

## Treatment of acute atrial fibrillation: ventricular rate control and restoration of sinus rhythm

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Atrial fibrillation (AF) is a familiar arrhythmia seen in the emergency department and the general population. In the past it was treated in the majority of cases by controlling the ventricular rate, whether the AF is acute or chronic. However, ventricular rate control alone does not address the underlying problem and the patients still remain in AF, cardiac output and symptoms have not been optimally corrected. There is definite risk of thromboembolism. Restoration of sinus rhythm is the only way of resuming the normal conduction physiology of the heart and correcting these problems. This article provides a review of the two major principles of rhythm treatment of acute AF: rate control and restoration of sinus rhythm. Transthoracic electrical cardioversion is the mainstay of treatment in haemodynamically unstable AF, whereas in stable AF, there is a choice between rate control and restoration of sinus rhythm, or they can be carried out in conjunction with each other. (*Hong Kong j.emerg.med.* 2000;7:85-95)

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### Introduction

Atrial fibrillation (AF) is the most common arrhythmia encountered across in the emergency department and the general population. It is present in 0.5% of the whole population, 2 to 5% of those aged over 60 years,<sup>1,2</sup> and constitute 34.6% of emergency hospitalisation of patients with arrhythmias in the United States.<sup>3</sup> It is usually haemodynamically tolerable and probably as a result of this, the treatment is often suboptimal both in the acute and chronic setting.

If left untreated, AF will lead to a variety of complications. It causes asynchronisation of atrial and ventricular contraction, impaired atrial contribution to ventricular filling, poor ventricular diastolic filling and increased myocardial oxygen consumption from fast heart rate. Patients may be

asymptomatic or symptomatic. Circulatory failure and shock are uncommon but sometimes recognised in those with Wolff Parkinson White syndrome, tight mitral stenosis or pre-existing conditions that markedly jeopardised left ventricular function.<sup>4-7</sup>

It is well established that AF is a risk factor in the development of stroke. Besides stagnation of blood flow in the dilated atria, it is claimed that abnormal clotting profile with elevated D-dimer concentration in the blood, rise in atrial natriuretic peptide and hematocrit also account for the higher risk of embolism in AF.<sup>8,9</sup> The risk of stroke is 5% per year,<sup>5,10</sup> and becomes higher if there are concurrent left atrial dilation, mitral stenosis, hypertension, coronary heart disease and left ventricular dysfunction. Old age itself predicts a greater likelihood of cerebral thromboembolic events, up to 23.5% in those 80 to 89 years old.<sup>5</sup>

The incidence of stroke is probably not higher in patients under 60 years old with lone AF. AF of recent onset (less than 48 to 72 hours) does not impose an excessive risk of atrial thrombus formation and stroke.<sup>11-13</sup> Table 1 summarises the complications of untreated AF.

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**Table 1.** Complications of untreated AF.

Complications	Underlying rationale
Reduced cardiac output	Atrioventricular asynchronisation
Ischaemic stroke	Stagnation of atrial blood flow; concurrent risk factors: left atrial dilatation, mitral stenosis, left ventricular dysfunction, hypertension, coronary heart disease, old age
Perpetuation of AF	Electrophysiologic remodelling
Tachycardiac cardiomyopathy	Fast and irregular ventricular response of AF

AF will perpetuate AF by means of inducing electrophysiological remodelling in the atria.<sup>11,14</sup> Chronic AF does not respond well to therapy and is unlikely to revert to sinus rhythm spontaneously.

Persistent AF will result in tachycardiac cardiomyopathy which in turn makes the patients more prone to AF, creating a vicious cycle.<sup>4,7</sup> The importance is that this cardiomyopathy is reversible with restoration of sinus rhythm and proper ventricular rate control.

When a patient presents with uncontrolled AF, accomplishment of haemodynamic stability is the primary goal, followed by ruling out emergent primary cause like acute myocardial infarction, acute surgical abdomen and gastrointestinal bleeding. Afterwards, the main modalities of treatment include control of ventricular rate, restoration of sinus rhythm and anticoagulation. Anticoagulation is not as urgent as the others in the emergency department and is seldom necessary in AF of recent onset. This will not be discussed here.

