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Hyperintense basal ganglia on T1-weighted MR in a patient with Langerhans cell histiocytosis.

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LETTERS

Hyperintense Basal Ganglia on T1-Weighted MR in a Patient with Langerhans Cell Histiocytosis

We read with great interest the article by Poe et al (1) in the November 1994 *AJNR*. They described demyelinating and gliotic cerebellar lesions in patients with Langerhans cell histiocytosis. In one of their patients, T1-weighted magnetic resonance (MR) images also demonstrated hyperintensities involving the basal ganglia and internal capsules. This patient had no neurologic dysfunction referable to these areas. The authors do not know what accounts for these hyperintense signals and propose that they may represent an uncommon manifestation of calcification or demyelination/remyelination of axonal sheaths.

Increased signal intensity in the basal ganglia and internal capsules on T1-weighted MR images has also been described in patients with chronic hepatic failure (2), in patients with portal-systemic encephalopathy (3), and in patients with long-term parenteral nutrition therapy (4). The basis for these signal changes in the case of long-term parenteral nutrition therapy may be deposition of intravenously administered paramagnetic trace elements, especially manganese, and/or an astrogliotic reaction to such deposition (4). In patients with liver disease, it may be speculated that hepatic dysfunction or the presence of portosystemic collateral vessels may result in bypassing of the regulatory mechanisms that clear paramagnetic substances such as manganese and prevent their deposition in brain tissue under normal conditions (4).

Liver involvement is common in Langerhans cell histiocytosis. Histiocytic infiltration of the liver may lead to sclerosing cholangitis with progressive liver failure and portal hypertension (5). Data on the liver function of the patient of Poe et al were not available, but possible liver involvement could be responsible for the increased signal intensity in the basal ganglia and internal capsules on T1-weighted MR images of their patient.

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Reply

We are pleased that Dr Miaux and colleagues found our patients stimulating enough to prompt an interesting question, and we thank them for their comments. We were previously aware of the research cited in their references regarding hyperintensity in the basal ganglia in patients with hepatic failure or receiving parenteral nutrition. However, our patient did not exhibit hepatosplenomegaly or liver dysfunction and did not receive parenteral nutrition. It is possible that the signal disturbance was related to the transitory presence of lipid-laden macrophages or released free radicals. The cause of these disturbed signals in our patient remains a mystery to us. We are dissatisfied that more than one inconclusive sign in our patients could not be explained. This seems almost "expected" in a disease that has undergone different classifications over the years and until only recently was considered neoplastic.

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Comment

The description by Poe et al of elevated signal intensity on T1 images within the basal ganglia of a patient with Langerhans cell histiocytosis is of interest, especially the absence of hepatic dysfunction (1) or the use of parenteral nutrition (2). Although the primary message of the article is that changes in cerebellar white matter signal intensity on T1- and T2-weighted images may occur in association with this disorder, increased signal intensity within the basal ganglia on T1-weighted images is also described. These alterations in the basal ganglia are, however, said to be associated with increased signal intensity on T2-weighted images.

In our own experience, increased T1 signal intensity in the globus pallidus, putamen, mesencephalon, and pitu-

itary gland most commonly occurs in adult patients with non-Wilsonian chronic hepatic dysfunction (1). In these patients there is no perceptually increased signal intensity on T2-weighted images. Similar findings have subsequently been demonstrated in pediatric patients with chronic hepatic dysfunction (J. A. Brunberg, unpublished data). Additionally, among 127 patients with Wilson disease, MR regions of increased signal intensity on T1-weighted images have been demonstrated in 11 of 127 patients, generally correlating with the presence and severity of hepatic dysfunction (J. A. Brunberg, unpublished data). In this group of patients, T2-weighted images often demonstrated significant associated alteration in signal intensity of the globus pallidus and putamen, as previously reported (3). Resolution of these regions of high signal intensity on T1-weighted images in the basal ganglia has been demonstrated in patients with chronic hepatic dysfunction after appropriate medical treatment (4) or liver transplant (5) and has been observed in our own patients with Wilson disease after institution of appropriate management of copper metabolism. In Wilson disease, we expect that this resolution relates to an improvement in hepatic function and not to a direct effect of brain copper on MR signal intensity.

