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Can atrial flutter be converted to sinus rhythm by antiarrhythmic drugs or should atrial pacing be performed?

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Can atrial flutter be converted to sinus rhythm by antiarrhythmic drugs or should atrial pacing be performed?

Ch. Hief, A. Podczec, C. Klösch, K. Steinbach

There are serious deficiencies in our knowledge of the susceptibility of atrial flutter to drug therapy. Therefore feasibility and efficacy of antiarrhythmic drugs in patients with atrial flutter were evaluated in a prospective study.

A stepwise protocol was applied in 37 consecutive patients (30 men, 7 women, mean age: 65 ± 12 years) with permanent atrial flutter type 1. Step 1: digitoxine, 0.75 mg i.v., followed by 0.1 mg orally. Step 2: propafenone, 750 mg per day orally (3–4 days). Step 3: sotalol, 400 mg per day orally (44 days). Step 4: burst stimulation of the right atrium.

In 3 patients termination of atrial flutter by pacing was immediately needed because of haemodynamic compromise. In 22 of 37 patients (59 %) the stepwise approach was feasible. Atrial flutter converted to sinus rhythm on drug therapy in 12 patients (32 %). In 5 patients sinus rhythm was achieved with step 1, in one patient with step 2 and in 6 patients with step 3. In 10 patients atrial pacing was carried out resulting in sinus rhythm in 8 of them. Due to side effects of the drug, lack of compliance or haemodynamic compromise, the protocol was not completed in 12 patients. In 10 of these 12 patients burst stimulation was performed and restored sinus rhythm in all of them. Thirty-three of 37 (89 %) patients were discharged in sinus rhythm.

Our data showed that the success rate of antiarrhythmic drug therapy given orally in patients with stable type I atrial flutter was low, only 12 patients converted to sinus rhythm under drug therapy. *J Clin Basic Cardiol* 1999; 2: 78–80.

Key words: atrial flutter, antiarrhythmic drugs

Atrial flutter complicates a variety of cardiovascular diseases and may be secondary to various pathologic situations like atrial fibrillation in thyroid disease, alcoholism and following cardiac surgery. Therefore, most clinicians make little distinction between atrial fibrillation and atrial flutter [1, 2]. Consequently, little is known about the pharmacological drug effects on atrial flutter itself. Almost all the evidence comes from studies in which patients with atrial fibrillation and atrial flutter were pooled and in most of these studies the drug effects on atrial flutter cannot be analyzed separately. In only few studies was the efficacy of intravenous antiarrhythmic drugs in atrial flutter evaluated in small groups of patients [3–8]. One study [9] showed a conversion rate of ibutilide, a new class III antiarrhythmic drug, of 63 %. Therefore, we evaluated the efficacy of a drug regimen in patients with documented sustained atrial flutter. Antiarrhythmic drugs were given orally.

Methods

Our study population consisted of consecutive patients seen from November 1993 to December 1995, in whom sustained atrial flutter type I was documented for the first time. Atrial flutter was characterized by negative flutter waves on the ECG in leads II, III, aVF and by a stable cycle length of 250 ms to 170 ms. According to the criteria described by Wells et al. [10], all patients were considered to have type I atrial flutter. Patients with atrial flutter persisting for more than three hours were included. Exclusion criteria were Wolff-Parkinson-White syndrome, sick sinus syndrome, acute myocardial infarction and hyperthyroidism. The complete medical history, physical examination, routine biochemical laboratory testing, chest X-ray and 12 lead electrocardiogram were obtained before entering a patient into the study. Each patient underwent two-dimensional echocardiography.

Drugs were given in a stepwise protocol. First, digitoxine (0.75 mg intravenously) followed by oral administration was administered. If atrial flutter persisted, propafenone was given orally at a dose of 750 mg per day for 3 to 4 days. If propafenone

did not terminate atrial flutter, after a pause of 24 hours sotalol was given orally for 4 to 6 days, at a target dose of 400 mg per day. If atrial flutter did not convert to sinus rhythm on drug therapy, burst stimulation was performed from the high right atrium by a pacing electrode catheter which was introduced into a cubital vein or a femoral vein under fluoroscopy. A Medtronic 5326 stimulator was used and burst pacing was done over 5–10 sec, starting with a pacing rate of 10 % above atrial stimulation performed with an amplitude of 2 mA. The pacing rate was then increased in increments of 50 beats/min until atrial flutter was terminated or a maximum pacing rate of 800 beats/min was reached. We chose this high pacing rate because we preferred atrial fibrillation as an endpoint rather than continued atrial flutter. If no conversion occurred, the catheter was repositioned and the procedure was repeated, at a maximum of 3 sites. In patients with haemodynamic instability during atrial arrhythmia and a systolic blood pressure < 90 mm Hg, uncontrolled heart failure or symptoms of unstable angina or side effects of antiarrhythmic drug therapy, the stepwise protocol was stopped and burst stimulation was performed immediately. Data are presented as means \pm SD and $p < 0.05$ was considered significant. An unpaired t-test was used for analysis.

