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MR field strength: unanswered questions.

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LETTERS

MR Field Strength: Unanswered Questions

With the increasing pressure for cost containment in health care, big-ticket items such as magnetic resonance (MR) are getting attention. Can 0.5-T state-of-the-art systems be as clinically efficacious as 1.5-T systems? It is therefore worthwhile to compare contrast enhancement between the field strength as was discussed in the *AJNR* article "Effect of Dose and Field Strength on Enhancement with Paramagnetic Contrast Media" (1).

Such a study requires a fair comparison. First, T1 relaxation times differ at 0.5 T and 1.5 T. Should not the authors have first selected the optimal repetition time for each field strength and then made the comparison? Second, the echo times used were not comparable. In "Methods," the authors state that the echo times ranged between 10 and 20 milliseconds. In fact, the clinical cases at 0.5 T from Figures 1 and 2 are annotated as 25 milliseconds. Were the echo times actually set for 25 milliseconds at 0.5 T because of the gradient limitations of that system but were 10 to 20 milliseconds on their 1.5-T system?

It is currently very difficult to make a truly valid comparison between field strengths, because many other hardware differences exist even within the same vendor's systems. In addition to more powerful gradients often being available on 1.5-T systems, radio frequency coils, software, and so forth, also generally differ.

Based on my own experience with different field strengths and vendors, there will continue to be some applications for which high-field systems will significantly outperform middle- and particularly low-field systems. The issue of contrast enhancement and image contrast's dependency on field strength remains an unanswered question in my mind.

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Reference

1. Lindsey RO, Yetkin FZ, Prost R, Haughton VM. Effect of dose and field strength on enhancement with paramagnetic contrast media. *AJNR Am J Neuroradiol* 1994;15:1849–1852

Reply

We appreciate Dr Weiner's interest in the study that we reported. Dr Weiner suggests in his letter that: (*a*) the clinical efficacy of MR imagers of different field strengths should be compared, (*b*) the comparison requires optimization of techniques at each field strength, (*c*) the hardware differences between scanners in our study account for some differences we observed, and (*d*) our paper does not answer the issue of contrast enhancement dependency on field strength.

The clinical efficacy of 0.5 T versus 1.5 T was not our focus. Readers interested in a comparison of clinical efficacy are referred to the results of a study in progress by Brian Rutt, MD, of the University of Western Ontario. Two Signa scanners with different field strengths are compared.

Choosing optimal techniques for comparing two scanners at different field strengths is not trivial. For example, the selection of repetition time involves more than merely an adjustment for the T1 relaxation times, which vary widely with field strength. In practice, tradeoffs are made between repetition time, number of sections, receiver bandwidth, and echo time. The number of sections obtained for a specific repetition time is related to the bandwidth, sampling time, and signal-to-noise ratio. The limitation in number of sections is more severe at 0.5 T than at 1.5 T, given the intrinsically decreased T1 dispersion at 0.5 T. Dr Weiner advocated that in our imaging at 0.5 T, repetition time could be shortened below the times that we used to improve gray-white matter contrast. Although shorter repetition time may improve tissue contrast, signal-to-noise ratio would be adversely affected, and conspicuousness may be decreased. Echo time also was selected to optimize image quality. The 25 milliseconds echo time was chosen for the clinical images to minimize a reconstruction artifact that arises from the nonhermitian nature of k-space in fractional-echo imaging at lower receiver bandwidths.

The hardware and software for the 0.5-T Signa and the 1.5-T Signa systems are well known to one of us (R.W.P.), lead designer of the original 0.5-T Signa system. The gradients in both systems are identical, as are the transceivers, host computers, software, operator interfaces, and coil geometries. The suggestion that the two scanners differ in their gradient and radio frequency coils is incorrect.

Our objective was to emphasize the relationship of contrast enhancement to T1:

$$T1_{observed} - T1_{tissue} = (T1^{tissue})^2/(T1_{contrast\ medium} + T1_{tissue})$$

Our measurements at 0.5 T and 1.5 T are consistent with the formula. The clinical examples selected appear to be consistent with the formula as well. The relationship between field strength and contrast enhancement is well established on theoretical and empirical grounds. (The suggestion of Scott Rand, MD, PhD, to derive the formula used in this letter from the formula contained in the original paper is gratefully acknowledged.)

Dr Weiner's letter raises important questions. We have attempted to clarify how our paper relates to those questions.

