# Guidelines for Potassium Channel Blocker Use

Anne M. Gillis, MD, FRCPC, FHRS

**KEYWORDS**

• Atrial fibrillation • Atrial flutter • Potassium channel blockers • Practice guidelines
• Ventricular arrhythmias

**KEY POINTS**

- The choice of antiarrhythmic drug is based on the efficacy and safety profile and influenced by the presence or absence of structural heart disease.
- Because of its adverse side-effect profile, amiodarone is recommended for the management of atrial fibrillation only when other agents have failed or are contraindicated.
- Antiarrhythmic drugs do not improve survival in those with ventricular arrhythmias or at risk of sudden cardiac arrest.
- For treatment of symptomatic ventricular arrhythmias in the setting of coronary artery disease or cardiomyopathy, amiodarone is generally the preferred agent, although sotalol may be considered in patients with mild ventricular dysfunction.

## INTRODUCTION

This article summarizes recommendations for the clinical use of antiarrhythmic drugs for the treatment and prevention of atrial and ventricular arrhythmias based on the current guideline and consensus documents. By the nature and process of guideline/consensus document development, the role of the novel potassium channel blockers that are currently under clinical investigation, some of which are discussed in earlier articles, have not yet been determined. It is also important to emphasize that many of the antiarrhythmic drugs currently available for clinical use have effects on multiple ion channels, including potassium ion channels, which contribute to their efficacy.

## ATRIAL FIBRILLATION/ATRIAL FLUTTER

Guidelines for the management of atrial fibrillation (AF) and atrial flutter have recently been updated by the Canadian Cardiovascular Society,[1–3] the European Society of Cardiology[4,5] and the American College of Cardiology/American Heart Association/Heart Rhythm Society (ACC/AHA/HRS).[6] Randomized clinical trials have failed to show a superiority of a rhythm control strategy compared with a heart rate control strategy on survival or stroke prevention.[7–9] Accordingly, the choice of a rhythm control strategy should be individualized based on the severity of symptoms, the impact on patients' quality of life, the desire to improve clinical outcomes, as well as patient preferences.[1–6] Choices of antiarrhythmic drug therapy are based on the presence or absence of significant structural heart disease and a history of congestive heart failure as well as the safety and efficacy profile of the drugs.

## PHARMACOLOGIC CARDIOVERSION OF RECENT-ONSET ATRIAL FIBRILLATION

The choice of antiarrhythmic drugs for pharmacologic cardioversion of recent-onset AF are shown

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Department of Cardiac Sciences, Libin Cardiovascular Institute of Alberta, University of Calgary, 3280 Hospital Drive Northwest, Calgary, Alberta T2N 4Z6, Canada
*E-mail address:* amgillis@ucalgary.ca

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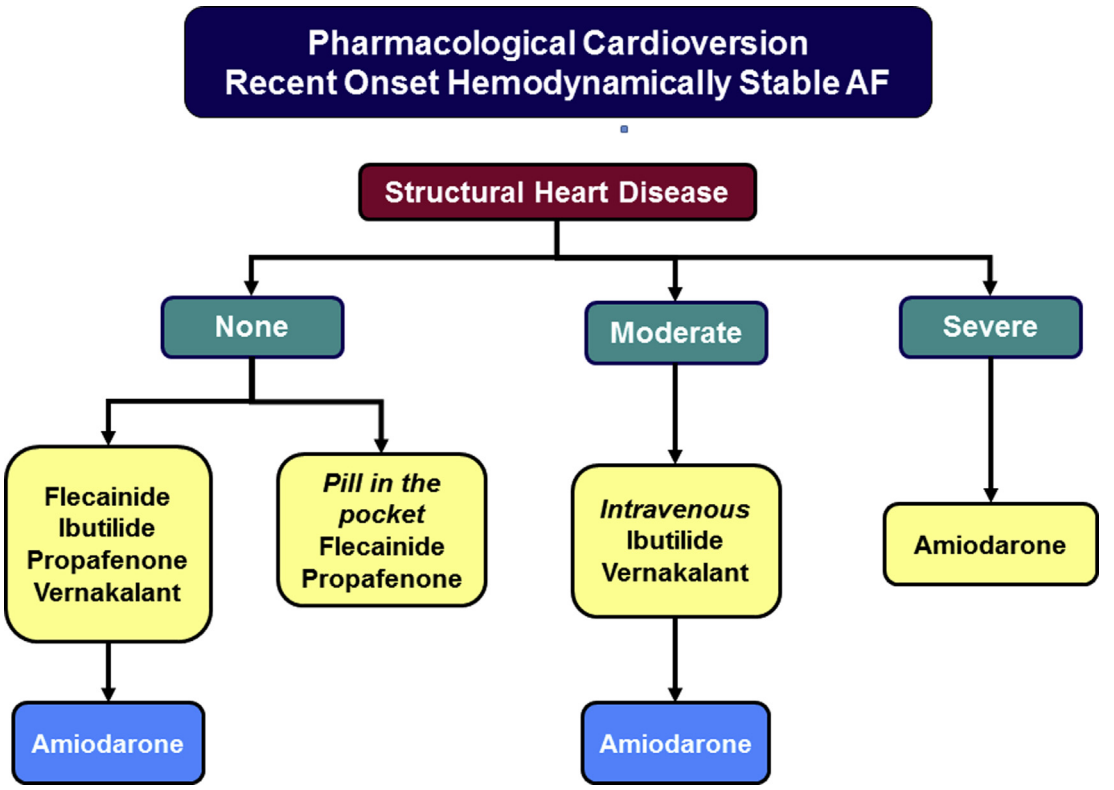


**Fig. 1.** Drug choices for pharmacologic conversion of recent-onset AF are based on the presence or absence and severity of structural heart disease.

in Fig. 1 and Table 1. The ACC/AHA/HRS' guidelines recommend the use of flecainide, dofetilide, propafenone, or intravenous ibutilide for cardioversion of AF or atrial flutter if contraindications for the selected drug are absent.[6] It is also recommended that dofetilide therapy should be initiated in hospital under continuous electrocardiogram (ECG) monitoring because of the risk of marked QT interval prolongation. The European Society of Cardiology's guidelines recommend

**Table 1**
**Drugs for pharmacologic cardioversion of atrial fibrillation**

| Intravenous Drugs | Dose |
|---|---|
| Flecainide | 2 mg/kg |
| Ibutilide | 1 mg over 10 min may repeat once (0.001 mg/kg if weight <60 kg) |
| Propafenone | 2 mg/kg |
| Amiodarone[a] | 150 mg over 10 min then 1 mg/min × 6 h then 0.5 mg/min for 18 h or switch to oral dose |
| Vernakalant | 3 mg/kg over 10 min; 2 mg/kg after 10 min if AF persists |
| **Oral Drugs** | **Dose** |
| Amiodarone | 400–800 mg in divided doses to a total of 10 g then 100–200 mg/d |
| Flecainide | 200–300 mg single dose |
| Propafenone | 450–600 mg single dose |
| Dofetilide | 125–500 mg bid based on creatinine clearance and QT interval |

[a] Amiodarone is less effective for early termination of AF.