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*Atrial fibrillation (AF) is an extremely common arrythmia which has increased prevalence with advancing age. Chronic AF is a major risk factor for stroke and increased mortality. Numerous etiologies exist for AF, including both cardiac and noncardiac causes. AF is responsible for more physician office visits, emergency department (ED) visits, admissions, and hospital days than any other dysrhythmia. It accounts for one-third of all dysrhythmias presenting to the ED.*

*ED presentations may vary from mild shortness of breath and fatigue to overt congestive heart failure (CHF) and hypotension. AF is usually easily diagnosed on the basis of the typical irregular irregularity of the ventricular response, since AF typically is the only rhythm that produces this pattern. A more pressing and familiar challenge for emergency physicians is control of the ventricular rate, although understanding the pathophysiology, etiologies, and various treatment modalities are essential for the emergency physician managing patients with AF.*

*The first part of this in-depth issue will discuss these points, with particular emphasis on drug selection and dosage for rate control. Part II will cover anticoagulation and restoration of normal sinus rhythm with an emphasis on pharmacological management.*

*— The Editor*

# Atrial Fibrillation

## Part I: Etiologies and Strategies for Ventricular Rate Control

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## Introduction

AF is the most common sustained arrhythmia, with a prevalence of approximately 0.4-1.0% in the general population, or an estimated 2.2 million Americans,[1] and more than 160,000 new cases each year.[2] It is the most common dysrhythmia presenting to the ED. The prevalence of AF increases with age and is estimated to be present in 5% of those older than age 65, 10% of those older than 70, 14% of people older than 84, and 22% of those older than 91.

The indicence of AF in older Americans (ages 62-90 years) has been reported to be 50-90 per 1000[3]. In one report, more than 10% of patients older than 90 years of age were in AF.[4] Approximately 50% of patients with AF are older than age 75.[1,5,6] Because the incidence of AF increases with age and average life expectancy has climbed in recent decades, it can be expected that AF will become a more significant part of every emergency and geriatric physician's practice in upcoming years.

Chronic AF is a major risk factor for stroke, carrying a risk of 5-7% per year in patients with coronary artery disease (CAD) or CHF who are not therapeutically anticoagulated. The presence of AF in the older age group indicates a high likelihood of coexisting cardiovascular or systemic disease.[7]

Patients with AF have a 1.3-2.6 time increased risk of mortality,[5] especially those with increased age, aortic valve disease, CAD, or CHF. Mortality is increased 12 times in patients with hypertension, and 17 times if mitral stenosis is present.

AF complicates 3-20% of myocardial infarctions (MIs). Patients with AF have a 4-7 time greater risk of stroke than the

general population.[8-10] Factors that increase risk of stroke include age, the presence of rheumatic heart disease, the presence of CHF, dilated left atrium (LA), previous MI, and hypertension. AF causes 45% of embolic strokes. Non-rheumatic AF causes 75,000 strokes/year in the United States. Framingham data suggest that stroke may be attributed to AF in more than 25% of events in patients older than age 80.[8] AF has been identified as the rhythm disturbance responsible for more than 85% of systemic thromboembolic events originating from the heart.[11]

## Risk Factors

There is an increased incidence of AF in people with diabetes mellitus; valvular heart disease; rheumatic heart disease; CAD; hypertension; CHF; hypertrophic and dilated cardiomyopathies; inflammatory or infiltrative diseases of the atria; pericarditis;[12] congenital heart disease; cardiac contusion; accessory pathways, including Wolff-Parkinson-White (WPW) syndrome; and those post cardiac surgery. *(See Table 1.)*



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Noncardiac disorders that place patients at risk include thyroid disease, malignancy, ethanol intoxication and withdrawal, electrolyte disturbances, sarcoidosis, amyloidosis, pheochromocytoma, and pulmonary embolism. Factors that may predispose a patient to AF include: cocaine abuse, theophylline toxicity, and increased sympathetic or parasympathetic activity as frequently occurs in trauma, peri-operative states, acute blood loss, or hypothermia.[7] The 10% of patients with AF and no associated disease are considered as having lone AF. AF also is more common in men than in women (2:1).

