

New onset atrial flutter termination by overdrive transoesophageal pacing: effects of different protocols of stimulation

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Aim We evaluated the effect of different stimulation protocols on atrial flutter interruption by transoesophageal pacing.

Methods and Results Eighty patients with new onset atrial flutter were randomized into four groups. Pacing was attempted under the following conditions: with short bursts (5 s), without treatment (group A) and after oral administration of propafenone 600 mg (group B); with prolonged bursts (30 s), without treatment (group C) and after oral administration of propafenone 600 mg (group D). Pacing interrupted atrial flutter in 20% of patients in A, 55% in B, 50% in C and 85% in D. The use of longer bursts gave better results both in patients without treatment ($P < 0.05$: C vs A) and in patients with propafenone ($P < 0.05$: D vs B). Comparing groups with the same stimulation protocol, we

observed a better response in patients treated with propafenone ($P < 0.05$: B vs A and D vs C). In the groups without treatment the use of shorter bursts was associated with a lower induction of stable atrial fibrillation (three vs nine episodes), in the groups on propafenone no differences were observed (one vs one episode).

Conclusions We conclude that the association of propafenone with long bursts gives the best result for interruption of new onset atrial flutter by transoesophageal pacing. (**Europace 2000; 2: 292–296**)

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Key Words: Propafenone, atrial flutter, transoesophageal pacing.

Introduction

Atrial flutter is a common arrhythmia, relatively resistant to antiarrhythmic drugs. In the past, synchronized external cardioversion and endocardial pacing have been considered the best treatment for the restoration of sinus rhythm^[1–5].

Recently, various reports have demonstrated the efficacy of transoesophageal atrial pacing in the interruption of atrial flutter^[6–15]. The influence of antiarrhythmic drugs on the success of the transoesophageal pacing has long been controversial. Two recent studies^[16,17] have shown that the administration of a class IC antiarrhythmic drug, propafenone, may facilitate the procedure. The best stimulation protocol

remains, however, debatable, particularly regarding the duration of pacing^[18].

In this study we evaluated, using a controlled, randomized and prospective method, the efficacy of a short versus a long duration transoesophageal pacing protocol for the termination of new onset atrial flutter and the effects of propafenone administration on both stimulation protocols.

Methods

Patients

Eighty patients (mean age 56 years), admitted to hospital with new onset type I atrial flutter^[19]. These patients had the following cardiovascular pathologies: chronic coronary artery disease in 20 patients; dilated cardiomyopathy in five patients; hypertensive heart disease in 31 patients; valvular heart disease in 18 patients; the remaining patients had no underlying

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Table 1 Clinical data

	Group A Short burst without treatment (n=20)	Group B Short burst with propafenone (n=20)	Group C Long burst without treatment (n=20)	Group D Long burst with propafenone (n=20)	P value
Mean age (years) (range)	56 (42–67)	55 (40–68)	55 (42–70)	56 (41–68)	ns
Arrhythmia duration					
(1 day)	5	4	6	5	ns
(1–3 days)	10	11	9	11	ns
(3–7 days)	5	5	5	4	ns
Atrial flutter cycle length (ms)*	226 ± 22	220 ± 21	225 ± 22	219 ± 15	ns
Left atrial size (mm)*	45 ± 3	44 ± 4	46 ± 4	44 ± 3	ns
Left ventricular ejection fraction (%)*	51 ± 7	52 ± 9	53 ± 5	52 ± 9	ns

*Values expressed as mean ± SD.

cardiovascular disease. The duration of atrial flutter varied from a few hours to 7 days. There were no patients on antiarrhythmic drug therapy; five patients were on rate limiting drugs. The patients were randomized into four groups, matched for age, existing organic heart disease, duration of arrhythmia and baseline atrial flutter cycle length. Mean left atrial size and left ventricular ejection fraction were, respectively, 44 ± 4 mm and $52 \pm 8\%$, with no significant differences in the four groups (Table 1). We performed transoesophageal atrial pacing in order to interrupt the arrhythmia in the four groups, in the following manner: with short pacing bursts (5 s), without treatment (group A) and 2 h after oral administration of propafenone 600 mg (group B); with prolonged pacing bursts (30 s), without treatment (group C) and 2 h after oral administration of propafenone 600 mg (group D). We did not anticoagulate the patients. Written informed consent was obtained from all patients.

