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Atrial Fibrillation and Acute Decompensated Heart Failure

John P. DiMarco, MD, PhD

Atrial fibrillation (AF) is a common rhythm in patients with acute decompensated heart failure (ADHF). Registry and trial data^{1,2} indicate that 20% to 35% of patients with ADHF who are admitted to the hospital will be in AF at presentation. In about one third of these patients, the AF will be of recent onset. Despite the high frequency with which the combination of AF and ADHF is encountered, there are few published data that specifically address this problem. AF and worsening heart failure interact in a dangerous pattern. The adverse effects of AF in patients with heart failure may include loss of atrial transport, rapid and irregular ventricular rates, and toxic effects of antiarrhythmic drug therapy. Worsened heart failure, in turn, leads to increased atrial stretch and heightened sympathetic tone. These latter changes make the AF more resistant to treatment using either a rate-control or a rhythm-control strategy (Figure 1).

The immediate general goals of therapy in ADHF are to improve symptoms, restore oxygenation, improve organ perfusion, and limit cardiac and renal injury.³ In patients with sinus rhythm and ADHF, the appropriate use of vasodilators, oxygen, diuretics, positive inotropic agents, and mechanical devices to support ventilation or cardiac output forms the cornerstone of therapy. In patients who present with AF, however, additional treatment decisions are required.

Initial Assessment

The clinical history provides critical information that should be used to guide treatment. Five key questions that should be asked before starting therapy are listed in Table 1. Many patients who present with ADHF will have implantable cardioverter defibrillators (ICDs) in place. Inappropriate ICD therapy during AF episodes is both undesirable and dangerous. If the patient has an ICD, interrogation and reprogramming of the ICD to minimize the risk for inappropriate therapy should be performed as soon as possible after presentation. Usually, the rate and duration of arrhythmia that triggers ventricular tachycardia or ventricular fibrillation detection should be increased, and supraventricular arrhythmia discriminators should be activated if they are available. With dual-chamber or biventricular devices, the atrial

tachyarrhythmia pacing response should be set to eliminate inappropriate high-rate atrial tracking.

Rate Control Versus Rhythm Control

AF episode duration will affect decisions about the advisability and potential for spontaneous, pharmacological, or electric cardioversion and selection of agents for rhythm or rate control. Three scenarios are commonly encountered. Some patients will present shortly after the onset of AF. Either the AF episode itself has rapidly precipitated heart failure in a previously stable patient or worsening heart failure has triggered an acute episode of AF. In these patients, the potential for successful early restoration of sinus rhythm is high if the heart failure symptoms can be controlled. A second pattern is seen when a patient develops an episode of AF for which the patient is either unaware or does not seek medical attention. During the ensuing days and weeks, the patient gradually slips into ADHF and then presents with severe symptoms. These patients will probably not convert spontaneously but may be candidates for a later cardioversion attempt. Finally, some patients with permanent AF that is usually well rate controlled will develop progressive heart failure and then present emergently with rapid ventricular rates due to the stress of the episode. Long-term restoration of sinus rhythm will rarely be possible in this latter group.

A careful medication history is important to guard against overdose and adverse drug interactions when drugs for rate control or rhythm control are considered. Anticoagulation status must be known before any cardioversion attempt, unless the episode is definitely known to be of <48 hours duration. Ventricular function strongly affects appropriate rhythm-control and rate-control drug choices. Concomitant disorders (eg, renal or pulmonary disease, infection) may limit drug therapy options and may also require specific therapy.

Immediate cardioversion should rarely be the first step in therapy for a patient with AF and ADHF. Although a shock may transiently restore sinus rhythm, the expected recurrence rate in the still-decompensated patient will be very high.⁴ Therefore, it is usually better to start with a rate-control strategy. If the patient has not been previously anticoagulated

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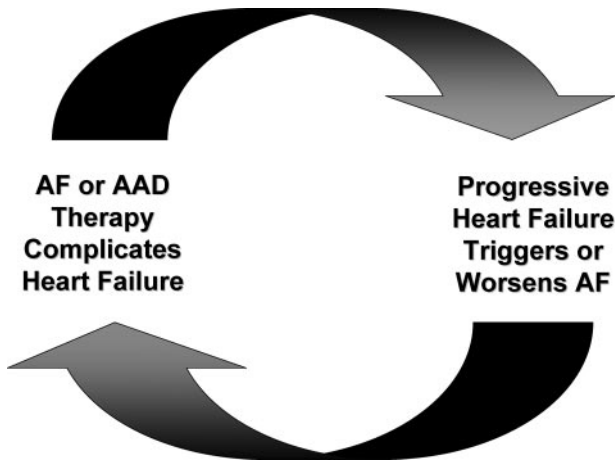


Figure. Adverse interactions between AF and heart failure. AAD indicates antiarrhythmic drug.

and there are no contraindications, heparin should also be started.

