NON-PHARMACOLOGICAL MANAGEMENT OF CARDIAC ARRHYTHMIAS

D.H. Birnie[*]
S.M. Cobbe[†]

Non-pharmacological treatment options for cardiac arrhythmias include radiofrequency ablation (RFA), surgical treatment, external cardioversion and implantable cardioverter defibrillators (see Table 1). This review largely focuses on their role in the management of supraventricular arrhythmias; the use of implantable cardioverter defibrillators in malignant ventricular tachyarrhythmias has been covered in a recent review in this journal.[1] The surgical treatment and RFA of ventricular tachycardia (VT) is currently limited to a few selected cases, although its role may increase in the future.

TABLE 1
The non-pharmacological treatment options for cardiac arrhythmias (second line = second to medical therapy).

Technique	Rhythm	Indication
Radiofrequency ablation	Atrioventricular re-entry tachycardia	
	Overt	Consider first line
	Concealed	Second line (?first)
	Atrioventricular nodal re-entry tachycardia	Second line
	Atrial flutter	Second line (?first)
	Atrial tachycardia	Second line
	Atrial fibrillation	
	AV Node ablation/ pacemaker.	Last resort
	AV nodal modification	Last resort
	Curative RFA	Experimental
Surgical	Ventricular tachycardia treatment	A few selected cases
	Atrial fibrillation	Experimental
External cardioversion	All sustained tachyarrhythmias	In emergency where tachyarrythmia is associated with haemodynamic compromise or angina
	Atrial flutter/ fibrillation	Electively
Implantable cardioverter defibrillators	Atrial	Experimental
	Ventricular	First line in selected cases

RADIOFREQUENCY ABLATION

The aim of catheter ablation is to destroy that area of myocardium which is integrally involved in the initiation or maintenance of a cardiac arrhythmia. High- and low-energy direct current shock, lasers, cryotherapy and microwave energy have all previously been tried, but the greatest success has been achieved with radiofrequency energy. The technique was developed in the late 1980s and is now regarded as the treatment of choice for symptomatic Wolff-Parkinson-White Syndrome, and as an early alternative to drug therapy for atrioventricular nodal re-entrant tachycardia. RFA also has an increasing role in the management of atrial flutter, atrial tachycardia, ventricular tachycardia and as palliation in severely symptomatic paroxysmal and sustained atrial fibrillation. More recently curative RFA for atrial fibrillation has been tried, but in this context RFA remains at an investigational stage.[2-7]

RFA is a specialised procedure which requires an operator fully trained in interventional electrophysiology. The equipment required includes adequate radiographic screening, a programmable intracardiac stimulator, and a computerised system for simultaneous acquisition, display and recording of multiple surface ECG leads and intracardiac electrograms. RF energy is delivered from an external generator to the myocardium via an endocardial electrode and a patch electrode on the patient's skin. Irreversible destruction of myocardium depends on generating local temperatures $\geq$50 °C.

RADIOFREQUENCY ABLATION FOR ATRIOVENTRICULAR RE-ENTRY TACHYCARDIA

Patients who have an accessory pathway of conduction between atrium and ventricle may develop atrioventricular re-entry tachycardia. Accessory pathways may occur anywhere around the atrioventricular ring. If an accessory pathway is associated with overt pre-excitation on the surface 12 lead ECG ('delta waves'), this is defined as the Wolff-Parkinson-White (WPW) syndrome. Patients with WPW syndrome and atrioventricular re-entry tachycardia may develop atrial fibrillation, probably as a result of rapid atrial reactivation during the tachycardia. In some instances, atrial fibrillation may arise as the primary arrhythmia. When atrial fibrillation is conducted down an accessory pathway

[*]*Specialist Registrar and* [†]*Walton Professor of Medical Cardiology, Department of Medical Cardiology, Glasgow Royal Infirmary, 10 Alexandra Parade, Glasgow G31 2ER*

then very fast ventricular rates can occur with the possibility of ventricular fibrillation. In view of this risk, radiofrequency ablation is the treatment of choice for patients with WPW and symptomatic atrioventricular re-entry tachycardia. Asymptomatic patients with evidence of pre-excitation on the ECG are at low risk of developing pre-excited atrial fibrillation, and are not normally considered candidates for ablation, except for when this is required to safeguard the patient's occupation (e.g. pilots, vocational driving licence holders). Patients with concealed accessory pathways are not at risk of pre-excited atrial fibrillation and sudden death. However, they should be considered for radiofrequency ablation if symptoms of atrioventricular re-entry tachycardia are not controlled by medical therapy, or if long-term drug therapy is not acceptable.

