Letters to the Editor

Duration of Nonvalvular Atrial Fibrillation and Stroke

To the Editor:

The epidemiologic data from the Framingham Study concerning atrial fibrillation (AF) included surprising evidence that stroke preceded the onset of chronic AF to a statistically significant degree.1 As noted in the accompanying editorial,2 it is more likely that paroxysmal AF, which later became chronic, was responsible for these antedating strokes, or that persistent (nonparoxysmal) AF resulted in stroke before its detection on routine biennial exam.

In their original publication, the Framingham investigators concluded that the occurrence of embolic stroke is unrelated to the duration of nonvalvular AF "with no evidence of a particularly vulnerable period."3 By their methodology, patients with AF and stroke who did not have AF on previous routine biennial exam were excluded from consideration. 3 If recent-onset AF is associated with increased stroke risk, most such patients with stroke would be excluded unless fortuitously due for routine biennial exam. Even excluding these patients, the Framingham data support a particularly high stroke incidence in the months following onset of chronic AF if considered in person-years at risk: 4 of 20 events occurred in 78 patients in the initial 6 months for a rate of 102 events per 1000 patient-years compared to 36 events per 1000 patient-years after 6 months (increase of 2.8).

There are three other studies addressing the issue of duration of nonvalvular AF and stroke risk. 4-6 In Fisher’s clinical study (which included 83 patients with nonvalvular AF and 17 patients with valvular AF), it is not possible to calculate the patient-year risk, but 27% of strokes occurred within 6 months of the diagnosis of AF. In Hinton et al’s autopsy study of 333 AF patients (29% had valvular disease), there were 3 embolic events in 40 patients with AF of less than 3 weeks duration — an extrapolated incidence of 100% per year. Aberg’s autopsy study reported embolism in 37% of 126 patients with nonvalvular AF of less than one week’s duration.4 The extremely high incidence of the latter two autopsy studies probably reflects the severity of underlying cardiac disease contributing to death.

It is undoubtedly true that increased risk of embolic stroke persists indefinitely after the development of chronic, nonvalvular AF. However, the available data and our anecdotal experience support the notion that the risk of embolism is particularly high in the months following onset of this dysrhythmia. This high early risk should be carefully considered in attempts to prevent the commonly devastating stroke, unheralded by TIA, that accompanies chronic, nonvalvular AF.

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References

To the Editor:

The authors have clearly suggested that those with recent onset AF are a highly susceptible cohort for stroke and they offer a plausible explanation for stroke preceding the onset of chronic AF in the Framingham biennial format.1

The data from our initial AF paper2 contained too few events to submit to statistical analysis. The thirty year follow-up of the Framingham cohort, recorded in this issue of Stroke deals with recent onset as well as chronic AF and risk of stroke.2 The “notion” of Drs. Hart, Easton and Sherman “that the risk of embolism is particularly high in the month following onset is amply borne out by epidemiologic data collected prospectively. Not only does initial stroke occur more often during the early months following onset of AF, but recurrence also occurs early and sooner in those with the arrhythmia.

The authors are to be congratulated for their perceptive articulation of this pertinent question.

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Does the Natural History of Transient Ischemic Attacks (TIAs) Justify Surgery?

To the Editor:

The extremely low incidence of completed stroke after TIA, cited by Muuronen and Kaste in Stroke,1 commands the attention of all physicians who treat patients with cerebrovascular disease. Their findings bring to light important therapeutic and epidemiologic issues and emphasize once again the continued need for properly designed randomized studies when advocating new strategies for managing patients with TIA. Many clinicians base therapeutic recommendations on the assumption that 25-40% of persons with TIAs will experience cerebral infarction within five years after onset of symptoms and that of these 40% occur during the first year.2, 3 In contrast, Muuronen and Kaste report an overall incidence of stroke after TIA in 314 patients to be 4.8% with mean follow-up of 7.8 years. Of 15 strokes only 20% (3/15) occurred within the first year after onset of TIA. Accepted criteria for identifying TIA were used, and this low incidence cannot be attributed to any specific form of therapy. If, indeed, the risk of stroke following TIA is only 4.8%, there would be little reason to subject patients to the potentially serious complications of angiography or surgery since the reported risk from this intervention in most centers approximates this figure.4 Furthermore, two recent studies, one from the U.S.5 and the other from Sweden,6 of patients treated by carotid endarterectomy report a greater overall incidence of stroke, even if analysis is confined to the territory of the operated artery.6

We wonder what accounts for the benign prognosis in the patients studied by Muuronen and Kaste. Is TIA more benign in some populations or are the differences methodological? Several important differ-
ences appear when the results of Toole et al.,7 are compared with those of Muuronen and Kaste (table). Muuronen and Kaste report (1) a young age structure, (2) lower incidence of associated risk factors and (3) a lower overall mortality. Furthermore, the prognosis for stroke and survival is better in both the Muuronen and Kaste and Toole studies when compared to the outcome in the population from Rochester, Minn.3 These discrepancies lead one to question whether the nature of vascular pathology, age distribution, and scientific way to evaluate better therapies for such a complex biological process as threatened stroke.

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To the Editor:

It is certainly a pleasure to read the letter of Dr. Shah and others. They seem to share our concern about the uncritical use of comparative “historical” controls when advocating new stroke therapies. Randomization is the only way to ensure that the treatment group and the control group are comparable. Whatever treatment is evaluated the criteria to include or exclude patients for that trial entail selecting a special group. Accordingly no two patient groups have identical “natural history.”

The main reason for the good outcome of our patients is most likely the young age structure. This also may explain the low incidence of associated risk factors and overall mortality. Furthermore, the outcome in the older groups in our study does not differ from other studies.3,4

The differences in the outcome in different patient groups underline the possibility that there are subgroups of TIA patients with different pathophysiologic factors. This once again stresses the importance of prospective randomized trials.5 If one wants to compare subgroups of patients then randomization stratified by prognostic features makes it possible, not so-called historical controls.6,7

Difficulties in clinical science do not make alternative, less scientific methods acceptable. Randomized trials alone provide the most ethical and scientific way to evaluate new stroke therapies.

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References


Cardiac Sequelae of Acute Stroke

To the Editor:

Myers’ and co-workers very interesting study on “Cardiac sequelae of stroke”1 supports earlier investigations indicating a causal relationship between cerebral lesions and an increased tendency to cardiac arrhythmias. I’m not convinced, however, regarding the conclusion that arrhythmia occurrence was independent of co-existing heart disease. The prevalence of coronary heart disease which may be expected to be more often associated with ventricular arrhythmias than e.g. m.v. hypertension, is not given in the paper. Were patients with atrial fibrillation excluded from the study?

The number of patients with different arrhythmias should preferably have been added to “arrhythmia hours” to make comparison easier. If serious arrhythmias (here ventricular tachycardia, couplets, VPB’s 5+/min and heart block second and third degree) were redefined as arrhythmias liable to initiate treatment i.e. ventricular tachycardia (of some length) and complete heart block, it seems as if these arrhythmias were almost only seen in stroke patients with known heart disease. It
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