Comment on the Letter by B. Eber on Anticardiolipin Antibody and Stroke: Possible Relation of Valvular Heart Disease and Embolic Events

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Thank you very much for allowing us to read and comment on the very thoughtful letter of Dr. Eber. Dr. Eber raises several important issues. In addition, however, we believe that some of Dr. Eber's concerns may be based on a misunderstanding of our data, specifically regarding cardiac and non-cardiac stroke risk factors. These were noted in detail in the results section of our article (pp 102-103, table 2). As indicated, 10 of 14 patients were known to be in normal sinus rhythm at the time of stroke and, although information from the time of stroke was not available in the 4 others, none was subsequently in atrial fibrillation, and we had no reason to suspect atrial fibrillation at the time of stroke. As for noncardiac risk factors, 1 patient was on the oral contraceptive pill at the time of the first stroke, but off the pill at the time of six subsequent strokes; 2 patients had a history of high blood pressure, but were normotensive at the time

of stroke, 3 patients had borderline hypertension, and only 1 patient was markedly hypertensive at the time of stroke. This patient had a further stroke and was subsequently found to have cardiac vegetations. In addition, we did not understand Dr. Eber's concern about carotid stenoses since, as we noted (p 104), angiograms were performed in 6 patients and showed no evidence of carotid stenosis.

Regarding lupus anticoagulant, although tests specifically for this factor were not done, partial thromboplastin times were available in 11 of 14 patients with stroke. Partial thromboplastin time was elevated in 9 of these 11, suggesting the presence of a lupus anticoagulant, as noted on p 105. We believe that Dr. Eber's appropriate concern about anticardiolipin antibody (Acla) levels is not applicable to our patients since, as we noted (p 100), all patients had levels > 5 SD above normal. For completeness, however, it should be noted that 13 of 14 patients had an IgG level $\geq$ 40 GPL units, 9 of 14 $\geq$ 60 GPL units, and 6 of 14 $\geq$ 80 GPL units on at least one occasion. Dr. Eber's interest in IgM Acla is shared by us: in this series, IgM was available in 10 patients, and was elevated in 6. In a subsequent manuscript, reporting results of a prospective assessment, we have dealt more completely with the issue of IgM Acla.

We suggested an association between Acla, stroke and valvular heart disease since the prevalence of valvular disease was very high among our patients with Acla and stroke. It was the existence of this relationship that led us to suggest the possibility (p 105) that Acla is a risk factor for stroke among patients with valvular disease. Certainly, however, Dr. Eber is

correct that this possibility cannot be accepted unequivocally without prospective data demonstrating that Acla elevation precedes stroke. As

we noted on p 107, our data do not prove a causal relationship between Ada, cardiac valvular disease and stroke. However, the strength of association we have reported suggests the possibility of this relation, which has been strengthened by results of our recent prospective studies in valvular and coronary diseases [1,2]. Clearly, as Dr. Eber suggests, additional studies will be needed to fully elucidate the relation among Acla, valvular disease and stroke.

References

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