Especially interesting has been the occasional finding of an identical pattern of alteration in T1 signal intensity in patients without hepatic dysfunction. In our experience, the most common occurrence of this finding is in lupus patients with clinically suspected encephalopathy. Only one such patient has had either a history of or current laboratory evidence of hepatic dysfunction. The possibility that an autoimmune process relates to the presence of such altered signal intensity in this group of patients, as Poe et al postulated for the more extensive MR changes in patients with Langerhans cell histiocytosis, is interesting but speculative. Finally, we continue to encounter 2 to 3 patients each year who have the above-described MR finding on T1-weighted images, but have no clinical or laboratory evidence of underlying disease. In several of these patients, extensive evaluations for laboratory evidence of hepatic dysfunction, autoimmune disease, calcium dysmetabolism, and altered manganese levels have been unrevealing.

Persistent attempts to obtain appropriate brain autopsy material for quantitative chemical analysis from patients with this pattern of increased T1 signal intensity have, in our hands, been uniformly unsuccessful. Such information, both from patients with hepatic dysfunction and from patients with similar MR alteration unassociated with hepatic dysfunction, will be essential for understanding this striking MR alteration.

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Cut-Off Fragments of Rubber Caps of Bottles of Contrast Material: Foreign Bodies in the Drip Infusion System

Our purpose is to warn of the possibility that small fragments of the rubber cap from contrast material bottles may enter the vascular system during administration. Our study results prompt us to recommend using a drip infusion set *with a filter* for all radiologic examinations.

The drip infusion system is used worldwide for parenteral administration of dextrose water or other medical substances in radiology as well as in other departments. The majority of the bottles for these systems have rubber caps pierced with needles that accompany the set. During intravenous administration of contrast material for cranial computed tomography (CT), we noted two small fragments of a rubber cap (product a, manufactured by company 1) in the dripping column of a drip infusion tube (Fig 1). This observation led to the following investigation. The names of the products and of the manufacturers are masked in this letter.

For a 3-month period at institution A, we inspected various contrast infusions for the presence of rubber fragments in the administration systems in more than 500 sequential radiologic examinations that used drip infusion sets. The radiographic examinations included infusion

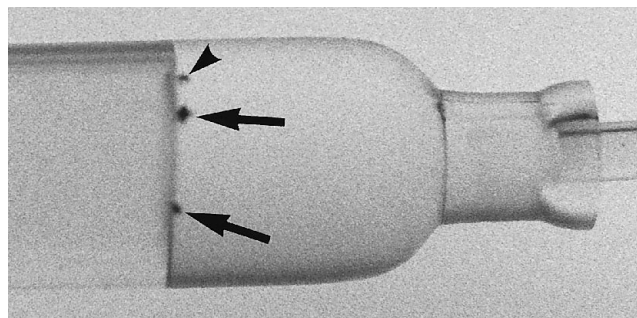


Fig 1. Two small fragments are floating in the drip column of the drip infusion set (arrows). These fragments were collected and found to be fragments from the rubber cap of the bottle. The other object is a small air bubble (arrowhead).

urography, drip infusion cholangiography, and intravenous digital subtraction angiography. Six different products manufactured by 3 separate companies were inspected. Company 1 manufactured a (n = 100) and b (n = 30). Company 2 manufactured c (n = 100), d (n = 25) and e (n = 5). Company 3 manufactured f (n = 240). At institution B, we performed the study in 100 contrast-enhanced cranial CT examinations using only product c (n = 100).

At institution A, we did not observe any fragments in the drip infusion set. At institution B, rubber fragments were found in 4 of the 100 bottles of contrast, although none of these fragments entered the drip infusion set tubes. We noted that the same nurse had set up the 4 drip infusion sets that contained the rubber fragments.

Almost all bottles of contrast material have a rubber cap that is penetrated by the drip infusion set needle when administered, so that the possibility always exists that rubber fragments could enter the bottle or the needle core. When the possibility becomes a reality, what are the causes? A number of factors, either alone or in varying combinations, appear to have relevance, including (a) the type of needle used, (b) the type of rubber cap used, (c) the manner in which the needle pierces the rubber cap, (d) the technique of the person administering the drip infusion, (e) the penetrating speed of the needle piercing the rubber cap, and (f) the rotational force of the needle piercing the rubber cap.

The set (JMS drip infusion dry #200, Japan Medical Supply, Hiroshima) we currently use has a needle with a side hole to prevent "coring," but fragments have still been observed in the bottles. Most tubes are now equipped with a filter (40 to 50 μ) that prevents the injection of foreign material into the vascular system; however, drip infusion sets without filters are still commercially available. A severe complication could result if a system without a filter is used for administration of contrast or flushing of catheters in conventional angiography or, especially, in neuroangiography. We strongly recommend using a drip infusion set with a filter to guard against the possibility of such complications.

