Letters to the Editor


Acute stress and ventricular arrhythmias

James et al. in their study demonstrated an increase in QT dispersion as a result of psychological stress in patients with coronary artery disease [3]. The authors suggest that ischaemia is a critical component of the response to psychological stress and mention beta-blocker therapy as one of the underlying issues. Yet in the studied patients the increase in QT dispersion was observed both in patients taking beta-adrenergic blockers and in those not using these drugs.

In our opinion this clearly underlines the need for further evaluation of non-pharmacological therapeutic approaches to psychological stress in coronary artery disease. The working group of Hoffman et al. demonstrated reduced sympathetic nervous system responsivity associated with the reduction of mental stress in the studied patients the increase in QT dispersion was observed both in patients taking beta-adrenergic blockers and in those not using these drugs.

In our clinic, all inpatients with cardiac arrhythmias and ischaemia during mental stress testing. Patients assigned to a 4-month programme of stress management had significantly fewer cardiac events and ambulatory ischaemia in the follow-up of 4 years [3]. Relaxation techniques were also successfully implemented in situations which are associated with an increased risk of cardiac arrhythmias, such as cardiac surgery [4]. James’s study further points to the therapeutic potential that lies in the reduction of mental stress in patients with coronary artery disease.

In our clinic, all inpatients with cardiac arrhythmias and coronary artery disease are treated with relaxation techniques; patients appreciate it as an instrument for self-help and show excellent compliance. There is, however, a need to compare the already evaluated methods of mental stress testing [3,5,6] in order to better select patients for stress therapy. Furthermore, intervention trials should not only focus on beta-adrenergic blockers but also evaluate the efficacy of relaxation techniques and stress reduction programmes in cardiac patients at risk.

This will hopefully be useful in closing the gap between theory and clinical practice in stress-reduction treatments in patients with coronary artery disease.

A. MICHALSEN
G. DOBOS
Department of Internal Medicine, Kliniken Essen Mitte, Essen, Germany

References


Use of low-molecular-weight heparin as bridge anticoagulation therapy in patients with atrial fibrillation undergoing transoesophageal echocardiography guided cardioversion

Anders Roijer and colleagues [1] have presented an interesting study using a transoesophageal echocardiography guided low-molecular-weight-heparin (dalteparin) approach to immediate cardioversion of low-risk patients with atrial fibrillation or flutter. They used transoesophageal echocardiography to exclude from cardioversion, patients with thrombus and other thromboembolic risk factors, such as severe spontaneous echo contrast and lower Doppler flow states (<0.25 m s⁻¹). Patients with risk factors (group B) received ‘conventional’ therapy in the form of prolonged warfarin. Importantly, no bleeding or embolic events occurred in the immediate low-risk cardioversion group (Group A) in whom dalteparin bridge therapy was used [3].

This study may be an important step in testing the safety and feasibility of low-molecular-weight heparins as bridge therapy for patients in atrial fibrillation undergoing cardioversion. However, only two-thirds of the patients in the study were assigned to the dalteparin early cardioversion group (Group A) using clinical criteria that render this group a very-low-risk study population. In clinical practice, it has been safe to perform early cardioversion in patients who are shown free of thrombus by transoesophageal echocardiography, but who may have severe spontaneous echo contrast and low left atrial appendage flow velocities, as long as they are therapeutically anticoagulated pre- and post-cardioversion. This may offer cardioversion convenience and expediency to a larger patient population. There may be an intermediate group with so-called ‘sludge’ or viscid spontaneous echo contrast in the left atrial appendage that have a similar risk potential to thrombus and thus should be excluded for early cardioversion.

Stellbrink and Hanrath [2], in an accompanying editorial, point out that the feasibility and safety of the transoesophageal echocardiography guided approach has been demonstrated by the Assessment of Cardioversion Using Transoesophageal Echocardiography (ACUTE) Pilot Study [3]. The ACUTE studies were designed to determine the relative efficacy of a transoesophageal echocardiography guided approach compared to the conventional approach [1,2]. Stellbrink and Hanrath note that the larger ongoing ACUTE clinical trial [4] will answer many of our questions on the appropriate indications for transoesophageal echocardiography. The ACUTE Multicenter Study preliminary findings for major outcomes at 8 weeks have been released [3]. There were no differences found in embolic events between the two groups and the ACUTE trial indicated that the transoesophageal echocardiography guided approach is a good alternative to conventional therapy because it lowers total time to cardioversion and
reduces bleeding complications. We agree that many of the questions on the use of transesophageal echocardiography in atrial fibrillation will be addressed through secondary analyses of the ACUTE data.

Anders Roijer and colleagues courageously used dalfopristin as a single dose in patients with atrial fibrillation[1]. There is no previously published safety data on the use of low molecular weight heparin in this patient population with transesophageal echocardiography. Stellbrink and Hanrath correctly point out that there should be a randomized study addressing the role of low-molecular-weight heparin in atrial fibrillation[2]. We would like to point out that a randomized pilot study is now underway in the United States. The ACUTE II study is specifically designed to compare the feasibility, general safety, and economics of a transesophageal echocardiographic-guided approach for patients in atrial fibrillation undergoing immediate cardioversion[3]. The ongoing pilot study will follow 200 patients over a 5-week period. Safety outcomes include ischaemic stroke, transient ischaemic attack, systemic embolization, major and minor bleeding, cardioversion success, clinical haemodynamic instability, and mortality. Importantly, the study will measure relative length of hospital stay, patient quality of life/utility, and treatment costs. Savings from a reduction in hospital stay and improved quality of life are expected to offset the higher cost of the low-molecular-weight heparins in North America[4]. The results of this pilot study will have further important clinical and economic implications for the arrhythmotbic management of patients with atrial fibrillation undergoing transesophageal echocardiography-guided cardioversion.

R. D. MURRAY1
S. R. DEITCHER2
A. L. KLEIN3

1Department of Cardiology,
Department of Vascular Medicine,
Cleveland Clinic Foundation, Cleveland, Ohio, U.S.A.

References

The wider use of magnesium

The Editorial ‘Magnesium: an antiarrhythmic drug, but only against very specific arrhythmias’ Eur Heart J 2000; 1116, concludes that: ‘Magnesium . . . given intravenously will remain the main drug in the treatment of torsade de points, but no other indications exists for it as an antiarrhythmic drug’.

This very subjective statement has to be questioned, as in digitalis toxicity-induced tachyarrhythmias, clinical data show a clear indication for magnesium[5–9], even though we lack controlled studies for obvious reasons. In 1992 England et al. documented a significant reduction of perioperative ventricular and supraventricular arrhythmias with magnesium[10]. These findings have been confirmed in recent studies[6–9].

The management of patients with ventricular arrhythmias not responding to class III antiarrhythmic drugs is a well known clinical dilemma. Although data showing cardioversion rates of 30% in patients with ventricular arrhythmias[6,10], magnesium is not first-line therapy in ventricular tachycardia. However, the fact that there is no interaction between magnesium and conventional antiarrhythmic drugs[11] justifies the use of magnesium as salvage therapy in patients refractory to class III antiarrhythmics.

Even oral magnesium has been shown to have antiarrhythmic potential. Zehender and co-workers documented a 17% reduction of premature ventricular systoles and significantly fewer ventricular arrhythmias in magnesium and potassium treated patients[12]. In a controlled study by Feyertag et al., oral magnesium reduced arrhythmias by 57%[13].

In my opinion, magnesium should be considered a safe and cheap alternative, or additive antiarrhythmic agent, in several clinical situations.

H. G. STUEHLINGER
Department of Emergency Medicine, University Hospital of Vienna, Vienna, Austria

References