REPLY

We thank Drs De Cocker and Hendrikse for their interest in our article "Cerebellar Watershed Injury in Children"[1] and acknowledge their extensive contributions to the present understanding of cerebellar ischemia, particularly of small cerebellar infarcts in adults.

Although the proximate pathophysiology of cerebellar hypoxic-ischemic injury in children and small-vessel thrombotic or thromboembolic occlusion in adults is likely quite different, there are many key points highlighted by the authors' research that serve to support the conclusions we make in our article.

First, the authors' study of the precise anatomic distribution of small cerebellar strokes highlights the propensity for predominantly cortical injury in cerebellar ischemia,[2] with relative sparing of the immediate subcortical white matter.

Second, their analysis of embolic infarct patterns supports the likelihood that the apices of the cerebellar fissures represent the end zones of perfusion of the penetrating cerebellar arterial branches.[3]

Third, the authors' use of superselective pseudocontinuous arterial spin-labeling to delineate vertebrobasilar end artery perfusion elegantly demonstrates the variability of the cerebellar vascular territories and their intervening borderzones.[4]

Taken together, the authors' conclusions support the supposition that cerebellar watershed injury would preferentially affect the foliar gray matter at the depths of the cerebellar fissures along the borderzones between the large-vessel territories. This pattern was exactly that observed in our cohort of pediatric patients. In addition, it helps to further validate the variable distribution of presumed watershed cerebellar injury in our series.

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Last, we appreciate the interesting notion that the authors raise regarding the possibility of greater overlap between the underlying pathophysiology of hypoperfusion injury and small embolic infarcts than is generally appreciated. Although the theory, as advanced by Caplan and Hennerici[5] and Caplan et al[6] is largely supported by examples of overlap in the setting of large-vessel steno-occlusive disease in adults, the importance of remaining open to the possibility of similar overlap in children is well-taken.

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