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Variable Appearances of Subacute Intracranial Hematomas on High-Field Spin-Echo MR

John M. Gomori[1,2]
Robert I. Grossman[1]
David B. Hackney[1]
Herbert I. Goldberg[1]
Robert A. Zimmerman[1]
Larissa T. Bilaniuk[1]

Subacute intracranial hematomas have variable appearances on high-field MR images. They are hyperintense on T1-weighted images owing to methemoglobin, but have variable intensities on T2-weighted images. Observation of the different high-field spin-echo MR intensity patterns of five subacute hematomas suggests that further subcategorization into different methemoglobin states may be possible. In particular, undiluted intracellular methemoglobin is hyperintense on T1-weighted images and markedly hypointense on T2-weighted images, undiluted free methemoglobin should be hyperintense on T1-weighted images and isointense or slightly hypointense on T2-weighted images, and dilute free methemoglobin is hyperintense on both T1- and T2-weighted images. However, it appears that certain regions of subacute hematomas may be difficult to differentiate, by intensity patterns alone, from melanotic melanomas or fat.

We believe that, despite some limitations, MR is useful in dividing subacute intracranial hematomas into their respective methemoglobin states, and also that further subcategorization is possible.

Early experience with MR imaging of intracranial hematomas at 1.5 T has revealed three characteristic signal-intensity patterns [1]:

1. Acute hematomas (<1 week old) are isointense to gray matter on T1-weighted images and markedly hypointense on T2-weighted images. This is ascribed to a selective T2 proton relaxation enhancement (relaxation time shortening) by intracellular deoxyhemoglobin.

2. Subacute hematomas (about 1 week to <1 month old) are initially hyperintense at the hematoma periphery on T1-weighted images. The hyperintensity on T1-weighted images proceeds to fill the whole hematoma and eventually (by 2 weeks) becomes observable on T2-weighted images as well. This is attributed to the formation of intracellular methemoglobin, which is paramagnetic, and to subsequent cell lysis and watery dilution by resorption of the resultant free methemoglobin.

3. In subacute and chronic hematomas, the adjacent brain parenchyma is slightly hypointense on T1-weighted images and markedly hypointense on T2-weighted images. This is because hemosiderin deposits produce a selective T2 relaxation enhancement.

We report our observations on the previously unappreciated variability of the high-field MR intensities of subacute hematomas and discuss the underlying relaxation mechanisms and relevant differential diagnoses.

Subjects and Methods

Over a period of 1 year, we observed five patients with subacute intracranial hematomas from various causes. All hematomas were diagnosed and staged by clinical and CT findings. Two were confirmed surgically and pathologically. MR imaging was performed on a GE 1.5-T superconducting unit. Spin-echo pulse sequences were obtained with 5-mm-thick slices, two excitations, and 256 × 128 acquisition matrixes. T1-weighted images were obtained with

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[1] Department of Radiology, Hospital of the University of Pennsylvania, 3400 Spruce St., Philadelphia, PA 19104. Address reprint requests to R. I. Grossman.

[2] Permanent address: Department of Radiology, Hadassah Hospital, P.O. Box 12000, Jerusalem, Israel 91120.

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TABLE 1: MR Intensities of Subacute Hematomas

Case No.	Type of Subacute Hematoma	Age of Hematoma	CT Density	Image Intensity Relative to Gray Matter		
				T1-Weighted	Proton-Density-Weighted	T2-Weighted
1	Spontaneous (normal angiography)	5 days	Increased	Increased	Unchanged	Decreased
2	Small AVM (surgically confirmed)	12 days	Unchanged Unchanged	Increased Increased	Increased Slightly increased	Increased Unchanged
3	Traumatic	6 days	Increased Decreased	Increased Slightly increased	Unchanged Increased	Decreased Increased
4	Hypertensive	9 days	Increased	Increased	Unchanged	Decreased
5	Traumatic, subdural (surgically confirmed)	7 days	Decreased Unchanged	Increased Increased	Increased Unchanged	Increased Decreased

Note.—AVM = arteriovenous malformation.