Technique

If there is overt pre-excitation on the 12 lead ECG then the likely site of the accessory pathway can be assessed using various validated algorithms.[8-11] The accessory pathway is further localised by mapping the site of earliest ventricular activation during sinus rhythm or atrial pacing, or by mapping the earliest atrial activation during ventricular pacing. Left-sided pathways are approached retrogradely via the aorta or via a trans-septal puncture. A successful ablation is seen by loss of 'delta waves' during radiofrequency application in overt pre-excitation (see Figure 1).

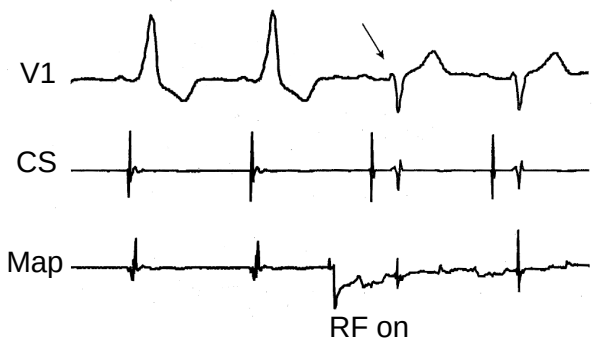

FIGURE 1
Radiofrequency ablation of Wolff-Parkinson-White Syndrome showing loss of delta waves on surface ECG (see arrow).
RF on = beginning of radiofrequency application.
CS = coronary sinus electrograms.
Map = electrograms at site of RF application.

Results and complications

From the results of a recent American survey, [12] rates of successful ablation varied between 82%and 91% depending on accessory pathway site. Significant complications (heart block, cardiac tamponade, pneumothorax, myocardial infarction, venous and arterial embolism and local vascular problems) were reported in 94 of 4,521 patients (2.1%), and 13 procedure-related deaths were recorded in 4,521 patients studies (0.2%). In Europe, the complication rate was 4.4%, with three deaths in 2,222 patients (0.14%).

RADIOFREQUENCY ABLATION FOR ATRIOVENTRICULAR NODAL RE-ENTRY TACHYCARDIA

Atrioventricular nodal re-entry tachycardia is the most common cause of paroxysmal supra-ventricular tachycardia. Moe *et al* in 1956[14] first described dual atrioventricular nodal transmission and it is now estimated that up to 10% of the population have the substrate for this arrhythmia. Typical atrioventricular nodal re-entry consists of anterograde conduction down a 'slow pathway' with retrograde conduction via a 'fast pathway'. The precise anatomical nature of these pathways remains controversial.[15] Atrioventricular nodal re-entry tachycardia is a benign arrhythmia[16] and RFA is reserved for patients with significant symptoms which cannot be controlled by tolerable drug therapy. Increasingly, patients are being offered RFA as a potentially curative procedure as an alternative to life-long anti-arrhythmic therapy.

Technique

Initially 'fast pathway' ablation was favoured but this led to an unacceptable incidence of heart block (6.8%)[16] and ablation is now targeted at the 'slow pathway'. Two techniques have been described for localisation of the 'slow pathway'. The anatomical approach uses His bundle and coronary sinus catheters as landmarks. The mapping catheter is manipulated initially into a posterior position well away from the His catheter. Radiofrequency applications are performed with successively more anterior positions until the tachycardia is no longer inducible. The alternative approach looks for specific endocardial potentials. Jackman *et al* described a 'sharp potential'[17] and Haisseguere *et al*[18] described a 'slow potential'. The occurrence of junctional rhythm with persistent retrograde fast pathway conduction during radiofrequency application is also used as a marker of satisfactory electrode position.[15] Many centres use a combination of these approaches.

Results and complications

Overall the success rate for RFA of atrioventricular nodal re-entry tachycardia is 96-100% with a late recurrence rate of 1-6% and a reported risk of complete atrioventricular block of between 0 and 3% (see Reference 15 for review). No procedure-related deaths were recorded in either the recent American or European surveys (3,867 patients).[12,13]

ATRIAL FLUTTER

Atrial flutter is caused by a single right atrial macro-reentrant circuit.[19] This proceeds in a counterclockwise direction around the atrium, passing through the narrow isthmus between the inferior vena cava and the tricuspid annulus. The objective of radiofrequency ablation is to interrupt conduction through this isthmus. Typically the flutter cycle-length is close to 200 ms, and 2:1 or 3:1 conduction is common, resulting in ventricular rates of around 150 and 100/min respectively. Occasionally 1:1 conduction can occur with haemodynamic collapse. Atrial flutter responds poorly to drug therapy and the relative ease and increasing success rates makes RFA an attractive early option.

