Use of Intravenous Dofetilide in Atrial Flutter
With Hemodynamic Instability

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Paroxysmal atrial flutter is a drug-resistant supraventricular tachyarrhythmia that is often accompanied by hemodynamic instability due to an excessive ventricular response. We report here a case of atrial flutter with 1:1 atrioventricular conduction, in which we were able to rescue the patient from a state of cardiogenic shock by using intravenous dofetilide, a new class III antiarrhythmic agent. Dofetilide was suitable for the treatment because it immediately increased atrioventricular nodal refractoriness without any negative inotropic action. (Jpn Circ J 1996; 60: 67–69)

Paroxysmal atrial flutter (AFL) is a drug-resistant supraventricular tachyarrhythmia that often involves hemodynamic instability even in normal or almost normal cardiac function. We report here a case of AFL with 1:1 atrioventricular (AV) conduction, in which we were able to rescue the patient from a state of cardiogenic shock by using intravenous dofetilide, a new class III antiarrhythmic agent. This treatment succeeded in reducing the ventricular rate and in immediately improving hemodynamics, although it failed to restore the sinus rhythm.

A 37-year-old female suffered palpitations and shortness of breath on the fifth day after resection of a thyroglossal cyst in the Otorhinolaryngology Department. She had no history of any cardiovascular disease. In the Inpatient Cardiology Department, she was generally in good health, but complained of palpitations and had a pulse of 130/min. Her blood pressure was 116/72 mmHg. An electrocardiogram revealed AFL with a cycle length of 200 msec, typical sawtooth morphology in leads II, III, and aVF, and 2:1 AV conduction. However, after repeated transesophageal pacing and intravenous administration of 100 mg of disopyramide, the AFL changed to another type; the F wave showed a cycle length of 240 msec with a round upper and lower end in lead V1. The QRS wave showed incomplete right bundle branch block. In addition, the AV conduction accelerated to 1:1 with a heart rate of 250/min, resulting in an abrupt fall in systolic blood pressure to 72 mmHg (Fig 1A). Although direct-current cardioversion (DC) was successful, AFL returned 5 min after its application.

Finally, after obtaining informed consent, we administered a 5 μg/kg intravenous dose of dofetilide in 5 min to increase AV nodal refractoriness. After approximately half of the dose had been injected, a complete right bundle branch block appeared (Fig 1B). AV conduction was suppressed to 2–3:1 after the injection was complete, although...
A) Before iv AF (1:1) + CRBBB HR 250 BP 72/-

B) Dofetilide iv (5 μg/kg/5min) 2'30"

C) AF (1:1) + CRBBB → AF (2:1) 2'50"

D) AF (3:1) BP 128/- 4'00"

E) After dofetilide & cardioversion (150J)

Fig 1. Electrocardiograms (leads V1 and II) in a case of atrial flutter (A–E). Panel A) shows atrial flutter at a cycle length of 240 msec with 1:1 atrioventricular conduction and incomplete right bundle branch block morphology. With intravenous dofetilide, the electrocardiogram showed complete right bundle branch block, B), and suppression of atrioventricular nodal conduction, C) and D). Note the QT prolongation after direct current cardioversion following administration of intravenous dofetilide. Abbreviations: AF, atrial flutter; BP, blood pressure; CRBBB, complete right bundle branch block; HR, heart rate; ICRBBB, incomplete right bundle branch block; and iv, intravenous injection.

AFL was observed, AFL with 1:1 AV conduction did not occur after these drugs (Fig 1E). Thyroid function was normal, but echocardiography disclosed left atrial and left ventricular dilatation (left atrial diameter = 38 mm, and diastolic and systolic left ventricular diameters = 50 and 40 mm, respectively) suggestive of dilated cardiomyopathy.

DISCUSSION

AFL is a drug-resistant supraventricular tachyarrhythmia in which it is advisable to restore normal sinus rhythm because of hemodynamic instability1,2 However, the efficacy of class I antiarrhythmic drugs is limited3. Moreover, as we stated above, disopyramide, a class IA agent, may cause the ventricles to respond in a 1:1 fashion, since 1) it has vagolytic action, 2) it has negative inotropic action on the left ventricle, which increases sympathetic tone, and 3) it slows the atrial rate by blocking sodium channels4. Even though DC and transesophageal/intracardiac atrial pacing are alternative treatments for eliminating AFL, they are not preventive and require special equipment. DC also needs general anesthesia which might exacerbate hemodynamic instability. Once attempts at restoring sinus rhythm have failed, digitalis, verapamil, or beta-adrenergic blockers can be used as alternative therapies to reduce the ventricular response1,2 However, it is inappropriate to use them in cases of cardiogenic shock, since digitalis is not quick-acting, and verapamil and beta-adrenergic blockers suppress cardiac function.

Dofetilide is a newly-developed antiarrhythmic drug that is classified in class III according to the Vaughan Williams classification4,5. Dofetilide increases the refractoriness of myocytes by blocking the delayed rectifier potassium channel. It can be administered intravenously and may be superior to other conventional antiarrhythmic drugs in that it increases AV nodal refractoriness immediately without any negative inotropic action. In our case, we assumed that dofetilide increased the refractoriness of both the AV node and the right bundle branch, and that it probably prolonged the reentrant circuit of the AFL by increasing the refractoriness of atrial myocytes. It could be used
safely and was well suited to the acute treatment of hemodynamically-significant AFL, despite its failure to restore sinus rhythm. However, when dofetilide is used, care should be taken to avoid potentially excessive QT prolongation.

We concluded that intravenous dofetilide can be a first-line therapy in patients suffering refractory paroxysmal atrial flutter with hemodynamic instability.

REFERENCES