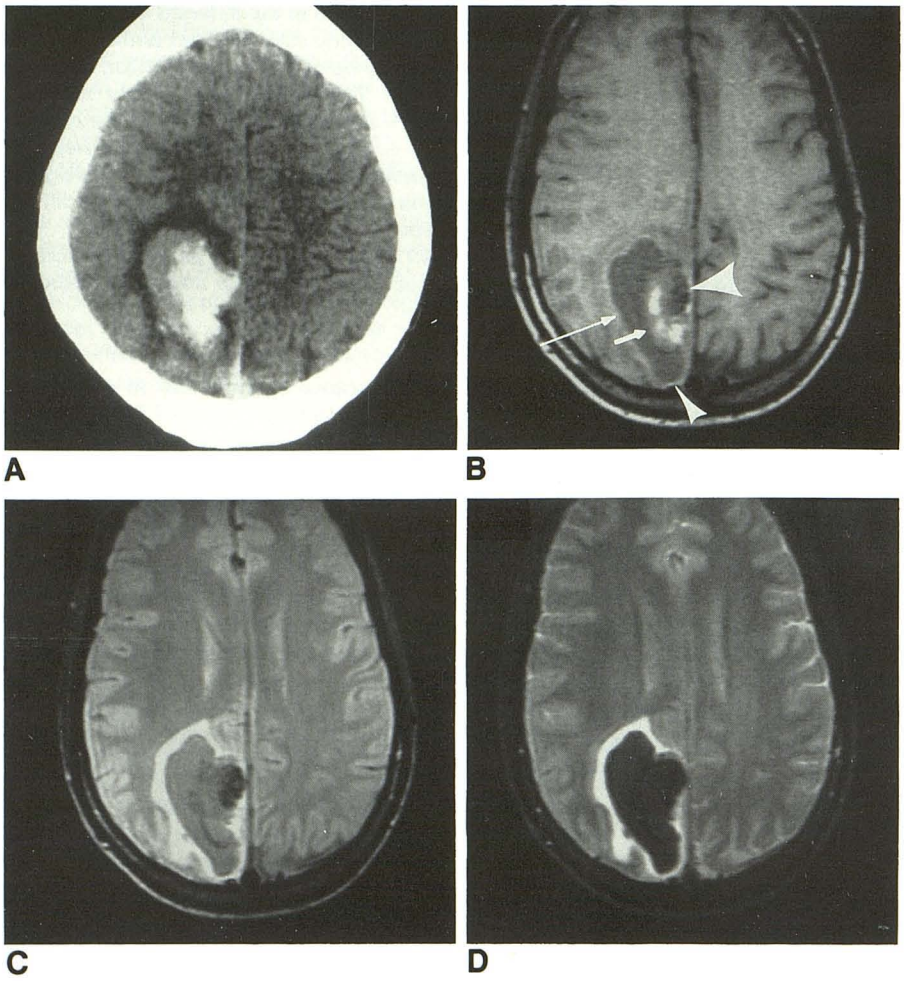

Fig. 1.—Case 1: 5-day-old multicompartmented spontaneous hematoma (normal angiography).

***A*, Unenhanced CT scan (4 days after hemorrhage) shows hyperdense hematoma medially with isodense portion laterally.**

***B*, T1-weighted image, TR = 600 msec, TE = 25 msec. Large lateral segment of hematoma (*long arrow*), slightly hypointense to gray matter, represents intracellular deoxyhemoglobin. Medial to it (*short arrow*) is hyperintense region representing intracellular methemoglobin; medial to this (*large arrowhead*) is small collection of intracellular deoxyhemoglobin more hypointense than lateral segment. Note thin rim of hyperintensity (*small arrowhead*) surrounding whole hematoma representing intracellular methemoglobin.**

***C*, Proton-density-weighted image, TR = 2500 msec, TE = 30 msec. Regions of hyperintensity have become slightly hypointense.**

***D*, T2-weighted image, TR = 2500 msec, TE = 80 msec. All regions have become markedly hypointense. Lateral and medial segments correspond to intracellular deoxyhemoglobin. Medial segment has higher deoxyhemoglobin concentration and higher hematocrit. Hyperintense regions on T1-weighted image are isointense on proton-density-weighted image and markedly hypointense on T2-weighted image because of undiluted intracellular methemoglobin. Note that subcutaneous and diploic fat is hyperintense on T1-weighted image, isointense on proton-density-weighted image, and hypointense on T2-weighted image.**

a repetition time (TR) of 400–800 msec and echo time (TE) of 20–25 msec. Proton-density-weighted images were obtained with a TR of 1500–2500 msec and TE of 20–35 msec. T2-weighted images were obtained with a TR of 1500–2500 msec and TE as a second echo of 40–120 msec.

Results

All subacute hematomas had regions of hyperintensity on T1-weighted images (Table 1 and Figs. 1–5). On proton-density-weighted images these regions were either isointense

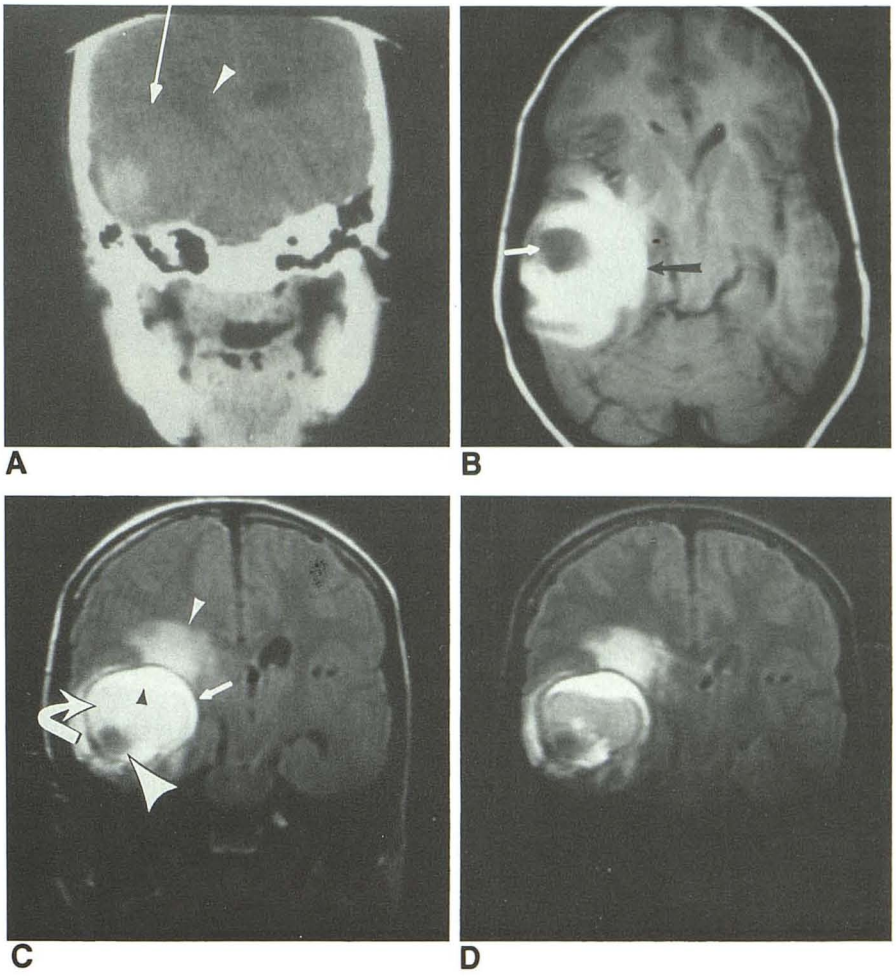

Fig. 2.—Case 2: concentric 12-day-old and 2-day-old hematomas caused by small vascular malformation (surgically confirmed).