Technique

An 'halo' catheter with upwards of ten pairs of electrodes is positioned in the right atrium around the tricuspid annulus. RF is applied sequentially along a line in the low right atrium to create a line of block in the re-entrant circuit. The success of RFA is shown by a change in the circumannular activation sequence during coronary sinus pacing, and inability to reinitiate atrial flutter by programmed stimulation.

Results and complications
Acute success rates of between 73 and 100% have been reported (see Reference 20 for review) but with recurrence rates of up to 44%; recurrences are significantly rarer with newer techniques. From the American survey,[12] 371 patients underwent ablation for atrial tachycardia and flutter with three significant complications and no deaths. In the European survey,[13] the complication rate was 5%.

ATRIAL TACHYCARDIA
'True' atrial tachycardia is a rare form of supraventricular tachycardia. Re-entrant atrial tachycardia occurs more commonly in the setting of structural heart disease, specifically following prior atrial surgery, and surgical scars are often involved in the circuit. Atrial tachycardia in the setting of a structurally normal heart may arise from an automatic intrinsic focus. Although this may be situated anywhere in the left or right atrium, the commonest sites are in the atrial tissue which extends into the proximal pulmonary veins or in the *crista terminalis* of the right atrium. The outlook of this arrhythmia is usually benign but occasionally tachy-cardiomyopathy can occur. There is only limited experience with RFA for atrial tachycardia but results are promising. Presently, RFA is limited to patients whose symptoms cannot be controlled on medical therapy.

Technique
The technique depends on the mechanism of the tachycardia. If it is shown to be macrore-entry, a similar approach to atrial flutter ablation is adopted, requiring creation of a line of conduction block to interrupt the circuit. Focal atrial tachycardia is ablated by delivering radiofrequency current at the site of earliest atrial activation.

Results and complications
Success rates of up to 92% have been reported.[21]

ATRIAL FIBRILLATION.
Atrial fibrillation is the most common cardiac arrhythmia. It affects 2% of people aged 65-74 and 5% of those older than 75 years, and causes very significant morbidity. Most atrial fibrillation is thought to be due to multiple simultaneous re-entry wavelets.[22] There are a number of non-pharmacological options for the treatment of atrial fibrillation and these are likely to be increasingly indicated.

Atrioventricular node ablation and permanent pacemaker implantation
Some patients with chronic or paroxysmal atrial fibrillation have severe symptoms resistant to drug therapy. A few small studies[23-27] have shown that atrioventricular node ablation combined with permanent pacemaker implantation is superior to drug therapy in improving symptoms. However, the procedure is irreversible and some patients feel worse during chronic pacing than in atrial fibrillation. Thus the procedure should be considered to be palliative rather than curative. For these reasons and the potential complications of pacemaker implantation, this technique is a last resort after a range of anti-arrhythmic medication have been tried. The decision to ablate the atrioventricular node is easier in patients with 'sick sinus syndrome' who need or already have a pacemaker.

Technique. A catheter is manipulated to a site where a large atrial electrogram and small His bundle electrogram are recorded. RF energy is applied until complete atrioventricular block is achieved and is continued for an additional 30 seconds. After atrioventricular node ablation, a dual chamber pacemaker with a mode-switching algorithm is implanted in patients with paroxysmal atrial fibrillation. During bouts of fibrillation, the mode switches from atrioventricular synchronous pacing to ventricular demand pacing, thus avoiding rapid irregular stimulation of the ventricles. Patients in permanent atrial fibrillation receive a rate-responsive ventricular demand pacemaker (VVIR).

Results and complications. In the American survey, atrioventricular nodal ablation was successful in 95% of 1,600 patients, with significant complications occurring in 21 (1.3%) and two procedure-related deaths (0.1%).[12] In Europe, the complication rate was 3.2%, and there was one death in 900 patients.[13] A lack of improvement or even deterioration in symptoms occurred in 7-14%.[23,26,27] One complication unique to atrioventricular node ablation is bradycardia-related ventricular arrhythmia and patients are usually prophylacticly paced at 90 beats per minute for one month after the procedure.

