Successful intracardiac electrical conversion of atrial flutter in patients with complex congenital heart disease

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Abstract

Sixteen patients presenting on 21 occasions with atrial flutter in association with complex congenital heart disease were treated by intracardiac stimulation techniques combined with activation mapping. Nineteen episodes of atrial flutter were successfully converted to sinus rhythm. In the remaining two episodes atrial fibrillation was induced with spontaneous conversion to sinus rhythm within 12 hours in one episode and immediate DC cardioversion to sinus rhythm in the other. Intracardiac stimulation techniques were highly successful in this group and allowed reliable conversion to sinus rhythm without general anaesthesia and high energy cardioversion. In patients with atrial flutter associated with congenital heart disease intracardiac stimulation techniques should be tried first.

Atrial flutter is common after surgical repair of complex congenital cardiac malformations and it can have profound haemodynamic consequences, particularly when ventricular function is impaired. Both drug treatment and external cardioversion with high energy DC shock have disadvantages.¹⁻³

Pharmacological agents can either allow the ventricular response to be controlled or may achieve chemical conversion to sinus rhythm. The conversion rate is low in these patients with congenital heart disease and residual lesions, especially when flutter has been present for a long time.

Even when the ventricular rate is controlled the circulation still lacks the atrial contribution to cardiac output and this may predispose the patients to worsening of atrioventricular valve regurgitation, particularly in hearts with one functioning ventricle, deteriorating right ventricular function in transposition of the great arteries, or in tetralogy of Fallot with pulmonary regurgitation. In general, the symptoms get worse when atrial flutter becomes established.

External DC cardioversion is effective in restoring sinus rhythm but it requires a general anaesthetic and the ventricular myocardium is liable to be damaged if repeated cardioversions are necessary. So it may be better to avoid cardioversion in a young patient with an abnormal ventricle. An alternative approach is to use intracardiac stimula-

tion techniques to restore sinus rhythm. Rapid overdrive pacing⁴⁻⁷ and long duration single pulses ("atrioversion")⁸ have both been successful.

We examined the results of these techniques in a group of young patients presenting with atrial flutter in association with a complex congenital cardiac malformation and deteriorating clinical state.

Patients and methods

METHODS

Patients were brought to the electrophysiological laboratory unsedated and in the post-absorptive state. Routine premedication for electrophysiological studies was used. The patients were routinely anticoagulated at the time of study. In most the study was performed as an elective procedure after the patient had been anticoagulated with oral anticoagulants for 4-6 weeks. In these patients warfarin was stopped for 24 hours before the study or until the International Normalised Ratio fell below 2. In those in whom the study was performed as an urgent procedure half dose heparinisation was used at the start of the catheterisation.

Under local anaesthesia we introduced one or more quadripolar electrode catheters (with 5 or 10 mm interelectrode spacing) from a femoral or subclavian vein and advanced the catheter(s) to the right atrium. In three episodes the catheter entered the left atrium because of an atrial septal defect or absent interatrial septum.

A surface electrocardiogram and intracardiac electrograms were recorded on a multichannel inkjet recorder. Endocardial stimulation was performed by manipulating the catheter(s) into various atrial sites. Four surface electrocardiographic leads were simultaneously recorded. Atrial sites were classified as high, mid, or low and as anterior, lateral, posterior, or septal in the right atrium. Left atrial stimulation was not performed.