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Editor's note.—*Dr Weiner's letter was forwarded to Dr Elster for further comment.*

Comment

In his letter to the Editor, Dr Weiner addresses a wide assortment of issues concerning image quality, cost-effectiveness, and contrast enhancement in low-field versus high-field MR systems. In only a few paragraphs allotted here, I cannot possibly provide a coherent analysis of the many factors involved in deciding, "Do we buy a 1.5-T or a 0.1-T scanner today?" I will address and defend, however, the fundamental premise of the article by Lindsey et al, that contrast enhancement of brain lesions is directly related to field strength, with less apparent enhancement at low field than at high field.

Having worked on a variety of MR systems over the years (with field strengths of 0.15 T, 0.35 T, 0.5 T, 1.0 T, and 1.5 T), I admit to being a "high-field aficionado." I fully understand and agree with Dr Weiner, however, that field strength by itself is only *one* determinant of overall image quality (just as engine horsepower is only one determinant of an automobile's performance). Nevertheless, my specific opinions about field strength and gadolinium enhancement are based neither on "personal preference" nor on the recent paper by Lindsey et al; they are based on established scientific principles and tissue relaxometry experiments that transcend the sales pitches of MR vendors.

I have recently reviewed these established biophysical principles and empirical data about the field-strength dependence of gadolinium and tissue relaxation times in an *AJNR* commentary (1). Without repeating the complete explanations here, I will list the basic conclusions once again: (*a*) The T1 relaxation time of the brain is dependent on field strength. (*b*) The relaxivity of gadolinium also depends on field strength, but in a fashion differing from that of brain. (*c*) The relaxation time of {gadolinium + brain} is a nonlinear function of field strength, with a fractionally greater effect at high field than at low field. Again, I must stress that these conclusions follow from basic theory confirmed by carefully performed laboratory investigations at multiple institutions; they are general principles of biophysics whose truth transcends anecdotal clinical experience.

Would a different choice of imaging parameters have produced a less obvious discrepancy between the high-field and low-field images? Possibly. But I sincerely doubt that merely by adjusting repetition time, echo time, or field-of-view, the contrast enhancement on the low-field images would have proved superior to those of the high-field study. Of course, this is not to say that "optimal" pulse sequences for the detection of gadolinium enhancement have yet been developed for MR scanners of *any* field strength. For example, we have demonstrated recently how the use of magnetization transfer suppression techniques can dramatically increase the conspicuity of contrast enhancement in a variety of diseases, sometimes revealing enhancement where none was seen before (2, 3). Other differences between specific MR scanners, including gradient strength, homogeneity, and coil design, also may have an effect on the degree of contrast enhancement observed.

As a final argument in support of my position, it should be noted that besides Lindsey et al, two other groups of investigators have come to similar conclusions about field strength and gadolinium enhancement. Chang and colleagues (4) recently compared contrast enhancement in 31 patients with brain tumors as a function of dose and field strength. For both single-dose and double-dose studies, the lesion-to-brain contrast-to-noise ratios were nearly a factor of 2 higher at 1.5 T than at 0.5 T. Similar conclusions also were drawn by Prager et al (Prager J, Rosenblum J, Kin D, et al, "Field Strength and Gadolinium Enhancement in the Clinical Setting," *Symposium Neuororadiologicum (XV) Proceedings,* Kumamoto, Japan: Springer-Verlag, 1994;552–553), who measured enhancing cranial lesions at 0.1 T, 0.3 T, and 1.5 T.

In conclusion, although one may argue the advantages and disadvantages of high-field versus low-field instruments ad nauseam, I firmly believe that the biophysical principles I have outlined are correct and place fundamental limits on the detectability of contrast enhancement as a function of field strength.

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Flow-Related Enhancement in the Vertebral Plexus Mimicking an Intramural Hematoma

MR has proved useful in the diagnosis of vertebral artery dissection (1–3). Lévy et al (2) reported a specificity of 98% for MR in diagnosis of vertebral artery dissection. Vertebral dissection is characterized by a narrowed eccentric signal void surrounded by a semilunar signal hyperintensity. The narrowed signal void corresponds to the residual lumen surrounded by the mural hematoma (1). False-positive results of MR are attributable to misinterpretation of the high-signal intensity of veins and fat with intramural hematoma at the craniocervical junction (2) or to intraluminal flow-related phenomena in the vertebral artery (1). We report here another possible false-positive of MR attributable to misinterpretation of flow-related enhancement in the vertebral venous plexus surrounding an occluded vertebral artery.