Atrioventricular nodal modification
Modification of the atrioventricular node to control rapid ventricular rates in atrial fibrillation was first described in 1994.[28,29] This procedure is only considered where medical therapy has failed because of the high incidence of requirement for a permanent pacemaker.

Technique. Ablation is directed at a similar area to the slow pathway in atrioventricular nodal re-entry tachycardia (see above). If an application does not reduce the ventricular response to less than 100 then successive burns are applied progressively more anteriorly until this is achieved.[30]

Results and complications. Rapid atrioventricular conduction may return after an apparently successful modification procedure.[30-32] Inadvertent complete heart block requiring permanent pacing occurs in between 5 and 16%.[30,32] Overall success rates (in up to 31 months follow-up) varies from 32-72.5%.[28,30-32] Two recent studies have compared this technique with atrioventricular node ablation and permanent pacing.[30,33] In the first study in patients with atrial fibrillation and left ventricular dysfunction, only 32% of the atrioventricular nodal modification procedures were successful compared to 100% of the complete atrioventricular nodal ablations.[30] Complete atrioventricular nodal ablation improved quality of life and left ventricular ejection fraction whereas modification did not.[30] In a second study[33] both procedures improved quality of life but complete ablation did so to a greater extent.

Curative ablation for atrial fibrillation. Curative ablation for atrial fibrillation is the biggest challenge remaining for interventional electrophysiologists. The multiple re-entrant wavelet hypothesis of atrial fibrillation was originally postulated in 1964 by Moe *et al*[34] and is now widely accepted. Moe *et al*[34] also suggested that suitably sited inexcitable areas might interrupt these wavelets and thus prevent atrial fibrillation. This is the basis of the surgical 'Maze' procedure

(see below); 'catheter Maze' type procedures have recently been attempted in animal models (see Reference 2 for review). In 1994 the first two successful procedures in humans were performed.[3,4] In 1996 Haissaguerre *et al* followed this with a series of 45 patients.[5] Initially they concentrated on producing lines of radiofrequency lesions in the right atrium, with disappointing results. Subsequently, in a subset of ten patients, they added left atrial burns with a success rate of 60%. The procedures are very prolonged and there is a significant risk of proarrhythmia, in particular new atrial flutter, and of thromboembolic complications.

More recently Haissaguerre's group have reported the successful RFA of an unusual focal atrial fibrillation.[6] Further in August 1998,[7] in a landmark paper, they demonstrated ablation of foci of atrial ectopic beats that had been shown to initiate atrial fibrillation. They studied 45 patients with drug-resistant paroxysmal atrial fibrillation with episodes occurring at least every two days. The initial electrophysiology study involved intra-cardiac mapping of isolated ectopic beats. The focus was localised according to the earliest atrial activity relative to the onset of the ectopic P wave, and ablation was performed at this site. A single focus of ectopics was identified in 29 patients and two to four sites in the other 16. Four of the foci were localised to atrial muscle and 65 (94%) to the pulmonary veins. Successful ablation of ectopic foci was achieved in 38 patients. During 8+/-6 months of follow-up, atrial fibrillation was eliminated in 28/45 (62%) of patients, i.e. in 28/38 (74%) of patients who had successful ablation of ectopic foci. No adverse events occurred. This is an exciting result but further studies are required to assess the long-term efficacy of this approach. In addition it is possible that they studied an unusual subset of atrial fibrillation patients. The high prevalence of foci in the pulmonary veins may reflect an excitation site which is particularly drug-resistant rather than the prevalence in the general population.

SURGICAL TREATMENT OF ATRIAL FIBRILLATION

Guiraudon *et al*[35] in 1985 described the 'corridor' operation in which an insulated sleeve of right atrial tissue connecting the sinoatrial and atrioventricular nodes was created. This operation was accompanied by a high incidence of other atrial arrhythmias and need for permanent pacing and this operation is now rarely performed. In 1991 Cox *et al* described the 'Maze' procedure[36] in which multiple incision lines are created to interrupt the re-entrant circuits. They recently reported their entire clinical experience (178 patients over 8° years).[37] 93% of patients were arrhythmia-free on no treatment. Of 107 patients with normal pre-operative sinus node function only one has required a pacemaker subsequently. Despite these promising results the procedure is performed in only a few centres in the world and remains experimental.[38] It is likely to be superseded by endovascular techniques.

ELECTRICAL CARDIOVERSION

Beck, in 1947, was the first to perform ventricular defibrillation.[39] In 1962, Direct Current cardioversion was described by Lown.[40] The electrical shock, by depolarising all excitable myocardium and possibly by prolonging refractoriness, establishes electrical homogeneity that terminates re-entry.