## Significance of Duration

Approximately 30-60% of patients convert spontaneously to sinus rhythm within 24 hours. Persistence of the dysrhythmia produces electrophysiologic changes in the atria, called "remodeling," which increases the likelihood that the rhythm will continue.[13]

In addition, the risk of thrombus formation is related to the duration of the dysrhythmia. Patients in AF for more than 48 hours are at risk of developing a left atrial thrombus, and therefore, are not candidates for elective chemical or electrical cardioversion unless they have been adequately anticoagulated. Patients with brief intermittent episodes of AF carry a much lower risk of cardioembolic stroke.[14]

## Pathophysiology

During AF the atria have disorganized, rapid irregular electrical activity exceeding 400 beats per minute. The atria do not contract effectively, and intra-atrial clotting is promoted. With subsequent resumption of atrial contraction, an embolism can occur. AF has been proposed to occur in three distinct clinical circumstances: as a primary arrhythmia in the absence of structural heart disease, as a secondary arrhythmia in the presence of a systemic abnormality, and as a secondary arrhythmia associated with cardiac disease that affects the atria. When AF is associated with structural heart disease, there may be atrial dilatation and patchy fibrosis, including evidence of destruction of the sinoatrial node.

Electrophysiologic mechanisms of AF have been proposed. The most widely accepted is the multiple wavelet hypothesis proposed by Moe.[15] This claims that multiple reentrant impulses of various sizes wander through the atria, creating continuous electrical activity. These multiple reentrant wavefronts propagate randomly in the atria. The wavelets have a functionally determined area of conduction block at the center, preventing their collapse and extinction. The minimum number of wavelets required for the perpetuation of AF is proposed to be six.[16] Longer wavelets cause coarse AF; smaller wavelets are found in fine AF. Wavelengths can be prolonged by antiarrhythmic drugs and shortened by increased parasympathetic tone or intra-atrial conduction abnormalities.

Reentry is more common in patients with sinoatrial node dysfunction. It is found more often with high atrial pressures, as from pulmonary hypertension, mitral stenosis, hypoxic/ischemic atrial tissue, and atrial distension or enlargement. AF results from small reentrant circuits activating multiple

areas of the atrial mass at the same time. The wavelets are constantly forming, collapsing, and reforming in the atria as refractory atrial tissue is encountered and bypassed. AF continues until these cycles of wavelets disappear or fail to regenerate.

AF may be due to autonomic dysfunction as well. With vagally mediated AF, it has long been recognized that increased parasympathetic tone predisposes otherwise normal hearts to the onset of AF. Activation of the muscarinic potassium channel in atrial muscle shortens the refractory period of atrial tissue. Increased vagal influence shortens the atrial refractory period and increases atrial dispersion with no change in atrial conduction velocity. Nocturnal, postprandial, or bending-associated onset is very suggestive of this mechanism. On the other hand, adrenergic-mediated AF is due to increased sympathetic tone, as occurs with pheochromocytoma. The clinical presentation may be due to paroxysmal daytime episodes of AF, which usually are associated with exercise, stress, or acute clinical states. Since AF may be initiated or maintained by different mechanisms in different patients, it is clear that no single antiarrhythmic drug could be uniformly effective in the management of AF.

With acute onset of AF, there is loss of atrial contraction, which ordinarily provides 10-40% of ventricular filling. An increased and irregular ventricular rate ensues. Diastole becomes shorter and erratic. It is notable that an irregular ventricular rhythm was found to be associated with a 15% decline in cardiac output compared with a regular rhythm at the same pacing rate.[17] With time, dilatation of both atria occurs. As higher atrial pressure becomes necessary to preserve forward flow from atrium to ventricle, atrial compliance decreases and intra-atrial pressures increase. Atrial remodeling may make future cardioversion more difficult with time. Because of the decrease in atrial compliance, ventricular volume increases, and ventricular diastole shortens. A rise in left atrial pressure may result in pulmonary edema. A shorter diastolic filling period leads to decreased stroke volume and decreased cardiac output, a so-called tachy-cardia-mediated cardiomyopathy.[18] Unlike other cardiomyopathies, this is reversible when ventricular rate is controlled.[19] Dilation of the atria is associated with stagnation of blood and increased incidence of thrombus.

