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D. Dai and D.F. Kallmes

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Reply:

We greatly appreciate the correspondence from Meng et al regarding our recent article about the basilar bifurcation in rabbits following carotid ligation.¹ We remain absolutely confident that the focal excrescences along the basilar apex in our model represent branch arteries and not aneurysms. Dr. Meng and colleagues raised concern that de- and re-staining our histologic sections might have compromised the ability to identify the internal elastic lamellae. However, the article they cite regarding such loss of accuracy was for immunohistochemical techniques,² not the histochemical staining, Verhoeff Elastic-Van Gieson (VVG), used in our study. We have re-reviewed all of our slides in detail. There are no aneurysms.

We are grateful to Dr. Meng and colleagues for providing new histologic images in their letter, different from those published previously.³ We note with interest that in Fig 1A in their letter, the artery denoted as the basilar artery is much much smaller than the P1 segment (the first segment of the posterior cerebral artery). From our experience, we have found that it is very easy to lose track during histologic processing of which branch is the basilar and which are the P1s; as such, simple rotation of the slide by 90° would place the bifurcation of the superior cerebellar artery/P1 at the “basilar tip.” Thus, the “aneurysm” noted in their Fig 1 looks similar to the origins of superior cerebellar arteries in a subject from our lab (Fig 1).

Given the disparate results between our study and their previous work, we speculated in our article that the apparent aneurysms noted by them might in fact have been branch arteries. Certainly there may be other potential explanations for different results between our study and theirs. In any event, because they identify aneurysms in 100% of cases, it should be relatively easy for them to confirm, by using complementary methods in addition to serial histologic sections, that they have induced aneurysms. These complementary and potentially confirmatory techniques could include angiographic or micro-CT imaging,^{4,5} or casting techniques such Microfil perfusion (MTS Medication Technologies, St. Petersburg, Florida).^{6–9} In addition, we would assume that the microaneurysms induced in their model might, with time, grow to become large and thus intuitively obvious to all observers, including us.

Perhaps the most efficient way to move forward in clarifying the apparent disparity in conclusions between our 2 research groups would be to provide the histologic data to an independent vascular pathologist. We would be delighted to supply all of our serial histologic slides to such an expert in hopes of advancing this important field.

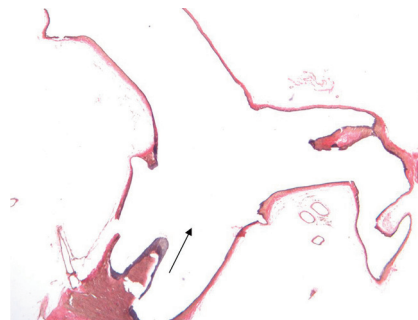


Fig 1. A subject at 8 weeks after right common carotid artery ligation in the elastase aneurysm model. Photomicrograph shows the basilar artery, superior cerebellar artery bifurcation, and the posterior cerebral artery at a low magnification. Arrow indicates the blood flow direction (VVG, original magnification $\times 40$).

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D. Dai

D.F. Kallmes

Neuroradiology Research Laboratory

Department of Radiology

Mayo Clinic

Rochester, Minnesota

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