Brain iron and T2 signal.

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LETTERS

Unusual Coil-Release Method

We would like to report an unusual method of releasing a coil stuck at the end of a catheter during an embolization procedure.

During transvenous embolization of a superficial temporal arteriovenous fistula, one of the coils became partially stuck in the catheter with the free end in the target vein. Numerous attempts to push the coil through were unsuccessful. Subsequently, the free end of the coil under the scalp was palpated and compressed by one operator, while the other operator withdrew the catheter, successfully releasing the coil in the target vein.

This method can obviously be used only under limited circumstances (superficial vascular procedures) but adds to the "treasure chest" of ways to get out of trouble during an interventional procedure.

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Brain Iron and T2 Signal

Several conclusions drawn in the paper by Thomas et al, "MR Detection of Brain Iron," (1) are not supported by the authors' data. We would like to bring our arguments to your attention for two reasons. We wish first to defend papers that we have previously coauthored (2, 3), and second, to emphasize that although we do not dispute that iron can shorten T2, we do not feel that it has been proved that iron is the *most* important factor in causing signal reduction in the basal ganglia. Our objections to the paper by Thomas et al are as follows:

1. They state that they have shown a relationship between "the age-dependent accumulation of iron coinciding with the rate of change of T2 signal." However, they did not measure T2 and iron of the same samples. Therefore at best they have shown that T2 decreases in some brain regions with age and that iron increases in these regions with age. A causal relationship between iron and T2 shortening is not supported by their data. They do not comment on the possibility that there may be other tissue variables, albeit as yet undetermined, that might contribute to the T2 decline.
2. With the exception of those patients under age 10 years, the graphs of their data raise questions about *any* relationship between the T2 and iron. The scatter of the data points on their graphs is tremendous. For instance, in the red nucleus and substantia nigra of patients over 60 years of age, iron concentration ranges from 500 to 2000 nmol/g (a 400% variation), but T2 ranges from only 50 to 60 milliseconds (a 20% variation). If T2 were closely related to iron, a much greater variation of T2 values would be expected. Furthermore, the error inherent in measuring T2 with a 4-echo clinical sequence, which we estimate from previous experiments on an identical MR scanner to be greater than 10%, is overlooked. Thus the variation in T2 reported by the authors can be almost completely accounted for by measurement error alone.
3. Their experiments with iron-containing agarose phantoms demonstrate no dependence whatsoever between ferrous iron concentration and T2, contrary to the statement in their conclusions. The T2 of the phantom with 0.25 mm iron is 40 milliseconds and the T2 of the phantom with 2.0 mm iron is also 40 milliseconds. This is an 800% change in ferrous iron concentration with no change in T2! With ferric ions there is a weak trend, a decline from 25 milliseconds to 19 milliseconds when ferric concentration increases from 0.5 mm to 2.0 mm, but in the absence of error bars the reproducibility of the measurements and the statistical significance of this trend is questionable.

Thus, for the reasons above and for the reasons raised in the commentary by Gomori and Grossman (4), we agree with Gomori and Grossman that Thomas et al's data do not support the hypothesis that brain iron contributes to the decreased T2 signal in the basal ganglia. Also, we partially agree with Gomori and Grossman in their reference to the experimental technique used to measure T2 in our papers (2, 3) to the extent that our technique was not exactly analogous to the clinical situation, but it was not, as they refer to it, "flawed." We used a 2.5-millisecond interecho delay with a minimum of 25 echoes, whereas clinically interecho delays are typically 20 to 40 milliseconds with 2 to 4 echoes. Our T2 measurement accuracy is better than 3%; the technique they propose has an error of 10%. Thus, if we had used their technique, which they correctly identify as being more sensitive to T2* effects, or, more accurately, apparent T2 because of diffusion in the presence of magnetic field inhomogeneities, we would have introduced a significantly larger error.