***A*, Enhanced coronal CT scan shows acute hyperdense right temporal hematoma surrounded by isodense subacute hematoma. Subtle thin ring of enhancement surrounds hematoma (*arrow*) and hypodensity of edema is seen superomedially (*arrowhead*).**

***B*, T1-weighted image, TR = 800 msec, TE = 20 msec. Central acute hematoma (*white arrow*) consisting of intracellular deoxyhemoglobin is slightly hypointense to gray matter. Surrounding subacute hematoma is markedly hyperintense (*black arrow*).**

***C*, Proton-density-weighted image, TR = 1500 msec, TE = 20 msec. Central acute hematoma (*large white arrowhead*) has remained mildly hypointense to cortex. Surrounding subacute hematoma now shows mildly hyperintense inner region (*curved arrow*) representing dilute intracellular methemoglobin and markedly hyperintense outer region (*black arrowhead*) representing free methemoglobin. Thin peripheral ring of parenchymal hypointensity (*straight arrow*) corresponds to hemosiderin deposition. Surrounding edema (*small white arrowhead*) has become hyperintense.**

***D*, T2-weighted image, TR = 1500 msec, TE = 40 msec. Acute hematoma has become more hypointense. Inner region of subacute hematoma has become isointense to gray matter, while outer region remains markedly hyperintense. Hemosiderin ring appears more hypointense and wider. Surrounding edema remains hyperintense. Acute central hematoma is composed of intracellular deoxyhemoglobin. Outer region of subacute hematoma corresponds to free dilute methemoglobin. Inner region of subacute hematoma corresponds to dilute intracellular methemoglobin.**

or hyperintense. On T2-weighted images, the regions that were isointense on the proton-density images were variably hypointense (Figs. 1 and 3–5), and the regions that were hyperintense on the proton-density-weighted images were variably hyperintense (Figs. 2, 3, and 5). One subacute hematoma (Fig. 1) was only 5 days old, indicating that the hyperintensity on T1-weighted images may appear as early as 5 days.

Discussion

The mechanism for the selective T2 relaxation enhancement of intracellular deoxyhemoglobin, intracellular methemoglobin, and of hemosiderin deposits is the dephasing of water molecules diffusing across field gradients owing to the heterogeneity in the distribution (intracellular or intralysosomal) of the paramagnetic substance (Fe^{2+} with four unpaired electrons for deoxyhemoglobin and Fe^{3+} with five unpaired electrons for methemoglobin, ferritin, and hemosiderin) [2]. In the absence of unpaired electrons, the protons of water molecules relax (realign with the magnetic field) via fluctuations in their local magnetic field caused by the motion of adjacent protons. The presence of unpaired electrons (for example, five unpaired electrons per methemoglobin molecule) creates fluctuations 1000 times larger owing to the electrons' larger magnetic moment. These larger fluctuations enhance the relaxation of protons. This is called the proton-electron dipolar-dipolar proton relaxation enhancement. The hyperintensity of T1-weighted images of subacute and chronic hematomas was ascribed to the proton-electron dipolar-dipolar proton relaxation enhancement of methemoglobin. This interaction drops off extremely rapidly with distance; the water proton must approach within 3Å of the unpaired electron for the interaction to occur. Unlike methemoglobin, the unpaired electrons of deoxyhemoglobin, ferritin, and hemosiderin are inaccessible to water protons for the dipolar-dipolar interaction. Intracellular methemoglobin, which has both relaxation mechanisms, causes significant T1- and T2-relaxation-time shortening. Cell lysis and the dilution of the liberated methemoglobin by resorption leads to a decrease in the T1 and T2 relaxation enhancement of the proton-electron dipolar-dipolar mechanism, and the cessation of the selective T2 relaxation enhancement caused by the intracellular (heterogeneously distributed) methemoglobin.

Heterogeneity in the distribution of any paramagnetic substance (for example, intracellular deoxyhemoglobin or methemoglobin) results in heterogeneity of magnetic susceptibility, which produces field gradients when an external magnetic field is applied. On spin-echo MR, diffusion of water protons