Indications

In general, any tachycardia that produces haemodynamic compromise, heart failure, or angina, and is resistant to prompt medical management should be terminated electrically. Most supraventricular arryhthmias, with the exception of atrial flutter and fibrillation, are usually responsive to medical therapy. Atrial flutter often responds poorly to drug therapy, thus DC cardioversion should be considered at an early stage.

The selection of patients with chronic atrial fibrillation for cardioversion is more difficult. Although there have been no randomised comparative studies, electrical rather than chemical cardioversion is generally chosen in this situation although this position may change with the development of new anti-arrhythmic agents. The decision to cardiovert depends on two factors: the likelihood of success in the long term, and the perceived advantages of maintaining sinus rhythm. The likelihood of success depends most importantly on the length of time in atrial fibrillation.[41] Increased age, left atrial size and impaired left ventricular function are also important factors reducing the likelihood of success. It is generally accepted that cardioversion for atrial fibrillation of greater than three years duration or with left atrial size greater than 60 mm is unlikely to succeed. An attempt should be made to restore sinus in most other patients,[42] especially if they are under 70 years. The potential benefits of restoration of sinus rhythm are improvement of symptoms and reduction of complications of atrial fibrillation. The latter has been presumed but not proven, and is the subject of a number of ongoing studies.[42]

Technique

It is not necessary to withhold digoxin before elective cardioversion unless there is clinical evidence of digoxin toxicity. Firm pressure should be applied to the paddles to reduce transthoracic impedance. A synchronised shock on the R wave is used for all cardioversions except for ventricular fibrillation. There is no evidence that any paddle position is more effective.[42] It is normal to start with the anterior-apex position, and if this is unsuccessful despite increasing energy delivered, then another position, usually antero-posterior is tried. For cardioversion of atrial fibrillation, shocks should begin at 100 J and for atrial flutter at 50 J.[42] For atrial fibrillation a commonly used sequence is 100 J/200 J/360 J/360 J and then one or two attempts with a different paddle position using 360 J.

Results and complications

The initial success rate of cardioversion for atrial fibrillation is 70-90%[42] but only about 20% will remain in sinus rhythm at 12-months follow-up.[41] Similar factors to those influencing likelihood of initial success determine the risk of relapse. Using additional anti-arrhythmic therapy can improve maintenance of sinus rhythm. For example, 50-70% of patients using amiodarone are still in sinus rhythm at 12 months.[43,44] Using a serial cardioversion and anti-arrhythmic drug approach, approximately one third of patients can be maintained in sinus rhythm at four years.[41] Direct current cardioversion has a low incidence of side-effects. Arrhythmias induced by the cardioversion generally are caused by inadequate synchronisation. Occasionally, a properly synchronised shock can produce ventricular fibrillation. To reduce thrombo-embolic complications patients should be anticoagulated for at least four weeks

before and after cardioversion unless the atrial fibrillation has been present for less than two days.[45] Although atrial flutter is probably of lesser thrombo-embolic risk, some authors advocate a similar anti-coagulant regime.[45] Anticoagulation for other atrial arrhythmias is not necessary. Direct current cardioversion does not lead to elevation in cardiac specific enzymes.[46,47]

There has been some interest in internal cardioversion with shock delivery between a catheter placed just inferior to the bundle of His and a backplate. Levy *et al*[48] compared internal with external cardioversion for atrial fibrillation. Internal cardioversion was initially more successful (91% versus 67%) but there was no difference in maintenance of sinus rhythm between the groups. Internal cardioversion may occasionally be tried if external shocks have failed.

INTERNAL ATRIAL DEFIBRILLATORS

Implantable defibrillators have been shown to improve survival in malignant ventricular arrhythmias[49,50] and are an accepted treatment option. Implantable atrial defibrillators are at a much less advanced stage of development. Their improvement faces the problems of accurate detection of atrial fibrillation, efficacy of termination, patient tolerability and the potential for pro-arrhythmia.[51] Atrial electrograms are of lower amplitude than those in the ventricle, and hence are harder to detect.[51] In addition there is a potential for confusion with low amplitude atrially detected ventricular electrograms.[51] The lowest defibrillation thresholds are achieved with shocks delivered between electrodes in the distal coronary sinus and right atrium.[52] In some patients intolerable pain can result from discharges <1 J.[53,54] Concerns regarding the possibility of induction of malignant ventricular arrhythmias have lead to the development of dual chamber defibrillators as well as 'stand-alone' atrial devices.[51] In March 1997 the first dual chamber atrial and ventricular defibrillator was implanted.[55] The first multicentre trial of 'stand-alone' implantable atrial defibrillators is underway.[56]