### Ventricular rate control

Decreasing the ventricular rate in uncontrolled AF will certainly improve symptoms, enhance ventricular diastolic filling and lower the chance of neurological complications, in particular those with pre-existing impaired cerebral blood supply.

#### **Calcium channel blockers**

Diltiazem in 0.25 mg/kg intravenously over 2 minutes is effective in slowing the ventricular response. If that fail, a second dose of 0.35 mg/kg is

administered 15 minutes later. A continuous infusion of 5 to 15 mcg/kg/min adjusted according to the ventricular rate is recommended in the light of its short duration of action. Diltiazem is effective in 80%<sup>15-18</sup> of cases in reducing the ventricular rate by 30% within 3 to 4 minutes after bolus injection.<sup>12,14-17</sup> It produces less negative inotropic effect when compared to verapamil, but its usage should still be precluded in conditions of hypotension and significant left ventricular dysfunction. Severe bradycardia or high grade atrioventricular block may be induced in patients with sick sinus syndrome.

Verapamil as a 2.5 to 5 mg intravenous injection over 2 to 4 minutes (the lower dose and slower rate of injection if elderly or borderline low blood pressure) is another calcium channel blocker in use but it has more negative inotropic effect although it cost less. The effectiveness and precautions are similar to that of diltiazem.

#### **Beta blockers**

The efficacy of beta blockers are similar to calcium channel blockers. They are deemed particularly effective if the arrhythmia results from heightened sympathetic tone and endogenous catecholamine release, for example, thyrotoxicosis. Post myocardial infarction AF is another situation where beta blockers are indicated. Beta blockers should be avoided in the settings of hypotension, congestive heart failure, bronchospastic disease or concomitant intravenous calcium channel blockers administration.

The non-cardioselective propranolol is administered in 1 mg intravenously over 2 minutes and repeated

if necessary up to total 5 mg at intervals of 2 minutes. Examples of cardioselective beta blockers include metoprolol and esmolol. The dosage of metoprolol is 5 mg intravenously given over 2 minutes and repeated every 5 minutes until a cumulative dose of 15 mg. The dosage of esmolol is 250 to 500 mcg/kg given over 1 minute intravenously with a subsequent maintenance infusion starting at 25 to 50 mcg/kg/min to a maximum of 300 mcg/kg/min titrated according to ventricular rate.<sup>19</sup> The ultra-short elimination half life of esmolol at 9 minutes is the result of its poor lipid solubility making any side effects like hypotension or heart failure rapidly reversible once it is discontinued. On the other hand, recurrence of arrhythmia may happen and this explains the necessity of the maintenance infusion.<sup>11,12,18</sup> Its cost is higher than propranolol and metoprolol.

### **Digoxin**

Digoxin has a long-standing history in the therapy of AF, in both acute and chronic form. Its positive inotropic effect causes it to be valuable in AF associated with heart failure. The onset of action is delayed even after intravenous administration. The therapeutic effect becomes apparent after 30 minutes and does not reach a peak in the first 2 hours. The action of digoxin relies predominantly on the vagotonic effect on the atrioventricular node. This mechanism of action is exemplified by its disappointing effectiveness in hyperadrenergic clinical states, for instance, fever, thyrotoxicosis, pain and many other situations of shock.<sup>7,11,14</sup>

There were inadequate well controlled clinical trials in the past assessing the efficacy of digoxin in the suppression of ventricular rate in uncontrolled acute AF. This traditional role of digoxin has recently been questioned.<sup>20,21</sup> In the chronic setting, digoxin slows down the resting heart rate but does not offer protection against acute exacerbation of AF during exertion when the sympathetic discharge overcomes the vagal tone.