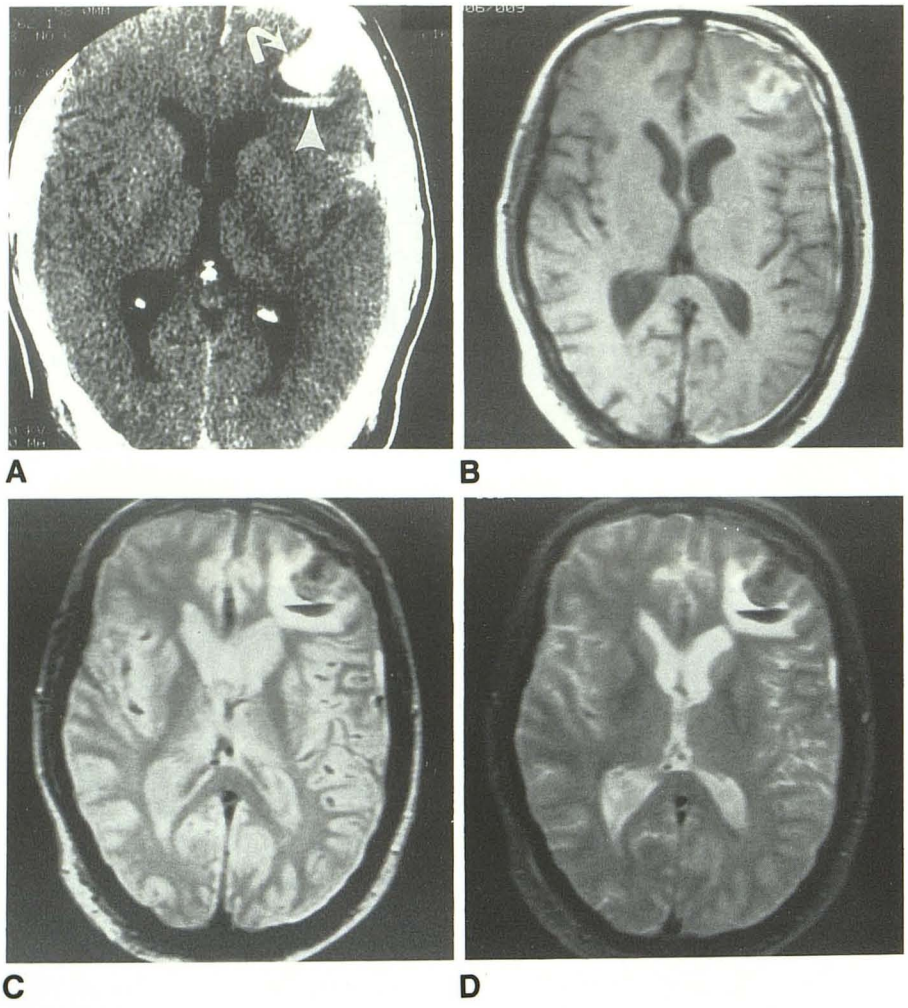

Fig. 3.—Case 3: traumatic 6-day-old hematoma.

A, Axial unenhanced CT scan (2 days after trauma) shows hyperdense left frontal hematoma (*arrow*). Posterior to it is hematoma region with fluid-fluid level (*arrowhead*) consisting of hypodense supernatant and hyperdense dependent layer. There is also a left temporal contusion.

B, Axial T1-weighted image, TR = 800 msec, TE = 25 msec. Hematoma has hyperintense periphery and isointense center. In fluid-fluid layer supernatant is slightly hyperintense and dependent layer is isointense to gray matter.

C, Proton-density-weighted image, TR = 2500 msec, TE = 35 msec. Hematoma center is hypointense to gray matter and periphery is isointense. Supernatant is markedly hyperintense and dependent layer is hypointense.

D, T2-weighted image, TR = 2500 msec, TE = 70 msec. Hematoma center is quite hypointense, representing intracellular deoxyhemoglobin. Periphery is hypointense, representing intracellular methemoglobin. Supernatant is very hyperintense, representing very dilute-free methemoglobin, and dependent layer is markedly hypointense, representing intracellular deoxyhemoglobin.

across these magnetic field gradients causes selective T2 relaxation enhancement, which increases as the square of the heterogeneity of the magnetic susceptibility and as the square of the magnetic field strength [1, 2]. It also increases with lengthening of the interecho interval [2]. The proton-electron dipolar-dipolar relaxation enhancement is not dependent on heterogeneity of magnetic susceptibility. It is unaffected by interecho interval and minimally affected by field strength or red cell lysis.

Recently, we studied the in vitro MR relaxation times of blood and their variations with field strength, oxidation state, and cell integrity [2]. That study confirmed the hypothesis of selective T2 relaxation enhancement for intracellular deoxyhemoglobin and methemoglobin and of proton-electron dipolar-dipolar proton relaxation enhancement for methemoglobin. The study also confirmed that the selective T2 relaxation enhancement of intracellular deoxyhemoglobin and of intracellular methemoglobin increases as the square of the magnetic field strength and as the square of the heterogeneity of magnetic susceptibility. The selective T2 relaxation enhancement also increased with prolongation of the interecho interval and was not present after cell lysis. The study demonstrated that the proton-electron dipolar-dipolar relaxation enhancement of methemoglobin was unaffected by field strength, the length of the interecho interval, or cell integrity.

The variable intensities of subacute hematomas on proton-density-weighted images are from differences in dilution. The variable intensities on the T2-weighted images are from both differences in dilution and variations in the selective T2 relaxation enhancement. The variability in the T2 relaxation enhancement is primarily caused by different degrees of cell lysis, resulting in a variable amount of selective T2 proton relaxation enhancement by the intracellular methemoglobin [2]. Cell lysis is more effective in decreasing the selective T2 relaxation enhancement of intracellular methemoglobin than is simple dilution because the methemoglobin released from the lysed cells increases the extracellular magnetic susceptibility. Thus, cell lysis decreases the differences in the magnetic susceptibility between the inside and the outside of the remaining intact RBCs containing methemoglobin. This decrease in the heterogeneity of magnetic susceptibility causes a marked decrease in the selective T2 relaxation enhancement, because the T2 relaxation enhancement is proportional to the square of the variation (heterogeneity) of the magnetic susceptibility [1, 2]. However, a change in the concentration of RBCs containing methemoglobin by dilution with a nonparamagnetic fluid such as plasma causes mild changes in the heterogeneity of magnetic susceptibility until the hematocrit exceeds 90% or is under 10% [1]. Therefore, dilution without lysis of intracellular methemoglobin does not greatly influence the selective T2 relaxation enhancement until it is under a hematocrit of 10%.

