Editorials

The right atrial/transoesophageal approach for conversion of atrial fibrillation: a hybrid method for compromise?

Conversion of atrial fibrillation to normal sinus rhythm should be a highly successful procedure. The method should be safe, take only a few minutes, without the need for general anaesthesia or deep sedation, and on an outpatient basis. Methods of conversion in clinical cardiology do not fulfil these criteria at the present time, as demonstrated by the guidelines of working groups and consensus conferences recommending inhospital use of drugs and/or electrical conversion only and post-cardioversion intensive care observation of patients[1]. Increasing limitations of the resources of health care systems mean that alternative methods for conversion of atrial fibrillation to sinus rhythm are needed.

External cardioversion of atrial fibrillation using direct current shock results in a higher conversion rate of sinus rhythm and needs less time in comparison to antiarrhythmic drug conversion. It is today the preferred procedure and is well established in short-term persistent lone atrial fibrillation or longer-term atrial fibrillation, after a transoesophageal echocardiogram, with warfarin before and after the treatment shock[1]. Recommendations range between low and titrated energy[2] and high power up to 6·0–6·60 J kg⁻¹[3]. Newer guidelines, however, prefer high power initially[1] with an upper limit of 360 J under general anaesthesia to prevent severe discomfort and pain. High power often results in a local cutaneous flush, burning and inflammation but rarely in skeletal and cardiac myocyte enzyme depletion. It is often followed by atrial stunning with the risk of thrombus formation and embolism, but rarely by ventricular stunning with intermittent left heart failure and pulmonary oedema in cases of severe ‘electric intoxication’. Titration of direct current cardioversion power may result in several shocks in cases with a high defibrillation threshold. Therefore a general decrease in power seems mandatory, possibly with sedation instead of general anaesthesia during the procedure. However, atrial stunning and thromboembolism cannot be avoided using lower power direct current conversion.

Several approaches to solve this problem have been investigated. Different diameters and locations of the paddles were investigated during external conversion via anterior–posterior or right anterior–left posterolateral routes. Changes in shock waveforms, double and triple shocks, biphasic and triphasic shocks and changes in tilt have been tested. However, a substantial decrease in power output resulted in a reduced conversion rate. General anaesthesia could not be avoided using different external paddle locations and alternative shock waveforms.

Intracardiac approaches, proposed after experimental testing[4], need less power and often result in sinus rhythm with an internal cardioversion rate of 93% compared with 79% using the external method in 187 patients with 217 conversion attempts[5]. Limitations in the success rate of external electric cardioversion of atrial fibrillation to sinus rhythm and higher energy result from weak electric fields in the posterior and cranial left atrial free wall. However, internal cardioversion is more invasive, time-consuming and needs radiation exposure for lead control. The lowest defibrillation threshold was demonstrated using high right atrial and anterior free wall vs coronary sinus locations of defibrillation coils with 6·5±3·0 J in 22 out of 25 patients[6]. A randomized comparison of the coronary sinus vs pulmonary artery lead demonstrated 4·1±2·3 J in 35/38 patients of the coronary sinus group vs 7·2±3·1 J in 39/42 patients of the pulmonary artery group[7]. A comparison of four different coil positions in the right atrium and axilla (20·1±7·4 J), the left pulmonary artery (9·9±7·7 J), and the coronary sinus (2·0±7·5 J) demonstrated the minimum defibrillation threshold to the coronary sinus/left pulmonary artery[8]. A further decrease, to one third of the internal defibrillation threshold, using two sequential shocks delivered over two current pathways in an experimental model has been reported[9].

The oesophagus is in close contact with the posterior left atrium and left ventricle and has been used
for many years in left atrial pacing\textsuperscript{10} and defibrilla-
tion\textsuperscript{11,12}. From the anatomical point of view, the
oesophagus is located where the weak electrical field limits a further decrease of energy during
direct current conversion of atrial fibrillation. In contrast to the coronary sinus approach, which is
located close to the left atrioventricular groove around the mitral annulus and therefore reflects a
much more inferior left atrial approach, the coil in the esophagus has a more oblique course to the
left atrium and includes upper and lower parts of the left atrium. If the location of the defibrillation
coils are in the upper and lower parts of both atria and a posterior left atrial–anterior right atrial
approach is used then the defibrillation threshold should be at a minimum. The location of the leads in
the right atrium and oesophagus, as described in the paper by Santini et al.\textsuperscript{13}, would be similar to the
above.