Through one pair of electrodes we delivered stimuli with a pulse width of 2 ms at twice diastolic threshold. Single stimuli were introduced after variable programmed delays from the previous spontaneous flutter electrogram to scan the atrial flutter cycle. The initial delay was 10 ms less than the flutter cycle length; this was reduced until refractoriness was encountered. The duration of the post-stimulation (return) flutter cycle was noted. A non-compensatory return cycle resulted in the flutter being reset (type I response (fig 1)). A

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Figure 1 An example of reset (type I) and compensatory (type II) responses to atrial stimulation during flutter. (A) Three surface and three intracardiac leads (two from mid-anterior right atrium (MARA) and low lateral right atrium (LLRA)). Flutter cycle length is 230 ms. A premature stimulus at the low lateral site produced a cycle of 180 ms, followed by a non-compensatory return cycle of 230 ms (a type I response). (B) Four surface leads and one intracardiac lead from mid-posterior right atrium (MPRA). Flutter cycle length was 215 ms. A premature stimulus resulted in a cycle of 175 ms followed by a compensatory return cycle of 255 ms ($175 + 255 = 2 \times 215$) (a type II response).

compensatory pause in the return cycle did not result in resetting (type II response). Further programmed stimuli (up to a total of four) were added if a type I response was found. Each additional stimulus was initially added at a coupling time 10 ms less than the flutter cycle length and reduced until refractoriness was encountered or conversion achieved.

If conversion was not achieved rapid atrial overdrive pacing was attempted with a modification of the protocol suggested by Waldo *et al.*⁷ Pacing was begun 10 beats per minute faster than the flutter rate, initially for two seconds then if necessary for a further burst duration of four, eight, 15, or 30 seconds. If flutter persisted, the pacing rate was increased by 10 beats per minute and the sequence of burst durations was repeated. It is known that pacing rates that exceed 135% of the flutter rate are more likely to result in atrial fibrillation,⁸ and these rates were not exceeded unless pacing had failed at lower rates at more than one site.

If conversion was not achieved a further type I atrial site was sought and the full stimulation protocol was repeated. If a suitable type I site could not be found the full conversion protocol was carried out at a type II site.

In one patient (two episodes) a new technique (Atrioversion⁸) was used instead of rapid atrial pacing, as part of an experimental protocol. A single constant current pulse with a duration 20 ms longer than the flutter cycle length was delivered 10 ms after the refractory period (as defined by a single stimulus) with an amplitude that was initially twice the diastolic threshold and was doubled to a maximum of 20 mA.

ELECTROCARDIOGRAPHIC DEFINITION OF ATRIAL FLUTTER

The diagnosis of atrial flutter was made from the surface electrocardiogram. The diagnosis required the presence of regular, rapid atrial activity with some degree of atrioventricular block, either spontaneously or during carotid sinus massage. An atrial rate of >200 beats per minute was required with clear evidence of classic flutter waves in at least one surface electrocardiographic lead. This rate is lower than the generally accepted lower rate limit for atrial flutter (240–250 beats per minute) because previous cardiac surgery and congenital abnormalities of the atrium can slow the flutter rate.

In episodes in which the atrial rate was below 200 beats per minute flutter was diagnosed only if the patient was receiving antiarrhythmic drug treatment likely to slow the atrial rate and if an earlier electrocardiogram had showed flutter at a rate greater than 200 with an identical flutter wave configuration before drug treatment.

Atrial flutter was classified as being of the “common” form if negative sawtooth deflections were seen in the inferior surface electrocardiogram leads and uncommon if the flutter waves were positive in leads I and II.

PATIENTS

Twenty one procedures were carried out on 16 patients (eight male, and eight female, aged 15–43 years, mean 26). Table 1 shows the clinical details. All patients had had previous corrective surgery for their congenital lesions on at least one and up to four occasions. Eleven patients had one procedure and five had two on separate hospital admissions. The duration of flutter before attempted conversion ranged from six hours to five years (median 14 days). At the time of study, the atrial rate was 158–363 beats per minute (mean 229) and the ventricular rate was 63–172 beats per minute (mean 100). 2:1 atrioventricular block was present in seven episodes, 3:1 block in four episodes, 1:1 conduction in one episode, and a variable block in the remainder.

The current antiarrhythmic drug treatment was amiodarone in seven episodes, digoxin in two episodes, digoxin and quinidine in two episodes, digoxin and disopyramide in two episodes, quinidine in one episode, amiodarone and digoxin in one episode, digoxin and flecainide in one episode, and xamoterol in one episode. In four episodes the patient was receiving no antiarrhythmic drug treatment at the time of study.