These changes may account for pulmonary edema and systemic emboli. After either electrical or chemical cardioversion, atrial mechanical function does not return immediately (atrial stunning) and may require minutes to six months before atria return to normal function.

**Medical Causes of Atrial Fibrillation.** Medical illness associated with or causing AF may be divided into cardiac and noncardiac etiologies.

*Cardiac causes include:*

- Ischemic heart disease (CAD, unstable angina, variant angina, MI);
- Pericarditis/myocarditis/endocarditis;
- CHF;
- Restrictive or hypertrophic cardiomyopathy;
- Sick sinus syndrome;
- WPW syndrome (pre-excitation);
- Hypertension;
- Post-operative cardiac and noncardiac surgery;
- Mitral valve disease: prolapse annular calcification, mitral stenosis, or mitral regurgitation;
- Rheumatic heart disease (mitral stenosis, mitral regurgitation);
- Left ventricular hypertrophy (aortic stenosis);
- Congenital heart disease (atrial septal defect [ASD], ventricular septal defect [VSD], transposition of great vessels, Ebstein's malformation);
- Infiltrative disorders: sarcoid, amyloid; and
- Atrial myxoma, atrial thrombi.

*Noncardiac causes include:*

- Hypoxia;
- Pulmonary (pulmonary embolism, pneumonia, chronic obstructive pulmonary disease [COPD], pulmonary hypertension, hypoxia of any cause);
- Endocrine (thyrotoxicosis, pheochromocytoma, hypoglycemia);
- Neurologic (head injury, intracranial hemorrhage, ischemic stroke, intracranial tumors);
- Alcohol intoxication and withdrawal ("holiday heart");
- Drugs (atropine, digoxin, heroin, theophylline, cocaine and other sympathomimetics, adenosine, antidepressants, anesthetics, caffeine);
- Physical (lightning, electrical shock, hypothermia, thoracic trauma);
- Metabolic (hypokalemia, hypomagnesemia, hypercalcemia);
- Neuromuscular disease (Friedrich's ataxia, muscular dystrophy);
- "Lone" AF (structurally normal heart); and
- Miscellaneous (exercise, nicotine gum, malignancy involving atria, stress).

## Classification

New onset AF usually includes acute onset and a duration of less than 24-48 hours. Approximately 50% of these episodes terminate spontaneously within 24 hours without antiarrhythmic therapy.[20]

Lone AF is AF in the absence of overt cardiovascular disease or precipitating illness; isolated AF is AF associated with an acute self-limited disorder (e.g., pericarditis, pulmonary embolus).

Paroxysmal AF has self-terminating episodes, which usually last less than 48 hours. Persistent AF is not self-terminating. Chronic AF is defined as continuous AF, which makes it difficult to maintain sinus rhythm after conversion and forces the physician to anticipate and prevent complications.

## Clinical Presentation

**The History.** Patients with AF may be asymptomatic. They may present with symptoms related to ventricular rate, including palpitations, or chest pain from cardiac ischemia. Patients may have shortness of breath, orthopnea, decreased exercise tolerance, dyspnea on exertion, or CHF with frank pulmonary edema. Loss of atrial contraction or

## Table 1. Atrial Fibrillation: Risk Factors

CARDIAC DISEASE: CLINICAL AND ECHOCARDIOGRAPHIC RISK FACTORS

- Congestive heart failure (CHF)
- Rheumatic heart disease
- Hypertensive heart disease
- Mitral valve disease: stenosis, regurgitation, prolapse, annular calcification
- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- Pericarditis/serositis
- Congenital heart disease
- Infiltrative disorders: sarcoid, amyloid

EXTRACARDIAC CONDITIONS

- Acute ethanol intoxication or withdrawal
- Hyperthyroid state
- Cholinergic drug use
- Surgery
- COPD
- Sepsis
- Hypoxia
- Substance Abuse: theophylline, cocaine, atropine, antidepressants, caffeine, heroin, nicotine gum
- Hypothermia
- Pheochromocytoma
- Pulmonary embolus
- Trauma
- Electrolyte disturbances
- Lightning, electrical shock
- Beta-blocker withdrawal
- Diabetes

atrioventricular (AV) synchrony may account for some of these symptoms as well, especially in patients with valvular or myocardial disease. The disruption of normal ventricular filling may lead to rapid onset of heart failure, hypotension, and/or syncope. Syncope also may be caused by other dysrhythmias such as bradytachy syndrome, symptoms from accessory pathways, ventricular tachycardia, or sick sinus syndrome.