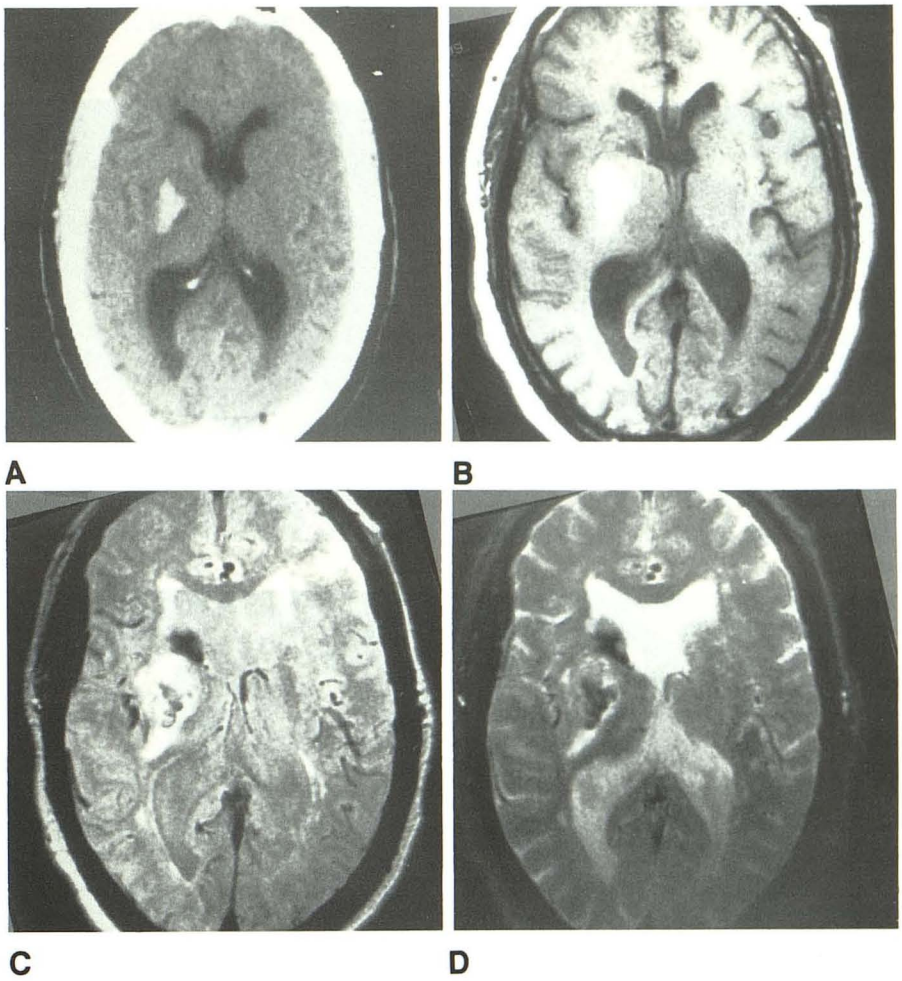

Fig. 4.—Case 4: 9-day-old hypertensive right basal ganglia hematoma in patient who apparently had a previous right caudate hemorrhage.

***A*, Unenhanced CT scan 6 days after hemorrhage shows hyperdense right basal ganglia hematoma.**

***B*, T1-weighted image, TR = 600 msec, TE = 25 msec, shows hyperintense hematoma.**

***C*, Slightly T2-weighted image, TR = 2500 msec, TE = 40 msec. Hematoma is hyperintense in periphery and isointense in center. Caudate is hypointense.**

***D*, T2-weighted image, TR = 2500 msec, TE = 80 msec. Hematoma has become more hypointense, representing intracellular methemoglobin. Caudate is quite hypointense, probably representing hemosiderin deposits of previous caudate hemorrhage.**

Intracellular methemoglobin has a powerful selective T2 relaxation enhancement in addition to its proton-electron dipolar-dipolar relaxation enhancement [2]. The proton density of intracellular methemoglobin or deoxyhemoglobin (hematocrit of 90% after plasma resorption) is similar to that of gray matter [1]. Therefore, purely intracellular methemoglobin is hyperintense on T1-weighted images, isointense or proton-density-weighted images, and quite hypointense on T2-weighted images (Fig. 1).

Panhyperintensity (on all spin-echo sequences) (Fig. 2) is a frequent finding in subacute and chronic hematomas. It is from the proton-electron dipolar-dipolar proton relaxation enhancement of dilute free methemoglobin. This relaxation causes a mild shortening of the T1 and T2 relaxation times, and the diluteness of the solution is responsible for a high proton density. The TRs used in spin-echo MR imaging are much longer than the TEs and usually are not much longer than the T1 relaxation times. The TEs are usually shorter than the T2 relaxation times. Therefore, the T1 shortening and high proton-density effects of dilute methemoglobin will dominate the spin-echo intensity pattern, resulting in panhyperintensity. Free, unbound Fe^{3+} ions also cause proton-electron dipolar-dipolar proton relaxation enhancement [2] and may also play a role in the panhyperintensity of dilute chronic hematomas.

Hyperintensity on T1-weighted images and isointensity on both proton-density- and T2-weighted images indicate a short T1 relaxation time and normal proton density and T2 relaxation time; that is, a proton-electron dipolar-dipolar proton relaxation enhancement without selective T2 proton relaxation enhancement or dilution. Free undiluted methemoglobin should have these properties.

If the hematocrit of intracellular methemoglobin is reduced by dilution without cell lysis (dilute intracellular methemoglobin) then the proton density will be elevated. The decrease in the proton-electron dipolar-dipolar relaxation enhancement is "compensated" by the increase in proton density until there is a great degree of dilution. The decrease in the selective T2 relaxation enhancement will only be evident if the hematocrit is drastically reduced (<10%) [1]. Therefore, the elevation of the proton density will be the most noticeable effect; that is, the T1-weighted image will remain hyperintense, the proton-density and T2-weighted images will become more intense (depending on the degree of dilution), but the T2-weighted image will still be significantly hypointense to the proton-density-weighted image (Fig. 2).