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Radiculopathy versus Referred Pain in Diskography

I would like to take exception to the terminology used by Nathan H. Lebowitz in his commentary, "Diskography for the Diagnosis of Radiculopathy without Nerve Root Compression" (1). Radiculopathy or radicular pain must be distinguished from referred pain or pseudoradicular pain (2). Radicular pain is sharp and often intense, radiat-

ing from the central position to some part of the lower or upper extremities. Coughing and sneezing or straining will often evoke this sharp pain. Also, any maneuver that stretches the nerve will provoke the pain. The pain must follow the distribution of the root, and paresthesias or sensory loss in the dermatomal distribution of the root usually accompanies radicular pain. Reflex loss, weakness, and/or muscle wasting are common findings in true radiculopathy. Referred pain in general has a more aching quality, does not as a rule project much below the knee, and is not accompanied by neurologic change other than vague numbness without demonstrable sensory impairment.

The radiating pain that Millette et al described in their article "Radiating Pain to the Lower Extremities Caused by Lumbar Disk Rupture without Spinal Nerve Root Involvement" (3), as mentioned in their discussion, is almost certainly referred pain and not true radicular pain, although because there are no neurologic findings described or, presumably, neurologic examinations performed, it is difficult to tell for certain. To conclude as Dr Lebowitz has that "this study supports the existence of a syndrome in which radiculopathy is caused not by compression of a nerve root, but rather by morphologic abnormality of the intervertebral disk, most commonly an annular tear," without the authors' documenting the neurologic findings in their patients is in error. This error of confusing referred pain with radicular pain is common in clinical practice and probably leads to many unnecessary lumbar spine surgeries. As far as the role of diskography in patients with back and leg pain, as mentioned in the commentary, there is no known proved treatment for patients who have back and leg pain and who have other imaging studies that do not demonstrate nerve compression, so one must wonder who the diskography is benefitting, the patient or the diskographer.

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Reply

We appreciate Dr Olivero's interest in our article. Since his main comments are directed to Dr Lebowitz, we will let him justify his use of the word *radiculopathy*. As for us, we certainly agree with Dr Olivero that it is a common error to confuse referred pain with radicular pain and, oddly

enough, one of us has recently written a letter to the editor of another journal, stressing the importance of making this distinction (1). The "Discussion" portion of our paper, as Dr Olivero appreciated, indicated clearly that the patients in our series presented referred pain and not radicular pain. We take exception, however, to Dr Olivero's statement that referred pain does not "as a rule" project much below the knee: it has been our experience that more than 50% of patients with referred pain to the lower extremities caused by a ruptured lumbar disk describe pain extending down to the calf or foot.

We disagree with the final statement in Dr Olivero's letter suggesting that because there is no known proved treatment for those patients, diskography benefits more the diskographer than the patient. Diskography is a time-consuming and tedious procedure, unpleasant for both patient and diskographer. In Quebec, radiologists receive from the universal health care system an honorarium equivalent to US \$90 for performing a four-level diskogram, a procedure that generally takes about 1 hour of the physician's time, and extra billing is prohibited. Not exactly a gold mine! Instead of being exposed to scattered radiations, sweating under a lead apron trying to get needles in the right places while trying to reassure a terrified patient, it would be less arduous and more financially rewarding to sit back in a comfortable chair reporting CT or MR studies.

We have acquired exceptional expertise with diskography, despite the fact that very little spine surgery is performed in our institution, precisely because radiologists have no incentive to learn this technique, and we get referrals from all over a province of 7 million people. We strongly believe, as we have stated in our article, that this procedure should be reserved for exceptional problematic chronic cases. Some patients are referred to us by spine surgeons, who are considering the feasibility of a lumbosacral fusion, in order to determine how many disks are diseased, and some are referred by psychiatrists for a trial of intradiskal and anterior epidural steroid injection, when all other conservative treatments have been ineffective. Most patients, however, are referred to us because previous imaging studies, including MR, have been unsuccessful in demonstrating the cause of their symptoms.