Study protocol

All patients were studied in a post-absorptive and non-sedated state. Patients given propafenone were kept on bed rest and monitored for 2 h before the procedure. All patients underwent transthoracic echocardiography to measure left atrial anteroposterior diameter and ejection fraction. We anaesthetized the nasopharynx with 2% lidocaine jelly. A quadripolar catheter (FIAB Florence model 25145) with an interpolar distance of 30–15–15 mm was advanced via the nostril and positioned at the point where we observed the largest amplitude of transoesophageal atrial deflection; the lead was then taped to the nose. Surface electrocardiographic leads I, III and V_1 and a bipolar transoesophageal electrogram were recorded at a paper speed of $50 \text{ mm} \cdot \text{s}^{-1}$ using an oscilloscopic recorder (Hellige EVR-130). Pacing was performed by connecting two of the four electrodes to a programmable stimulator (Medtronic 5328 programmable stimulator, Medtronic, Inc, Minneapolis, MN, U.S.A.). The pulse duration of the stimulator was

set at 9.9 ms. Pacing was initiated using a stimulus intensity of 16 mA and increased by 2 mA increments until stable atrial capture was achieved. We considered that atrial capture was achieved when the morphology of the atrial flutter waves changed on the surface electrocardiogram. Often visualization of atrial activity on the surface electrocardiogram was disturbed by pacing artifacts; in these circumstances evidence of atrial capture was determined by a change in ventricular response during pacing. In the short stimulation protocol (groups A and B) this was the usually-applied criterion. Pacing was initiated at a pacing cycle length 20 ms shorter than the atrial flutter cycle length and maintained, respectively, for 5 s in groups A and B, and for 30 s in groups C D. If atrial flutter was not interrupted the pacing cycle was decreased in steps of 20 ms until atrial flutter was terminated or a cycle of 150 ms was reached. If the arrhythmia continued, or atrial fibrillation was induced, direct current shock was performed after 1 h if sinus rhythm did not spontaneously occur. Transoesophageal atrial pacing was considered effective if atrial flutter was converted immediately, or after a transient period (less than 1 h) of atrial fibrillation, to sinus rhythm.

We analysed the following variables: rate of success of the procedure, episodes of atrial fibrillation induced, cycle length change after propafenone administration, left atrial size, left ventricular ejection fraction.

Data are expressed as mean ± SD for continuous variables and rates (%) for categorical variables. Statistical analyses were performed using the paired Student's t-test for continuous variables and chi-squared analysis for categorical variables. A *P* value less than 0.05 was considered significant.

Results

Transoesophageal atrial pacing was effective in interrupting atrial flutter in 20% (4/20) of patients with short pacing bursts without treatment (group A), 55% (11/20) of patients with short pacing bursts and propafenone (group B), 50% (10/20) of patients with long pacing

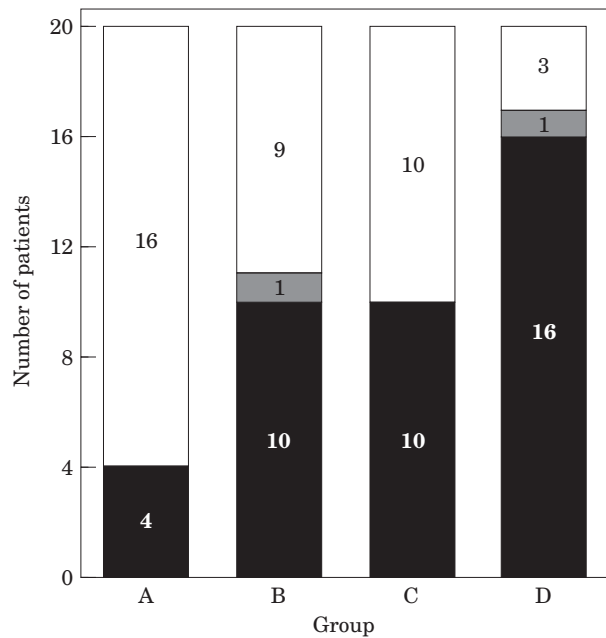


Figure 1 Results of transoesophageal pacing in the four groups expressed as number of patients in whom sinus rhythm restoration was obtained immediately (■), obtained after a transient period of atrial fibrillation (◻) or not obtained (□), respectively. Group A=short bursts without treatment; Group B=short bursts after oral propafenone; Group C=long bursts without treatment; Group D=long bursts after oral propafenone.

