Anticoagulation and atrial fibrillation

EDITOR.—We would like to congratulate McNulty and colleagues on their excellent clinical audit of antithrombotic therapy in atrial fibrillation. Their approach to the methods of audit has been impeccable, comparable in audit cycles 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), 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. In this analysis, moderate or severe left ventricular dysfunction on two dimensional echocardiography best predicted significant abnormality which appeared to be an independent risk factor for thromboembolic stroke in non-valvular atrial fibrillation. 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.

Regarding other risk factors, hormone replacement therapy use may also increase risk while alcohol consumption may be protective.

McNulty and colleagues do not specifically address the problem of paroxysmal atrial fibrillation, which has occasionally been seen to contribute to thromboembolic stroke in non-valvular atrial fibrillation, especially in the presence of clinical risk factors. In general, our recommendation is to use warfarin in patients with paroxysmal atrial fibrillation unless they can be classified as having “lone” atrial fibrillation or there are contraindications to the use of anticoagulation, where aspirin should be used instead.

The safety and tolerability of long term anticoagulation titrated to conventional levels (international normalised ratio (INR) 2–3) is less clear in the very elderly (age older than 75 years), which is the age group encompassing perhaps half of the atrial fibrillation associated stroke patients. The elderly are also prone to more co-morbidity, polypharmacy, cognitive problems and frailty; indeed, biological age in some ways is more important than chronological age, and the decision must be based on the risk-benefit ratio, as with many things in clinical medicine. There have also been suggestions that an INR range of 1.6–2.5 can provide substantial, if partial efficacy (estimated to be nearly 90% of the highest intensities), and could be used for elderly patients to minimise haemorrhagic complications, although this has not been verified by any prospective study. Given the uncertainty about the safety of INRs >2.5 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, 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.

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

ASSSESS 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 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 feasible 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.

LETTERS TO THE EDITOR

Anticoagulation and atrial fibrillation


time orally anticoagulated patients in their audit were in therapeutic INR range or on the incidence of thrombotic stroke and major haemorrhage in this patient group. As implementation of antithrombotic management in atrial fibrillation patients in their hospital remains suboptimal, even on follow up audit, INR control in the orally anticoagulated patient group could also be inadequate, thus jeopardising the hoped for reduction in strokes in this high risk atrial fibrillation group. In addition, the increase in patients on oral anticoagulation in the period between the two audits may have adversely affected the delivery of acceptable INR control by increasing demands on time and resources in a presumably already overstretched district general hospital haematology laboratory.

We suggest that, in future, similar audits of management of atrial fibrillation should include information on the quality of INR control in orally anticoagulated patients. If such patients are found to be inadequately maintained in therapeutic INR range, increased resources should be allocated to improve the efficacy of INR control. Possible areas of improvement in anticoagulant control might include the upgrading of laboratory coagulation equipment, better communication between clinicians and the anticoagulation clinic, adequate laboratory, clerical, nursing and medical staffing, ongoing more effective patient and hospital staff education, computerised dosage systems and, where selected cases, patient self-monitoring and self-management.

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Implementation of antithrombotic management in atrial fibrillation

EDITOR,—We read the above original article by Gupta et al and are very concerned about the possible word of warning. The questions they raise concern regarding the increased resources needed to deliver the current published 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.

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Dear Drs McNulty, Hutchinson, and Hardy: 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 audit predated the publication of this revision.1 When the audit was conceived we did debate whether to include paroxysmal atrial fibrillation (PAF) as well as sustained atrial fibrillation 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 1999 the audit was completed.

Finally, the response from Murphy and Casey seems to raise two questions and a possible answer. The first was the questions as to 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 the 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.2 The last five prothrombin readings were analysed, and it was found that only 9% of these patients were not adequately warfarised; 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 guidelines. We agree that treating more patients with warfarin for atrial fibrillation 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.

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

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Implementation of antithrombotic management in atrial fibrillation

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