Diskography is more sensitive than MR to radial annular tears, which are very often responsible for chronic low-back pain with referred pain to one or both lower extremities. In our experience, MR fails to suggest any abnormality in 15% of these patients. However, in the majority of patients MR T2-weighted studies show that some disks have lost their normal signal, with or without bulging of their posterior outline. Unfortunately, these disks are qualified in most radiologic reports as "degenerated bulging disks," and the patients are told that these are trivial, part of the normal aging process, and do not explain their low back pain—certainly not pain down the leg, since there is no evidence of nerve root compression. These patients are then told that, because no "herniated disk" has been shown, they must have a simple sprung back, which should resolve in a few weeks. Unfortunately, because a

chronic inflammation process often results from annular tears (2), these patients frequently cannot resume their previous work activities, and will have relapsing symptoms for years, often resulting in litigation with worker's compensation boards or private insurance companies. They become quite frustrated because their complaints are not taken seriously. Even though diskography is an unpleasant and often quite painful procedure, these patients are always very grateful when they are told that the cause of their symptoms has been identified by an objective test, proving that they are not hypochondriacs or malingerers. Referring physicians tell us that establishing a clear diagnosis, and explaining to those patients the exact nature and prognosis of their lesions, is the best way to stimulate their cooperation in a rehabilitation program including proper work reinsertion. The conviction that diskography serves a useful purpose for these patients has given us the necessary motivation to carry on over the years, despite the major controversies surrounding the use of this procedure.

Correlation of diskography with MR images should make radiologists realize that their reporting of lumbar MR studies is often inadequate, underestimating the potential clinical importance of certain observations. We are hoping that, in the near future, a better understanding of disk pathology and real significance of MR images will improve the anatomic accuracy and clinical usefulness of MR reports in such a way that there will be fewer indications for diskography.

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Reply

I thank Dr Olivero for his comments, and accept his criticism of my use of the word *radiculopathy*. A more precise phrase (but rather awkward) would have been "pain similar and sometimes indistinguishable from the pain of radiculopathy." There is no disagreement about the clinical features of patients with nerve root compression. Indeed, dermatomal distribution of pain and sensory abnormality, reflex and motor loss, and exacerbation by nerve stretch and Valsalva's maneuver are all features of true nerve root compression. Similarly, many patients with lumbar spine abnormalities present with referred pain to the buttock and thigh, without abnormalities on neurologic

exam. There is little confusion in these clinical situations. However, not all patients with true nerve root compression in the lumbar spine present with pain going all the way to the foot. Similarly, a patient sometimes reports pain radiating below the knee in a dermatomal distribution, but has no radiographic evidence of root compression (1), and no neurologic abnormality on physical examination. It is in these patients that disk injection will often reproduce their extremity pain. Milette et al and others have shown that a common morphologic feature in this circumstance is an annular tear.

Milette et al included all patients with pain radiating below the gluteal fold in their study. No data were presented to determine the percentage of patients with pain in the thigh as compared with those with pain radiating as far as the foot. The text refers to patients with "radiating pain to the leg," "sciatica," and "radiating pain to the buttock, thigh, lower leg, and foot." Using the term *radiculopathy* to describe these symptoms is perhaps incorrect, but the message of Milette et al's paper should not be lost in this semantic controversy. Radiating symptoms in the lower extremity, sometimes mimicking the pain of nerve root compression, can occur without nerve root compression. In these patients, disk injection can reproduce symptoms of radiating pain, and annular tears are commonly found.

I agree with Dr Olivero that the treating physician must differentiate between radiating pain caused by nerve root compression and radiating pain from other causes. Management and prognosis differ dramatically in these two groups of patients.

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Comment

Dr Olivero makes an important point in his letter about the difference between radiculopathy and referred pain. He is absolutely correct. The comment is unnecessary in the context of the original manuscript, however, because Milette et al repeatedly state that the pain they observed was referred, not radicular, indicating they know the difference. Dr Lebowitz may have been somewhat casual in his terminology, but terminology is really of secondary importance with respect to the article and commentary. The issue at hand is the usefulness of diskography. The usefulness of any test is determined by its sensitivity, specificity and predictive value. Calculations of these require a standard of reference. This is precisely the problem. There is little