Intermediate states may exist. The intensity patterns of these states will vary between the patterns of the individual components. A combination of intracellular methemoglobin and deoxyhemoglobin will be variably hyperintense on T1-weighted images. The proton-density- and T2-weighted images will not be affected because the proton densities and the T2 relaxation times are similar for intracellular methemoglobin and intracellular deoxyhemoglobin. Similarly, different

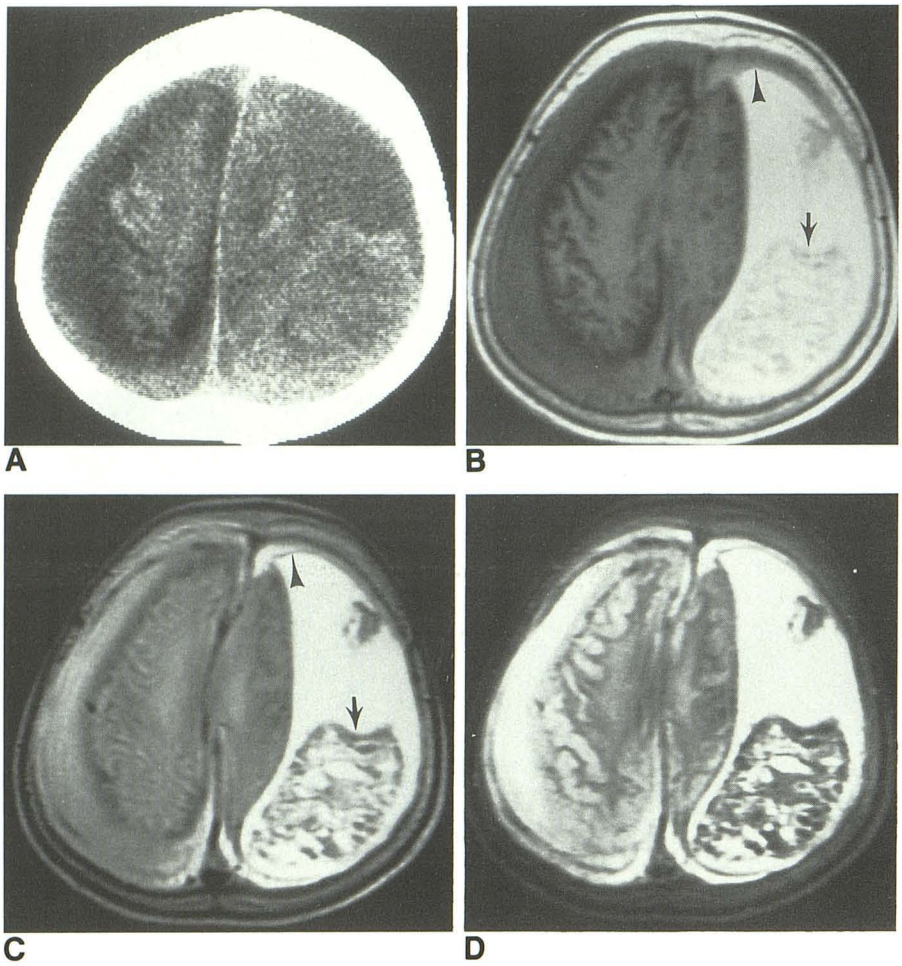

Fig. 5.—Case 5: subacute left subdural hematoma (surgically confirmed) of uncertain age in 3-year-old patient with seizure disorder and recurrent head trauma.

***A*, Unenhanced CT scan shows left subdural hematoma with slightly hypodense supernatant and isodense dependent portion. Small chronic subdural hygroma is on right.**

***B*, T1-weighted image, TR = 600 msec, TE = 20 msec, 7 days after CT study. Supernatant of left subdural hematoma is hyperintense and dependent portion (*arrow*) is less hyperintense with multiple pools of hyperintensity in it. There is small similar region superiorly (*arrowhead*). Right subdural hygroma is isointense.**

***C*, Proton-density-weighted image, TR = 2500 msec, TE = 30 msec. Supernatant remains hyperintense, and dependent (*arrow*) and superior (*arrowhead*) regions are now isointense with multiple pools of hyperintensity. Right subdural hygroma is slightly hyperintense.**

***D*, T2-weighted image, TR = 2500 msec, TE = 80 msec. Supernatant (free dilute methemoglobin) remains very hyperintense. Dependent and superior regions are hypointense (intracellular methemoglobin) with pools of hyperintensity (free dilute methemoglobin). These regions represent areas of clot lysis. Right subdural hygroma is now hyperintense.**

degrees of lysis of intracellular methemoglobin will result in variable T2-weighted image hypointensities because of the change in selective T2 proton relaxation enhancement caused by the variable decreases in the heterogeneity of the magnetic susceptibility. The T1-weighted and proton-density-weighted images will be relatively unaffected because both intracellular and undiluted free methemoglobin have similar proton-electron dipolar-dipolar proton relaxation enhancement and proton density. Various degrees of free methemoglobin dilution will cause the T1-weighted image to vary from hyperintensity to hypointensity, the proton-density-weighted image to vary from isointensity to hyperintensity and then back to isointensity, and the T2-weighted image to vary from isointensity to hyperintensity. As the free methemoglobin is diluted it loses its proton-electron dipolar-dipolar enhancement and its proton density increases to that of CSF. CSF has such long T1 and T2 relaxation times that on conventional spin-echo MR images with TRs less than 3000 msec it will be hypointense on T1-weighted images, mildly hypointense or isointense on proton-density-weighted images, and hyperintense on T2-weighted images. Of course other combinations may occur as well; for example, undiluted free deoxyhemoglobin and free methemoglobin. In this case there will be variable hyperintensity on T1-weighted images with isointensity on proton-density- and T2-weighted images.

