A 55-year-old man was first evaluated for mild angina pectoris of 2 years’ duration. A recent myocardial perfusion study performed with dipyridamole and technetium sestamibi demonstrated ischemia of the inferior wall of the left ventricle. His history indicated the presence of marked obesity, hypertension, and osteoarthritis. He was told that he had had “asthmatic bronchitis.” He was told several years ago that he also had a transient bout of a cardiac arrhythmia, possibly atrial fibrillation (AF), although this was poorly documented.

Coronary cineangiography demonstrated multivessel coronary disease with 95% obstruction of the apical portion of the left anterior descending artery with 60% involvement of this vessel at its midportion. There were minor lesions in the obtuse marginal branch of the circumflex with 90% narrowing in the midportion of the proximal circumflex artery. The right coronary artery demonstrated 60 to 70% proximal obstruction with a 70% narrowing in the posterior descending branch. The left ventriculogram demonstrated mild inferolateral hypokinesis with an ejection fraction exceeding 55%. Left ventricular pressure was 160/15 prior to the ventriculogram.

Because of the mild nature of symptoms and normal left ventricular systolic function, the patient was treated medically with a combination of nifedipine, hydrochlorothiazide, and triamterene.

The symptoms of angina pectoris completely disappeared for the next 6 months. He noted, however, on the day of admission to the hospital that he suddenly felt weak and that his heart beat was rapid and irregular. He was seen in the emergency department at that time and found to be in AF with a rapid ventricular response in the range of 140 to 160 beats per minute. At the time he was admitted, he had been symptomatic for about 12 h. Examination at the time of admission, revealed a markedly obese man with a height of 5’10” and a weight of 308 lb. Blood pressure was 150/80. The lungs were clear. Cardiac examination revealed no abnormal sounds or murmurs. There was no evidence of neck vein distention or edema. The abdominal examination was negative, and the liver was not palpable. Electrocardiogram disclosed atrial fibrillation with a rapid ventricular response but was otherwise normal limits.

Transthoracic echocardiogram was performed and demonstrated a normal-sized left ventricle with slightly increased wall thickness. The wall motion appeared low normal without focal abnormalities. The left atrium was mildly dilated, measuring 4.6 cm in diameter. There were no valvular abnormalities, and no masses or thrombi were identified.

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Questions for Consultants

1. How would you manage this patient?
2. Would you recommend anticoagulants and with what agent? If these are not recommended, under what conditions would you recommend such treatment?
3. How would you proceed with pharmacologic agents to:
   a) Control the ventricular response rate?
   b) Restore normal sinus rhythm?
4. Under what circumstances would you require hospital admission with telemetric cardiac monitoring?
5. Are there any circumstances which would require performing transesophageal echocardiography?
6. When would you employ electrical countershock to restore normal sinus rhythm? Are there conditions which could allow this procedure to be accomplished as an outpatient?
7. Assuming that normal sinus rhythm is restored, what drugs, if any, would you use to maintain normal sinus rhythm?

Comments by S. Mark Sopher, MD, and A. John Camm, MD

Here is a man who seems to have been designed to make decisions regarding management of his AF particularly difficult! He tolerates the arrhythmia reasonably well, and he certainly does not require cardioversion as an emergency. He has both ischemic and hypertensive heart disease which increase the chance of recurrent AF following successful cardioversion and which may enhance the risks of antiarrhythmic agents, particularly of class Ic. His vague history of “asthmatic bronchitis” suggests that he may not tolerate a beta-blocker and increases the concern to avoid amiodarone-induced pulmonary fibrosis. Furthermore, lung hyperinflation and his obesity would combine to increase the transthoracic impedance and thus reduce the chance of successful cardioversion by direct current (DC) shock. Despite all these complications, he would benefit from a simple stepwise approach to management.

First, it is necessary to look for any other possible etiologic factors or precipitants of AF such as hyperthyroidism, metabolic or electrolyte disturbance, pneu-
monia, or excessive alcohol intake (whether acutely or chronically). The first major management decision concerns immediate cardioversion; given the short duration of AF, young age, nonrheumatic etiology, and relatively small left atrial diameter, the probability of success is high with either DC shock or an IV infusion of an antiarrhythmic drug. Cardioversion of AF of less than 48-h duration is distinguished by the lack of a requirement for anticoagulation prior to cardioversion, and by the high success rate with IV antiarrhythmic drugs, and, in the absence of reversible precipitating factors for AF, should be attempted.

In Europe, IV antiarrhythmic drugs are frequently used to cardiovert acute AF, and the choice between these and transvenous DC shock is often one of logistics. While continuous ECG and blood pressure monitoring is mandatory at least during drug administration, DC shock requires a short-acting general anesthetic or heavy sedation, and an anesthesiologist must be available. DC shock (including 360 J with paddles in the anterior-posterior positions) is generally preferred unless anesthesia is contraindicated, but if it fails, an infusion of up to 150 mg flecainide over 30 min could be tried. The patient should be admitted for these initial procedures, but any future elective attempts at cardioversion could be performed as a “day-case.”