In five episodes the atrial rate was below 200 beats per minute. In four of these the patient was receiving amiodarone and had previous evidence of atrial flutter with an atrial rate of over 240 beats per minute before amiodarone and an identical flutter wave configuration. In the remaining episode, the flutter rate was 188 when the patient was receiving disopyramide and it accelerated to 330 in a subsequent episode when the patient's treatment was changed to xamoterol.

Conversion of atrial flutter was attempted only during episodes in which there was

Table 1 Data on patients

Case	Age	Sex	Basic diagnosis	Previous surgery	Residual lesions	Previous arrhythmias	Previous treatment	Result of conversion and follow up
1	17	M	VSD, infundibular pulmonary stenosis	VSD closure and resection of pulmonary stenosis (age 14)	Very small VSD, mild pulmonary stenosis	AFI + syncope (age 17) (requiring cardioversion)	Quinidine then amiodarone	Sinus rhythm, flutter recurred age 20, repeat conversion to sinus rhythm
2	33	F	ASD (ostium secundum)	ASD repair (age 33)		SVT + cerebral embolism (age 32)	Nil	Sinus rhythm
3	20	F	ASD (ostium secundum)	ASD repair (age 5)		Persistent atrial flutter since age 5	Digoxin + amiodarone	Sinus rhythm—reverted to atrial flutter 1 month; controlled by digoxin
4	28	M	Tetralogy of Fallot	L Blalock total repair (age 9)	Small VSD, pulmonary regurgitation, pulmonary stenosis, cardiomegaly	Atrial tachycardia (age 26), atrial flutter (age 28)	Amiodarone, digoxin, disopyramide	Sinus rhythm, flutter recurred age 31, repeat conversion to SR
5	31	M	VSD, infundibular stenosis, pulmonary hypertension	VSD closure infundibular resection (age 11)	Pulmonary hypertension, right heart failure	Atrial flutter (age 31)	Digoxin	Sinus rhythm, heart/lung transplant age 32
6	31	F	Tricuspid atresia, ASD	Pott's operation (age 18 months)	Tricuspid atresia, ASD	Atrial fibrillation (age 27), complete heart block (age 30) (pacemaker insertion)	Amiodarone, digoxin	Sinus rhythm
7	34	M	Tetralogy of Fallot, ASD, anomalous origin of LAD	Pott's operation (age 3) and total repair + ASD closure (age 14)	Pulmonary regurgitation, pulmonary stenosis, PA stenosis, anomalous origin LAD	Syncope age 30, ventricular extrasystoles	Digoxin	Sinus rhythm, flutter recurred age 35, repeat conversion to sinus rhythm
8	22	F	Tricuspid atresia, ASD, Fontan at age 12	Glenn's operation (age 8 months), L Blalock (age 8), Fontan (age 12), redo Fontan (age 22)		VT (age 19), ?SVT/VT (age 20)	Disopyramide, amiodarone	Sinus rhythm, flutter recurred age 23, repeat conversion to sinus rhythm
9	22	F	TGA, VSD, pulmonary hypertension	Blalock-Hanlon (age 2 months), banding of PA (age 18 months), palliative Mustard (age 8)	Pulmonary hypertension, VSD, tricuspid regurgitation	SVT (age 17)	Amiodarone	Sinus rhythm
10	15	M	Mitral atresia, doublet outlet LV, ASD	Blalock-Hanlon			Amiodarone	Sinus rhythm
11	19	F	PDA, ostium primum ASD, coarctation	PDA ligated (age 10 months), ASD repaired (age 5)	Coarctation of aorta	AVNRT/SN disease (age 11), antitachycardia pacemaker removed age 17 (infection)	Digoxin	Atrial fibrillation, immediately converted to sinus with 75 J DC
12	25	M	Tricuspid atresia	Fontan shunt			Amiodarone	Sinus rhythm
13	31	M	Univentricular heart, tricuspid atresia	Fontan (age 20), closure VA leak (age 28)	Calcified RA clot	Recurrent AFI since 2nd op	Quinidine, digoxin, disopyramide	Sinus bradycardia treated with xamoterol. AFI recurred after 2 months—repeat conversion to sinus rhythm
14	24	M	Tetralogy of Fallot	Total repair (age 8)	Pulmonary regurg, pulm stenosis	Recurrent AFI since age 18	Amiodarone	Sinus rhythm
15	43	F	Pulmonary stenosis	Pulmonary valvotomy (age 7) open pulmonary valvotomy + PFO closure (age 16)	Infundibular hypertrophy	Recurrent AFI since age 41	Digoxin, flecainide, verapamil	Sinus rhythm
16	33	F	ASD	ASD repair	Pulmonary stenosis		Digoxin	Sinus rhythm