In a 39-year-old woman with a left cerebellar infarction, the digital angiogram (Fig 1) demonstrated an occluded left vertebral artery. Axial spin-echo T1-weighted (500/12 [repetition time/echo time]) MR images, 5-mm thickness, 1-mm intersection gap, were obtained from the level of the first thoracic vertebra to the level of C3-4, and from the level of the fifth cervical vertebra to the level of the skull base in two separate multisection acquisitions. The acquisition matrix was 256 × 256, for a field of view of 200 mm. A saturation pulse was placed inferiorly to suppress arterial inflow signal.

On the two first sections of the lower multisection stack (Fig 2A), at the level of C3-4, the left vertebral artery showed a narrowed eccentric signal hypointensity surrounded by a semilunar signal hyperintensity that could correspond to a residual lumen surrounded by a mural hematoma. On the lower sections, there was no "mural hematoma." The foramen transversarium was normal in size. On the corresponding images of the higher multisection stack (Fig 2B), there was no "mural hematoma," but a concentric slightly higher signal than in the center of the artery. This semilunar signal hyperintensity present on the two first sections of the lower multisection stack could be interpreted as a flow-related enhancement in the vertebral venous plexus that surrounds the vertebral artery in the transverse canal (4). There also was flow-related enhancement in the cerebrospinal fluid (CSF) of the posterior subarachnoid spaces. Flow-related enhancement (entry section phenomenon) is commonly observed with slow flow (5) and may appear in veins and in the CSF of the subarachnoid spaces, as in our case. It is observed for initially encountered sections of a multisection acquisition and decreases or is eliminated on sections deeper in the imaging volume. We supposed that the good visibility of the vertebral plexus on the left side was attributable to dilatation of this plexus secondary to the absence of flux in the occluded vertebral artery.

In conclusion, elevation of signal intensity in the periphery of an occluded vertebral artery might correspond to an intramural hematoma, but also could be attributable to a flow-related enhancement in the dilated concentric verte-

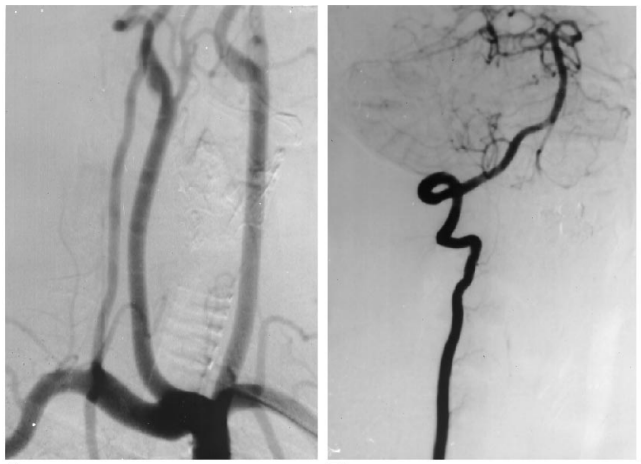

Fig 1. Digital substraction angiograms in a 39-year-old woman with a left cerebellar infarction.

A, Digital substraction aortic arch angiogram demonstrates absence of opacification of the left vertebral artery.

B, Digital substraction right vertebral angiogram shows retrograde filling of the distal part of the left vertebral artery and posterior inferior cerebellar artery.

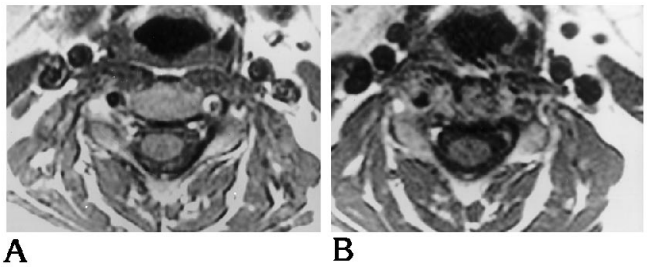

Fig 2. T1-weighted axial spin-echo MR images at the level of C3-4, originating from two multisection stacks acquired with a saturation pulse placed inferiorly.

A, In this case of angiographically demonstrated vertebral occlusion, the first section of the lower multisection stack demonstrated normal flow void in the right vertebral artery, but the left vertebral artery showed a narrowed eccentric signal hypointensity surrounded by a semilunar signal hyperintensity that could correspond to a residual lumen surrounded by the mural hematoma.