Many radiologists believe that the different degrees of darkness in the basal ganglia on T2-weighted images can be attributed to and equated with differences in iron concentration in these regions. Although we agree that tissue iron does reduce magnetic resonance signal, it is our opinion that at the concentrations of iron found in the basal ganglia, the dependence of T2 on total iron concentration is weak. Thus, the dominance of iron's effect, relative to the multiple other tissue constituents that can affect magnetic resonance signal, is unproved. Radiologists should

not equate basal ganglia signal differences with differences in iron concentration.

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References

1. Thomas LO, Boyko OB, Anthony DC, Burger PC. MR detection of brain iron, *AJNR Am J Neuroradiol* 1993;14:1043–1048
2. Chen JC, Hardy PA, Clauberg M, et al. T2 values in the human brain: comparison with quantitative assays of iron and ferritin. *Radiology* 1989;173:521–526
3. Chen JC, Hardy PA, Kucharczyk W, et al. MR of human postmortem brain tissue: correlative study between T2 and assays of iron and ferritin in Parkinson and Huntingdon disease. *AJNR Am J Neuroradiol* 1993;14:275–281
4. Gomori JM, Grossman RI. The relation between regional brain iron and T2 shortening. *AJNR Am J Neuroradiol* 1993;14:1049–1050

Editor's note: This letter was sent to Dr Thomas and her colleagues for reply. No reply has been received.

MR of Pineal Cysts

In the January issue of the Journal, Fleege et al reported a most interesting group of 19 patients with symptomatic pineal cysts, all surgically confirmed (1). Pineal cyst was the preoperative diagnosis in only three cases (15%). However, they report that 41% were isointense with cerebrospinal fluid on T1- and T2-weighted scans, the typical appearance of a pineal cyst.

The authors argued that the T2 signal, enhancement, and hydrocephalus noted in many of their cases were atypical for pineal cyst. They stated, "On T2-weighted images they [typical pineal cysts] are hyperintense relative to brain and CSF" and cite an early paper (2). In that paper we described a variable contrast of pineal cysts with cerebrospinal fluid on T2-weighted scans obtained on scanners at different field strengths. It would seem quite reasonable that less contrast was noted on these scans obtained at 1.5 T than in that earlier experience at 0.15 and 0.5 T. The authors suggested that the nodular enhancement of the cysts in their series varied from that described in the literature (3). In the paper referenced we stated, "In two cases [out of six], small areas of focal enhancement were seen within the cyst . . . This enhancement may be inhomogeneous at first but becomes homogeneous on later scans." It was our intent with that paper to emphasize that the enhancement of benign pineal cysts may cause them to be mistaken for pineal tumors. Hydrocephalus in association with benign pineal cyst has been reported frequently and correlates with the size of the cyst.

Fleege et al noted that three cysts with fluid-fluid levels and suggest that this finding reflects recent hemorrhage. However, these fluid-fluid levels can also be seen in asymptomatic pineal cysts. One was illustrated in our 1986 paper. Their observation that this correlated with hemosiderin in the cyst wall is of considerable interest but not convincing for recent hemorrhage in all cases as suggested.

It is important at the outset to recognize that although pineal cysts are common, Fleege et al described a unique subset of patients. The cysts they described were on average 1.6 cm in size and, obviously, all were sufficiently symptomatic to require surgery. The authors concluded, "The MR appearance of pineal cysts is variable, ranging from that of an uncomplicated cystic mass to a mass associated with hemorrhage, enhancement, or hydrocephalus. This variability may make them indistinguishable from other pineal-region tumors". We are not suggesting that all of these cases should have been diagnosed before surgery. The two patients in whom the cyst was isointense or hyperintense with brain on T1 were atypical and could not be confidently diagnosed before surgery. However, the authors do not choose to emphasize that many if not most of the remaining 17 cases had entirely typical imaging features of pineal cyst.

Their paper emphasizes the many pitfalls these lesions provide. However, we would argue that enhancement, fluid-fluid levels, and hydrocephalus should not subvert the diagnosis of pineal cyst in those cases with otherwise typical magnetic resonance imaging features. Certainly, in patients with symptoms of midbrain compression or hydrocephalus, surgery is usually necessary regardless of whether the lesion is a tumor or benign cyst. But in the more frequent situation of a patient with the presenting symptom of headache without hydrocephalus, it is essential to be familiar with all the imaging features of pineal cysts.

