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Subdural hematoma mimicking epidural hematoma.

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Correspondence

Letters to the Editor

Diffuse Cerebral Angiomatosis

I read with interest the paper by Wagle et al. [1] in which the working diagnosis of diffuse capillary telangiectasis of the brain was based on the angiographic and MR findings. In the differential diagnosis of a case with diffuse cerebral angiomatosis, the syndromes of Divry–Van Bogaert [2] and Sneddon [3] should be considered.

Divry–Van Bogaert syndrome is manifested by noncalcified diffuse leptomeningeal angiomatosis and livedo reticularis. The syndrome may be sporadic or familial. The neurologic picture that appears around the third decade of life includes multifocal ischemic attacks, dementia, epileptic seizures, and pseudobulbar and extrapyramidal syndromes. Carotid angiography can show either extensive angiomatosis localized mainly in the distal territories of anterior and middle cerebral arteries [4] or distal, fine angiomatous circulation from tiny collateral vessels in distal arterial occlusions [3]. Postmortem examinations have shown leptomeningeal angiomatosis, multifocal infarcts, and parietal thickening of the leptomeningeal arteries with thrombosis and recanalization.

The clinical and radiologic findings in Sneddon syndrome are almost identical to those of Divry-Van Bogaert syndrome, so at present it is difficult to distinguish between them. The livedo usually precedes the first neurologic symptom but may appear afterward. In any case, if a diagnosis of Sneddon syndrome is to be considered, it would be worth performing angiography on the hand to see if the characteristic narrowing and dilatation in the common and proper palmar digital arteries are present.

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Neuroimaging and the Lesion of Multiple Sclerosis: An Addendum

In late November 1986, a second MR image was obtained of the patient previously described in the May/June 1987 issue of *AJNR* [1]. This procedure was performed on a much newer machine and resulted in an image that was technically far superior. The second MR image was compared with the double-dose delayed contrast-enhanced CT scan originally obtained in January 1984 (Fig. 1 on next page). The comparison dramatically confirmed and reinforced our original observation that few if any of the areas of impaired blood-brain barrier developed into areas of increased signal intensity that may represent plaques of multiple sclerosis. Close examination of the new MR image suggests that most, if not all, of the plaques located in the periventricular white matter did not enhance in January 1984 and thus probably antedate that particular exacerbation. Clinically, the patient has continued to maintain a completely stable condition.

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Subdural Hematoma Mimicking Epidural Hematoma

A recent article in *AJNR* [1] reported two cases of acute subdural hematoma "mimicking" an epidural hematoma. However, in both cases, the extraaxial hematomas clearly can be seen to cross nearby adjacent calvarial suture lines, suggesting a subdural location.

It is well established that an epidural hematoma rarely dissects past a calvarial suture [2–4]. Dissection usually is seen in association with a fracture and dural laceration involving a dural sinus (e.g., superior sagittal sinus, transverse sinus). In neither of the cases presented is the extraaxial collection adjacent to a significant dural sinus.

In addition, the hematoma on the right side of case 1 clearly shows a crescentic "tail," which is concave toward the brain. This is a reliable differential feature in favor of subdural hematoma. Ironically, this is a distinguishing feature that the authors describe in their discussion.

Acute subdural hematomas are seen most often in association with other injuries, especially cortical contusions. Both these patients had thrombocytopenia, and a hemorrhagic diathesis has been reported in patients with isolated acute subdural hematoma [5].