AFI, atrial flutter; ASD, atrial septal defect; AVNRT, atrioventricular nodal reentrant tachycardia; LAD, left anterior descending coronary artery; LV, left ventricle; PA, pulmonary artery; PDA, patent ductus arteriosus; PFO, patent foramen ovale; RA, right atrium; SN, sinus node; SR, sinus rhythm; SVT, supraventricular tachycardia; TGA, transposition of great arteries; VA, ventriculoatrial; VSD, ventricular septal defect; VT, ventricular tachycardia.

evidence of regular atrial flutter on the surface electrocardiogram. Irregular atrial activity or atrial fibrillation was excluded. In 16 episodes, flutter was of the “common” form. In five episodes, flutter was classed as “uncommon”.

Results

Table 2 shows the data gathered at electrophysiological study. All stimulation sites were in the right atrium. There was a type I response to atrial stimulation site in 16 episodes (81%) at 24 atrial sites. No response to attempted conversion was found at eight sites (mid-lateral 1,

low lateral 1, high anterior 2, mid-anterior 2, mid-septal 1, mid-posterior, 1). The distribution of the 16 type I sites at which conversion occurred was: high lateral 3, mid lateral 2, low lateral 2, high anterior 2, mid-anterior 3, low anterior 1, mid-septal 1, high posterior 1, mid-posterior 1. In addition, three type II sites were found in these 17 episodes (mid-septal 1, low septal 1, high anterior 1). Conversion was not attempted at these latter sites.

Only type II responses were found at nine sites in five episodes. Conversion did not occur at four sites (mid-lateral 1, high anterior 1,

Table 2 Electrophysiological data on individual episodes

Case	Age	Sex	Current treatment	Duration of flutter	Flutter type	Atrial rate	Ventricular rate	Stimulation site in RA
1	17	M	Quinidine	2 days	Common	304	152	Mid-lateral
2	18	M	Amiodarone	1 day	Common	221	110	Mid-lateral
3	33	F	Nil	3 wk	Common	271	135	High lateral
4	20	F	Nil	5 yr	Common	218	109	Low lateral
5	15	M	Amiodarone	8 h	Uncommon	181	80	High anterior
6	31	F	Digoxin + quinidine	2 mnth	Uncommon	363	110	Low lateral
7	22	F	Nil	Unknown	Uncommon	206	103	Mid lateral
8	34	M	Nil	3 mnth	Common	252	126	Low septal
9	22	F	Digoxin + amiodarone	Unknown	Common	166	83	Low posterior
10	28	M	Amiodarone	18 mnth	Common	219	109	Mid posterior
11	31	M	Amiodarone	4 days	Common	176	88	High posterior
12	22	F	Amiodarone	2 days	Common	300	150	Low lateral
13	31	M	Digoxin + quinidine	2 wk	Common	206	75	High lateral
14	25	M	Amiodarone	Unknown	Common	172	172	High anterior
15	34	M	Digoxin + disopyramide	7 days	Common	237	79	Mid-anterior
16	31	M	Digoxin + disopyramide	7 days	Common	188	63	Mid-anterior
17	19	F	Digoxin	45 days	Uncommon	263	98	High anterior
18	31	M	Xamoterol	14 days	Common	330	110	Mid-anterior
19	24	M	Amiodarone	3 mnth	Common	158	79	High anterior
20	43	F	Digoxin + flecainide	4 mnth	Common	233	82	Low anterior
21	33	F	Digoxin	6 mnth	Uncommon	256	128	High lateral