CONCLUSIONS

The non-pharmacological treatment of supra-ventricular arrhythmias has expanded rapidly over the last decade and will continue to do so. Non-pharmacological options are likely to be selected earlier as expertise and equipment improve, and complication rates continue to decrease. The non-pharmacological treatment of arrhythmias offer the benefit of cure and thus the avoidance of long-term drug therapy with the attendant inconvenience, side-effects, and risk of pro-arrhythmia. The curative ablation of atrial fibrillation is the biggest challenge remaining for interventional electrophysiologists, but significant advances are being made.

TABLE 2
Results and complications of radiofrequency ablation.

Rhythm	Success rate	Complications
Atrioventricular re-entry tachycardia	82-91% [12,13]	2.1-4.4% [12,13]
Atrioventricular nodal re-entry tachycardia	96-100% [15]	0-3% incidence of heart block [15]
Paroxysmal atrial flutter	73-100% [20]	0.8-5% [12,13]
Atrial tachycardia	92% [21]	
Atrial fibrillation		
AV node ablation and pacemaker	32-72.5% [28, 30-32]	5-16% incidence of heart block [30,32]
AV nodal modification	95% [12]	1.3-3.2% [12,13]

REFERENCES

1 Adgey J, Johnstone PW, Mathew TP. Sudden cardiac death - a predictable, avoidable and treatable event. *Proc R Coll Physicians Edinb* 1998; **29:**350-6.

2 Haissaguerre M, Shah DC, Jais P *et al.* Role of catheter ablation for atrial fibrillation. *Curr Opin Cardiol* 1997, **12:**18-23.

3 Haissaguerre M, Gencel L, Fischer B *et al.* Successful catheter ablation of atrial fibrillation. *J Cardiovasc Electrophysiol* 1994; **5:**1045-52.

4 Swartz JF, Pellersels G, Silvers J *et al.* Catheter-based curative approach to atrial fibrillation in humans. *Circulation* 1994*;* **90(Suppl 1):**I335.

5 Haissaguerre M, Jais P, Shah DC *et al.* Right and left atrial radiofrequency catheter ablative therapy of paroxysmal atrial fibrillation. *J Cardiovasc. Electrophysiol* 1994; **7:**132-44.

6 Jais P, Haissaguerre M, Shah DC *et al.* A focal source of atrial fibrillation treated by discrete radiofrequency ablation. *Circulation* 1997; **95:**572-6.

7 Haissaguerre M, Jais P, Shah DC *et al.* Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med* 1998; **339:**659-66.

8 Xie B, Katrittsis D, Heald SC *et al.* Localisation of accessory pathways from the 12 lead ECG using a new algorithm. *Am J Cardiol* 1994; **74:**161-5.

9 Fitzpatrick AP, Gonzales RP, Lesh MD *et al.* New algorithm for the localisation of accessory atrioventricular connections using a baseline electocardiogram. *J Am Coll Cardiol* 1994; **23:**107-16.

10 Milstein S, Sharma AD, Guiraudon G *et al.* An algorithm for the elecrocardiographic localisation of accessory pathways in the Wolff-Parkinson-White Syndrome. *PACE* 1987; **10:**555-63.

11 Linsay BD, Crossen KJ, Cain ME. Concordance of distinguishing electrocardiographic features during sinus rhythm with the localisation of accessory pathways in the Wolff-Parkinson-White Syndrome. *Am J Cardiol* 1987; **59:** 1093-102.

12 Scheinman M. Patterns of catheter ablation practice in the United States: Results of the 1992 NASPE survey. *PACE* 1994; **17:**873-83.

13 Hindricks G, on behalf of the Multicentre European Radiofrequency Survey (MERFS) Investigators of the Working Group on Arrhythmias of the European Society of Cardiology. The Multicentre European Radiofrequency Survey (MERFS): Complications of radiofrequency catheter ablation of arrhythmias. *Eur Heart J.* 1993; **14:**1644-53.

