Surg Neurol 1985;23:201–4

We have reviewed the article written by Dr. Aoki and Dr. Mizutani in the *Journal of Neurosurgery* and agree that primary subarachnoid hemorrhage in moyamoya is exceedingly rare. We thank the authors for drawing this point to our attention.

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Cerebral Hemorrhage

To the Editor:

I read with interest the article "Moyamoya Disease and Cerebral Hemorrhage" by Hardy and Williams in the May issue of SURGICAL NEUROLOGY (Surg Neurol 1984;21:507–10), in which they reported a case of a 54-year-old man with cerebral hemorrhage secondary to "moyamoya disease." As the authors state, it is evident that moyamoya disease causes intraparenchymal hemorrhage as seen in their patient and in cases reported by us as well [1]. However, in regard to the features of the cerebral angiography shown in their case, it is highly doubtful whether this case may belong to moyamoya disease: The right and left common carotid angiograms reveal a long tubular stenosis beginning at the bifurcation of the common carotid artery and extending into the cranial cavity, where it is occluded. Their angiograms do not demonstrate any characteristics of moyamoya disease.

Furthermore, on the left vertebral angiogram, nearly normal-appearing internal carotid, middle cerebral, and anterior cerebral arteries are opacified through retrograde flow from the posterior communicating artery; this opacification is sufficient to exclude the diagnosis of moyamoya disease. It is, therefore, hardly acceptable that this case meets the criteria of moyamoya disease [2].

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1. Aoki N, Mizutani H. Does moyamoya disease cause subarachnoid disease? Review of 54 cases with intracranial hemorrhage confirmed by computerized tomography. J Neurosurg 1984;60:348–53.
2. Guideline for the diagnosis of moyamoya disease: annual report of the Research Committee on the Progressive Occlusive Disease of the Circle of Willis. Tokyo: 1978:132.

Reply. We would like to take issue with Dr. Aoki's comments regarding the diagnosis of our patient. There has been considerable discussion regarding the absolute etiology of moyamoya disease. Some authors have considered it to be postviral while others have assumed it to be a primary vasculopathy. Still others have considered the possibility of arteriosclerotic disease [1] as the primary occlusive event in the older population with this pattern.

We would agree with Debrun et al [2] on the etiologic nonspecificity of the pattern. Although the Japanese may now require proximal occlusion of the anterior cerebral and middle cerebral arteries for inclusion into their syndrome complex, the complex "fibrillar" collaterals arising from deep perforating choroidal, lenticulostriate, and thalamic branches must be explained and have been well defined in moyamoya. These have often been grouped with the moyamoya disease complex. They all share the same clinical syndrome of intracranial occlusive disease with the fine perforating collaterals. Our case fits nicely into this category, and we believe it is only a matter of semantics that Dr. Aoki is pursuing.

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1. Hinshaw DB, Thompson JR, Hasso AN. Adult arteriosclerotic moya-moya. Radiology 1976;118:633–6.
2. Debrun G, Sauvegrain J, Aicardi J, et al. Moya-moya, a nonspecific radiological syndrome. Neurology 1975;8:241–4.

Rerupture of Intracranial Aneurysms

To the Editor:

Concerning the paper "Analysis of 223 Ruptured Intracranial Aneurysms with Special Reference to Rerupture" by N. Ayoagi and I. Hayakawa (Surg Neurol 1984;21:445–52), I have several comments and questions.

In the abstract it is stated, "The incidence of rerupture was increased in patients with narrowly localized vasospasm and acute hydrocephalus." These conclusions cannot be made from the data presented. Table 5 (p. 449) suggests that rerupture carries a higher incidence of severe spasm (and higher morbidity and mortality) but does not imply that severe vasospasm leads to higher rate of rerupture. (A problem arises in that no mention is made as to when in the clinical course angiography was performed.) Furthermore, the statement is made (p. 449) that ". . . patients with ruptured aneurysms in whom the perfusion pressure does not change (i.e., little or no vasospasm) are at risk for rerupture." These statements are contradictory to one another. Also, on page 450 it is stated ". . . of patients with a single ruptured aneurysm 19% developed acute hydrocephalus after the hemorrhage compared with 54% with rerupture." Again, this does *not* say that acute hydrocephalus leads to higher rerupture rate!

The discussion of rerupture rates (p. 448) is also not clear. Following ". . . of 223 patients, the rerupture rate was 17%, 29%, and 30% for patients with Hunt–Hess grades 0–II, III, and IV, respectively," the authors go on to suggest ". . . of 46 patients with rerupture, 50%, 33%, and 17% had grades 0–II, III, and IV, respectively, indicating an inverse relationship between the Hunt–Hess grade and the incidence of rerupture." This is both artificial and misleading. It simply restates the fact that many more patients in this series were of clinical grades 0–II than III or IV and has little to do with an "inverse relationship" between true incidence and clinical grade.