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CONSENSUS CONFERENCE ON ATRIAL FIBRILLATION IN HOSPITAL AND GENERAL PRACTICE HELD IN THE UNIVERSITY OF ST ANDREWS ON 3–4 SEPTEMBER 1998

FINAL CONSENSUS STATEMENT

Atrial fibrillation (AF) may be classified as paroxysmal, persistent (if it does not spontaneously terminate but reverts to sinus rhythm with electrical/pharmacological intervention), or permanent. This arrhythmia affects about 5% of the UK population over 65 years of age, with prevalence rising to 10% in those over 75.

AF is associated with valvular and ischaemic heart disease, heart failure, hypertension, and diabetes. It occurs with acute myocardial infarction, thyrotoxicosis, pulmonary embolism, toxins such as alcohol, acute respiratory infections, cardiac surgery, and chest injury and can develop whenever the left atrium is dilated. However, 50% of all patients with paroxysmal AF, and 20% with persistent or permanent AF, have otherwise normal hearts ('lone AF'). AF is not benign. It is a major cause of morbidity, and doubles mortality. Better treatment of AF could substantially reduce the burden of illness in society, especially through prevention of stroke. The fast and irregular heart rate of AF can cause palpitations, dizziness, malaise, anxiety, and heart failure; but the most serious consequence is ischaemic stroke, due to embolism from the left atrium. AF is found in 15% of all stroke patients and 2-8% of patients with transient cerebral ischaemia. The risk of ischaemic stroke in AF without rheumatic heart disease is about 5% per annum but varies according to the presence of specific factors in a given individual. After an initial embolism the stroke recurrence rate is 12% per year and the annual risk of death is 5%.

Optimal assessment of the patient with AF includes a full history and examination, a 12-lead electrocardiogram, an echocardiogram (to determine whether or not valvular disease and left ventricular systolic dysfunction are present), and other investigations to identify underlying causes and guide treatment.

The treatment of AF can be complex, and may include the use of potentially hazardous therapies; thus it requires close collaboration between the primary and secondary health care teams. Here we review four questions related to control of symptoms and prevention of thromboembolism in non-rheumatic AF.

HOW DO WE MAINTAIN SINUS RHYTHM IN PAROXYSMAL AF?

If a patient has 'lone AF' with infrequent attacks that are well tolerated, antiarrhythmic drug therapy is not indicated. When the symptoms are troublesome, either beta-blockers or class Ic antiarrhythmic drugs (e.g. flecainide, propafenone) should be used as first-line treatment. Class Ic antiarrhythmic drugs, sotalol, and amiodarone must only be started under hospital supervision. In cases where there is evidence of underlying heart disease such as hypertension or coronary disease, beta-blockers have particular advantages; but when heart failure is present, amiodarone is the drug of choice.

Digoxin is not effective since it does not prevent paroxysms or control the ventricular rate when they occur. There are electrophysiological interventions that may be helpful in patients whose symptoms do not respond to drug therapy.

HOW DO WE RESTORE SINUS RHYTHM IN PERSISTENT ATRIAL FIBRILLATION?

A patient with very recent onset AF requires immediate assessment and treatment with heparin. Therapy to restore sinus rhythm is most successful when given early.

If it is certain that AF has been present for two days or less, cardioversion should be attempted electrically or pharmacologically. Warfarin therapy is not required in these patients if cardioversion is successful. Flecainide is the drug of choice in these circumstances provided that left ventricular systolic function is normal; in cases of impaired left ventricular function, amiodarone should be used.

If AF has been present for more than two days, electrical cardioversion should be employed because pharmacological cardioversion is less likely to succeed, and anticoagulation is essential. To reduce the risk of thromboembolism, warfarin should be given to achieve an international normalised ratio (INR) in the range 2.0–3.0 for three weeks before cardioversion and continued for at least four weeks after cardioversion. With this strategy the risk of thromboembolism early after the procedure is reduced from 5-7% to 1-2%. Whether warfarin should be given beyond four weeks is uncertain, but this treatment may be considered in patients with a continuing high risk of recurrence of AF (large left atrium, poor left ventricular function, hypertension) or previously symptomless AF.

If persistent AF is of less than three months' duration, we recommend an attempt at restoration of sinus rhythm. Cardioversion is especially appropriate where a precipitant has been corrected (e.g. a chest infection, thyrotoxicosis, alcohol binge) and the patient has a structurally normal heart.