Neurologic symptoms related to thromboembolism, may include mental impairment, sleep loss, memory loss, irritability, syncope or near-syncope, or focal neurologic deficits.

Possible nonspecific complaints include fatigue, dizziness/lightheadedness, somnolence, tiredness, general ill health, or shortness of breath.

Important factors in the evaluation of the patient in AF include the presence of alcohol or illicit drug use, as well as risk factors for CAD. Any symptoms of possible ischemic heart disease or CHF should be elicited.

**The Physical Examination.** In the absence of underlying conduction system disease, the ventricular response will, in general, be rapid—approximately 100-160 beats/min. In the absence of accessory pathways, the ventricular response will seldom be more than 200 beats/min. Since the atrial rate may be 350-600 beats per minute, with a rapidly conducting accessory pathway, ventricular rates may exceed 300 beats/min and precipitate ventricular fibrillation.[2] Since some of the ventricular contractions may not transmit a pressure wave to the radial artery due to inadequate filling times, there may be a pulse deficit. In all cases, the pulse will be irregularly irregular.

In patients with slow rates that are not attributable to medications inhibiting AV node conduction, there may be underlying conduction system disease that will increase the risk of bradycardia upon cardioversion. Younger patients may have a more rapid ventricular response than elderly ones due to absence of disease in the conduction system. Normalization of the rate in a patient with pre-existing AF on digitalis may indicate the presence of atrial tachycardia, junctional or idioventricular rhythms, or ventricular tachycardia.

Specific physical findings in patients with hemodynamically stable AF, considered to be present when ventricular rate is less than 140 beats/min, may include:

- Soft or absent $S_1$;
- Irregularly irregular pulse;
- Pulse deficits;
- Loss of jugular A waves; or
- Physical signs of thyrotoxicosis, pulmonary embolism, pericarditis, CHF, or valvular heart disease.

Physical findings in unstable AF, arbitrarily defined as ventricular rate greater than 140 beats/min, may include:

- Distended neck veins;
- Pulmonary rales;
- Hypotension;
- Cardiogenic shock; or
- Physical signs of thyrotoxicosis, PE, CHF, pericarditis, or valvular heart disease.

## Diagnostic Studies of Use in the ED

Recommended serum laboratory studies may include: CBC, routine chemistry (consider electrolytes and magnesium), and PT/PTT. If the patient is taking warfarin, the therapeutic INR should be between 2 and 3. Thyroid function tests may be indicated. Consider toxicology screen for ethanol, theophylline, sympathomimetics, and digoxin as appropriate.

Chest x-ray (CXR) may reveal cardiomegaly, CHF, pneumonia, COPD, tumor, or evidence for PE.

Electrocardiogram (ECG) will confirm the diagnosis. AF shows an irregularly irregular rhythm with no visible P waves. Fibrillatory (F waves) appear as isoelectric or fine. If there is a coarse baseline, the rhythm may be mistaken for atrial flutter. QRS complexes typically are narrow. A rapid ventricular response (> 160 beats/min) with wide complex is suggestive of an accessory pathway. Ashman's phenomenon may be present, which may be manifested by a transient bundle-branch block that produces a wide QRS complex when a long R-R interval is followed by one that is short.

Patients taking digitalis may have characteristic ST depression; patients on class 1A antiarrhythmic agents may show QT prolongation. Evidence for ischemia, pericarditis, old or new infarction, or ventricular hypertrophy should be sought.

