Catheter ablation by low energy DC shocks for successful management of atrial flutter

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Abstract

Objective—To assess the effects of low energy ablation of the substrate for atrial flutter.

Design—Initial retrospective analysis of patients undergoing low energy ablation of the atrioventricular node for refractory atrial flutter (group 1) was followed by a prospective assessment of low energy ablation in the postero-inferior right atrium for the same condition (group 2).

Setting—Tertiary referral centre for management of cardiac arrhythmias.

Patients—Seven men (aged 50–67 years) with refractory atrial flutter.

Interventions—Multiple (3–10) low energy DC shocks with a cumulative energy of 100–245 J in the region of the atrioventricular node in group 1 and 12–15 low energy DC shocks (cumulative energy 110–235 J) guided by the anatomical landmarks of the triangle of Koch and applied directly to the atrial wall.

Main outcome measure—Freedom from recurrence of atrial flutter.

Results—In group 1 despite initial complete atrioventricular block in three patients, atrioventricular conduction had resumed in all by one month. All four, however, were in sinus rhythm at follow up six to 13 months later. Two of the three patients in group 2 were free of atrial flutter at follow up three to four months after ablation.

Conclusion—Ablation of the atrial flutter substrate with low energy DC shocks is feasible. Precise electrophysiological mapping is not necessary.

small but significant morbidity and mortality.^{1,2}

A more attractive approach to the long term management of atrial flutter has been to tackle the arrhythmia substrate directly.^{3–5} This has been made possible by the improved understanding of the mechanisms of flutter gained from animal models of the arrhythmia and from human intracardiac electrophysiology studies.^{6–8} It seems from such studies that the typical form of atrial flutter is due to a macro-reentry circuit within the right atrium with an area of slow conduction postero-inferiorly in the triangle of Koch, between the os of the coronary sinus and the atrioventricular valve ring. Cryoablation of this area, guided by intraoperative epicardial mapping, has been successfully used to prevent recurrence of the arrhythmia.³ More recently high energy endocardial DC ablation has been performed guided by endocardial mapping techniques. We have used low energy DC shocks for endocardial ablation because high energy discharges have been shown to cause significant barotrauma to cardiac structures remote from the intended site of injury. We achieved long term remission of atrial flutter in four patients with chronic atrial flutter in whom the aim of the procedure had been to achieve atrioventricular block. This serendipitous abolition of atrial flutter suggested to us that precise mapping of the area of slow conduction was not necessary for successful ablation of the atrial flutter circuit. We therefore prospectively used multiple low energy shocks guided by the anatomical landmarks of the triangle of Koch in an attempt to abolish the arrhythmia substrate.

Patients and methods

PATIENTS

We studied seven men (age 50–67 years) with either chronic or paroxysmal atrial flutter that was refractory to medical treatment (table 1). All had severe symptoms from their arrhythmia; during exercise three patients had experienced pre-syncope caused by 1:1 conduction of the arrhythmia to the ventricle. The cause of the flutter was known in three patients (ischaemic heart disease, alcoholic cardiomyopathy, and dilated cardiomyopathy). In the remainder, though the aetiology of the arrhythmia was unknown, three had evidence of impaired left ventricular function caused by primary myocardial disease or perhaps a response to chronically rapid ven-

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Accepted for publication
13 June 1991

Atrial flutter is a common arrhythmia for which no entirely satisfactory treatment is available. Though DC cardioversion is often successful in restoring sinus rhythm in the short term, relapses are common. Despite the growing number of pharmacological agents available for arrhythmia prophylaxis many patients are either intolerant of drug treatment or achieve inadequate relief from their symptoms. In this group atrioventricular node/His bundle ablation has been the treatment of choice. This is not an ideal solution, however, because it requires the insertion of a permanent pacemaker, the atrial arrhythmia is not abolished, and the procedure itself has a

Table 1 Patient details

Case No	Age/sex	Type	Flutter cycle length (ms)	Duration (mnth)	Medications	Aetiology	LV EDD/ESD (cm)	LA size (cm)
Group 1:								
1	50/M	Chronic (type I) 1:1	220	17	Di	DCM	6.5/5.9	4.8
2	64/M	Chronic (type I)	250	21	A, D, Di, F, P, S, V	Lone	6.0/4.5	4.0
3	67/M	Chronic (type I)	210	36	A, At, Di	IHD	6.1/4.8	4.4
4	39/M	Chronic (type I) 1:1	240	39	A, Di, F, P, S	Lone	5.5/4.2	4.8
Group 2:								
5	62/M	Chronic (type I)	280	11	A, At, Di	Alcoholic	5.2/4.2	3.8
6	58/M	Paroxysmal (type I)	225	7	D, F, P, S	Lone	5.3/3.4	4.2
7	58/M	Paroxysmal	250	8	A, At, D, F, V	Lone	6.1/4.8	4.6