Results

Patient characteristics are summarized in Table 1. Systolic left ventricular function was normal in 31 patients (84 %) and reduced in 6 patients (16 %). Left atrial size was normal in 54 % of patients.

Treatment protocol (Fig. 1)

The protocol was not performed on 3 patients (8 %) due to haemodynamic compromise. In these patients burst stimulation was carried out immediately. In 12 patients atrial flutter converted to sinus rhythm during drug therapy and in 10 patients the protocol was completed through all steps. In 12 patients (32 %) the protocol was not completed due to side ef-

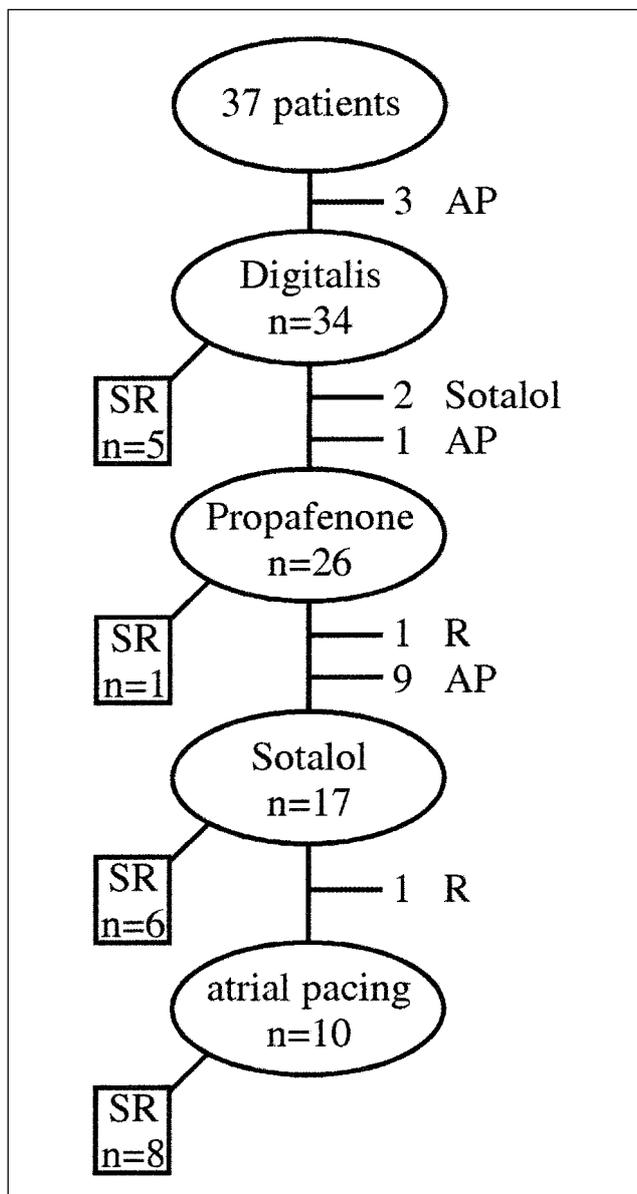


Figure 1. Efficacy of oral antiarrhythmics in patients with sustained atrial flutter. A step-wise protocol was performed (AP: atrial pacing was performed and terminated atrial flutter in sinus rhythm in all patients; R: patient refuses additional therapy).

fects of drug therapy (3 patients), due to haemodynamic compromise (4 patients) and to patient refusal (2 patients) or the doctors' refusal (3 patients).

Conversion rate (Table 2)

The success rate was 15% with digitalis, 4% with propafenone and 35% with sotalol. Burst stimulation was performed in 23 patients with continuation of atrial flutter. Three patients had no drug therapy, 1 patient received digitalis, 9 patients propafenone and 10 patients sotalol. Twenty-one of these patients (91%) converted to sinus rhythm. One patient was discharged with atrial fibrillation and one patient with atrial flutter.

Influence of atrial size and left ventricular function on the efficacy of drug therapy (Table 3)

Antiarrhythmic drug therapy was efficient in 40% of patients with a normal size of the left atrium and in 24% of patients with a left atrium which was greater than 40 mm. Eleven of

Table 1. Clinical characteristics of 37 study patients with first documented sustained atrial flutter type I

Mean age (yrs)	65 ± 12
Gender (male/female)	30/7
Etiology of heart disease	
No heart disease	n = 20 (54%)
Coronary artery disease	n = 7 (19%)
Hypertensive heart disease	n = 5 (14%)
Dilated cardiomyopathy	n = 3 (8%)
Constrictive cardiomyopathy	n = 1 (2.5%)
Valvular heart disease	n = 1 (2.5%)
Left ventricular function	
EF ≥ 60%	n = 31 (84%)
EF < 60%	n = 6 (16%)
Left atrial size (mm)	
≤ 40 mm	n = 20 (54%)
> 40 mm	n = 17 (46%)

Table 2. Efficacy of antiarrhythmic drugs in converting atrial flutter to sinus rhythm

	No. of patients	Efficacy n (%)
Digitalis	34	5 (15%)
Propafenone	26	1 (4%)
Sotalol	17	6 (35%)
Burst stimulation	23	21 (91%)

Table 3. Influence of atrial size and left ventricular function on the efficacy of drug therapy

	No. of patients	Efficacy n (%)
EF ≥ 60%	31	11 (35%)
EF < 60%	6	1 (17%)
LA > 40 mm	17	4 (24%)
LA ≤ 40 mm	20	8 (40%)

the patients (35%) with normal left ventricular function converted to sinus rhythm with drug therapy compared to one of the patients (17%) with reduced ventricular function. Drug treatment tended to be more effective in patients with normal left atrial size and normal left ventricular function, but this was not statistically significant.