The authors demonstrated that the introduction of the lead into the oesophagus is less time-consuming,
independent of radiation exposure and may be controlled by electrogram observation only, in
comparison to the coronary sinus coil location. Both positions include the option of post-shock
pacing in selected patients with sick sinus syndrome, using a pre-automatic sinus pause in
comparison to external shock using the paddles. On the other hand, the oesophageal location of the
coil reflects a ‘true’ anatomical posterior position, independent of any cardiac abnormality, such as
rotation, atrial or ventricular enlargement or transversal axis of the heart. The distance between the
intra-oesophageal coil and the left atrial muscle is
versal axis of the heart. The distance between the
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lead may be avoided by use of the surface QRS
complex synchronization. Identical electrical results
have been demonstrated by a single lead with dual
coils in comparison to the best intracardiac positions
in the right atrium and coronary sinus\textsuperscript{14}. However,
when a double-coil lead is introduced and the
surface QRS complex is used for synchronization, the
potential problem of venous puncture in warfarin-
treated patients remains. This technique should be
compared in a randomized study with the method of
Santini et al. using an intra-oesophageal coil.
Before the use of the proposed technique in public
hospitals with limited technical competence and out-
patient cardiological care, further evaluation is
needed.

D. PFEIFFER\textsuperscript{1}
B. LUDERITZ\textsuperscript{2}
\textsuperscript{1}University of Leipzig, Leipzig, Germany
\textsuperscript{2}University of Bonn, Bonn, Germany

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The unresolved mystery of stress-induced ST segment elevation after myocardial infarction

See page 814 for the article to which this Editorial refers

Studies of the relationship among stress-induced ST segment elevation, myocardial viability and ischaemia after myocardial infarction have gained increasing interest, particularly in the thrombolytic era, as a large proportion of patients retain viable myocardium which may improve spontaneously or after revascularization[1–4]. It is important to identify viable myocardium in order to match patients to the most appropriate strategy for improvement of left ventricular function.

In this issue, Mezilis et al. studied the value of dobutamine- and exercise-induced ST segment elevation shortly after acute myocardial infarction in the prediction of improvement of regional and global left ventricular function after revascularization in 39 patients[5]. They concluded that there is a strong association between dobutamine- and exercise-induced ST segment elevation and functional recovery following revascularization. Exercise-induced ST segment elevation in more than three leads, the development of ST segment elevation in both tests and a biphasic response during dobutamine echocardiography accurately predicted functional recovery. This study confirms the value of electrophysiographic studies during stress testing and shows that a simple method with little interpretation bias can be as useful as sophisticated methods in the detection of myocardial viability. The relationship between dobutamine-induced ST-segment elevation and late functional recovery after acute myocardial infarction is consistent with our previous report as well as those of other investigators[1,2,4].

One of the limitations of this study is the exclusion of patients with severe left ventricular dysfunction, as studies of myocardial viability are most useful in such patients.

Many studies have concluded that stress-induced ST segment elevation is not a specific sign of regional myocardial ischaemia after myocardial infarction as opposed to patients without previous infarction[1,3,6]. Most of these studies demonstrated a similar prevalence of ischaemia in patients with and without ST segment elevation. It is difficult to explain the apparent contradiction between the positive relationship with myocardial viability and the lack of correlation with regional ischaemia. One of the possible explanations is that ST segment elevation may be related to myocardial stunning rather than myocardial hibernation. In fact, most of the studies that demonstrated a higher prevalence of viability in patients with ST segment elevation were conducted after acute myocardial infarction[1,2,4,5]. In these patients, late spontaneous recovery of function may occur consistent with myocardial stunning as the major mechanism of reversible dysfunction. The absence of regional ischaemia in this case is in favour of spontaneous recovery of function as ischaemia may interfere with functional improvement or induce myocardial hibernation. However, in the study of Mezilis et al.[5], ST segment elevation was associated with a higher prevalence of a biphasic response, which is presumably a sign of myocardial ischaemia. It is not clear from this paper how many patients developed ischaemia in normally contracting myocardium at rest in the distribution of the infarct-related artery. The possibility that late functional recovery was due to spontaneous changes of myocardial perfusion and function rather than revascularization in some patients cannot be excluded[7].