Echocardiogram, either transthoracic (TTE) or transesophageal (TEE) can determine atrial size and measure left ventricular function and dimensions (chamber and wall). It can evaluate the mitral and aortic valves, search for left atrial "smoke" from stasis or thrombus, and assess mechanical atrial function after cardioversion. Left atrial enlargement (> 5.0 cm) is a specific marker for reduced likelihood of achieving and/or maintaining sinus rhythm,[19] although it may be of more prognostic value if rheumatic valvular disease is present.[21]

## Therapy

It has been demonstrated that "atrial fibrillation begets atrial fibrillation."[22] That is, the longer a patient is in AF, the more likely it is that he or she will remain in AF. Long-standing AF reduces the odds of successful cardioversion and of maintenance of sinus rhythm.[23] This results from a series of electrophysiologic and anatomic changes that occur in the atrium during AF and facilitate reentrant impulses within the atria. Simply put, the longer the atria remain in AF, the more prone they are to stay in AF or convert back to AF, even after conversion to sinus rhythm.[24] Chronic AF (> 12 months) has been cited as a marker of reduced likelihood of achieving or maintaining sinus rhythm.[19] Thus, the main thrust of the management of acute or paroxysmal AF is to control the ventricular rate and restore sinus rhythm as soon as it is feasible.

Points that are debated include whether the rhythm should be accepted and the rate controlled, or whether to strive for the restoration and maintenance of sinus rhythm. The patient may have an acute, treatable illness such as pneumonia, pericarditis, pulmonary embolus, or thyrotoxicosis,[25] in which the AF is likely to resolve upon correction of the underlying disease. Current trials (e.g., Atrial Fibrillation Follow-up Investigation in Rhythm Management [AFFIRM] and Pharmacological Intervention in Atrial Fibrillation [PIAF]) will attempt to address this issue from the perspectives of cost, efficacy, adverse effects, and benefits of attempting to maintain sinus rhythm.[26] Since the therapeutic end points involve rate control, conversion to sinus rhythm, and prevention of thromboembolic complications, these will be addressed in turn. It has not been shown yet that antiarrhythmic treatment of AF prolongs survival.[19] In fact, only the benefits of anticoagulation are supported by substantial evidence.[27] Disease nature and severity are major factors determining the likelihood and severity of proarrhythmic risk. Therefore, treatment strategies will vary by patient.

In all cases, the underlying cause(s) of AF should be determined and addressed. Infection, hypoxia, hypovolemia, and electrolyte disturbances should be treated. The patient should be placed on a cardiac monitor, and have an intravenous line established, oxygen administered as needed, and vital signs monitored closely.

**Medical Management.** Medical management of AF, therefore, centers on the following three guidelines:

- Control of ventricular rate;
- Prevention of thromboembolism; and
- Restoration and maintenance of normal sinus rhythm.

These issues should be addressed in turn, always with an eye toward improving quality of life, minimizing iatrogenic problems, and achieving prolongation of life.[28]

Some of the questions regarding the relative merits of rate control with anticoagulation, as opposed to rhythm restoration, may be resolved by ongoing studies such as the NIH-sponsored AFFIRM trial.[29]

### Table 2. Emergency Electrical Cardioversion

- Choose a short-acting, intravenous anesthetic, or amnestic agent: Propofol, etomidate, methohexital, and midazolam are options.
- Paddle placement: Hand-held right parasternal at 2nd, 3rd intercostal space and left lateral at cardiac apex, or defibrillator pad-left parasternal and left posterior chest.
- Energy: 200 J initially, if unsuccessful wait 3 minutes, then 300 J. If still unsuccessful, 360 J. *Synchronization on the R wave to prevent ventricular fibrillation.*

## Controlling the Ventricular Rate

Ventricular rate in AF is typically 130-200 beats/min if the conduction system is intact. Thus, younger patients have higher ventricular rates compared to the elderly, who usually have AV nodal disease. While the accepted goal for the ventricular rate has been less than 100 beats/min, this may vary with clinical status. For example, patients with poor left ventricular (LV) function benefit from a higher heart rate for maintenance of cardiac output. On the other hand, a lower heart rate is advantageous for patients with diastolic dysfunction because ventricular filling is dependent on the length of diastolic period, which is inversely related to the heart rate. Non-invasive studies have shown that maximal cardiac output may be achieved at a heart rate of 120 beats/min in patients with AF.[30] A heart rate during AF which is slightly higher than that found during sinus rhythm may be acceptable, to compensate for the loss of atrial contribution to cardiac output.[31]