In patients with AF persisting beyond three months or where the duration of AF is uncertain, it is unclear whether or not rhythm control (i.e. restoration of sinus rhythm and maintenance for as long as possible) is more effective than rate control combined with anticoagulation at reducing long-term morbidity and mortality. These approaches are being compared in three randomised trials, and at present the choice of treatment should be based on physician judgment and informed patient preference.

Electrical cardioversion is initially successful in three-quarters of patients but relapse is frequent (25-50% at one month and 70-90% at one year). Treatment with an antiarrhythmic drug before and after cardioversion can increase both the initial success rate and the proportion of patients maintaining sinus rhythm in the longer term (to 50-60% at one year). It is not certain which patients will benefit from this approach. Those most likely to require antiarrhythmic therapy for maintenance of sinus rhythm are patients with longstanding AF, structural heart disease, and hypertension. We do not know for how long treatment should be continued after cardioversion or which drug or drug sequence is most effective; amiodarone is commonly used.

HOW DO WE ACHIEVE OPTIMAL CARDIOVASCULAR FUNCTION IN ATRIAL FIBRILLATION?

Haemodynamic impairment in AF results principally from the rapid ventricular response. Optimal cardiovascular function is best obtained by restoration and maintenance of normal sinus rhythm, but for many patients this is not achievable, and the goal of treatment is then to limit the ventricular rate to 90/minute at rest and 180/minute on exercise.

Digoxin alone may control the ventricular rate in a resting patient, but it is less effective at controlling the heart rate during exercise. Consequently, a beta-blocker (e.g. atenolol) or a rate-limiting calcium channel blocker (e.g. verapamil) should be considered as first-line treatment, particularly in patients with coexisting hypertension or coronary disease. In heart failure, digoxin remains the treatment of choice for rate control.

If monotherapy is ineffective then combination therapy should be considered (e.g. digoxin and beta-blocker, or digoxin and a rate-limiting calcium channel blocker). When verapamil is prescribed less digoxin is required. Beta-blockers should not be

given in combination with verapamil.

For selected patients in whom medical therapy has failed or is not tolerated, electrophysiological interventions, such as radiofrequency ablation of the atrioventricular node together with implantation of a rate-responsive pacemaker, may be indicated.

HOW DO WE PREVENT THROMBOEMBOLISM IN ATRIAL FIBRILLATION?

The following comments apply to all forms of AF.

Warfarin therapy decreases the risk of ischaemic stroke by about 65%, aspirin by only 20%. Warfarin and aspirin should only be given together in special circumstances.

Patients with high risk of ischaemic stroke (one or more of: previous stroke or transient cerebral ischaemia; >75 years of age; hypertension; diabetes mellitus; coronary artery disease; congestive heart failure; left ventricular dysfunction) should receive warfarin therapy unless it is contraindicated. Patients aged 65–75 years without other risk factors are at moderate risk and can be treated with warfarin or aspirin. Isolated left atrial enlargement is not an independent risk factor for thromboembolism.

In general, a target range of 2.0–3.0 for the INR gives satisfactory protection while minimising the risks of major haemorrhage. There is uncertainty about the safety of higher ratios in patients >75 years of age, for whom a target of 1.6–2.5 may represent a better balance between risk and benefit.

For patients at moderate or high risk of stroke in whom warfarin is contraindicated, aspirin should be prescribed. Contraindications to warfarin include risk of bleeding due to coexisting medical conditions, and any tendency to falls or other exposure to trauma. Another consideration is the likelihood of poor concordance (compliance), which will be influenced not only by the patient's ability to manage medications but also by local facilities for control of the INR. Special care must be taken with other drugs that interact with warfarin, including amiodarone. Patient preference is important.

Low-risk patients with AF should be treated with aspirin 75 mg–300 mg.

Anticoagulant therapy after an acute cerebral ischaemic event should be delayed until most of the deficit has resolved or, in the case of more severe strokes, more than two weeks has elapsed.

COMMENTS

We have considered only the four questions we were asked to address. Detection and management of underlying conditions and associated risk factors are important.

Patient participation is integral to good management. Patients must be informed of the risks and benefits of any of the treatments offered, and also of alternatives that may be available locally or elsewhere.

Therapeutic decisions in AF should take into account the need to maximise health gains from limited resources by use of the most cost-effective therapies.

There are many remaining areas of uncertainty about the management of AF, especially in relation to the first three questions. There is more evidence on the fourth question, although elderly patients have been under-represented in clinical trials. Further research is essential.

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