### **Amiodarone**

Amiodarone exerts its Class III effect during chronic oral therapy. When given intravenously in acute condition, it behaves like beta blocker or calcium channel blocker, which together with its sodium and potassium channel blocking properties, and its

antiadrenergic mechanism, slows the ventricular response by its action on the atrioventricular node. It decreases the ventricular rate by 25% in 1 hour and does not impose obvious haemodynamic adverse effects.<sup>22</sup> The negative inotropic effect is not significant and is the drug of choice in the setting of significant structural heart disease. Amiodarone is indicated when control of the AF is unsuccessful with other antiarrhythmic medications.

### **Magnesium**

When compared with digoxin alone, intravenous magnesium sulphate given in combination with digoxin may be useful in achieving faster and greater ventricular rate control through its action on atrioventricular node with no significant side effects.<sup>23,24</sup> Magnesium is probably a weaker drug than verapamil. There are by far few studies designed to evaluate the role of magnesium in AF treatment. Table 2 summarises the various drugs used intravenously to control the ventricular response rate in acute AF.

### **Restoration of sinus rhythm**

Ventricular rate control alone still leaves the patients in AF. It is only the restoration of sinus rhythm that restore the normal electrophysiology of the heart and prevent the complications of the original arrhythmia. The heart rate is regularised at a physiological rate so that the diastolic filling of the ventricles is normalised and the atrial contribution is optimised from the atrioventricular synchronisation. Cardiac output will then be augmented and subsequently improves the haemodynamic parameters and symptoms like palpitation, dyspnoea and malaise. Exercise tolerance and quality of life have shown to improve as a result.

Restoration of sinus rhythm interrupts the vicious cycle of electrophysiologic remodelling induced by AF. If AF is allowed to persist, it becomes more difficult to treat. In AF a higher atrial pressure is generated to compensate for the poor ventricular diastolic filling arising from irregular and fast ventricular rate. The resultant dilation of atria on its own may precipitate both AF and atrial thrombus formation. Through regularisation of the heart rate

**Table 2.** Various intravenous drug treatment for controlling the ventricular rate of acute AF.

Drugs	Remarks
Calcium channel blockers: diltiazem, verapamil	-Effective -Both have similar efficacy but diltiazem possesses less negative inotropic effect
Beta-blockers	-Effectiveness similar to calcium channel blockers -Particularly indicated in thyrotoxicosis and acute myocardial infarction
Digixin	-Delayed onset of action -Less effective in hyperadrenergic states -Few well controlled studies documenting its value
Amiodarone	-Slow onset of action -Effective and indicated after failure of other medications
Magnesium sulphate	-Few studies conducted -May potentiate digoxin's therapeutic effect

as well as lessening of the degree of atrial dilation, sinus rhythm restoration will definitely reduce the risk of thromboembolism and ischaemic stroke. The risk of stroke is lowered by two-third in patients who have been anticoagulated.<sup>25</sup> However, anticoagulation results in bleeding complications (1.3% of major haemorrhage and 2.8% of minor bleeding),<sup>4</sup> requires regular monitoring causing inconvenience to the patients and medical staff, and careful attention is needed to avoid drug interaction. It is clear that converting AF back to sinus rhythm will obviate the need and complications of anticoagulation.

Uncontrolled AF and other forms of tachycardia are postulated to cause a reversible condition called tachycardiac cardiomyopathy especially if the AF is prolonged, and is further complicated by the poor result of conversion back to sinus rhythm. The tachycardia leads to left ventricular dysfunction which perpetuates the AF. Apart from the fast ventricular response, the irregular rhythm in AF may play a role in contributing to the cardiomyopathy. Restoration of sinus rhythm enables correction of the rate and regularisation of the ventricular contraction.<sup>4,7</sup>

Early after restoration of sinus rhythm, cardiac function in terms of stroke volume, and left ventricular ejection fraction is improved due to increased end-diastolic volume, decreased end-

systolic volume and increased mean cycle length.<sup>25</sup> However, the cardiac contractility does not return to normal immediately despite a normal electrical rhythm. It is often delayed by days to months (longer for longstanding AF) for the gradual resolution of the tachycardiac cardiomyopathy. This resultant atrial stunning creates a vulnerable period after cardioversion for the development of atrial thrombus and cerebral embolism.