B, On the corresponding image of the higher multisection stack, there was no "mural hematoma," but a concentric slightly higher signal than in the center of the artery. The semilunar signal hyperintensity present on the first section of the lower multisection stack could be interpreted as a flow-related enhancement in the vertebral venous plexus that surrounds the vertebral artery in the transverse canal. Note also a flow-related enhancement in the CSF of the posterior subarachnoid spaces.

bral plexus, as demonstrated in this case. To eliminate this phenomenon, it could be useful to use a superiorly placed saturation pulse to negate venous flow. Another possibility, as was done in our case, is to obtain an additional multisection acquisition positioned such that the lower sections of the second sequence overlap the entry sections of the first, to eliminate flow-related enhancement in these sections.

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Editor's note.—*The letter of Dr Miaux was referred to Michael Brant-Zawadzki for his comments, which follow.*

Comment

I have reviewed the letter by Dr Miaux et al, and find that the case presented raises some perplexing issues. First of all, this 39-year-old woman with a left cerebellar infarction in whom an occlusion of the left vertebral artery was demonstrated angiographically may well have suffered a vertebral artery dissection leading to complete occlusion of the vessel. In that case, if this is a correct assumption, the question of why the vertebral artery lumen appears low in signal intensity is raised. This may be because of the conversion of oxyhemoglobin to deoxyhemoglobin in an acutely occluded, dissected vertebral artery. If so, the case does not truly represent a "false-positive."

Second, the discrepant signal intensity in the left foramen transversarium at the C3-4 level on the two separate acquisitions may have several explanations. The author of the letter is correct in suggesting one potential reason: the flow-related enhancement produced by unsaturated blood entering the venous plexus surrounding the vertebral artery. It is interesting to note that according to the case report, the T1-weighted acquisition started inferiorly, and proceeded superiorly. One might expect the greatest flow-related enhancement to appear in end sections of a countercurrent acquisition, which may be the case here if one assumes cephalocaudad direction of flow in the venous plexus. Thus, this explanation is quite valid. Other possible causes of discrepancy would be asymmetric signal of the fat in the foramen transversarium, caused by coil positioning and centering. This is clearly a less likely explanation. It would have been interesting to test the likely explanation of flow-related enhancement by administering intravenous gadolinium. Clearly, in some cases differentiation of hematoma from fat or flow-related enhancement can be quite difficult. Fat-saturated techniques and paramagnetic intravenous contrast can help resolve these difficulties in most instances of persistent indecision.

In any case, the most interesting issue in this case still is the explanation of the small but hypointense vertebral artery itself and the cause of the entire syndrome. Nothing in this case excludes dissection as the underlying cause of the clinical and MR presentation, thus the term "false-positive" may not apply.

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Encephalopathy with Bilateral Thalamotegmental Lesions? Japanese Encephalitis

We read with great interest the paper "Acute Encephalopathy with Bilateral Thalamotegmental Involvement in Infants and Children: Imaging and Pathological Findings" by Yagishita et al (1) in the March 1995 issue of *AJNR*. In this report, five children presenting with fever, vomiting, convulsion, altered sensorium, decerebration, or decortication were reported. This clinical picture is consistent with an encephalitic syndrome. The computed tomography scan of these patients revealed bilateral thalamotegmental hypodense lesions, and MR showed hemorrhagic changes in these areas. The authors postulate a postviral or postinfectious encephalopathic illness. To us, these clinical and radiologic features appear characteristic of Japanese encephalitis (2). CSF was abnormal in one patient only, and revealed 140 mg/dL protein. None of the patients had CSF pleocytosis in their study. A normal CSF, however, does not exclude viral encephalitis (3). In our study of Japanese encephalitis, CSF was normal in one of six patients in the acute stage (2). The authors do not indicate the timing of CSF examination in their study, which may influence the CSF results. Although influenza antibody titers have been reported, there is no mention of Japanese encephalitis antibody titers, which is more important in their patients. In the autopsy study, the lesions corresponded to the area of involvement in Japanese en-

cephalitis. Lack of cellular response in an autopsy specimen has been taken as a point against encephalitis. On the contrary, the commonly encountered histopathologic feature of Japanese encephalitis is almost complete absence of accompanying cellular and exudative response in the brain. In the leptomeninges the cellular reaction is not prominent (4). Although the incidence of Japanese encephalitis has declined substantially in Japan, patients with Japanese encephalitis continue to be reported (5). The authors discuss the differential diagnosis of bilateral thalamotegmental lesions at length, but the discussion on Japanese encephalitis is conspicuously missing. We also note that the authors did not refer to our recent paper with identical clinical and radiologic features (2), and earlier Japanese studies have been omitted (6, 7). In our opinion, the authors should investigate the possibility of Japanese encephalitis in their patients.

