LETTERS TO
THE EDITOR

Anticoagulation and atrial fibrillation

EDITOR,—We would like to congratulate McNulty and colleagues on their excellent clinical audit of antithrombotic therapy in atrial fibrillation.1 Their approach to the methods of audit has been impeccable, completing this audit cycle by instigating a process of reaudit after the results from the original data set had been intensively presented to hospital colleagues, itself leading to a highly effective intervention.

We would, however, appreciate this opportunity to provide a brief update in a few aspects of the current knowledge of stroke prevention in atrial fibrillation. The Lip and Lowe algorithm used for risk stratification was first published in 1996 and a refinement has since been proposed (box 1),1 which has been adapted for use in local and national guidelines.

The essential changes relate to the contribution of echocardiography and paroxysmal atrial fibrillation. In the Atrial Fibrillation Investigators overview on echocardiographic risk factors for thromboembolism, left atrial size per se does not appear to be an independent risk factor on multivariate analysis and is no longer used in the risk stratification schema; indeed, left atrial dilatation rarely occurs independent of “other” pathology, such as hypertension or heart failure which themselves constitute high risk features.4 In this analysis, moderate or severe left ventricular dysfunction on two dimensional echocardiography was the only significant abnormality which appeared to be an independent risk factor for thromboembolic stroke in non-valvular atrial fibrillation.5 Other studies using echocardiography for risk stratification have rarely found that the investigation significantly contributed to antithrombotic therapy management decisions, as many patients already had clinical risk factors allowing effective risk stratification.6

Regarding clinical risk factors, hormone replacement therapy use may also increase risk while alcohol consumption may be protective.7 McNulty and colleagues do not specifically address the problem of paroxysmal atrial fibrillation,8 which appeared to be an independent risk factor on multivariate analysis and is now considered significant in the Lip and Lowe algorithm as part of a refined risk stratification schema.9

We would, however, appreciate this opportunity to present the current understanding of stroke prevention in atrial fibrillation. The present recommended international normalised ratio (INR) therapeutic range of 2–3 for patients with atrial fibrillation over the age 75, a target INR of 2.0 (range 1.6–2.5) may be a reasonable compromise between toxicity and efficacy for this age group, pending further data about the safety of higher intensities. Further information from our ongoing Medical Research Council funded BAFTA (Birmingham Atrial Fibrillation Treatment Assessment) study in elderly patients aged 75 years, with non-valvular atrial fibrillation in primary care, would provide further information.

McNulty and colleagues extrapolate from their data the potential savings both in hospital admission days and financial cost,1 which may be achievable by a nationwide consensus approach to the problem. We wholeheartedly support this, and agree with their call for improved stroke prevention in atrial fibrillation, especially as there is now evidence the benefits and risks of anticoagulation seen in the clinical trials can realistically be translated into everyday clinical practice.1

D G CONWAY
G Y H LIP
Haemostasis Thrombosis and Vascular Biology Unit, University Department of Medicine, City Hospital, Birmingham B18 7QH, UK G.Y.H.LIP@bham.ac.uk


Box 1: Risk stratification and anticoagulation in non-valvular atrial fibrillation

ASSESS RISK AND REASSESS REGULARLY:

(1) High risk (annual risk of CVA = 8%–12%)
• All patients with previous transient ischaemic attack or CVA.
• All patients aged 75 or over with diabetes and/or hypertension.
• All patients with clinical evidence of valve disease, heart failure, thyroid disease, and/or impaired left ventricular function on echocardiography.
(2) Moderate risk (annual risk of CVA = 4%)
• All patients aged under 65 with no clinical risk factors: diabetes, hypertension, peripheral arterial disease, ischaemic heart disease.
• All patients aged over 65 who are not in high risk group.
(3) Low risk (annual risk of CVA = 1%)
• All patients under 65 with no history of embolism, hypertension, diabetes, or other clinical risk factors.

TREATMENT

• High risk: give warfarin (target INR 2.0–3.0) if no contraindications and reasonable in practice.
• Moderate risk: either warfarin or aspirin 75–300 mg. In view of insufficient clear cut evidence, treatment may be decided on individual cases. Referral and echocardiography may help.
• Low risk: give aspirin 75–300 mg daily.