Cardioversion is not without risk, restoration of sinus rhythm leads to improved cerebral blood flow, carrying emboli to the cerebral vasculature.<sup>26</sup> The resumption of efficient atrial contraction may also dislodge the thrombus formed during the period of AF. As the return of atrial mechanical function may be delayed up to a few weeks, events of systemic embolism is possible within this period. It is not sure whether the post-cardioversion anticoagulation should be extended to cover such a long period, although by experience most of the embolism occur within the first week.<sup>26</sup> Patients with recent onset AF (less than 48 to 72 hours) do not have higher chance of atrial thrombus formation and systemic embolism. Attempting cardioversion in this group of patients does not require prior anticoagulation. Otherwise a 3 week anticoagulation with warfarin is recommended in AF of longer duration.<sup>11,12,27</sup> Transoesophageal echocardiography provides an excellent view of the left atrial appendage where thrombus is likely to occur and transthoracic

echocardiography does not visualise well. It is a valuable imaging technique of reassuring the clinicians the absence of atrial thrombus before proceeding to cardioversion.<sup>12,14,28,29</sup>

Cardioversion is classified into electrical and pharmacological. Each has its merit and drawback in stable AF, but electrical cardioversion is additionally indicated in haemodynamic instability such as hypotension, congestive heart failure, impaired sensorium and ischaemic chest pain. Table 3 summaries the benefits and risks of cardioversion of AF.

## Electrical cardioversion

### *Transthoracic electrical cardioversion*

This is the usual choice in electrical cardioversion. It is immediately effective in nearly 90% of cases in achieving sinus rhythm restoration.<sup>13,30,31</sup> Long duration of AF (the paramount patient factor, particularly more than 3 years), large atrial diameter (worst in those more than 6 cm on parasternal long axis view), old age and preexisting mitral stenosis with severe rise in left atrial pressure are the factors hindering the efficacy of cardioversion.

Appropriate sedation is required, aiming at adequate pain relief and providing amnesia. Electrodes should be placed widely apart to avoid shunting of electric current. The breast of female patients and bony structures like the scapula have high electrical impedance and electrode placement over them is not recommended. The starting energy level is 100 J (in the past it was 50 J) because the atria are relatively posterior structure. The energy level is

escalated in a stepwise fashion to 200, 300 and 360 J if necessary. Failed cardioversion will result from under or over electrical current. Refibrillation may be the rationale in the latter case.<sup>32</sup> Overall in patients with AF, 50 % of them will be cardioverted at 100 J and 85 % at 200 J.<sup>13</sup> If cardioversion is not attained with maximum energy level, the electrode position should be changed from the usual anterior-apex position to the less convenient anterior-posterior position (anterior electrode over left parasternal area, posterior electrode between thoracic spine and scapula) because more electrical current traverses through the ventricles rather than the atria in the former position.<sup>13,27</sup>

Deleterious effects due to transthoracic electrical cardioversion are usually few. According to the 3 studies each allocating 204 to 246 patients by Gelder, Aberg and Resnekov,<sup>31,33-35</sup> the complications were mainly arrhythmias and thromboembolism. The arrhythmias were: ventricular arrhythmia 0 to 1.6% consisting of nonsustained ventricular tachycardia and ventricular fibrillation; atrial arrhythmia including atrial tachycardia 0.8%, circus movement tachycardia 0.4%, atrioventricular nodal tachycardia 1.2%, asystole in those with underlying sick sinus syndrome 0.5 to 1.2%, 2<sup>nd</sup> degree sinoatrial block 0.4%, symptomatic atrioventricular nodal rhythm 0.8%. The arrhythmias were considered cardioversion related if they occurred up till 24 hours postcardioversion. Thromboembolism occurred from 0.4 to 1% within the first week after cardioversion. (Table 4)

Transthoracic electrical cardioversion was shown not to induce myocardial damage in recent studies using cardiac troponin T (TnT) as a marker for myocardial

