and T2-weighted SE and T1-weighted GE scans. Smaller or older hematomas had smaller or no central regions of bright signal. Such lesions were seen only as areas of decreased SI on T2weighted SE, and particularly on T1-weighted GE images (Fig. 6).

In addition to analyzing hematoma SI by arbitrary location in the hemorrhage (i.e., center and rim), we also evaluated the SI of hemorrhage independent of location in the hematoma. Table 1 summarizes SI patterns as a function of age of hemorrhage, independent of lesion region. Thirty-seven hematomas ( $80 \%$ ) had low SI somewhere within the lesion on T2-weighted SE images. This was seen with comparable frequency in hemorrhages of all ages. Twenty-eight hematomas ( $61 \%$ ) had increased SI on T1-weighted SE images; this was seen most frequently in subacute hemorrhages. Fortyfive hematomas had low SI on T1-weighted GE images. Only one hematoma, a 22 -hr-old hemorrhage imaged at 1.0 T , lacked this relatively distinctive SI. There were no known

blood or clotting abnormalities to account for this anomalous appearance.

## Discussion

The empirical observations of this study document the variable MR appearance of intracerebral hemorrhage on SE and GE images at 0.5 and 1.0 T . This finding is consistent with the more current literature. For instance, Gomori et al. [1] reported decreased central SI on T2-weighted SE images of acute hemorrhage, while our results and those of Zimmerman et al. [3] show some acute hematomas to have hyperintense central SI. Likewise, Gomori et al. [1] indicated that acute hematomas have isointense to low SI centrally on T1-
weighted images, while our results and those of Zimmerman et al. [3] show some hematomas with hyperintense SI.

Some authors have suggested that variations in the MR appearance of hematomas can be attributed to differences in field strength, pulse sequences, or other technical factors. For instance, Gomori et al. [5] suggested that acute hematomas have central low SI on T2-weighted SE images at "high" field strengths ( 1.5 T ), but not necessarily at lower field strengths. Zimmerman et al. [3] have reported such appearances at 0.5 T, and Sapponen et al. [2] and Bydder et al. [6] have seen similar low SI at 0.17 and 0.15 T , respectively. The variable pattern of hematomas on SE scans was recently confirmed at 0.5 and even 1.5 T by Seidenwurm et al. [7].

Our study also documents the variable, but sensitive, MR appearance of hemorrhage on GE images. Gradient echoes

Fig. 6.-6-year-old right thalamic hematoma.
$A-D$, Spin-density-weighted SE ( $A$ ), T2-weighted SE (B), T1-weighted SE (C), and T1-weighted GE (D) MR images at 1.0 T . T1-weighted GE image is most sensitive in detecting old hemorrhage.


TABLE 1: Signal-Intensity Changes Suggestive of Hemorrhage

| Age of <br> Hematoma | Total No. of <br> Hematomas | No. That <br> Decreased on <br> T2-Weighted <br> Spin Echo | No. That <br> Increased on <br> T1-Weighted <br> Spin Echo | No. That <br> Decreased on <br> T1-Weighted <br> Gradient Echo |
| :--- | :---: | :---: | :---: | :---: |
| Hyperacute | 8 | 5 | 2 | 7 |
| Acute | 19 | 16 | 10 | 19 |
| Subacute | 10 | 9 | 10 | 10 |
| Chronic | 9 | 7 | 6 | 9 |
| Total | 46 | 37 | 28 | 45 |

should be intrinsically more sensitive than spin echoes to magnetic susceptibility effects [7, 8]. However, there are many possible GE pulse sequences with an infinite variety of TRs, TEs, and flip angles. This study used a T1-weighted
sequence with a $90^{\circ}$ flip angle and TR and TE approximating those of the T1-weighted SE sequence. Although this GE sequence is not clinically popular, it was found to be very sensitive to magnetic susceptibility effects, and, importantly, it differs by a single variable from the T1-weighted SE image. Thus, the SI difference between tissues with stationary spins is due primarily to the magnetic susceptibility hetereogeneity of local tissue.