If the patient is hemodynamically unstable, electrical cardioversion is the therapy of choice. *(See Table 2.)* In the emergency setting, this is limited to unstable patients and seldom will be necessary. The effectiveness depends on how long a patient has been in AF. Failure rates are approximately 20-50% and this is an uncomfortable procedure. Patient sedation is desirable, while maintaining a systolic blood pressure of greater than 90 mmHg and intubating if necessary. Paddles may be placed on the anterior/posterior chest wall or at a position that corresponds to the apex/base of the heart, and synchronized cardioversion employed during the patient's respiratory expiration phase. Conversion success depends upon energy utilized: 100 J converts 50%, and 200 J converts approximately 85%. Additional shocks are administered at 360 J. Waiting three minutes between shocks reduces transthoracic impedance. In patients taking digoxin, less energy may be needed, but the patient should not be cardioverted if the AF is due to digoxin toxicity. Electrical cardioversion will be discussed further in the sec-

ond part of this two-part series. In general, emergency cardioversion from AF due to hypotension will be rare, but if the patient does not respond to a fluid challenge, cardioversion may be mandatory.

If the patient is hemodynamically stable, then the ventricular rate may be managed pharmacologically, or electrical cardioversion may be considered in a controlled setting.

Beta-blockers block the AV node, are negative inotropes, and may precipitate bronchospasm in patients with reactive airway disease. They may precipitate vasoconstriction in patients with peripheral vascular disease. They also are drugs of choice in AF associated with increased sympathetic tone, such as hyperthyroidism, alcohol withdrawal, fever, pain, or anxiety. They may be especially useful in patients who have sustained a previous myocardial infarction. Concomitant decreases in cardiac contractility may result in hypotension or heart failure.

Esmolol is a cardioselective beta-1-adrenergic blocking agent administered intravenously. It is desirable when there is a risk of heart failure or hypotension because of its rapid onset of action and its rapid metabolism by red cell esterases. Consequently, it has a short duration (T 1/2 = 9 minutes) of action. It is available only in IV form.

The loading dose is 0.5 mg/kg IV over one minute, and can be repeated as necessary. Ventricular rate response will occur within 15 minutes.

Maintenance dose range is 0.05 to 0.2 mg/kg/min IV (5-20 mg/h). Esmolol is very short acting, with complete disappearance of clinical effects within 20 minutes. Hypotension is encountered in 12-48% of patients.[32] The loading dose may be repeated as needed. *(See Table 3 for drug dosages.)*

Metoprolol also has beta-1 selectivity and a plasma half-life of 3-4 hours. The loading IV dose is 5 mg over 2-4 minutes, repeated up to three doses (maximum dose is 15 mg). The maintenance IV dose is 5-10 mg every six hours. The oral dose is 50-100 mg bid.

Atenolol has beta-1 selectivity, and is longer acting. The loading IV dose is 5 mg over five minutes; repeated in 10 minutes as needed. Maintenance PO dose is 25-100 mg once daily.

Propranolol is available intravenously or orally. The loading IV dose is 0.5-1.0 mg q 5 minutes, up to 0.15-0.2 mg/kg. Its half-life is 2-6 hours. Maintenance PO dose is 40-240 mg/day in 3-4 divided doses.

Use any beta-blocker, and especially the non-selective ones, with extreme caution in patients with severe CHF or bronchospastic disease.

Pindolol is a beta-blocker that has intrinsic sympathomimetic activity. It may, therefore, slightly increase the heart rate at rest, while slowing the ventricular response during periods when sympathetic tone is high. It has been proposed for use in AF associated with bradytachy syndrome,[19,33] and is dosed 5 mg bid up to 60 mg/day.

Calcium channel blockers cause a decrease in arterial smooth muscle tone and have the potential to cause negative inotropy. Since AV node conduction depends on the rate of recovery of the calcium slow channel, verapamil and diltizem both slow conduction though the AV node and can rapidly slow ventricular response in AF. Calcium antagonists as a class may precipitate CHF, AV block, and bradycardia. Hypotension may be due to vasodilation or negative isotropy.

Diltiazem blocks the AV node and is a negative inotrope, but less so than verapamil.[34,35] Therefore, it is safer to give to patients with borderline low blood pressure or mild-to-moderate CHF.[34] Its onset of action is rapid (< 5 minutes).