A, amiodarone; AT, atenolol; D, disopyramide; Di, digoxin; F, flecainide; P, propafenone; V, verapamil; DCM, dilated cardiomyopathy; EDD, end diastolic dimension; EF, left ventricular ejection fraction; ESD, end systolic dimension; IHD, ischaemic heart disease.

tricular rates. Two patients had associated atrioventricular conduction disease. Alcohol ablation of the atrioventricular node by a transcoronary approach had previously been considered in two patients but had been abandoned because of difficulty in cannulating the atrioventricular nodal artery. One further patient had previously undergone unsuccessful high energy ablation of the atrioventricular node which had been complicated by a period of hypotension.

In the group 1 patients the primary aim of the procedure was to ablate the atrioventricular node and His bundle. Group 1 patients were drawn from a total of six patients with atrial flutter/fibrillation in whom we had used a modified low energy power source to attempt abolition of atrioventricular conduction. Group 2 patients were those in whom selective ablation of atrial tissue in the triangle of Koch was planned.

METHODS

All ablation procedures were performed under general anaesthesia. A 6F bipolar pacing electrode was introduced via the subclavian vein and then positioned in the right ventricular apex. The 7F bipolar electrode that was to be used for delivery of the ablative shock was then introduced via the right femoral vein and placed across the tricuspid valve to record the His bundle potential. All recordings were displayed on a Siemens Elema Mingograf 82 jet recorder at a paper speed of 100 mm/s. In those patients undergoing His bundle ablation (group 1) the ablating catheter was gradually withdrawn until a large low right atrial signal was obtained while a His potential deflection

was just maintained. A low energy shock (<25 J) was then delivered with a specially modified energy source (Cardiac Recorders CR60) the characteristics of which have been previously described.^{9 10} If atrioventricular conduction resumed after the initial shock then further shocks were given until stable complete heart block was achieved (table 2). Temporary ventricular pacing was started and continued until a permanent pacing system was implanted.

In group 2 patients a quadripolar electrode was introduced via the subclavian vein and placed in the coronary sinus. This allowed accurate visualisation of the course of the vessel. In these patients the ablation catheter was again positioned to record a His potential. This localised the position of the His bundle and defined the apex of the triangle of Koch (fig 1). Then we withdrew this catheter using clockwise torque to rotate the tip posteriorly towards the os of the coronary sinus. Catheter manipulation was performed under single plane fluoroscopy in the anteroposterior projection to verify each position in a right anterior projection, which gave optimal visualisation of the low posterior right atrium. An initial shock of 5 J was delivered near the apex of Koch’s triangle at a site where the His bundle deflection was no longer visible (fig 2). If atrioventricular conduction was not compromised by this, a further shock of 10 J was delivered at the same site. Multiple further shocks were then delivered covering as much of the surface area of the triangle as was technically feasible. Particular care was taken to ensure that several shocks were delivered not only around the mouth of the coronary

Table 2 Ablation details

Case No	Shocks (J)	Cumulative energy (J)	Effect on atrioventricular conduction	Outcome	Follow up (mnth)
Group 1:					
1	30, 30, 40	100	3° block for < 1 mnth. 1° block long term	Sinus rhythm	14
2	20, 20, 20, 20, 20, 20, 20, 20, 20,	200	3° block for < 1 mnth. No effect long term	Sinus rhythm	7
3	5, 10, 20, 20, 20, 20, 25, 25, 50,	245	3° block for < 1 mnth. 1° block long term	Sinus rhythm	3
4	50 400, 400, 25, 25, 25	800 + 75	3° block for < 1 h. Nil long term	Sinus rhythm	7
Group 2:					
5	5, 5, 5, 5, 5, 10, 10, 10, 10, 10, 10,	110	Nil	Sinus rhythm	3
6	15, 15 5, 5, 5, 10, 10, 10, 15, 15, 15,	130	Nil	Developed atrial fibrillation at two mnth	2
7	15, 15 5, 10, 10, 10, 15, 15, 15, 15, 20,	235	1° block for < 10 beats	Recurrence of atrial flutter at one mnth	2
	20, 20, 20, 20, 20, 20				

Figure 1 Anatomical boundaries of the triangle of Koch. Apex = His bundle; sides = tendon of Todaro, and septal leaflet of tricuspid valve. The base runs through the coronary sinus. Broad arrows indicate the putative re-entrant circuit in type 1 atrial flutter.

sinus but also just within the os. Energies of up to 20 J were used at sites not immediately adjacent to the area of the atrioventricular node. The procedure was stopped when at least two shocks had been delivered at each attainable site. We then attempted to induce atrial flutter using single extrastimuli and rapid atrial pacing. All patients were followed up at one month and three months after discharge from hospital.