Following restoration of sinus rhythm, the issues of antiarrhythmic and antiembolic medication arise. I would wait for a recurrence of AF without precipitant before considering prophylactic antiarrhythmic medication. While sotalol has the potential advantages over a class I agent of having additional antihypertensive and antianginal effects, and possibly a lower risk of proarrrhythmia in the presence of ischemic heart disease, there is a history of “asthmaic bronchitis” and a risk of bronchospasm. However, if clear symptoms or signs to suggest significant obstructive airways disease were lacking, sotalol should be tried at 40 mg twice a day building up to 160 mg twice a day over a few weeks if tolerated, with a warning issued to stop the drug in case of breathlessness, cough, or wheeze. If sotalol was not tolerated, or if AF recurred, a class Ia agent such as quinidine or disopyramide should be tried. The use of amiodarone (at 200 mg/d) to maintain sinus rhythm should be considered only if there were recurrences of AF on at least two occasions while taking different antiarrhythmic agents, with regular monitoring of thyroid, liver, and pulmonary function tests. All antiarrhythmic agents could be started as an outpatient prior to DC shock to minimize the risk of early reversion to AF. With amiodarone, oral loading should start several weeks before DC shock in case this alone restored sinus rhythm and to increase the chance of successful cardioversion with the shock.

In regard to the question of anticoagulation in AF, a more careful assessment of his risk factors for thromboembolism, bleeding, and injury is needed. All cardioversion procedures should be covered with heparin unless oral anticoagulants are used to maintain an international normalized ratio of 1.7 to 2.5 both before and after the procedure. Following successful cardioversion of this first episode of acute AF, the patient should take aspirin at 300 mg/d and be told to present within 48 h of the recurrence of symptoms. At this time a repeat cardioversion could be attempted with subsequent anticoagulation with warfarin. However, for elective cardioversion of AF greater than 48-h duration by any method, anticoagulation with warfarin for at least 1 month both prior to and following cardioversion should be used. There is no reason to use transesophageal echocardiography to exclude left atrial thrombus unless the patient presented requiring cardioversion of AF greater than 48-h duration without prior full anticoagulation, or had a history of embolism.

If all attempts to restore and maintain sinus rhythm proved futile, long-term anticoagulation with warfarin is required in view of his hypertension, ischemic heart disease, and dilated left atrium. During AF, drugs should be used to control the ventricular rate if the mean at rest was greater than 90 beats per minute, or if he had symptoms of an excessive ventricular rate. Digoxin therapy can be guided by symptoms, plasma levels of digoxin, and both resting and exertional ventricular rates. The addition of a beta-blocking agent (if tolerated) or verapamil may improve rate control during exertion. However, because the patient appears to tolerate AF well even with an uncontrolled ventricular rate, he does not warrant nonpharmacologic methods such as cardiac surgery to maintain sinus rhythm. Nonpharmacologic methods such as a catheter ablation procedure designed to control the ventricular rate could be considered if it proved impossible to do this with drugs.

The final decisions relate to his ischemic heart disease: reversible risk factors need to be addressed including plasma lipids and smoking. The patient also requires a careful dietary history including alcohol consumption with strong advice and support to lose weight. A symptom-limited exercise test once sinus rhythm had been restored should be used to assess functional ischemia, but as long as the patient remains free of angina, revascularization is not indicated.

EDITOR’S COMMENTS

Following the hospital admission, this patient was given digoxin intravenously to slow the ventricular response rate. Within 12 h, the rate was slowed to less than 100 beats per minute. In the effort to restore normal sinus rhythm, an infusion of propranolol (2 mg/min) was begun after a loading dose of 6 mg/kg at
0.5 mg/kg/min was given. Within 2 h of this infusion, normal sinus rhythm was restored. He was then given propafenone orally (150 mg every 8 h), and cardiac rhythm was monitored for an additional 48 h in the hospital, during which normal sinus rhythm was maintained. He was subsequently followed up as an outpatient over the next 6 months. He continued to manifest normal sinus rhythm. Propafenone was continued in the same dosage, although the digitalis glycoside was discontinued 6 weeks after the hospitalization.

As Drs. Sopher and Camm have indicated above, the management of AF has continued to evolve and allows for considerable flexibility. In recently acquired atrial fibrillation, if the patient is hemodynamically stable, our first approach is to use rapidly acting drugs intravenously to slow the ventricular response rate. We prefer IV digoxin, although diltiazem or verapamil may be used if greater rapidity of response is required. If AF persists, we usually attempt to restore normal sinus rhythm with an IV drug such as procainamide, although electrical countershock may be used at any time, especially if the patient is markedly symptomatic or hemodynamically unstable. IV flecaïnide is not currently available in the United States. During the early hours of management, echocardiographic assessment is generally undertaken to evaluate cardiac status including atrial size, left ventricular wall thickness, and systolic function as well as valvular function. In addition, hematologic values are obtained including thyroid studies to search for underlying diseases predisposing to atrial fibrillation. After restoration of normal sinus rhythm, we generally use oral drugs to maintain a stable rhythm if there have been previous episodes of AF or if intrinsic cardiac abnormalities are present such as atrial enlargement or left ventricular hypertrophy or dysfunction. We believe that, in the absence of structural heart disease, one can generally begin these agents for a few days outside the hospital setting prior to elective countershock procedures. On the other hand, in the presence of disease such as left ventricular hypertrophy or dysfunction, our approach differs from that of Drs. Sopher and Camm, wherein we prefer to bring the patient into a hospital setting for cardiac monitoring for at least 2 to 3 days while oral antiarrhythmics are instituted. This is done to detect proarrhythmic effects which are generally more apt to occur in the setting of cardiac disease.

Anticoagulants are generally not necessary if the AF has persisted for less than 48 h. If anticoagulants are employed after a prolonged bout of AF, however, they should be continued for 4 to 6 weeks after conversion to normal sinus rhythm because of the delayed recovery of mechanical atrial function, a factor which may increase the likelihood of delayed thromboembolic events.

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