*Echocardiography: not needed for routine risk assessment but refines clinical risk stratification in case of impaired left ventricular function and valve disease. A large atrium per se is not an independent risk factor on multivariate analysis.

CVA = cerebrovascular accident.

www.postgradmed.com
Implementation of antithrombotic management in atrial fibrillation

EDITOR,—We read the above original article of McNulty et al with interest particularly the antithrombotic management algorithm in fig 1.1 There is no mention of the time when the retrospective study was conducted. As was submitted in January 2000 we assume that the study was conducted sometime in 1999. The algorithm originally published in 1998 should be considered outdated in 1999/2000 because: first, in atrial fibrillation an age of 75 years or older is a high risk factor for thromboembolism to qualify for long term anticoagulation.2 Secondly, a large left atrium in patients with atrial fibrillation but without mitral valve disease is not an independent risk factor for thromboembolism.3 Therefore left atrium size of more than 4.5 cm per se should not be used to decide for long term anticoagulation in these patients. Thirdly, patients with atrial fibrillation who are below 65 years of age but with hypertension/ischaemic heart disease/diabetes mellitus are at high risk and should be given warfarin, not aspirin.4 Fourthly, patients with atrial fibrillation at high risk of thromboembolism in whom warfarin and aspirin are contraindicated should be considered for clopidogrel or at least dipyridamole. In patients with atherosclerotic vascular disease, clopidogrel has been shown to be more effective than aspirin in reducing risk of ischaemic stroke, myocardial infarction or vascular death without increasing adverse effect.5 Patients intolerant of aspirin should be given modified release dipyridamole or clopidogrel.6 We believe that using such improved management protocol for atrial fibrillation either for audit purpose or clinical practice, is likely to further reduce the risk of ischaemic stroke.

Des McNulty, Hutchinson, and Hardy respond: We read the three letters above with much interest and would like to address the issues raised by each, in turn. We appreciate the update on current knowledge of stroke prevention provided by Conway and Lip, which we have followed as it appears in the literature. Obviously we were unable to use these revised guidelines as our original article preceded the publication of this revision.7 When the audit was conceived we did debate whether to include paroxysmal atrial fibrillation (PAF) as well as sustained atrial fibrillation (SAF) in our study but at that time the “jury was still out” as to whether PAF carried the same risk—so we chose to audit sustained atrial fibrillation only; again we appreciate the update on PAF.

In response to Lairikyengbam et al—as stated our paper was submitted in January 2000. The auditing process took over a year to complete, as it takes time to accumulate 370 patients with atrial fibrillation, retrieve and extensively review case notes, and analyse the data. It should also be noted that there was a gap of six months between the two audits reported to allow for an education programme. Therefore when the revised guidelines were published in July 19998 the audit was completed.

Finally, the response from Murphy and Casey seems to raise two questions and a possible word of warning. The questions regard whether our patients had therapeutic INR control and what was the incidence of major adverse events in our group—unfortunately audit (and reaudit, in the case of our report) consists of snapshots of current management. In the case of lifelong intervention with warfarin therapy, audit would be an inappropriate tool to measure their two concerns and perhaps a prospective study would be a more useful way of answering their questions. We note a recent report by Gupta et al studying elderly patients with atrial fibrillation on warfarin.9 The last five prothrombin readings were analysed, and it was found that only 9% of these patients were not adequately warfarinised; only four major bleeds occurred over 265 treatment years in this particular treatment group. They also raise concerns regarding the increased resources needed to deliver the current published recommended guidelines. We agree that treating more patients with warfarin would increase demands on haematology laboratory resources; however, we (like most people involved in this field) believe that this is offset by the potential reduction in the demands made upon stroke and general inpatient beds, physiotherapy, occupational therapy, and all the other resources which are consumed after a stroke.

9 Gupta A, Thomas P. Are atrial fibrillation patients receiving appropriate stroke prophylaxis? Br J Cardiol 2001;8:38–42.