**Table 3.** Benefits and risks of cardioversion of AF to sinus rhythm.

Benefits	Risks
Improved cardiac output	Postcardioversion atrial stunning and subsequent thromboembolism, usually in AF more than 48-72 hours duration
Interrupts electrophysiologic remodelling	Requirement and risks of preconversion anticoagulation in AF more than 48-72 hours duration
Reduced incidence of ischaemic stroke	
Avoids need and risks of anticoagulation	Complications peculiar to electrical cardioversion, side effects of drugs for chemical cardioversion
Decreases tachycardiac cardiomyopathy	

**Table 4.** Complications of transthoracic electrical cardioversion.

Complications	Percentage
a) Arrhythmias (within 24 h post-conversion)	
-ventricular arrhythmia	0 - 1.6
-atrial arrhythmia	
-atrial tachycardia	0.8
-circus movement tachycardia	0.4
-atrioventricular nodal tachycardia	1.2
-asystole (in preexisting sick sinus syndrome)	0.5 - 1.2
-2 <sup>nd</sup> degree sinoatrial block	0.4
-symptomatic atrioventricular nodal rhythm	0.8
b) Thromboembolism (within 1 week of post-conversion)	0.4 - 1

injury.<sup>36,37</sup> TnT is a regulatory protein controlling the actin-myosin of the myocardial contractile fibres. Other troponin subunits are troponin I (TnI) and troponin C (TnC). TnT is not detectable in the blood of normal individuals. It is very sensitive and more specific than the conventional cardiac markers such as total CK and CK-MB. Its ongoing tremendous release from the damaged myocardium accounts for its persistent peak in the blood for 3 to 5 days after acute myocardial infarction. Besides acute myocardial infarction, TnT is also elevated in unstable angina and bears prognostic implication in ischaemic heart disease. TnI is even more specific than TnT, without the false positive value of TnT in the presence of chronic renal failure and muscle disease. In the study by Neumayr, TnT remained undetectable after cardioversion with average and maximum energy of 223 J and 1370 J respectively.<sup>36</sup> Another study by Grubb revealed similar result, with no significant rise of TnT after cardioversion.<sup>37</sup>

#### **Transcatheter internal electrical cardioversion**

This treatment modality has increasingly been studied recently for conversion of AF that failed to respond to transthoracic electrical cardioversion. The success rate of the latter is limited in chronic AF, dilated atria, high impedance caused by obesity, strong body build and emphysema. The reported success rate is 70 to 93%.<sup>38-42</sup> These studies enrolled patients mainly with chronic AF, so the overall conversion rate may even be higher in the recent onset AF. The low energy requirement of internal cardioversion accounts for its safety and complications from defibrillation itself are rarely reported.

#### **Pharmacological cardioversion**

Pharmacological cardioversion is not a very popular treatment modality yet. Studies up till now usually involve sample less than 100 patients. Despite this, there are a number of well controlled studies proving it to be effective in AF especially those of recent onset.

##### **Flecainide**

Flecainide in 2 mg/kg intravenously over 10 minutes is effective in 59 to 92% of cases if the onset of AF is less than 24 to 72 hours (Table 5) Side effects are usually minor, like dizziness, nausea, transient hypotension and bradycardia. Occasionally the hypotension is marked and warrants discontinuation of treatment.

As mentioned before, the shorter the duration of AF, the higher is the success of cardioversion. This is illustrated by several trials using flecainide. (Table 6)

Capucci undertook a trial comparing single oral flecainide 300 mg to intravenous amiodarone and placebo, the conversion rate of flecainide within 8 hours was 91% which was superior to that of amiodarone (37%) and placebo (48%). The mean conversion time of oral flecainide was 190 ± 147 minutes, much longer than that of intravenous flecainide.<sup>50</sup>

##### **Propafenone**

Propafenone, another Class Ic antiarrhythmic drug

like flecainide, converted AF to sinus rhythm with a similar efficacy in controlled trials. (Table 7) The dosage is the same as flecainide, 2 mg/kg/10 min intravenously. Oral propafenone is also effective but the onset of action is slower. Side effects resemble that of flecainide.