Results

In group 1 all patients were in chronic atrial flutter at the time of the procedure. In three patients stable complete heart block was achieved after three to 10 shocks with a cumulative energy of 100–245 J. The single largest energy used was 50 J. All of these patients had permanent adaptive rate pacing systems implanted and at discharge did not need antiarrhythmic treatment. At subsequent follow up (range three to 14 months) these patients remained symptom free. All had evidence of a return of atrioventricular conduction with two showing first degree atrioventricular block. In patient 4 high energy ablation

of the atrioventricular node was attempted with two 400 J shocks. Only transient atrioventricular block was achieved but the patient developed hypotension with a systolic blood pressure of 60 mm Hg. The procedure was therefore stopped. Two days later attempted low energy ablation produced only temporary atrioventricular block. This patient was discharged on antiarrhythmic treatment to await surgical ablation of the atrioventricular node. However, he suffered no recurrences of his symptoms and his antiarrhythmic treatment was gradually withdrawn.

In group 2 patients 12–15 shocks were delivered with a cumulative energy of 105–235 J. The single maximum energy used was 25 J. During the procedure one patient developed evidence of transient first degree atrioventricular block that lasted less than 10 beats. At the end of the procedure atrial flutter could still be induced in two patients. At follow up of three to four months two patients remained free of atrial flutter. Of these, one patient had developed atrial fibrillation, an arrhythmia that had not been documented before ablation. In the third patient atrial flutter recurred a month after the procedure.

COMPLICATIONS

No patient developed hypotension or evidence of ventricular arrhythmia during low energy ablation. In group 2 one patient with alcoholic cardiomyopathy developed mild congestive heart failure one week after the procedure. This responded satisfactorily to diuretic treatment.

Discussion

Our experience suggests that the substrate for atrial flutter can be ablated by multiple low energy shocks in the triangle of Koch without the need for extensive endocardial mapping. In 1925 Lewis first suggested that atrial flutter was due to a macro-reentry circuit in the right atrium. There is now considerable evidence to support this hypothesis from both experimental models of the arrhythmia and from clinically based electrophysiology studies.^{7 11 12} The typical form of the arrhythmia (type 1) in humans is characterised by negative flutter waves in the inferior leads of the surface electrocardiogram. The arrhythmia can be entrained by rapid atrial pacing and reset by appropriately timed atrial extrastimuli, suggesting a reentry mechanism with an excitable gap. Endocardial mapping of the arrhythmia shows that activation proceeds in an anticlockwise direction from the os of the coronary sinus to the bundle of His, then to the high right atrium, and finally to the lateral wall of the atrium before returning to the os of the coronary sinus thereby completing the circuit^{13 14} (fig 1).

Waldo first developed the concept of entrainment of reentrant arrhythmias based on his observations of atrial pacing in patients with postoperative atrial flutter.¹⁵ Central to this concept of entrainment is the presence of both an excitable gap and an area of slow conduction within the reentrant circuit. The ability to

Figure 2 Triangle of Koch enlarged showing the approximate sites and magnitudes of energy delivered in joules.

entrain an arrhythmia is now widely accepted as proof that the arrhythmia is caused by a reentrant mechanism. However, direct proof that the circuit of atrial flutter contains an area of slow conduction has only recently been obtained.^{3 16 17} The most convincing evidence comes from Klein and colleagues who reported two patients with atrial flutter in whom intra-operative epicardial mapping confirmed the atrial activation sequence demonstrated by the intracardiac catheter approach. In addition there was a clumping of isochrones in the postero-inferior right atrium between the os of the coronary sinus and the atrioventricular node indicating slowed conduction in this region. After cryoablation of this area neither patient experienced a recurrence of their arrhythmia. Whether the posteroinferior right atrium is an obligatory part of the flutter circuit and consistently shows slow conduction has not been definitively established. It has been suggested that this area of slow conduction can be identified by mapping of the atrium during entrainment of atrial flutter and by assessment of the tachycardia response to attempted reset by atrial extrastimuli. This procedure termed entrainment mapping is one of the methods used to localise the area of slow conduction before ablation.^{17 18}