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References

1. Fleege MA, Miller AM, Fletcher GP, Fain JS, Scheithauer BW. Benign glial cysts of the pineal gland: unusual imaging characteristics with histologic correlation. *AJNR Am J Neuroradiol* 1994; 15:161–166
2. Mamourian AC, Towfighi J. Pineal cysts: MR imaging. *AJNR Am J Neuroradiol* 1986;7:1081–1086
3. Mamourian AC, Yarnell T. Enhancement of pineal cysts on MR images. *AJNR Am J Neuroradiol* 1991;12:773–774

Reply

We would like to thank Drs Mamourian and Towfighi for their careful review of our manuscript. However, we do not understand the point of their criticisms. It was our intention to describe the magnetic resonance findings in a large series of symptomatic patients with pathologically proven pineal cysts. Studies describing the magnetic resonance appearance of pathologically proven pineal cysts are rare, limited to individual case reports or small series. Drs Mamourian and Towfighi described the magnetic resonance findings in a group of asymptomatic patients with presumed pineal cysts; they have no pathologic verification (1, 2). In the absence of surgical or pathologic proof, we are not certain how they conclude that their findings are typical for pineal cyst or how they can compare their findings with ours. We cannot comment on differences caused by field strength because we do not know whether pathologically proved glial cysts look different at low field strength.

It is unfortunate that Drs Mamourian and Towfighi have missed the main point of our manuscript. We stand by our conclusion that the magnetic resonance appearance of pineal cysts is variable and that this variability may make them indistinguishable from other pineal region tumors. It is important to include glial cyst in the differential diagnosis of mass lesions of the pineal gland. Communication between the radiologist, surgeon, and pathologist plays a pivotal role in patient treatment. For further discussion of the clinical and histologic characteristics of symptomatic glial cysts, we recommend the manuscript by Fain et al (3).

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References

1. Mamourian AC, Towfighi J. Pineal cysts: MR imaging. *AJNR Am J Neuroradiol* 1986;7:1081–1086
2. Mamourian AC, Yarnell T. Enhancement of pineal cysts on MR images. *AJNR Am J Neuroradiol* 1991;12:773–774
3. Fain JS, Tomlinson FH, Scheithauer BW, et al. Symptomatic glial cysts of the pineal gland. *J Neurosurg* 1994;80:454–460

Lesions in Oculocerebrorenal Syndrome

In a recently published case report, Carroll et al (1) describe two distinct lesions occurring in oculocerebrorenal syndrome. The first lesion consists of confluent white matter abnormalities that are hyperintense on long-repetition-time magnetic resonance images. These have been previously reported (2, 3). The authors introduce a second lesion that consists of multiple cystic foci in the subcortical and deep white matter. Although not cited in the authors' references, we had previously described these cystic lesions that are visible on short and long repetition sequences (4). We agree with the authors that these findings suggest a more profound white matter involvement with this disease than originally may have been appreciated.

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References

1. Carroll WJ, Woodruff WW, Cadman TE. MR findings in oculocerebral syndrome. *AJNR Am J Neuroradiol* 1993;14:449–451
2. O'Tuama LA, Laster DW. Oculocerebrorenal syndrome: case report with CT and MR correlates. *AJNR Am J Neuroradiol* 1987;8: 555–557.
3. Gabrielli O, Pierleoni C, Barbato M, Cecconi M, Coppa GV, Giorgi PL. Study of the SNC using MRI in a case of Lowe's syndrome [Ital]. *Pathologica* 1989;81:643–645
4. Demmer LA, Wippold FJ II, Dowton SB. Periventricular white matter cystic lesions in Lowe (oculocerebrorenal) syndrome. *Pediatr Radiol* 1992;22:76–77

Reply

I was interested to find out that Demmer et al have described similar cystic foci in the deep and subcortical white matter in patients with oculocerebrorenal syndrome. Our findings corroborate their earlier findings and provide further evidence for the presence of two distinct lesions.

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