mid-anterior 1, high septal 1). Conversion occurred at the remaining five sites (mid-lateral 1, low lateral 1, high anterior 2, low posterior 1).

Single atrial stimuli did not convert any episode of flutter. Conversion to sinus rhythm was achieved with programmed stimuli in six episodes. Figure 2 shows an example of conversion with two stimuli. Rapid atrial pacing resulted in conversion to sinus rhythm in 11 episodes (figure 3 shows one) and to atrial fibrillation in two. One of these latter two episodes had the most rapid atrial rate (363 beats per minute) and endocardial mapping showed coexisting areas of atrial fibrillation and flutter not suspected from the surface electrocardiogram; sinus rhythm spontaneously resumed 12 hours later. In the other episode where atrial fibrillation ensued, DC cardioversion was carried out immediately because the patient had documented sinus node disease and the atrial catheter would have allowed backup atrial pacing if required.

The remaining two episodes in one patient were converted to sinus rhythm by a single long constant current pulse ("atrioversion"⁸). Fifteen of the 19 conversions to sinus rhythm were at type I atrial sites.

One conversion to sinus rhythm after rapid atrial pacing was followed by profound, sustained sinus bradycardia, requiring atrial support pacing and subsequently isoprenaline infusion to maintain an adequate rate. The patient was subsequently treated with oral xamoterol, but had a further recurrence of atrial flutter two months later and was again successfully converted to sinus rhythm. Xamoterol was stopped and an atrial demand pacemaker was implanted.

Overall, 19 episodes (90%) were returned directly to sinus rhythm during the procedure. One patient, who had coexisting areas of atrial fibrillation not suspected from the surface electrocardiogram and was converted to atrial fibrillation, subsequently reverted to sinus rhythm after 12 hours. Clinical success was thus achieved in 20 episodes (95%). In all patients the clinical state improved.

Discussion

In patients with one functioning ventricle, as in tricuspid atresia or double inlet ventricle, the onset of atrial flutter causes a considerable symptomatic deterioration. Atrial flutter is also dangerous in other complex lesions, such as

Figure 2 Conversion of atrial flutter to sinus rhythm by two premature atrial stimuli. Four surface electrocardiographic leads and two intracardiac electrograms showing atrial flutter with a cycle length of 220 ms. Two extrastimuli (coupling times 140 ms and 180 ms) caused immediate reversion to sinus rhythm.

Response type	Conversion method	Result of conversion	Details of conversion
Type I (reset)	Atrioversion	Sinus rhythm	
Type I (reset)	Atrioversion	Sinus rhythm	
Type I (reset)	Extrastimuli	Sinus rhythm	180/170 ms
Type I (reset)	Extrastimuli	Sinus rhythm	170/140 ms
Type II (non-reset)	Rapid atrial pacing	Sinus rhythm	CL 165 ms for 4 s
Type II (non-reset)	Rapid atrial pacing	Atrial fibrillation	CL 100 ms for 8 s
Type II (non-reset)	Extrastimuli	Sinus rhythm	270/250 ms
Type I (reset)	Extrastimuli	Sinus rhythm	155/140/140/140 ms
Type I (reset)	Extrastimuli	Sinus rhythm	270/210 ms
Type I (reset)	Rapid atrial pacing	Sinus rhythm	CL 200 ms for 4 s
Type I (reset)	Extrastimuli	Sinus rhythm	240/240/290 ms
Type I (reset)	Rapid atrial pacing	Sinus rhythm	CL 210 ms for 8 s
Type I (reset)	Rapid atrial pacing	Sinus rhythm	CL 200 ms for 4 s
Type II (non-reset)	Rapid atrial pacing	Sinus rhythm	CL 290 ms for 8 s
Type I (reset)	Rapid atrial pacing	Sinus rhythm	CL 160 ms for 4 s
Type I (reset)	Rapid atrial pacing	Sinus rhythm	CL 230 ms for 30 s
Type I (reset)	Rapid atrial pacing	Atrial fibrillation	CL 160 ms for 4 s
Type I (reset)	Rapid atrial pacing	Sinus rhythm	CL 150 ms for 8 s
Type I (reset)	Rapid atrial pacing	Sinus rhythm	CL 300 ms for 2 s
Type I (reset)	Rapid atrial pacing	Sinus rhythm	CL 170 ms for 4 s
Type I (reset)	Rapid atrial pacing	Sinus rhythm	CL 180 ms for 2 s

