

**1. In a case of AF, where the ventricular rate has been stabilized to a 60 bpm to 80 bpm range with negative inotropics or chronotropics, would it not be safer to discard warfarin in favour of clopidogrel, ticlopidine, or even ASA?**

Question submitted by: Dr. Ramgoolam, Winnipeg, Manitoba.

Atrial fibrillation (AF) poses a risk for stroke, independent of whether the ventricular rate has been stabilized or not, or whether the rhythm is permanent, persistent, or paroxysmal.

The precise risk of stroke is determined by factors such as advanced age, prior stroke or transient ischemic attack, hypertension, heart failure, and diabetes. The decision to administer an anticoagulant is usually based on the presence or absence of any of these risk factors.

Currently, there is insufficient information to support “discarding” warfarin in favour of an antiplatelet agent. Warfarin is unquestionably established to reduce the risk of stroke in at-risk individuals, provided the international normalized ratio (INR) is maintained between 2.0 and 3.0; acetylsalicylic acid (ASA) is almost certainly inferior to warfarin in such patients. Clopidogrel as monotherapy has not been investigated in this context. However, the combination of clopidogrel

and ASA is, at least hypothetically, a reasonable one which could be considered an alternative to warfarin in the future. There is an ongoing randomized, controlled trial (RCT) comparing the combination of clopidogrel and ASA to warfarin in a non-inferiority paradigm; it is an international study which began in mid-2003 called the Atrial fibrillation Clopidogrel Trial with Irbesartan for the prevention of Vascular Events (ACTIVE).

For the moment, stopping warfarin in favour of clopidogrel or ASA alone, or their combination, is not recommended, except as a consideration in the context of an RCT with full patient consent.

Answered by:

**Paul Dorian**, MD, MSc, FRCPC, professor, University of Toronto, and director, arrhythmia service, division of cardiology, St. Michael's Hospital, Toronto, Ontario.

## 2. What would be a practical schedule for following patients on amiodarone?


Question submitted by: Dr. Rosen, Toronto, Ontario.

Amiodarone is an effective antiarrhythmic drug which has a high incidence of adverse side-effects, such as cough, bradycardia, hypothyrea, and increased liver enzymes.

The treatment regime is complex. The initial setup and titration of the dose is best performed by an experienced cardiologist. In life-threatening ventricular arrhythmias, the therapy should be initiated in the hospital.

For atrial fibrillation, the starting dose is 600 mg to 800 mg daily, in two divided doses. A lower dose

should be considered for women and for patients with low body weight. After four weeks, the daily dose can be gradually reduced. Within six months, a maintenance dose of 100 mg to 300 mg daily is usually sufficient.

Table 1 shows a summary of recommended schedules based on various tests. 

Answered by:

**J. George Fodor**, MD, PhD, FRCPC, professor emeritus of medicine, and head of research, Prevention and Rehabilitation Centre, University of Ottawa Heart Institute, Ottawa, Ontario.

Table 1

### Recommendations for routine laboratory testing with chronic amiodarone therapy

<u>Test</u>	<u>Time</u>
Liver function tests	Baseline, and every six months
Thyroid function tests (Serum TSH and T <sub>4</sub> )	Baseline, three months, and then every six months
Serum creatinine and electrolytes	Baseline, and as necessary
Chest radiograph	Baseline, and yearly
Ophthalmologic evaluation	Baseline if visual impairment, and then for symptoms
Pulmonary function tests (DLCO)	Baseline, and for otherwise unexplained dyspnea, particularly in patients with underlying lung disease, and for abnormalities on chest radiology
Electrocardiogram	Baseline, and yearly

TSH=Thyroid-stimulating hormone; T<sub>4</sub>=Thyroxine; DLCO=Diffusing capacity for carbon monoxide

Adapted from:

Goldschlager N, Epstein AE, Naccarelli G, et al: Practical guidelines for clinicians who treat patients with amiodarone. North American Society of Pacing Electrophysiology. Arch Intern Med 2000; 160(12):1741-8.

Harjai KJ, Licata AA: Effects of amiodarone on thyroid function. Ann Intern Med 1997; 126(1):63-73.