The more popular GE gradient-recalled acquisition in the steady state (GRASS) sequences, which use a shorter TR and lower flip angle, are difficult to evaluate in terms of magnetic susceptibility influence because the relative spindensity, T1, and T2 contrast properties differ from those of the SE sequences. The GRASS type of GE sequence is not more sensitive in the detection of the magnetic susceptibility effects of hemorrhage than the T1-weighted GE sequence
used in this study. This opinion is based on our experience with in vitro blood clots and a limited number of clinical hematomas imaged with both types of GE sequences and on the comparability of these clinical results (decreased SI in 45 of 46 hematomas) with those of others using GRASS sequences $[1,3,7,8]$. However, both types of GE sequences are very reliable in detecting hematomas of any age.

T1-weighted GE scans showed mixed SI patterns with regions of hyperintensity simulating those noted on T1weighted SE and/or regions of hypointensity simulating those noted on T2-weighted SE. This reflects the sensitivity of T1weighted GE to both T1 and T2* effects. Hypointensity with this pulse sequence was also noted at the periphery of acute and subacute hematomas, immediately around the hematoma. This may represent a boundary zone between two areas of differing magnetic susceptibility (hemorrhage vs normal brain). This appearance may mimic the appearance of hemosiderin in chronic hemorrhage [8].

While our results and the literature show a variable appearance of intracerebral hemorrhage, a general pattern is found in over two thirds of cases. Initially, there is low SI on T1weighted GE scans, and shortly thereafter on T2-weighted SE. This appearance is followed by and mixed with areas of increased SI on T1-weighted SE. While this pattern is vague as to region and age of a hematoma, it is relatively specific in terms of determining evidence of hemorrhage. Thus, this general pattern is very sensitive for the detection of hemorrhage, which is usually the critical clinical issue.

In summary, there was no consistent temporal or regional SI pattern for intracerebral hematomas examined by a set of MR sequences (spin-density-, T1-, and T2-weighted SE and T1-weighted GE). However, there was a general pattern that effectively defined the presence of hemorrhage. We hypothesize that the appearance of a hematoma on MR is an indirect
measure of individual factors present in the hematoma and the surrounding brain. Thus, the diverse appearances of hematomas on MR may hold important diagnostic information about the surrounding brain. For this information to be more meaningful, the biological basis of the individually variable MR signals must be understood.

## ACKNOWLEDGMENTS

We thank Michael Mawad, Barry Horowitz, Randy Blackwell, and Marlin Sandlin at The Methodist Hospital for clinical help and Marge Eddy for help in manuscript preparation.

## REFERENCES

1. Gomori JM, Grossman RI, Goldberg HI, Zimmerman RA, Bilaniuk LT. Intracranial hematomas: imaging by high-field MR. Radiology 1985; 157:87-93
2. Sipponen JT, Sepponen RE, Tanttu JK, Sivula A. Intracranial hematomas studied by MRI at 0.17 and 0.02 T. J Comput Assist Tomogr 1985;9: 698-704
3. Zimmerman RD, Heier LA, Snow RB, Liu DPC, Kelly AB, Deck MDF. Acute intracranial hemorrhage: intensity changes on sequential MR scans at 0.5 T. AJNR 1988;9:47-57
4. Hayman LA, Taber KH, Ford JJ, Bryan RN. Mechanisms of MR signal alteration by acute intracerebral blood: old concepts and new theories. AJNR (in press)
5. Gomori JM, Grossman RI, Yu-lp C, Asakura T. NMR relaxation times of blood: dependence on field strength, oxidation state, and cell integrity. $J$ Comput Assist Tomogr 1987;11:684-690
6. Bydder GM, Pennock JM, Porteous R, Dubowitz LMS, Gadian DG, Young IR. MRI of intracerebral hematoma at low field ( 0.15 T ) using T2 dependent partial saturation sequences. Neuroradiology 1988;30:367-371
7. Seidenwurm D, Meng T-K, Kowalski H, Weinreb JC, Kricheff II. Intracranial hemorrhagic lesions: evaluation with spin-echo and gradient-refocused MR imaging at 0.5 and 1.5T. Radiology 1989;172:189-194
8. Edelman RR, Johnson K, Buxton R, et al. MR of hemorrhage: a new approach. AJNR 1986;7:751-756