The loading dose for diltiazem is IV 0.25mg/kg over two minutes, with a repeat dose of 0.35 mg/kg over two minutes 15 minutes later if the first dose was ineffective. The patient may be pretreated with 100 mEq calcium if hypotension is a concern.

The maintenance IV dose is 5-15 mg/hr. Oral dose is in the range of 90-360 mg/day in divided doses, and a conversion from intravenous to oral diltiazem has been described.[36] Rate may slow gradually over 4-5 hours of infusion.

Diltiazem may cause hypotension in 4-7%,[37,38] bradycardia, or CHF; it acts synergistically with digoxin or beta-blockers.

## Table 3. Dosages of Drugs for Rate Control in Atrial Fibrillation

| |
|---|
| **DIGOXIN** |
| Initial dose: 0.5 mg IV; then 0.25 mg in one hour; repeat in six hours up to a maximum of 1-1.5 mg/24-hours |
| **DIGOXIN AND MAGNESIUM** |
| Initial dose: 0.5 mg digoxin IV, 2 gm $MgSO_4$ bolus, then 1 gm $MgSO_4$/hr X four hours |
| **DILTIAZEM** |
| Initial dose: 0.25 mg/kg IV bolus, (typically 20 mg); give second bolus of 0.35 mg/kg15 min later if needed |
| Maintenance dose: 5-15 mg/hour |
| Oral dose: 60 mg-90 mg every six hours |
| **PROPRANOLOL** |
| Initial dose: 1 mg IV, repeat in five minutes if needed |
| Oral dose: 10 mg every six hours, up to 80 mg every six hours |
| **METOPROLOL** |
| Initial dose: 5 mg IV, repeat in five minutes up to three doses as needed |
| Oral dose: 50-100 mg bid |
| **ESMOLOL AND DIGOXIN** |
| Digoxin initial dose: 0.25-0.5 mg, repeated in two hours |
| Esmolol loading dose: 0.5 mg/kg IV over one minute, repeated if necessary. Esmolol maintenance dose: 5-20 mg/hour |
| **VERAPAMIL** |
| Initial dose: 2.5-5 mg IV, repeat in 30 minutes if needed |
| Maintenance dose: 0.05-0.2 mg/min |
| Oral dose: 120-360 mg/day (80-120 mg every 6 hrs) |
| **AMIODARONE** |
| Initial dose: 5-7 mg/kg over 10-40 minutes |
| Maintenance dose: 200 mg-400 mg PO daily |

Verapamil blocks the AV node, is a negative inotrope, and is best avoided in patients whose ejection fraction is less than 35%.[7] In patients with CHF, verapamil can cause profound hypotension due to diminished cardiac contractility and peripheral vasodilatation. Its rapid onset of action (less than 10 minutes) is similar to esmolol.[39] Its half-life is approximately four hours.

The suggested loading dose is 2.5-10 mg over two minutes IV; repeated q 30 minutes (pretreat with 100 mEq calcium to reduce risk of hypotension). A 5-15 mg bolus dose reduces the ventricular rate within five minutes. Maintenance dose IV is 0.05-0.2 mg/min, or an hourly maintenance of 0.125 mg/kg/hr. Oral dose is 120-360 mg/day in divided doses.

Verapamil may cause hypotension, bradycardia, constipation, or CHF. It increases the digoxin level and acts synergistically with digoxin. It decreases clearance of metoprolol, and should be avoided in AF associated with WPW syndrome. Due to bronchodilating properties, it may be useful in patients with COPD. It may delay atrial remodeling when used at the onset of AF.[40]

Digitalis praparations (digoxin) have been widely available for decades. The effect is mediated primarily through the vagus nerve and results in an increased effective refractory period of AV node and a decreased conduction velocity. The clinical result is a decrease in ventricular rate. Digitalis inhibits the active transport of sodium and potassium across cell membranes. Inhibition of the sarcolemma ATPase results in an increase in cytosolic calcium, thus increasing the force of myocardial contraction.

It is a positive inotrope and is the most commonly prescribed cardiac glycoside. It increases carotid baroreceptor responsiveness and thereby decreases sympathetic tone. The overall effect is a lower blood pressure and higher cardiac output. Digitalis exhibits a slow onset of action for rate control and requires incremental administration. It is used mainly to potentiate the AV nodal blocking effect of calcium channel and beta-blockers.

