Atrial fibrillation and new therapeutic strategies

See page 1951 for the article to which this Editorial refers.

When new therapy challenges a critical disease the clinical spectrum is changed. But although the implantable cardioverter defibrillator is at the forefront of ventricular arrhythmia treatment, the number of implantations per million population remains limited (Fig. 1). The reasons for this poor take up must be one of costs set against the benefits, and indeed, the implantable cardioverter defibrillator has been closely followed by the implantable atrial defibrillator, before the former has been generally accepted.

Once introduced, a new technology must be fully studied and its costs and benefits weighed. However, some would question the value of yet more implanted hardware to prevent atrial fibrillation. Are other therapeutic approaches not sufficient to preserve an acceptable quality of life, under the safeguard of anticoagulation? It will take more than a decade to solve these questions. The National Heart, Lung and Blood Institute has recently started the ‘Atrial Fibrillation Follow-up Investigation of Rhythm Management [AFFIRM] study’. Patients with a history of paroxysmal or chronic (≤6 months) atrial fibrillation will be randomized to either atrial fibrillation (maintenance with anticoagulation and rate control) or sinus restoration with cardioversion and antiarrhythmic drugs. Outcomes to be investigated include mortality and quality of life. In a recent study, many patients with chronic atrial fibrillation failed to respond to the serial electrical cardioversion strategy. However, in younger patients with a fair exercise tolerance and a duration of atrial fibrillation shorter than 36 months, this approach may be worthwhile.

Duckers et al. present a novel rate-smoothing algorithm. It reduces RR variability in atrial fibrillation, without altering the overall heart rate. The variability between consecutive RR intervals was reduced by 73% and the overall variability of RR intervals was diminished by 59%. The precise long-term effect in reducing symptoms or preventing tachycardiomyopathy remains to be investigated. Possibly, the effect could be clinically more significant when the mean heart rate is maintained at a lower frequency by adjuvant drug treatment.

Greenhut et al. have compared a ventricular rate stabilization pacing algorithm with a control VVI pacing at a fixed rate of 50 ppm. The mean ventricular rate increased from 79 to 82 beats min⁻¹ as...
a result of the ventricular rate stabilization algorithm. The ventricular rate variability was much less during ventricular rate stabilization, as indicated by a 50% decrease in the coefficient of variation (SD/mean). In this study, 12/15 patients were on digoxin. Further studies should address haemodynamics and the potential long-term quality-of-life benefits. Although for some patients, serving as their own control, a symptomatic improvement might be obtained, this software solution is probably too subtle for matching with hard end-points, such as mortality and thromboembolic complications.

In the exploration of new frontiers of atrial fibrillation, rate-smoothing algorithms are a software tool not requiring additional hardware. So, there is no objection to its inclusion in modern pacemakers.

The question is, must we look at atrial fibrillation in the same way as accessory pathways, atrioventricular nodal re-entrant tachycardia and ventricular arrhythmias? Although I agree that today there is no place for old age or ageing processes, it could be that in the atrium something irreversible happens in old people. I hope that trials, such as the AFFIRM study, will provide common sense results, such as those used to approach atrial fibrillation in clinical practice. Before re-naming atrial fibrillation 'a malignant entity', randomized trials are necessary to explore the natural history of the disease and its 'conventional therapies'. Until recently, compliance with conventional therapy seemed to be acceptable in clinical practice. When replacing conventional therapies by aggressive and expensive new strategies, cost/benefit considerations will require a comparison of hard end-points and quality-of-life measurements. Atrial fibrillation is acquiring a new aura, as it is waiting for magic devices.

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References

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Assessing prognosis after acute myocardial infarction

See page 1873 for the article to which this Editorial refers

Risk stratification after myocardial infarction aims to divide patients into those at high risk of sudden death or reinfarction who will benefit from cardiac catheterization and myocardial revascularization, and those at low risk who can be followed expectantly on medical treatment. Prognosis after myocardial infarction is directly related to residual myocardial ischaemia, the presence and extent of which can be assessed using either exercise electrocardiography, stress echocardiography or myocardial perfusion imaging.

Exercise electrocardiography is commonly performed for assessing prognosis after infarction, but the overall sensitivity of ST segment depression for future cardiac events is low, averaging only 27%[11]. This relative insensitivity reflects difficulty interpreting ST segment changes where the resting electrocardiogram is abnormal, coupled with the frequent use of submaximal rather than maximal exercise.

Bigi et al[2] compared exercise electrocardiography and stress echocardiography for assessing prognosis after myocardial infarction: neither test was predictive of cardiac death, non-fatal reinfarction