Influence of heart disease on the efficacy of drug therapy

The presence of organic heart disease did not affect the success rate of drug therapy. 35% of the patients with heart disease and 30% of the patients without heart disease, i.e. 6 patients in each group, converted to sinus rhythm with antiarrhythmic drug therapy.

Discussion

In patients affected by atrial flutter conversion to sinus rhythm is usually warranted. Treatment depends on the severity of symptoms. In patients with hypotension, heart failure or angina, emergency electrical cardioversion or rapid atrial pacing are necessary. The majority of patients with mild symptoms is at least initially treated with drugs. Whereas atrial fibrillation is a common tachyarrhythmia in humans, atrial flutter is relatively rare. Both arrhythmias complicate a variety of cardio-

vascular diseases. This is probably why in most of the studies evaluating the efficacy of antiarrhythmic drugs patients with atrial fibrillation and atrial flutter were pooled [1, 2]. In most of these studies atrial tachyarrhythmias can thus not be analysed separately. So there is little information on drug efficacy in atrial flutter. In only few studies was the efficacy of antiarrhythmic drugs in atrial flutter evaluated in small groups of patients. In all of these studies drugs were given intravenously. Suttrop et al. reported a 40 % success rate for propafenone in 2 of 5 patients and a 20 % success rate for flecainide in 1 of 5 patients [4]. In the study of Bianconi et al. 33 % of 15 patients with atrial flutter successfully converted to sinus rhythm on propafenone [3]. These previous studies of class Ic antiarrhythmic agents suggested that conversion of atrial flutter is rare and occurs less frequently than in patients with atrial fibrillation. Intravenous dofetilide, a pure class III antiarrhythmic agent, had a greater success rate in patients with atrial flutter (56 % conversion rate) than in those with atrial fibrillation (14 % conversion rate). The efficacy rate of dofetilide in patients with atrial flutter was higher than the conversion rate to sinus rhythm with class Ic drugs [8, 11, 12]. Seven of 10 patients converted to sinus rhythm with dofetilide [8] and Falk et al. reported a 56% efficacy rate with dofetilide in patients with atrial flutter [12]. Stambler et al. [9] showed a 63 % success rate of ibutilide in patients with atrial flutter. Several factors complicate the assessment of the drug's clinical usefulness. Ibutilide is available as a short-term intravenous agent, but cannot be used for maintenance therapy. There is a high recurrence rate of atrial tachyarrhythmias if sinus rhythm is not maintained by drugs. Synergistic effects with other antiarrhythmics and a potential prolongation of the QT interval with proarrhythmia limit the early administration of other antiarrhythmics. Proarrhythmia was seen in 8.3 % of patients treated with ibutilide, mostly during infusions, but also three hours later. 1.7 % of patients treated with ibutilide had sustained polymorphic ventricular tachycardia. The high frequency of ventricular arrhythmias is a matter of concern: Patients selected for this study carried a low risk for these complications. Patients with chronic heart failure, recent myocardial infarction or angina pectoris were excluded and careful attention was paid to normal potassium levels and normal baseline QTc. Our data reflect a cohort of patients with their first documented permanent so-called type I or common atrial flutter. To our knowledge, it is the first study in which a stepwise treatment protocol with oral antiarrhythmic drugs was used. The success rates achieved were 15 % with digitalis, 4 % with propafenone and 35 % with sotalol. Thus only 12 of 37 patients (32 %) converted to sinus rhythm on drug therapy. Overdrive pacing was effective in 91 %. The majority of patients who underwent overdrive stimulation in our cohort was on antiarrhythmic therapy (20 of 23 patients; 87 %). Compared to other studies, the success rate of pacing was very high [13–15]. Della Bella et al. reported a success rate of 20 % without drugs, of 50 % with oral disopyramide and of 70 % with intravenous disopyramide [13].

Heldal et al. showed a direct conversion to sinus rhythm by pacing in 75 % of patients treated with flecainide and in only 31 % without drug treatment [14].

Conclusion

Little information is available in the literature about the drug management of atrial flutter. Data are limited to small groups of patients. Our data pointed out that the success rate of class Ic and class III antiarrhythmic drugs given orally in patients with sustained atrial flutter type I was low and that continuation of the protocol was not feasible in one third of patients because of haemodynamic compromise, side effects of drug treatment or patient's or doctor's refusal.

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