Although the optimal resting heart rate during AF is between 60 and 100 bpm, rates below 100 bpm may not be achievable during AHDF until volume overload and hypoxia have been corrected. A more realistic target is to achieve a heart rate below 120 bpm during the first hours of treatment. Digoxin should be the first rate-control agent considered, but in patients with high persistently sympathetic tone, it may have little effect early in the course of therapy. If the patient has already been taking digoxin, additional doses may be dangerous and should be avoided, unless a low serum digoxin concentration (<0.5 ng/mL) can be confirmed. Cautious addition of small doses of an intravenous β -blocker, usually metoprolol in 2.5- to 5-mg increments or, if systolic function is preserved, diltiazem, to digoxin will often be required. If rate control along with relief of volume overload and dyspnea can be achieved, patients will frequently revert back to sinus rhythm if the AF episode is of recent onset. If the patient does not improve with these measures, meets anticoagulation criteria for conversion, and is not already on an antiarrhythmic drug, a trial of intravenous amiodarone may be helpful, because it may slow the ventricular rate and facilitate early conversion. Amiodarone can be reloaded in patients already on chronic, moderate doses (≤ 200 mg daily) but should not be added if the patient has been taking another antiarrhythmic drug that prolongs the QT interval, such as sotalol or dofetilide.

Table. Key Questions to Consider Before Starting Therapy

Does the patient have an ICD or pacemaker in place?
Does the patient have preserved or reduced systolic function at their baseline?
What is the duration of the AF episode?
Is the patient already on drugs for rhythm or rate control and anticoagulation?
What concomitant disorders are present?

If this approach fails and heart rates during AF remain elevated, cardioversion after a period of loading with an antiarrhythmic drug, usually amiodarone, is the next step. When the patient has not been adequately anticoagulated, a transesophageal echocardiogram followed by maintained anticoagulation may facilitate the early cardioversion attempt. If cardioversion attempts are unsuccessful and the patient remains symptomatic with elevated rates, atrioventricular junctional ablation, often with a biventricular pacing system, is an additional option that can be considered.

Once the ventricular rate has been at least partially controlled, the possible benefit of a cardioversion should be considered, unless the patient has known long-standing persistent AF. In the latter situation, the probability of restoring and maintaining sinus rhythm is low. Therefore, once the acute heart failure exacerbation has been corrected in such a patient, a continued rate-control strategy is appropriate. In patients with new- or recent-onset AF, an attempt at cardioversion and drug therapy is reasonable, with the final decision on a long-term strategy based on symptoms, drug tolerance, and the frequency of recurrent episodes. As was shown in the Atrial Fibrillation and Heart Failure Trial,⁵ there is no a priori benefit associated with a rhythm-control strategy, but individual patient responses vary widely, and I usually make at least 1 attempt to maintain sinus rhythm in any patient with more than mild symptoms associated with AF. In selected patients, left atrial catheter ablation may prove effective, but experience with this approach for patients in whom AF was not the primary cause for heart failure has been limited.

Disclosures

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References

- Adams KF, Fonarow GC, Emerman CL, LeJemtel TH, Costanzo MR, Abraham WT, Berkowitz RL, Galvao M, Horton DP. Characteristics and outcomes of patients hospitalized for heart failure in the United States: rationale, design, and preliminary observations from the first 100,000 cases in the Acute Decompensated Heart Failure National Registry (ADHERE). *Am Heart J*. 2005;149:209–216.
- Krum H, Gilbert RE. Demographics and concomitant disorders in heart failure. *Lancet*. 2003;362:147–158.
- Dickstein K, Cohen-Solai A, Filippatos G, McMurray JJV, Ponikowski P, Poole-Wilson PA, Stromberg A, van Veldhuisen DJ, Atar D, Hoes AW, Keren A, Mebazza A, Nieminen M, Priori SG, Swedberg K. ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2008. *Eur Heart J* 2008;29:2388–2442.
- Kanji S, Stewart R, Fergusson DA, McIntyre L, Turgeon AF, Hebert PC. Treatment of new onset atrial fibrillation in noncardiac intensive care unit patients: a systematic review of randomized controlled trials. *Crit Care Med*. 2008;36:1620–1624.
- Roy D, Talajic M, Nattel S, Wyse DG, Dorian P, Lee KL, Bourassa MG, Arnold MO, Buxton AE, Camm AJ, Connolly SJ, Dubuc M, Ducharme A, Guerra PG, Hohnloser SH, Lambert J, Le Heuzey J-Y, O'Hara G, Pedersen OD, Rouleau J-L, Singh BN, Stevenson LW, Stevenson WG, Thibault B, Waldo AL, for the Atrial Fibrillation and Congestive Heart Failure Investigators. Rhythm control versus rate control for atrial fibrillation and heart failure. *N Engl J Med* 2008;358:2667–2677.

KEY WORDS: antiarrhythmia agents ■ fibrillation ■ heart failure ■ atrial fibrillation