CL, cycle length.

tetralogy of Fallot. It is common in the third and fourth decades in both operated and unoperated patients with various defects of the atrial septum and anomalies of pulmonary venous drainage. As in all symptomatic arrhythmias two strategies are required. The first is prevention, which includes exclusion of a predisposing structural lesion, such as obstruction in a conduit connection used in the Fontan or right ventricular outflow reconstruction procedures. The second is restoration of sinus rhythm during the acute paroxysm. The major disadvantages of external DC cardioversion are that general anaesthesia is needed and that the myocardium of an already abnormal ventricle may be damaged. The attractions of endocardial conversion are obvious. The technique, however, requires that there is good venous access and that the atrial arrhythmia is regular. It cannot be applied to atrial fibrillation. In one of the two episodes in the present series where atrial fibrillation resulted from conversion, endocardial atrial mapping showed distinct

coexisting areas of flutter and fibrillation that were not evident on the surface electrocardiogram. Conversion to sinus rhythm was thus unlikely in this episode.

For this study we selected patients with regular and rapid atrial arrhythmia. Patients with anomalies of systemic venous drainage were excluded owing to difficulties of access. Otherwise, it is our policy to start anticoagulation with heparin and to proceed to electrical conversion as soon as is possible. Appropriate antiarrhythmic medication to prevent recurrences is instituted or continued after conversion. We advise patients to return immediately if a further paroxysm occurs.

In the five instances in which the atrial rate was below 200 beats per minute during flutter, concomitant drug treatment accounted for the relatively slow atrial rate. A gradual lengthening of the flutter cycle without a change in flutter wave configuration was seen on sequential electrocardiographic recordings after treatment was started. Because this effect was consistent with the electrophysiological effect of amiodarone and disopyramide we thought that the mechanism of these slower rhythms must be identical and that they should remain classified as atrial flutter.

As well as having a high success rate, endocardial paced conversion of atrial flutter allows immediate atrial support pacing when there is sinus arrest after conversion—a facility not available during external cardioversion. This proved essential in one episode where profound sinus bradycardia followed conversion.

All of the study group had had previous corrective surgery, usually at an early age. None had a history of atrial arrhythmias before operation, and it is probable that the surgery contributed in a major way to creation of the substrate for their atrial flutter.

The procedure was highly successful in this group of patients. The mean procedure time

Figure 3 Conversion of atrial flutter to sinus rhythm by rapid atrial overdrive pacing. Four surface electrocardiographic leads and two intracardiac electrograms from the low lateral right atrium (LLRA) show the last four beats of an 8 s overdrive pacing train (cycle length 210 ms). After the end of pacing there were two atrial depolarisations, the first of which may be sinus in origin, followed by clear resumption of sinus rhythm.

was an hour, and theoretically the procedure could be carried out at the bedside under echocardiographic control. We believe that intracardiac electrical conversion is the best treatment for atrial flutter in patients with congenital heart disease because it avoids general anaesthesia and external cardioversion.

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