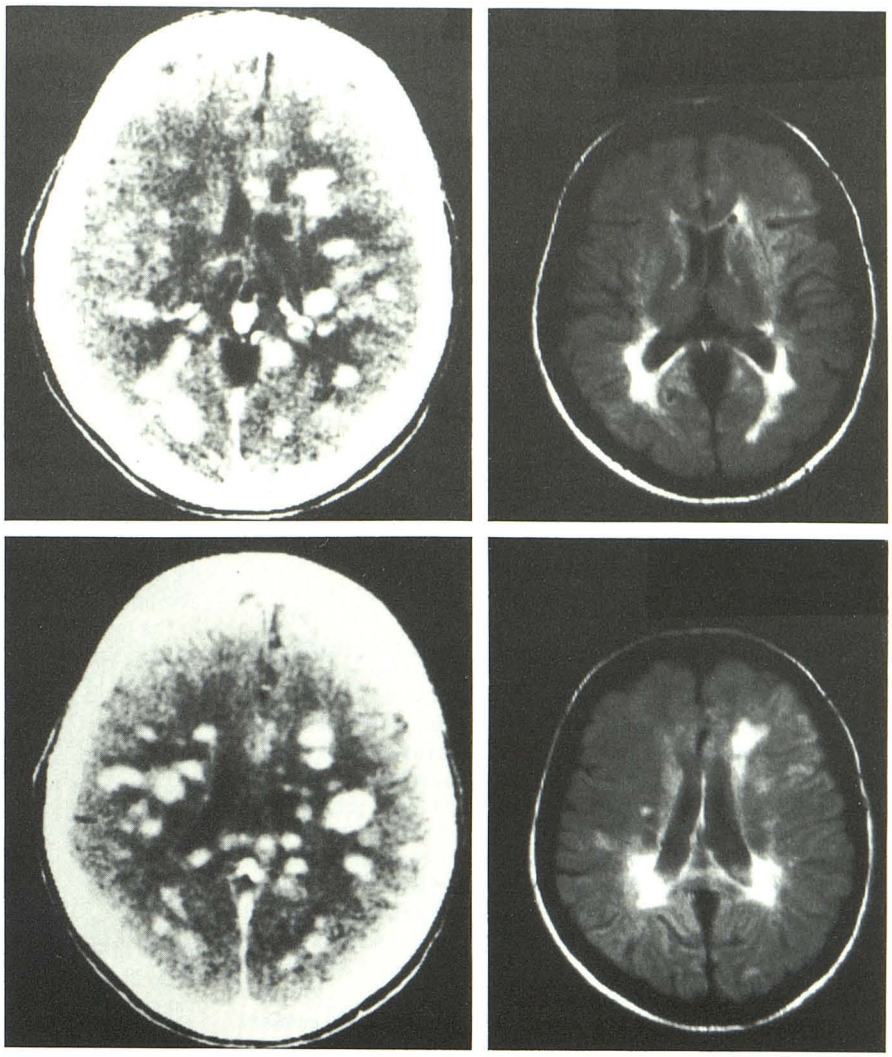

Fig. 1.—Double-dose delayed contrast-enhanced CT scan obtained January 1984 (*left*) and MR image (TR = 2.5 msec; TE = 30 msec) obtained November 1986 (*right*) of the brain of a woman with long-standing multiple sclerosis. (MR image courtesy of James Wallman, Lahey Clinic, Burlington, MA.)

This report emphasizes that subdural hematomas can produce a biconvex mass with an acute clinical presentation, but the differential features allow localization of the blood to the subdural space.

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* The author's views are not necessarily those of the Departments of the Army or Defense.

Reply

Dr. Smirniotopoulos mentions two features that may help to localize the described hematomas to the subdural space: crossing of calvarial suture lines and the presence of a crescentic tail at the periphery of the hematoma. We certainly agree that these signs may be very useful in establishing the correct diagnosis. However, these differential features are not present in every case. We were able to find a case of an epidural hematoma that dissected past a calvarial suture (coronal suture) without involvement of a dural sinus (Fig. 2 in [1]). In addition, one of the hematomas described by us did not cross any suture line (Fig. 1, left-sided hematoma, in [2]). A crescentic tail was not present in two of the three acute subdural hematomas reported by us. In such cases it may be very difficult or impossible to arrive at the correct preoperative diagnosis.

We think that this type of acute subdural hematoma cannot be judged always by the usual diagnostic criteria. The pathophysiology involved in these cases is different; namely, acute bleeding into a blocked subdural space. As a result, the hematoma is fundamentally

focal. The peculiar shape of this "hyperconvex subdural hematoma" may serve as a clue to the correct diagnosis because the arachnoid membrane surrounding it can be stretched and ballooned more easily than the dura mater. Epidural hematomas are usually more fusiform. Finally, when a hyperconvex extraaxial hematoma contains a fluid level, the diagnosis of acute subdural hematoma should be considered.