Figure 6 illustrates the four possible methemoglobin states and Table 2 presents the functional relationships between the methemoglobin states and the resultant relaxation enhancements and proton density. In Table 2, the (Hct)(100 − Hct) factor is the heterogeneity factor [1]. The 1/T2* on gradient-echo MR is proportional to the field gradient across the cell membrane and the 1/T2 of spin-echo MR is proportional to the square of the field gradient. The gradient across the cell membrane is proportional to the difference in magnetic susceptibility across the cell membrane and to the field strength. The fractions in the equations result from the fact that methemoglobin has five and deoxyhemoglobin has four unpaired electrons per iron atom, and that the magnetic susceptibility is proportional to the number of unpaired electrons. Table 3 presents the patterns of the relative relaxation times and proton densities occurring in the various types of regions associated with subacute intracranial hematomas. Of course, mixtures will have patterns intermediate to their individual components.

A report by Di Chiro et al. [3] demonstrated the evolution of intracranial hemorrhage in a monkey on high-resolution MR imaging at 0.5 T. Their images (interpreted in light of the importance of susceptibility heterogeneity) at days 2–3 were characteristic of intracellular deoxyhemoglobin; days 4–8, of intracellular methemoglobin; and days 5–15, of dilute free

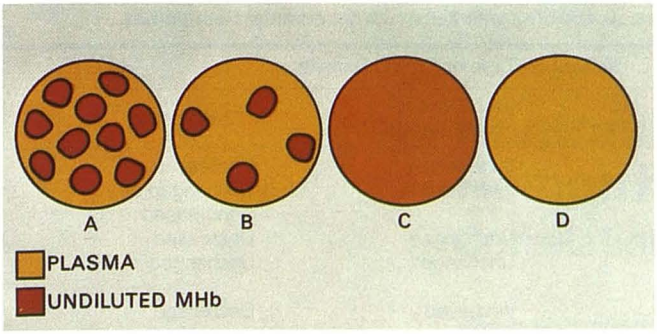

Fig. 6.—Four possible methemoglobin (MHb) states are combinations of methemoglobin and plasma. *Plasma*: no magnetic susceptibility; no dipolar-dipolar relaxation enhancement; high proton density. *Methemoglobin*: high magnetic susceptibility; has dipolar-dipolar relaxation enhancement; if undiluted, has average proton density.

***A*, Undiluted intracellular methemoglobin: has magnetic susceptibility heterogeneity (T2 shortening); has dipolar-dipolar relaxation enhancement (T1 shortening); has average proton density.**

***B*, Dilute intracellular methemoglobin: has some magnetic susceptibility heterogeneity; has some dipolar-dipolar relaxation enhancement; has high proton density.**

***C*, Undiluted free methemoglobin: has no magnetic susceptibility heterogeneity; has dipolar-dipolar relaxation enhancement; has average proton density.**

***D*, Dilute free methemoglobin: has no magnetic susceptibility heterogeneity; has some dipolar-dipolar relaxation enhancement; has high proton density.**

TABLE 2: Functional Relationships Between Hemoglobin States, Relaxation Rates, and Proton Density

$$\text{T2 PRE} \propto (\text{Hct})(100 - \text{Hct})\,[\,(\text{MHb}) + 24/35\,(\text{Hb}) - \text{MHb} - 24/35\ \text{Hb}]^2 \text{Bo}^2$$

$$\text{T2}^* \text{ PRE} \propto (\text{Hct})(100 - \text{Hct})\,[\,(\text{Mhb}) + 24/35\,(\text{Hb}) - \text{MHb} - 24/35\ \text{Hb}]^2 \text{Bo}$$

$$\text{PEDD PRE} \propto (\text{MHb}) + \text{MHb}$$

$$\text{PD}_w - \text{PD}_h \propto (\text{MHb}) + (\text{Hb}) + (\text{HbO}_2) + \text{MHb} + \text{Hb} + \text{HbO}_2$$

Note.—$\propto$ = approximately equal to; T2 PRE = 1/T2 on spin-echo MR owing to susceptibility heterogeneity; T2* PRE = 1/T2* on gradient-echo MR owing to susceptibility heterogeneity; PEDD PRE = 1/T1 and 1/T2 owing to proton-electron dipolar-dipolar proton relaxation enhancement; Hct = hematocrit; circled MHb = intracellular methemoglobin; MHb = extracellular (free) methemoglobin; circled Hb = intracellular deoxyhemoglobin; Hb = extracellular (free) deoxyhemoglobin; circled HbO_2 = intracellular oxyhemoglobin; HbO_2 = extracellular oxyhemoglobin; PD_w = proton density of pure water; PD_h = proton density of the subacute hematoma; Bo = strength of magnetic field. Magnetic susceptibility is proportional to n (n+2), where n = the number of unpaired electrons; hence, the fraction 24/35 for 4 and 5 unpaired electrons (deoxyhemoglobin and methemoglobin, respectively).

methemoglobin, as well as of hemosiderin at 2 months.

Melanin is a free-radical trap and can chelate paramagnetic metal ions. The unpaired electrons of the free radicals and of the chelated metal ions and probably of associated methemoglobin are the source of melanin's paramagnetism [4, 5]. The major paramagnetic effect of significance to spin-echo MR imaging is the proton-electron dipolar-dipolar interaction [6]. In deeply pigmented melanomas without gross pathologic evidence of hemorrhage this interaction can cause sufficient proton relaxation enhancement to mimic methemoglobin's hyperintensity on T1-weighted images (Fig. 7). However, melanotic melanomas are not as hyperintense on T2-weighted images as free dilute methemoglobin is nor as hypointense as undiluted intracellular methemoglobin. Thus, melanotic melanomas may be difficult to distinguish from certain regions of subacute hematomas. Fat is hyperintense on T1-weighted images, isointense on proton-density images, and hypointense on T2-weighted images at high field strengths (1.5 T) (Fig. 1). It may also be difficult to differentiate it from regions of subacute hematomas or from melanotic melanomas. However, associated MR and clinical findings usually allow the differentiation of melanotic melanoma, fat, and subacute hematomas.