14 Moe GK, Preston JB, Burlington H. Physiologic evidence for

a dual A-V transmission. *Circ Res* 1956; **4:**357-75.
[15] Zhu DHW, Maloney JD. Radiofrequency catheter ablative therapy for atrioventricular nodal re-entrant tachycardia. In: *Interventional electrophysiology* Williams and Wilkins, 1997; 275-316.
[16] Gaita F, Riccardi R, Calo L. Importance and implications of AV block following radiofrequency ablation. *Heart* 1998; **79:**534-5.
[17] Jackman HM, Beckman KJ, McCleland JH *et al.* Treatment of supraventricular tachycardia due to AV nodal reentry by radiofrequency catheter ablation of slow pathway conduction. *N Engl J Med* 1992; **327:**313-9.
[18] Haissaguerre M, Gaita F, Fisher *et al.* Elimination of AV nodal re-entrant tachycardia using discrete slow potentials to guide application of radiofrequency energy. *Circulation* 1992; **85:**2162-75.
[19] Olshansky B, Wilber DJ, Hariman RJ. Atrial flutter - update on the mechanism and treatment. *PACE* 1992; **15:**2308-35.
[20] Lesh MD, Kalman JM, Olgin JE. An electrophysiological approach to catheter ablation of atrial flutter and tachycardia: from mechanism to practice. In: *Interventional electrophysiology*. Williams and Wilkins, 1997; 347-82.
[21] Lesh MD, Van Hare GF, Epstein LM *et al.* Radiofrequency catheter ablation of atrial arrhythmias. Results and mechanisms. *Circulation* 1994; **89:**1074-89.
[22] Allessie MA, Koning K, Kirchhof CJ *et al.* Electrophysiological mechanisms of perpetuation of atrial fibrillation. *Am J Cardiol* 1996; **77:**10A-23A.
[23] Brignole M, Gianfranchi L, Menozzi C *et al.* An assessment of atrioventricular junction ablation and DDDR mode-switching pacemaker versus pharmacological treatment in patients with severely symptomatic paroxysmal atrial fibrillation. A randomised controlled study. *Circulation* 1997; **96:**2617-262.
[24] Kay GN, Bubien RS, Epstein AE *et al.* Effect of catheter ablation of the atrioventricular junction on quality of life and exercise tolerance in paroxysmal atrial fibrillation. *Am J Cardiol* 1988; **62:**741-4.
[25] Fitzpatrick AD, Kourouyan HD, Siu *et al.* Quality of life and outcomes after radiofrequency His-bundle catheter ablation and permanent pacemaker implantation: impact of treatment in paroxysmal and established atrial fibrillation. *Am Heart J* 1996; **131:**499-507.
[26] Marshall HJ, Harris ZI, Griffith MJ *et al.* Atrioventricular nodal ablation and implantation of mode switching dual chamber pacemakers: effective treatment for drug refractory paroxysmal atrial fibrillation. *Heart* 1998; **79:**543-7.
[27] Kamalvand K, Tan K, Kotsakis A *et al.* Is mode switching beneficial? A randomised study in patients with paroxysmal tachyarrhythmias. *J Am Coll Cardiol* 1997; **30:**496-504.
[28] Feld GK, Fleck RP, Fujmura O *et al.* Control of rapid ventricular response by radiofrequency catheter modification of the atrioventricular node in patients with medically refractory atrial fibrillation. *Circulation* 1994; **90:**2299-307.
[29] Williamson BD, Man KC, Daoud E *et al.* Radiofrequency catheter modification of atrioventricular conduction to control the ventricular rate during atrial fibrillation. *N Engl J Med* 1994; **331:**910-6.
[30] Twidale N, Mcdonald T, Nave K *et al.* Comparison of the effects of AV node ablation versus AV nodal modification in patients with congestive heart failure and uncontrolled atrial fibrillation. *PACE* 1998; **21:**641-51.
[31] Canby RC, Roman AC, Kessler DJ *et al.* Selective radiofrequency ablation of the 'slow' atrioventricular nodal pathway for control of the ventricular response to atrial fibrillation. *Am J Cardiol* 1996; **77:**1358-61.
[32] Morady F, Hasse C, Stickberger SA *et al.* Long term follow-up after radiofrequency modification of the atrioventricular node in patients with atrial fibrillation. *J Am Coll Cardiol* 1997; **29:**113-21.
[33] Lee SH, Chen SA, Tai CT *et al.* Comparisons of quality of life and cardiac performance after complete atrioventricular junction ablation and atrioventricular junction modification in patients with medically refractory atrial fibrillation. *J Am Coll Cardiol* 1998; **31:**637-44.