Recently several workers have suggested that fractionated or double spike electrograms may be a marker for the area of slow conduction.¹⁹⁻²¹ These can be recorded during spontaneous or entrained flutter in most patients and also occasionally during atrial pacing or sinus rhythm. They are found most consistently in the triangle of Koch but have also been recorded at other right atrial sites remote from this region. Although some workers suggest that these electrograms, particularly those with a double spike configuration, bracket the area of slow conduction on the circuit others argue that they represent colliding wavefronts within the refractory core around which the arrhythmia circulates. Whether these abnormal electrograms are simply epiphenomena or truly represent the area of slow conduction remains controversial. Both groups that have previously reported high energy DC ablation of atrial flutter regard the recording of these abnormal electrograms as the most important way of identifying the most suitable area for ablation. In some patients they recorded fractionated electrograms in more than one area of the right atrium. In practice they ablated only in the triangle of Koch. We achieved remission and possibly long term cure of the arrhythmia by delivering shocks in this region without resorting to complex endocardial mapping.

The fact that attempted His bundle ablation also prevented recurrences of atrial flutter in some patients suggests that shocks delivered at the apex of the triangle of Koch may alter the conduction characteristics within a wider area of the triangle thereby abolishing or favourably modifying the arrhythmia substrate. However, ablation in this area may have additional unwanted electrophysiological consequences. One patient in our group and three patients in the series of Saoudi *et al* developed previously

undocumented atrial fibrillation after ablation. The procedure may have disrupted but not abolished the arrhythmia substrate giving rise to recurrent episodes of unstable atrial flutter that degenerated into atrial fibrillation. Alternatively, modification of the surrounding atrial myocardium may have predisposed it to primary atrial fibrillation. Ablation in the right atrium therefore seems to alter the electrophysiological substrate in some patients in such a way that the flutter circuit can either no longer be engaged or followed but fibrillation, which does not require this discrete zone of slow conduction, may be facilitated.

Not all patients undergoing His bundle ablation either in our own or other series have shown abolition of atrial flutter. In some patients, but not all, this secondary effect on the flutter circuit may have been masked by successful interruption of atrioventricular conduction. This variable success in ablating the flutter circuit is probably attributable to differing extents of tissue damage. In some patients though atrial muscle at the apex of Koch's triangle had been ablated other areas within the triangle could provide alternative conduction paths for the circulating wavefront. Only by abolishing all potential paths through this isthmus would the area of slow conduction be adequately extirpated. Use of a single high energy DC shock in this area ablates not only a few millimetres around the catheter tip but also modifies the conduction characteristics of the surrounding tissue for several square centimetres.²² If low energy shocks were to be used then several lesions would be required throughout the triangle to ensure that all potential paths for the arrhythmia had been ablated.

The use of intracardiac high energy DC shock has been associated with early cardiac perforation and long term impairment of exercise capacity by right ventricular damage. This damage is probably mediated by arcing and barotrauma. Experimental work suggests that these are related to the maximum energy used, whereas useful tissue damage is related to peak voltage. The energy source that we used was designed to deliver capacitive discharges with a short time constant by an anodal discharge. The shorter time constant means that though the peak voltage and current achieved were unchanged the total charge and energy were reduced. When used with a specially designed ablation catheter with a contoured tip peak voltages of up to 1423 V can be achieved before arcing occurs.²³ This minimises gas formation, arcing, and barotrauma to the surrounding cardiac structures.²⁴ Most shocks delivered in our patient group, though well below the high energies of 150-300 J used by other groups, were above the arcing threshold for the system when used with a standard 7F ablation catheter. Therefore some degree of arcing and barotrauma must have occurred which, it could be argued, may have contributed not only to the overall effectiveness of the procedure but might also result in long term impairment of cardiac function. In addition, though it is possible, there is no conclusive proof that the baro-

trauma caused by multiple low energy shocks is less than that caused by a single high energy shock if the same cumulative energies are used. In future, improved power sources and catheter design may make ablation of the flutter substrate possible without causing any barotrauma. In the meantime, if our simplified approach to the procedure shows continued success in the long term, it will increase the applicability of the technique to a wider group of patients.

CONCLUSION

Catheter ablation in the low posterior right atrium can be successfully used to ablate the substrate of atrial flutter. The use of low energy shocks may help to minimise the effects of barotrauma. The performance of the procedure can be guided by the simple anatomical landmarks of the triangle of Koch rather than by using extensive endocardial mapping techniques.

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