

<<Braunwald心脏病学（套装上下）>>

图书基本信息

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## <<Braunwald心脏病学（套装上下）>>

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<<Braunwald/心脏病学 ( 套装上下)>>

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## 章节摘录

版权页：插图： Pharmacologic Rhythm Control The results of published studies on the efficacy of antiarrhythmic drugs for AF suggest that all of the available drugs except amiodarone have similar efficacy and are associated with a 50% to 60% reduction in the odds of recurrent AF during 1 year of treatment.<sup>29</sup> The one drug that stands out as having higher efficacy than the others is amiodarone. In studies that directly compared amiodarone with sotalol or class I drugs, amiodarone was 60% to 70% more effective in suppressing AF. However, because of the risk of organ toxicity, amiodarone is not appropriate first-line drug therapy for most categories of patients with AF. Because their efficacy is in the same general range, the selection of an antiarrhythmic drug to prevent AF often is dictated by the issues of safety and side effects. Ventricular proarrhythmia from class IA agents (quinidine, procainamide, disopyramide) and class III agents (sotalol, dofetilide, dronedarone, amiodarone) is manifested as QT prolongation and polymorphic ventricular tachycardia (torsades de pointes). Risk factors for this type of proarrhythmia include female gender, left ventricular dysfunction, and hypokalemia. The risk of torsades de pointes appears to be much lower with dronedarone and amiodarone than with the other class III drugs. The ventricular proarrhythmia from class IC agents (flecainide and propafenone) is manifested as monomorphic ventricular tachycardia, sometimes associated with widening of the QRS complex during sinus rhythm but not QT prolongation. Published studies indicate that the drugs most likely to result in ventricular proarrhythmia are quinidine, flecainide, sotalol, and dofetilide. In controlled studies, these agents increased the risk of ventricular tachycardia by a factor of 2 to 6. Adverse drug events resulting in discontinuation of drug therapy are fairly common with rhythm-control drugs. Withdrawal due to adverse effects was most common with quinidine, disopyramide, flecainide, sotalol, and amiodarone.<sup>29</sup> A review of studies in which 32 treatment arms received an antiarrhythmic drug for AF found that 10.4% of patients discontinued drug therapy because of an adverse drug event, most commonly gastrointestinal side effects and neuropathy. The best options for drug therapy to suppress AF depend on the patient's comorbidities. In patients with lone AF or minimal heart disease (e.g., mild left ventricular hypertrophy), flecainide, propafenone, sotalol, and dronedarone are reasonable first-line drugs, and amiodarone and dofetilide can be considered if the first-line agents are ineffective or not tolerated. In patients with substantial left ventricular hypertrophy (left ventricular wall thickness >13 mm), the hypertrophy may heighten the risk of ventricular proarrhythmia, and the safest choice for drug therapy is amiodarone. In patients with coronary artery disease, several of the class I drugs have been found to increase the risk of death, and the safest first-line options are dofetilide, sotalol, and dronedarone, with amiodarone reserved for use as a second-line agent. In patients with heart failure, several antiarrhythmic drugs have been associated with increased mortality, and the only two drugs known to have a neutral effect on survival are amiodarone and dofetilide (see Chap. 37).

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