Rapid imaging by gradient echoes rather than by spin echoes has recently been introduced. Its sensitivity to T2* allows the effects of susceptibility heterogeneity to be detected at much lower field strengths [7, 8], at the cost of lower signal to noise and increased sensitivity to field gradients because of the magnet and the macroscopic susceptibility heterogeneity and chemical shifts. Although subacute hematomas have not yet been carefully studied by gradient-echo techniques, they should have characteristics similar to those observed on high-field spin-echo MR. One additional effect seen on gradient-echo techniques can be confusing. It is the peripheral hypointensity caused by the border between macroscopic regions of different susceptibilities, for example, dense calcium–soft tissue; air–soft tissue; fat–water; and deoxyhemoglobin, methemoglobin, or hemosiderin and soft tissue. These must be differentiated from marginal hemosiderin deposits around hemorrhagic lesions. Marginal hemosiderin deposits have both T2* shortening and T2 shortening on spin-echo images because of both macroscopic and microscopic susceptibility heterogeneity.

Our current clinical MR observations and previous in vitro models indicate that further subcategorization of subacute hematomas is possible on the basis of T1-, proton-density-, and T2-weighted images. However, melanotic melanomas and fat may be difficult to differentiate, by intensity patterns alone, from certain regions of subacute hematomas.

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TABLE 3: Relative Relaxation Times and Proton Densities in Regions Associated with Subacute Intracranial Hematomas

Region	Relaxation Time or Proton Density		
	T1-Weighted	Proton-Density-Weighted	T2-Weighted
Dilute intracellular methemoglobin[a]	Decreased	Increased	Decreased
Dilute (free) lysate (free) methemoglobin[a]	Decreased	Increased	Unchanged or increased
Undiluted intracellular methemoglobin[a]	Decreased	Unchanged	Decreased
Undiluted (free) lysate (free) methemoglobin[a]	Decreased	Unchanged	Unchanged
Dilute intracellular deoxyhemoglobin	Unchanged or increased	Increased	Decreased
Dilute intracellular and lysate (free) oxyhemoglobin; lysate (free) deoxyhemoglobin; or any dilute protein solution, edema, tumor, or CSF	Unchanged or increased	Increased	Unchanged or increased
Intracellular deoxyhemoglobin or hemosiderin	Unchanged	Unchanged	Decreased
Gray matter (standard) or undiluted (free) lysate (free) deoxyhemoglobin or oxyhemoglobin, or undiluted intracellular oxyhemoglobin	Unchanged	Unchanged	Unchanged
Hemosiderin deposits ± calcifications	Unchanged	Decreased	Decreased
Very dense calcifications, dura, or flow void	Unchanged	Decreased	Unchanged

[a] Can be mimicked by fat or melanin.

Note.—Undiluted = concentration equivalent to hematocrit of 90%.

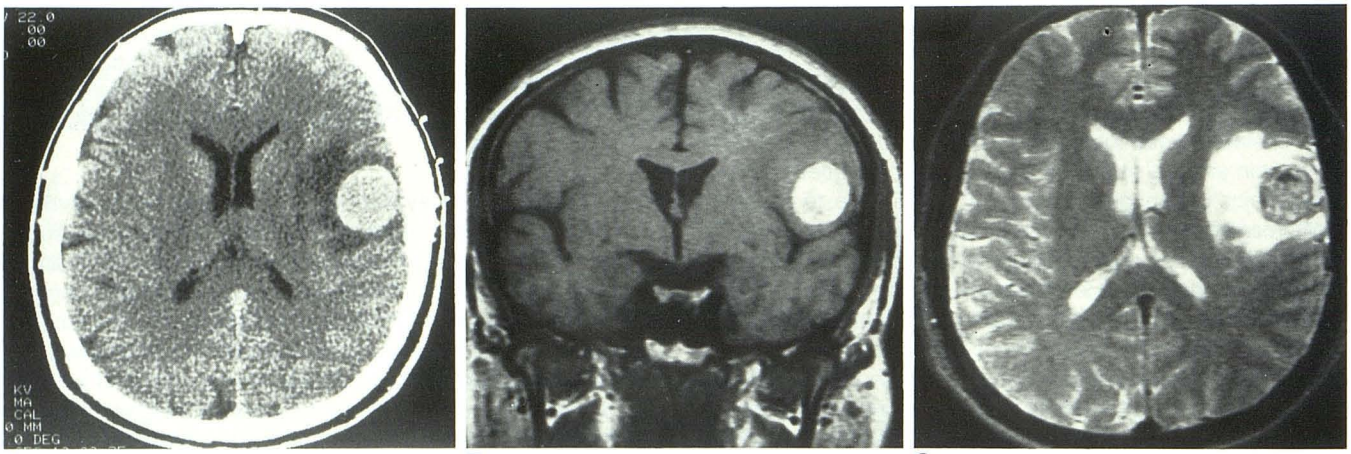

Fig. 7.—Nonhemorrhagic (pathologically proved) melanotic melanoma metastasis.

***A*, Unenhanced CT scan shows high-absorption left frontal mass surrounded by low-absorption regions (edema).**

***B*, T1-weighted image, TR = 600 msec, TE = 20 msec. Tumor is hyperintense because of proton-electron dipolar-dipolar proton relaxation enhancement of melanin.**

***C*, T2-weighted image, TR = 2500 msec, TE = 80 msec. Tumor is mostly isointense with some regions of hypointensity. There is no peripheral hemosiderin ring. Surrounding edema is hyperintense.**

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