bursts without treatment (group C) and 85% (17/20) of patients with long pacing bursts and propafenone (group D) (Fig. 1).

The use of longer bursts of pacing gave significantly better results both in patients without treatment ($P=0.046$: group C vs group A) and in patients in therapy with propafenone ($P=0.038$: group D vs group B). Comparing the groups with the same pacing protocol, a significantly better outcome was observed in patients on therapy with propafenone ($P=0.022$: group B vs group A and $P=0.018$: group D vs group C).

The mean number of unsuccessful efforts to terminate atrial flutter before successful attempts were statistically similar in the four groups (5.3 ± 0.8 group A; 5.1 ± 1.02 group B; 5 ± 0.97 group C; 5.05 ± 1.14 group D).

There was immediate reversion to sinus rhythm in four patients in group A, 10 patients in group B, 10 patients in group C and 16 patients in group D. In the other two patients (one in group B and one in group D) sinus rhythm was reestablished after a transient period of atrial fibrillation, ranging from 60 s to 18 min (Fig. 1).

Stable atrial fibrillation was induced in three patients in group A, one patient in group B, nine patients in group C and one patient in group D ($P<0.05$ group C vs group A) (Fig. 2).

Initially, the atrial flutter cycle lengths of the four groups were statistically similar: 226 ± 22 ms (group A), 220 ± 21 ms (group B), 225 ± 22 ms (group C) and 219 ± 15 ms (group D). A significant lengthening of the