disagreement that diskography is an excellent test to determine the morphology of the disk, although its accuracy has not yet been determined. This determination as such is of no clinical significance, because morphologic changes and pain are not directly related. Diskography does not compete with CT or MR in the diagnosis of a disk herniation, nor is it clinically used to determine the various patterns of disk degeneration that occur in both symptomatic and asymptomatic persons. For these reasons, the reproduction of pain is considered the key to determine whether diskography is positive or negative (1). The importance of pain reproduction is appropriately made by Milette et al. When using the test, adjacent disks must be injected and should not reproduce the patient's usual pain (2). Why the pain occurs is not entirely clear. However, it is clearly established that it is not caused by increased pressure on nerve roots nor by a chemical effect, because it occurs even when contrast material does not leak out of the disk. It has been argued that the pain is caused by a so-called internal disk disruption, defined as a radial fissure extending to the well-innervated outer third of the annulus. It is hypothesized that the annulus has been sensitized by the degenerative process and therefore responds to mechanical stimulation. This concept is clinically unproved. Indeed, it would seem impossible to prove. Vanharanta et al (3) did attempt to study this and reported that the size of the radial tear was a factor of importance in the reproduction of pain, but in only two thirds of disks. Because morphologic changes, in which determination of sensitivity, specificity, and predictive value against a standard of reference would be possible, are of no importance, how can we test the usefulness of diskography at all? Surgery provides no absolute standard of reference.

Clearly, we need to apply to diskography the measures we apply to all other tests. In other words, we need to determine its validity and utility through strict scientific evaluation. How we do this is the true determination. Let us not get lost in terminology, but focus on this important issue so that we can finally determine the place of diskography in clinical practice.

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Dangers of Endovascular Treatment of an Unusual Carotid Cavernous Fistula

We read with interest two recent articles (1, 2) describing arteriovenous fistula between the posterior communicating artery and the cavernous sinus, caused by head trauma. It is rare for the intradural cerebral arteries to have a communication with the cavernous sinus (3–5). We have also reported an arteriovenous fistula between the intradural internal carotid artery and the cavernous sinus; it had been first misdiagnosed as a direct carotid cavernous fistula (6). It was treated with transarterial balloon occlusion, which resulted in rupture of the pseudoaneurysm formed at the internal carotid artery.

We would like to point out that this type of fistula, either at the internal carotid artery or at the proximal portion of the posterior communicating artery, is formed by a pseudoaneurysm without the normal arterial wall structure. Although Kinugasa et al elegantly occluded the fistula, endovascular treatment to occlude the fistula, either with balloons or coils, can cause rupture of the pseudoaneurysm. Thus, the treatment of choice for this type of traumatic direct arteriovenous fistula is direct surgery to exclude the pseudoaneurysm and occlude the fistula (3–5). However, if this is not feasible, endovascular treatment might be the alternative. Even if endovascular occlusion of the fistula without exclusion of the pseudoaneurysm is carried out, there remains a chance that the pseudoaneurysm may bleed or enlarge to behave as a mass lesion.

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Reply

We thank Drs Komiyama and Yasui for their comments on our article. As they point out, we also thought about the possibility that this type of fistula might be formed by a

pseudoaneurysm without the normal arterial wall structure. However, we could not find such a pseudoaneurysm on MR imaging before intravascular surgery. Thus, we only performed occlusion of the fistula by coils, as shown in our report. We think that the orifice of the fistula of this case existed on the lateral wall of the cavernous sinus.

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Reply

The central theme of our article was of early recognition of a traumatic pseudoaneurysm of the posterior communicating artery to cavernous sinus fistula. However, a considerable amount of debate existed regarding appropriate therapy. It was both our opinion and that of our neurosurgeons that surgical intervention with clipping of the posterior communicating artery anterior and posterior to the aneurysm was the most appropriate therapy. A major concern was the uncertainty of the structural integrity of the aneurysm wall, coupled with the potential additional stress of hemodynamic changes during the endovascular procedure. This case was also presented at the Interventional Neuroradiology Conference in Wyoming. There it was also the informal consensus of opinion to proceed surgically. The subsequent surgery was not easy and a clip could only be placed occluding the posterior communicating artery anterior to the aneurysm.

A second surgery was planned to clip the posterior aspect of the posterior communicating artery. The patient, who had some decrease in her massive exophthalmos, declined further intervention. Consideration was also given to closing the fistula from an endovascular venous approach with coiling of the cavernous sinus. Because after surgery there is now only one very small pathway through the aneurysm, it was expected that with cavernous sinus coiling the aneurysm would also soon thrombose. The patient has also declined this procedure, but has only minimal exophthalmos 17 months after surgery.

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Comment

I have read with great fascination the letter from Drs Komiyama and Yasui warning of the dangers of treating a carotid cavernous fistula where the connection occurs from the supraclinoid carotid or posterior communicating artery and the cavernous sinus. Their own experience with an acute connection involving the supraclinoid carotid artery that had massive bleeding after endovascular treatment is compelling evidence of the dangers involved.

