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Re: Assessment of Carotid Artery Patency on Routine Spin-Echo MR Imaging of the Brain

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Commentary

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In this issue, Lane et al. [1] provide additional data for the value of spin-echo MR imaging in assessing the patency of the internal carotid artery. A number of previous studies, including our own, have suggested that alteration of the normal signal void of the internal carotid artery should be viewed with suspicion [2–7]. The current study by Lane and his colleagues further categorizes the different types of signal alteration that may be encountered, specifically when the studies are performed with cardiac-gated spin-echo technique without the use of gradient-moment nulling (flow-compensation) software. These authors also verify our experience that a normal signal void may be present in the face of severe carotid stenosis (63% of their patients with stenosis greater than 90% exhibited normal signal void in the intracranial segment of the internal carotid artery).

Seemingly, only a minor discrepancy arises in the results of Lane et al. compared with our own. Namely, these authors place a high degree of confidence in the observation that a *completely* isointense signal within the intracranial carotid artery is indicative of atherosclerotic occlusion of that artery, whereas we reported two cases in which isointense signal was seen with severe stenosis but incomplete angiographic occlusion. Lane et al. explain the discrepancy in the different techniques employed. As noted earlier, their study was performed with cardiac gaiting instead of gradient-moment nulling to achieve reduction of flow-propagated phase-encoding artifacts (incidentally, the TR parameters quoted in their results obviously must have varied somewhat from the 600-msec and 2000-msec values given in their methods, as the heart rate would determine the actual TR setting in any given individual).

It is highly likely that complete isointensity of the intracranial carotid artery may correspond to complete occlusion of the carotid artery angiographically in a high percentage of cases. Nevertheless, I would hold out the caveat that this is not likely to be 100% accurate. In our own observations, a slow rate of flow was noted angiographically in a carotid artery with isointense MR appearance. Serial frames from the angiogram demonstrated that the contrast propagation from the distal common carotid artery to the carotid siphon took 14 sec. I would submit that such a slow rate of flow is insufficient to induce the flow-related alterations of signal intensity observed by Lane et al. in their case material. I cannot totally dispute the fact that the combination of saturation pulses and gradient-moment nulling can combine to produce isointensity simulating complete occlusion in a slow-flow state. I would also predict that there may be instances in which a cardiac-gated technique is used where a combination of pseudogating and slow flow might induce an isointense picture.

Nevertheless, these are somewhat speculative points and one must bring the discussion back to the realm of the practical. Lane et al. state that their objectives "were to determine whether angiography can be obviated in cases of suspected occlusion. . . ." They also state that "a reliable diagnosis of atherosclerotic occlusion can be made when isointense intraluminal signal is observed." In their discussion, they state "such an appearance can obviate invasive angiographic confirmation in the appropriate clinical setting." The

This article is a commentary on the preceding article by Lane et al.

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key phrase in all of this is *appropriate clinical setting.* I would suggest that any patient in whom transient ischemic attacks or fluctuating ischemic symptoms occur will undergo conventional angiography, even if isointense signal within the ipsilateral carotid artery is identified on MR. Such patients require evaluation of the entire cerebral circulation, including collateral flow, even if complete occlusion of the ipsilateral carotid artery is present. Therefore, the value of intracranial carotid artery isointensity in obviating conventional angiography wanes in "the appropriate clinical setting."

It might be pointed out that even the advent of MR angiography, with time-of-flight and phase-contrast techniques, may not allow us to completely obviate conventional angiography. It must be remembered that MR images of the vasculature relate strongly to *velocity* information rather than to true *anatomic* information of a contrast-filled lumen, which is provided by conventional angiography. These two phenomena are not equivalent. Even MR angiographic techniques have a threshold of flow velocity below which the observed signal intensity may simulate that of stationary hydrogen nuclei. The vagaries of MR artifacts at the skull base, effects from flow in the cavernous sinus, nuances of software manipulations such as gradient-moment nulling, saturation pulses, and so forth can befuddle the observer in any given case.

In summary, Lane and his colleagues have elegantly confirmed the observation that replacement of normal signal void in the intracranial carotid artery should be viewed with suspicion. They also verify that the presence of normal signal void does not exclude significant carotid disease. Finally, we would all agree that although completely isointense signal within the carotid artery most often indicates occlusion of that vessel, in the appropriate clinical setting conventional angiography should still be performed to verify the complete occlusion (as pseudoocclusion can be treated surgically) and to provide additional information regarding pathways of collateral flow and possible compromise therein.

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