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Reply

We appreciate Dr Misra and Dr Kalita's interest in our report. It was important to know that MR images of patients with Japanese encephalitis showed T1 shortening in the thalami. However, we could not refer to their report, because it was published after submission of our manuscript. Acute encephalopathy that we have reported differs from Japanese encephalitis in the following respects.

First, clinical conditions are different. Japanese encephalitis is now rare in Japan and almost always occurs in summer, particularly in the period from early August to early September, because mosquito populations become large and invade residential areas only in summer (1–4). In contrast, the encephalopathy in our study occurred in the period from March to September. It was reported that the onset of the encephalopathy most often was in the winter (51% in December through February) (5). Infections of Japanese encephalitis in Japan occur much more often in the southwestern areas than in the northern areas (1, 4), whereas such regional difference in the incidence of the encephalopathy was not found. CSF pleocytosis was not obtained in any of 26 patients with the encephalopathy (6). The examinations were performed in the acute stage of the illness. No increased antibody titers for Japanese encephalitis were observed. Laboratory studies of Japanese encephalitis reveal uniformly abnormal CSF findings: pleocytosis as high as 1000 cells per milliliter has been noted, and the CSF frequently does not return to normal for 7 weeks (7).

Second, pathologic findings are different. In the encephalitis, the most severely affected regions are the thalamus, globus pallidus, substantia nigra, and pontine nuclei (8). In contrast, this encephalopathy did not affect the substantia nigra or pontine nuclei (6). On the contrary, the pontine tegmentum was involved in the encephalopathy.

Finally, imaging findings are different. MR images of four patients with encephalitis did not show T1 shortening in the thalami in a Japanese report (2). Furthermore, abnormalities in the pontine tegmentum were observed in all five patients with the encephalopathy in our study (6). However, in the study of Dr Misra et al, pontine lesions were observed in only two of six patients, although they did not describe whether the lesions were in the pontine tegmentum (9).

In summary, it is our belief that the acute encephalopathy described by us is not Japanese encephalitis.

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Is Fast Spin-Echo Superior to Gradient-Echo Imaging in Detecting Spinal Cord Lesions. . .or Not?

Great emphasis has been placed on fast spin-echo sequences and their potential to replace conventional spin-echo and gradient-echo acquisitions in spinal MR (1) (Sze G, "Fast Spin-Echo Imaging in the Evaluation of the Spine, presented at the 30th Annual Meeting of the American Society of Neuroradiology, St Louis, Mo, May 30–31, 1992). Recently, we observed a case that made us less confident about this point.

A 31-year-old man presented in November 1994 with sudden numbness in both arms. MR was performed to rule out multiple sclerosis. A sagittal fast spin-echo sequence failed to show any definite abnormality in the cervical spine (Fig 3A). Because of an instructional misunderstanding, an unrequested sagittal gradient-echo (700/30) sequence was then performed and clearly demonstrated a large area of increased signal in the cord at the level of C-3 (Fig 3B). An axial gradient-echo sequence (700/25) performed at the level C3-4, confirmed the presence of a plaque centrally located in the spinal cord (Fig 3C).

We do not have an answer to suggest to explain the possible technical reasons of this finding. The machine we use is a General Electric Vectra 0.5-T unit, and the pulse sequences are those recommended by the manufacturer. Because we observed this case, we perform both sagittal fast spin-echo and gradient-echo sequences in patients referred for evaluation of spinal cord lesions. Actually, we have not observed (yet) any case similar to the present case; nevertheless, in no case have gradient-echo sequences failed to show lesions detected by fast spin-echo sequences.

Therefore, the original question still remains: is fast spin-echo superior to gradient-echo imaging in detecting spinal cord lesions. . .or not?