Ganau performed a multicentre trial in the emergency departments in Italy with a sample of 156 patients. Intravenous propafenone 2 mg/kg/10 min was tested against placebo for AF of less than 72 hours. The conversion rate by 2 hours for propafenone and placebo was 70.3% and 17.3% respectively. Side effects were observed in 2.5% of all the patients, including hypotension, bradycardia, nausea, conversion to atrial flutter with 2:1 block. All responders were discharged home unless those in poor condition and with drug side effects.<sup>56</sup>

Direct comparison between intravenous flecainide and intravenous propafenone was conducted by

Suttorp.<sup>57</sup> Both drugs were instituted at 2 mg/kg/10 min. Flecainide was found to be better than propafenone in converting AF to sinus rhythm. For AF of less than or equal to 24 hours, the conversion rate for flecainide and propafenone was 93% and 57% respectively. The conversion rate for AF that was more than 24 hours was 83% for flecainide and 50% for propafenone. Adverse effects occurred more frequently in flecainide treated patients, 42% against 8% of the propafenone group. The frequency of side effect of flecainide was significantly higher than other studies, but they were all mild and did not interrupt the continuation of treatment.<sup>57</sup>

Oral flecainide 300 mg and oral propafenone 600 mg as a single dose were shown to possess comparable success for AF less than 7 days duration. The conversion rate at 3 hours was 59% for flecainide and 51% for propafenone. At 8 hours, it was 78% and 72% for flecainide and propafenone respectively. Both of them were more potent than placebo.<sup>58</sup>

**Table 5.** Conversion rate of flecainide compared with controls in recent onset AF.

	AF duration	Conversion rate	
		Flecainide	Control
Madrid <sup>43</sup>	Less than 24 hours	92%	65% (procainamide)
Crijns <sup>44</sup>	Less than 24 hours	77%	-----
Reisinger <sup>45</sup>	Less than 24 hours	69%	31% (sotalol)
Donovan <sup>46</sup>	Less than 24 hours	53%	34% (amiodarone)
			2% (placebo)
Suttorp <sup>47</sup>	Less than 24 hours	86%	6% (verapamil)

**Table 6.** Conversion rate of flecainide depending on the AF duration.

	Conversion rate (AF duration)	
Goy <sup>48</sup>	79% (<10 days)	38% (>10 days)
Borgeat <sup>49</sup>	86% (<10 days)	22% (>10 days)
Crijns <sup>44</sup>	77% (<24 hours)	0% (>24 hours)

**Table 7.** Conversion rate of propafenone compared with controls in recent onset AF.

	AF duration	Conversion rate	
		Propafenone	Control
Bianconi <sup>51</sup>	< 72 h	50%	25% (digoxin)
Botto <sup>52</sup>	< 72 h	57%	25% (digoxin)
Botto <sup>53</sup>	< 72 h	46-57%	17% (placebo)
Fresco <sup>54</sup>	< 72 h	58.5%	29.4% (placebo)
Lavanga <sup>55</sup>	< 72 h	56%	0% (placebo)

**Ibutilide**

Ibutilide is new Class III antiarrhythmic drug. It prolongs the action potential by means of blocking the rapid component of the outward delayed rectifier potassium current  $I_{kr}$ . Another action of ibutilide is activation of the slow inward sodium current during the plateau phase of action potential, a process independent of the  $I_{kr}$ . These decreased  $I_{kr}$  and increased sodium influx may be observed in the congenital long QT syndrome. Ibutilide induces prolonged QTc interval dependent on the dose and blood level. Any condition predisposing to torsade de pointes should be corrected before administering ibutilide.

Ibutilide undergoes extensive hepatic metabolism and the metabolites are largely excreted through the kidney. The necessity of dose reduction in face of hepato-renal impairment is unknown, but it is theoretically safer to monitor these patients longer after ibutilide treatment.