[34] Moe GK. On the multiple wavelet hypothesis of atrial fibrillation. *Arch Int Pharmacodyn Ther* 1962; **140:**183-8.
[35] Giraudon GM, Campbell CS, Jones DL *et al.* Combined sino atrial node atrio-ventricular isolation: a surgical alternative to His bundle ablation in patients with atrial fibrillation. *Circulation* 1985; **72** (suppl III)**:**III-220.
[36] Cox JL, Schuessler RB, D'Agostino JH *et al.* The surgical treatment of atrial fibrillation: III. Development of a definitive surgical procedure. *J Thoracic Cardiovasc Surg* 1991; **101:**569-83.
[37] Cox JL, Schuessler RB, Lappas DG *et al.* An 8^{+} year clinical experience with surgery for atrial fibrillation. *Ann Surg* 1996; **224 :**267-73.
[38] Doevendans PA, Wellens HJJ. Atrial dissection for atrial fibrillation: when, how much and where? *J Am Coll Cardiol* 1996; **28:**991-3.
[39] Beck CS, Pritchard WH, Feil HS. Ventricular fibrillation of long duration abolished by electrical shock. *JAMA* 1947; **135:**985-86.
[40] Lown B, Amarasinghem R, Neuman J. New methods for terminating cardiac arrhythmias: use of synchronised capacitor discharge. *JAMA* 1962; **182:**548-55.
[41] Van Gelder, Crijns HJ, Tieleman *et al.* Value and limitation of electrical cardioversion in patients with chronic atrial fibrillation - importance of arrhythmia risk factors and oral anticoagulation. *Arch Intern Med* 1996; **156:**2585-92.
[42] Van Gelder IC. Crijns HJ. Cardioversion of atrial fibrillation and subsequent maintenance of sinus rhythm. *PACE* 1997; **20:**2675-83.
[43] Gosselink ATM, Crijns HJ, Van gelder IC, *et al.* Low dose Amiodarone for maintenance of sinus rhythm after cardioversion of atrial fibrillation or flutter. *JAMA* 1992; **267:**3289-93.
[44] Horowitz LN, Spielman SR, Greenspan AM *et al.* Use of amiodarone in the treatment of persistent and paroxysmal atrial fibrillation resistant to Quinidine therapy. *J Am Coll Cardiol* 1985; **6:**1402-7.
[45] Mayet J. More RS. Sutton GC. Anticoagulation for cardioversion of atrial arrhythmias. *Eur Heart J* 1998; **19:**548-52.
[46] Neumayr G, Schratzberger P, Friedrich G *et al.* Effect of electrical cardioversion on myocardial cells in patients in intensive care. *BMJ* 1998; **316:**1207.
[47] Georges JL, Spentchian M, Caubel C *et al.* Time course of troponin I, myoglobin and cardiac enzyme release after electrical cardioversion. *Am J Cardiol* 1996; **78:**825-6.
[48] Levy S, Lauribe P, Dolla E *et al.* A randomised comparison of external and internal cardioversion of atrial fibrillation. *Circulation* 1992; **86:** 1415-20.
[49] The AVID Investigators. A comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from near fatal ventricular arrhythmias. *N Engl J Med* 1997; **337:**1576-83.
[50] Moss AJ, Hall WJ, Cannom DS *et al.* Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. *N Engl J Med* 1996; **335:**1933-40.
[51] Jung W, Luderitz B. Implantable atrial defibrillators: Quo Vadis? *PACE* 1997; **20:**2141-5.
[52] Ayres GM, Griffin JC. The future role of defibrillators in the management of atrial fibrillation. *Current Opinion in Cardiology* 1997; **12:**12-17.
[53] Saksena S, Mongeon L, Krol *et al.* Clinical efficacy and safety of atrial defibrillation using biphasic shocks and current nonthoracotomy endocardial lead configurations. *Am J Cardiol* 1995; **76:**913-21.
[54] Murgatroyd FD, Slade AKB, Sopher SM *et al.* Efficacy and

tolerability of transvenous low energy cardioversion of paroxysmal atrial fibrillation in humans. *J Am Coll Cardiol* 1995; **25:**1347-53.

[55] Jung W, Luderitz B. Implantation of an arrhythmia management system for ventricular and supraventricular tachyarrhythmias. *Lancet* 1997; **349:**853-4.

[56] Jung W, Kirchhoff PG, Lau CP for the Metrix System Clinical Investigational Group. Therapy delivery with the METRIX automatic atrial defibrillator system: threshold stability and shock safety. *J Am Coll Cardiol* 1997; **29:**78A.