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Editor's note.—*See the August 1995 issue of AJNR, pages 1559–1560, for Dr Sze's reply.*

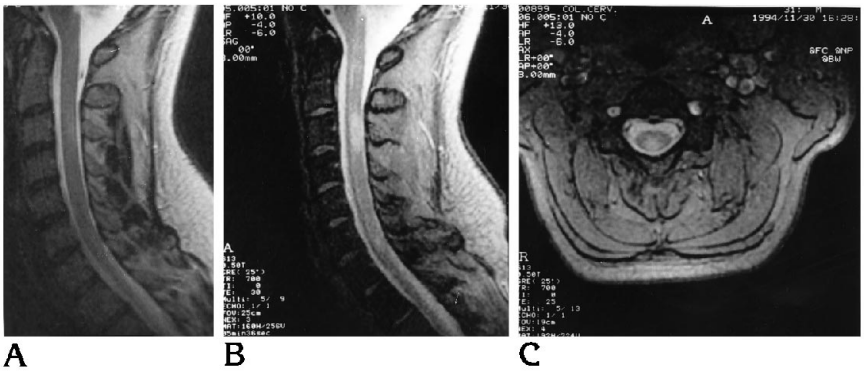

Fig 3. *A*, Sagittal fast spin-echo (4000/130; echo train, 10; echo spacing, 21 milliseconds) does not show any definite abnormality in the cervical spine.

B, Sagittal and *C*, axial gradient-echo images (700/30-25; flip angle, 25°) indicate the presence of a plaque centrally located in the spinal cord at the level C-3.

Neurovascular Conflict and Essential Arterial Hypertension: MR Evaluation

We read with great interest the article by Akimura et al (1). These authors investigated neurovascular compression of the ventrolateral low brain stem in essential hypertensive patients using MR. With the aim of detecting a neurovascular compression at the level of the root entry zone of cranial nerves IX and X, recently we used MR and MR angiography to investigate 30 young patients with essential arterial hypertension (group A) and 30 normotensive control patients (group B). In addition, we explored the neurovegetative pattern of both groups studying the short-term heart rate variability and the systolic arterial pressure variability in the frequency domain by power spectrum density at rest and after sympathetic activation (2–6). We performed the study in young subjects to reduce the incidence of abnormal arterial courses and loops caused by arteriosclerosis and prolonged hypertension. The results of our study were presented at the 10th World Federation of Neurological Societies Congress.

We agree with Akimura et al that MR is a useful method for the evaluation of a neurovascular compression, providing good spatial resolution and flow images simultaneously. Moreover, we believe that MR angiography can integrate the information provided by MR, showing the courses of vessels and revealing possible posterior fossa arterial loops. About 80% of our hypertensive patients showed neurovascular compression, in comparison with 30% of those in group B; this difference is statistically significant ($P = .0001$). Moreover, in accordance with the hypothesis of stimulation of the ventrolateral medulla by a neurovascular compression, with adrenergic and serotoninergic neuron activation and subsequent arterial hypertension, the markers of neurovegetative activity identified by spectral analysis showed significantly higher sympathetic activity in group A ($P < .0001$).

Contrary to previous observations (7, 8), in our series there was no evidence of left-sided dominance of neurovascular compression (about 55% right sided and 45% left sided), and we believe, in accordance with Akimura et al, that there is no neurophysiologic reason for a left-sided dominance.

It is presumed that a certain number of patients did not present neurovascular compression at the ventrolateral low brain stem, representing the true "essential" hypertensive patients. Moreover, it is advisable to follow clinically the asymptomatic cases with neurovascular compression (30% of our control group) because of the possibility of late onset of hypertension. We believe that larger prospective studies with MR and MR angiography and heart rate variability and systolic arterial pressure spectral analysis are necessary to confirm the neurovascular hypothesis of arterial hypertension. We also agree with Akimura et al that surgical correlation in selected cases is advisable to confirm the neuroradiologic and neurophisiologic findings.

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Reply

We appreciate the interest shown by Pierpaolo et al regarding our article. They also investigated neurovascular compression of the ventrolateral medulla at the level of the root entry zone of cranial nerves IX and X with MR, and with a larger control group, although they do not specify what sequences they used. Their findings are consistent with ours, although they found right-sided dominance of neurovascular compression in 55% of patients, whereas our results showed compression on the left side five times more often than on the right side. They also found signif-

icantly greater neurovegetative activity in the hypertensive group than in the control group; this result is probably related to activated vasopressor neurons in the hypertensive group. They evaluated younger patients, and we admit that it is better to do this kind of study with young subjects to exclude the factor of aging. However, we regard the duration of hypertension as another important factor, as mentioned in our article. We agree on the importance of MR angiography to reveal the looping of arteries in the vicinity of the root entry zone and rootlets of cranial nerves IX and X. Using three-dimensional fast low-angle shot imaging, we easily obtained MR angiography simply by applying the maximum intensity projection technique and could identify looping in some cases.

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