Ibutilide shows satisfactory results in relatively longstanding AF, (Table 8) but it appears that the data for recent onset AF is limited. The results in the present studies may be extrapolated to recent onset AF but this remains to be verified. An important side effect of ibutilide is torsade de pointes, with an incidence of 1.6 to 8.3%. The torsade de pointes are usually nonsustained and subsides spontaneously, but some of them require treatment like direct current cardioversion or magnesium sulphate injection. A post-ibutilide injection observation of 4 hours or until the return of baseline QTc is necessary as this is the period (particularly the first 40 minutes) that torsade de pointes may occur. Other adverse effects are rare. The current recommended dosage of ibutilide

is 10 mg over 10 minutes and that same dosage is repeated after 10 minutes if there is no response.<sup>59-61</sup>

**Procainamide**

This is a drug with long history but most of the studies are not well controlled to justify a reliable and consistent result.<sup>66-68</sup> It is less efficacious according to the studies comparing it with Class Ic drugs and ibutilide aforementioned. The unwanted effects of procainamide are hypotension, arrhythmogenicity and prolonged infusion up to 1 hour.

**Amiodarone**

It has not been shown convincingly to possess higher conversion rate than the comparison agents in several trials.<sup>69-73</sup> The Class III effect manifested in chronic treatment probably does not occur in acute intravenous administration.

**Discussion**

Based on the benefits of restoring sinus rhythm in patients with AF, such as alleviating symptoms, optimising cardiac output, obviating anticoagulation and preventing electrophysiological remodelling, the treatment may advance from the present predominantly rate control to targeting conversion to sinus rhythm as much as possible in the foreseeable future. If AF is encountered early in the onset, cardioversion should be attempted, if feasible, without delay in view of the non-necessity of anticoagulation and higher success rate.

Problems are still present and required to be tackled before recommending widespread practice of cardioversion of stable AF to sinus rhythm. The

**Table 8.** Comparison of ibutilide with controls in conversion of AF to sinus rhythm.

	AF duration	Conversion rate	
		Ibutilide	Control
Stambler <sup>62</sup>	23 days (mean)	32%	5% (procainamide)
Volgman <sup>63</sup>	3 h to 90 days	51%	21% (procainamide)
Stambler <sup>64</sup>	3 h to 45 days	46% (<7 days) 8% (>7 days)	2% (placebo)
Ellenbogen <sup>65</sup>	3 h to 90 days	42% (<30 days) 16% (>30 days)	3% (placebo)

ideal chemical agent for cardioversion has yet to be confirmed and accepted universally. The new Class III drug ibutilide is documented to be quite satisfactory in restoring AF of longer duration to sinus rhythm. After more studies specific in recent onset AF, ibutilide may be the option for this category of patients. Postcardioversion thromboembolism due to stunning of the atria may be a risk. The necessity of preventing this possible complication and the drug regimen to be used is not comprehensively delineated. The availability of transoesophageal echocardiography for ruling out atrial thrombus will be strongly appreciated by the clinicians. Unfortunately it is not widely available and requires expertise to operate it. AF will relapse in a certain proportion of patients after successful cardioversion, but the indication, choice and time of commencing the antiarrhythmic drugs for maintaining sinus rhythm are not well defined for the time being.

Although Ganau demonstrated a high conversion rate using propafenone with few side effects, this was the only single reviewed study performed in the setting of emergency department.<sup>56</sup> It is still too early to recommend using this to restore sinus rhythm in patients having haemodynamically tolerable AF of recent onset in the emergency department. Before putting it into practice in the emergency department, the following issues should be resolved: the drug of choice for chemical cardioversion, the length of post-conversion observation period, the role of the post-conversion anticoagulation, the role of the sinus rhythm maintenance therapy and the availability of early referral to the cardiologists for follow up.

Despite the problems listed, early cardioversion of AF to sinus rhythm is a beneficial and practical issue. It will revolutionise the treatment of AF into a new era provided more studies are conducted.

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