In our own experience with the treatment of more than 300 direct carotid cavernous fistulas we have not yet encountered such an unusual case. Therefore, our comments are based on extrapolation of experiences gained with pseudoaneurysms in other locations. We would certainly agree that if an embolic device is delivered into an acute pseudoaneurysm, especially one involved in a fistula, that there is a great risk of enlargement and rupture of the pseudoaneurysm. If a pseudoaneurysm associated with a supraclinoid connection is discovered in the acute phase, surgical therapy may be indicated. Pseudoaneurysms involving the cavernous sinus with a typical carotid cavernous connection can be and have been treated with detachable balloons with great success, in our experience. Because of the risk of fatal subarachnoid hemorrhage, pseudoaneurysms should be occluded as soon as they are discovered (1).

If a period of time has passed since the initial injury and the discovery of the pseudoaneurysm, usually at least 4 to 6 weeks, then the wall of the pseudoaneurysm may have matured enough to permit the safe delivery of an embolic device without the fear of rupture. It is therefore not surprising that Kinugasa and his associates were successful at transvenous coil embolization of a fistula between the posterior communicating artery and the cavernous sinus that was discovered 1 month after the injury. Tytle and his colleagues reported a similar fistula in the same location presumed to be from an injury that occurred 31 years earlier and caused clinical symptoms for at least 13 years. Surgical treatment, which Dr Komiyama and his colleague recommend in their letter for the treatment for this condition, failed to obliterate the connection. I suspect that transvenous coil occlusion would have been successful in this unusual fistula. We share Drs Komiyama and Yasui's concern about endovascular treatment for acute pseudoaneurysms in this unusual location, and their opinion regarding the need for prompt closure with surgical intervention. If the false aneurysm escapes detection and a sufficient time has passed, then endovascular occlusion is a viable alternative.

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MR Methods of Measuring Changes in Brain and Cerebrospinal Fluid Volume with Age and Menstrual Cycle

We read with interest the paper by Blatter et al, "Quantitative Volumetric Analysis of Brain MR: Normative Database Spanning 5 Decades of Life" (1). This appears to corroborate our own work to quantify change of brain and cerebrospinal fluid volume with age (2) which, though based on MR, used a very different nonplanimetric approach (3). We also used intracranial cavity volume as a normalizing factor for brain size. We found mean brain loss per decade to be 1.6% in men and 0.5% in women. These figures are comparable to those of Blatter et al, 0.92% and 0.8%, respectively, for the range of 25 to 55 years.

We would raise one word of caution about any comparison of individual female volumetric measurements with the normative data Blatter et al provide. A study we performed (4) showed that total intracranial cerebrospinal fluid volume in premenopausal female subjects increased before menstruation by 11.5 mL over a mean midcycle value of 101.3 mL ($n = 20$, $P < .0001$). Bearing in mind that skull cavity volume does not change during this period, the implication based on the modified Monro-Kellie doctrine (5) is that the volume of the brain must decrease in a commensurate fashion to compensate.

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Reply

We appreciate Drs Condon and Hadley's pointing out the agreement between our recently reported normative database for brain volume and their previous work. We are also aware of another recent MR-based study (1) showing an age-regression slope for total brain volume nearly identical to what we reported. Having multiple MR-based volumetric studies that are not only internally consistent but also show a high degree of correlation with large autopsy

studies (2) increases our confidence in the MR-based techniques.

Drs Condon and Hadley also refer to previous work comparing measurements of total intracranial cerebrospinal fluid volumes at two different points in the menstrual cycle in premenopausal female subjects. We have read this study with interest. Because we did not question the healthy female volunteers about their menstrual history, the data we obtained can neither directly corroborate nor refute Drs Condon and Hadley's conclusions.

However, in light of our data, two points are worth noting relative to the magnitude of any possible intracycle change in brain volume. First, if a significant intracycle variation in total brain volume occurred in premenopausal females, we would expect to observe a greater standard deviation of that volume measurement among female subjects in those decades. In fact, the standard deviation observed in the groups of female subjects in the 3 decades from 16 to 45 years was not different from that in the corresponding groups of males. Further, rather than a decrease in the standard deviation after menopause, we

observed an increase in the 55-to-65 decade. Second, the magnitude of the intracycle variation reported by Drs Condon and Hadley less than 1% of total brain volume. Because this possible variation represents only one third of one standard deviation in the measurement *after* correcting for population differences in total intracranial volume, we are doubtful that it poses a significant source of error.

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