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Reply:

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Reply:

We greatly appreciated the insightful comments of Mariën et al regarding our retrospective case-control study on posterior fossa syndrome (PFS).¹ We are aware of and recognize the significant work and contribution of our colleagues to the domain. The authors of the letter rightfully pointed out that cerebellocerebral diaschisis (CCD) has previously been proposed as a likely explanation for the pathomechanism of PFS, mostly in the context of sporadic case studies of patients with PFS by using single-photon emission tomography (SPECT) studies. For example, in 1998, 2 case studies used SPECT to demonstrate reversible hypoperfusion in the frontal cerebral cortex in patients who developed PFS.² In 2001, this was reiterated in a single case study demonstrating the same phenomenon.³ The authors of the letter also pointed out a review study on cerebellar function that appeared to link PFS to a diaschisis phenomenon via right-sided cerebellar damage.⁴ There was also a talk at the 41st Annual Meeting of the Academy of Aphasia on this topic.⁵

It is reassuring that there appears to be a growing consensus in the literature regarding this matter. As mentioned previously, the idea of CCD as a potential cause of PFS has been previously proposed from the interpretation of case observations, but the phenomenon had not yet been scientifically tested. Because of our study design, we can quite confidently state that patients who developed postsurgical PFS had specific changes in cerebral blood flow (very likely global, but dominantly frontal hypoperfusion) compared with those who did not develop this syndrome and that patients who developed PFS had a specific damage pattern to the proximal efferent cerebellar pathways compared with those who did not develop the syndrome. Additionally, our study appears to be the first to use dynamic susceptibility-weighted contrast-enhanced perfusion MR imaging techniques to approach the matter. It is our hope that our contribution can be used by others to further characterize the manifestations of this disorder.

We inferred from the conclusions of our study and the clinical signs of acute PFS that the acute manifestation of PFS appeared to be a speech apraxia rather than a dysarthria or cerebellar ataxia. Mariën et al cited an article related to the behavioral disorders and other manifestations in patients undergoing posterior fossa surgery.⁶ The description in the study of acute PFS is very similar to our own experience: "All had normal involuntary palatal, lip, and tongue move-

ments, but none of them could imitate tongue or lip movements on command."⁶ The inability to perform a task while the motor functions required to do that task are intact seems to indicate apraxic dysfunction. As Mariën et al astutely mentioned, this applies to the initial mutism and not the subsequent verbal output disorder. Our study did not evaluate the long-term sequelae of PFS, and we have no comment on the etiology of subsequent disorders.

Our esteemed colleagues will be pleased to learn that since the submission of our manuscript, we have continued our research and found further evidence to validate the emerging concept of the development of postoperative PFS, which we intend to publish in the near future.

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