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Physiologic Changes During High Field Strength MR Imaging

I read with interest the recent article by Kido et al. [1] concerning the physiologic changes during high field strength MR imaging, and I think that certain aspects of this paper need further clarification and explanation. In addition, I would like to offer some constructive criticism.

Kido et al. indicated that "the temperature increases and other physiologic changes observed during MR scanning . . . at RF powers of 0.2 and 0.8 W/kg were small and of no clinical concern." It should be noted that the investigators based their comments about temperature on measurements obtained from the axilla of their normal volunteers.

The purpose of the study was "to quantify changes in body surface temperature." However, the axilla is an unacceptable site for temperature measurement since it is not representative of surface (or skin) temperature, primarily because it is not an exposed surface, and the tissues of the upper arm radiate to one another. In thermophysiologic studies, numerous sites have been selected for assessments of changes in surface or skin temperature, but the axilla has never been one of them [2].

The authors also should realize that measurements obtained via contact techniques, such as the thermistor probe used in their study, are subject to considerable error because of the variations in skin temperature caused by pressure exerted from the device [3]. Conventional thermistors are also unacceptable for measurement of temperatures during exposure to RF radiation because the wire leads can distort the field and produce heating by electromagnetically induced currents [4]. High-resistance thermistors connected to leads with electrical conductivity characteristics that are about the same or less than the electrical conductivity of the surrounding medium (i.e., tissue) must be used [4]. It does not suffice to simply disconnect the thermistor during the MR scan and then connect it when the temperature measurements are taken, as was done in this study.

Nothing was mentioned about the environmental conditions (e.g., room temperature, relative humidity, airflow) in which these experiments were performed or whether the patients were allowed to adjust or "equilibrate" to these conditions. This is particularly important because temperature responses can be altered dramatically in relation to environmental changes. Were the environmental conditions controlled and the same for each subject in this study?

It was unclear whether the subjects undergoing the head scans had MR performed with a transmit/receive "head coil," as is usually the case in the clinical setting, or with the body coil. The indicated pulse parameters and specific absorption rates (i.e., up to 0.06 W/kg) suggest that the head coil was used; in that case, only a localized absorption of RF power occurs. The coil should be specified, particularly since the changes in temperature measured from the axilla cannot reveal to any substantial degree what is happening within the head coil. Because of the aforementioned issues, the significance of the information provided by this study is questionable.

Kido et al. indicated that their results (i.e., changes in axillary temperatures of 0.1°C for the 0.0 W/kg group, 0.2°C for the 0.2 W/kg group, and 0.5°C for the 0.8 W/kg group) were consistent with a study performed by Schaefer et al. [5] on RF radiation-induced heating. However, Schaefer et al. reported an average change in body temperature (measured in the esophagus) of only 0.3°C during a 20-min exposure at a specific absorption rate of 4.0 W/kg (approximately five times the level used in the study by Kido et al.). How can these results be considered comparable?

Another minor point, the bar graph in Fig. 1 does not appear to indicate the reported temperature changes of 0.1, 0.2, and 0.5°C for the subjects in the 0, 0.2 and 0.8 W/kg groups (i.e., the bars do not "line up" with the temperature scale).

The topic of temperature responses to MR imaging performed at specific absorption rates above the level recommended by the U.S. Food and Drug Administration (whole-body average specific absorption rate of 0.4 W/kg) is extremely important and has significant safety and economical implications. For these reasons, it is vital that investigations examining temperature changes resulting from the absorption of high RF power be conducted in a manner that is physiologically meaningful (i.e., measurements should be obtained at sites that are representative of skin and/or body temperatures) and clinically relevant (i.e., if transmit/receive surface coils are used, temperatures should be measured from the tissue that is contained within the coil). Only then will a thorough understanding of the thermal effects of RF radiation from MR imaging be understood so that safe levels of exposure and thresholds for adverse effects can be determined.

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Reply

We have attempted to answer the questions posed by Dr. Shellock in sequence. First, the axillary site was chosen as a compromise for