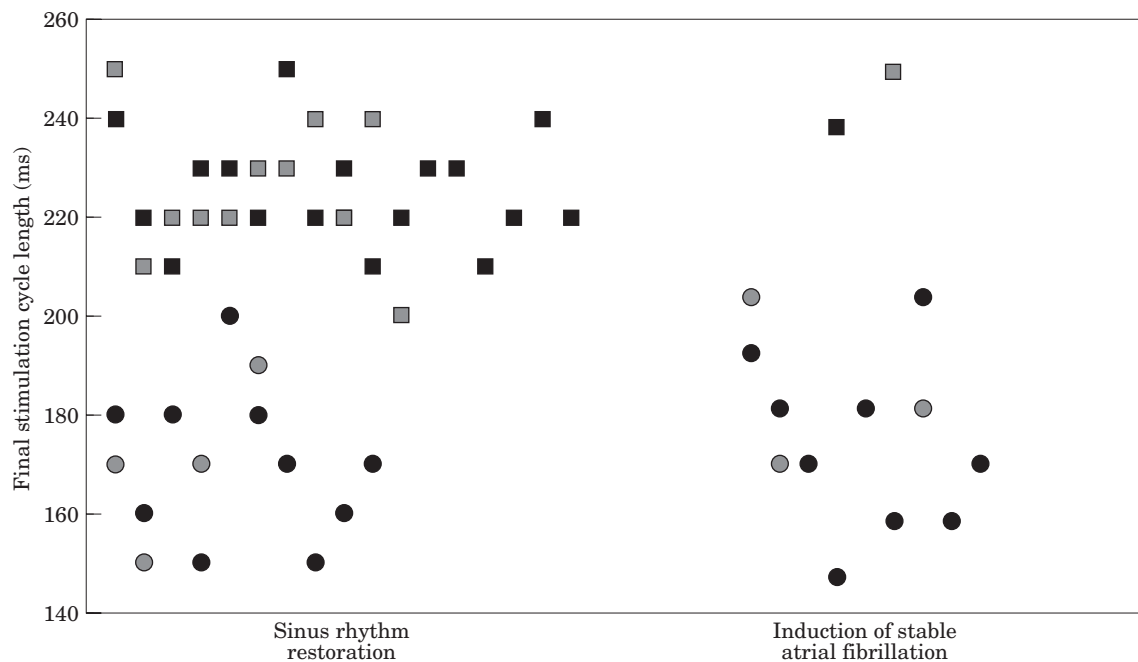


Figure 2 Stimulation cycle lengths, in the different groups, leading to sinus rhythm restoration or induction of stable atrial fibrillation. Group A=short bursts without treatment (○); Group B=short bursts after oral propafenone (◻); Group C=long bursts without treatment (●); Group D=long bursts after oral propafenone (■).

flutter cycle was observed after treatment with propafenone: 264 ± 21 vs 220 ± 21 ms, $P < 0.0005$ (group B) and 258 ± 16 vs 219 ± 15 ms, $P < 0.005$ (group D). Reversion to sinus rhythm was achieved at a shorter paced cycle in patients without treatment than in those given propafenone (171 ± 16 vs 205 ± 21 ms, $P < 0.0005$). Pharmacological conversions to sinus rhythm were not observed.

Stimulation was well tolerated by all patients; occasionally they reported mild discomfort, described as 'a transient sensation of retrosternal burning', during pacing. The threshold for atrial capture was never higher than the pain threshold.

After treatment with propafenone the atrioventricular conduction ratio changed from 2.32 to 2.76. We did not observe any abrupt enhancement of atrioventricular conduction or hypotension in the patients studied. No embolic events were documented.

Discussion

In recent years several studies^[16-17] have been published to demonstrate the efficacy of rapid transoesophageal atrial pacing in converting atrial flutter to sinus rhythm. Most of these studies^[6-15], uncontrolled and not randomized, have given controversial results concerning the possible facilitating effect of antiarrhythmic drug treatment on the procedure.

Two recently published controlled and randomized studies^[16,17], showed that propafenone may be considered a useful adjunct in atrial flutter interruption by transoesophageal pacing.

The best stimulation protocol is still debatable. All the previous studies used short or long duration stimulation protocols, without comparing their efficacy in a controlled manner.

Our prospective, controlled, randomized study demonstrates that a long (up to 30 s) stimulation protocol gives a higher rate of restoration of sinus rhythm, both in groups without treatment and in those treated with propafenone.

These results may be explained by the fact that the wave front arising from the pacing site may take a relatively long time to overtake the atrial activation which results from a wave front arising from the flutter circuit. In this setting, a longer stimulation protocol may increase the possibility of entry of the pacing stimuli into the reentry circuit and cause its interruption^[23].

In our study the long stimulation protocol, as has already been shown in previous studies^[6,9,11,12,15,18], tended to precipitate a more stable atrial fibrillation. Propafenone administration allows atrial flutter interruption at a longer paced cycle length, which is less likely to induce atrial fibrillation and favours conversion to sinus rhythm of atrial fibrillation resulting from pacing. In this way, even using long stimulation protocols, propafenone reduces the incidence of stable atrial fibrillation, which is a major drawback for a procedure easily performed as a day case.

Moreover, comparing the groups that underwent the same stimulation protocol, propafenone appears to increase the rate of atrial flutter interruption. These data confirm that, in an anatomically defined reentry circuit, such as type I atrial flutter^[20-24] the administration of an antiarrhythmic drug which reduces conduction rate more than it prolongs refractory period, results in a wider excitable gap. This can favour atrial flutter termination by overdrive pacing, as shown for disopyramide^[25,26], procainamide^[27] and propafenone^[16,17]. The rate of success obtained in our study confirms that this positive effect is more clinically beneficial than the negative effect of propafenone on pulse conduction^[8-12], which can prevent the pacing stimuli entering the arrhythmia circuit and cause its interruption.

We, therefore suggest that the best transoesophageal stimulation protocol for new onset atrial flutter interruption, given the higher rate of restoration of sinus rhythm and the lower rate of induction of stable atrial fibrillation, is the association of a pacing burst up to 30 s with administration of propafenone.

In patients with a first episode of atrial flutter this protocol may eliminate the need for hospitalization.

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