Digitalis has demonstrated no effect on life expectancy in patients with CHF, but its use has clearly been shown to lessen symptoms and to decrease the likelihood of hospitalization.[41] It is best used in AF patients with moderate to severe CHF and for rate control in sedentary patients with chronic AF unlikely to undergo tachycardias due to exercise.

The loading IV dose for digitalis is 1 mg over 24 hours in 0.25-0.5 mg increments q 6-8 hours, with a maintenance IV or PO dose 0.125-0.25 mg qd.

Of all three classes of drugs, digitalis has slowest onset—up to 1-2 hours. Significant slowing of ventricular rate takes four hours or more—5.5 hours in one report.[42] Its effectiveness is attenuated with high catecholamine states (pain, respiratory distress, anxiety, alcohol withdrawal, fever, exercise, thyrotoxicosis, theophylline toxicity, postoperative states). Patients discharged home on digitalis may be aware of a rapid heart rate on physical exertion, as digitalis alone is ineffective in controlling ventricular rate during exercise.[31] Therefore, it has more use in sedentary older patients than in young, ambulatory people with AF, or when combined with a beta-blocking agent.[32]

## Table 4. Targeted Strategies for Rate Control in Atrial Fibrillation

| | |
|---|---|
| **PATIENTS WITH NO STRUCTURAL HEART DISEASE** | |
| Calcium channel blockers (Diltiazem, Verapamil) | Diltiazem easily converted to oral use. |
| Beta-blockers | Esmolol shortest acting. |
| **MILD OR MODERATE HEART FAILURE AND NORMAL TO HIGH BLOOD PRESSURE** | |
| Diltiazem IV | Rapid onset, follow with a continuous infusion. Use with caution if ejection fraction < 35%. |
| Digoxin IV | Does not cause hypotension. Significant slowing make take 4-6 hours. |
| Beta-blockers | Use with caution, consider esmolol. |
| **PATIENTS WITH SEVERE HEART FAILURE** | |
| *Consider electrical cardioversion.* | |
| Digoxin | Slow in onset for rate effect. |
| Amiodarone | Slight risk of extreme bradycardia. |
| **PATIENTS WITH SEVERE CHF AND SIGNIFICANT HYPOTENSION** | |
| *Consider electrical cardioversion.* | |
| Digoxin | Administer pressor agents. |
| Amiodarone | Administer pressor agents. |
| **PATIENTS WITH ISCHEMIC CHEST PAIN OR ACUTE MI** | |
| *Consider electrical cardioversion.* | |
| Beta-blockers | Have the advantage of decreasing mortality in acute MI. |
| Diltiazem IV | Titrate to effect. Some protection against ischemia. Avoid ejection fraction < 35%. |
| Ablation | Electrophysiology team to consider. |
| **WOLFF-PARKINSON-WHITE SYNDROME AND RAPID AF** | |
| Procainamide | Slows conduction through the bypass tract and may convert to sinus rhythm. Do not give digoxin or calcium channel blockers. |
| *If unstable or very rapid ventricular rate (> 250 beats/min), go directly to DC cardioversion.* | |
| **THYROTOXICOSIS** | |
| Beta-blocker IV | If the patient has concurrent high output CHF, choose esmolol because of its short half-life; otherwise may give propranolol. |
| **PATIENTS WITH RAPID RATES AND HYPERTENSION** | |
| Verapamil IV | Less expensive than diltiazem, but with more negative inotropy. |
| Diltiazem IV | |
| Beta-blockers | Metoprolol, atenolol, propranolol. |
| **ALCOHOL WITHDRAWAL** | |
| Beta-blockers | |
| **COPD** | |
| Verapamil | |
| Diltiazem | |
| Digoxin | |

Excretion may be impaired in elderly patients and those with renal dysfunction resulting in a long elimination half-life of 36-48 hours. Clinical benefit from digitalis in CHF is achieved at levels of 1 ng/mL, and optimal effect at levels of 1.3 ng/mL. The relationship between dose and response is not predictable. Patients may be clinically toxic at levels of 2-3 ng/mL. Symptoms of toxicity include: visual changes (intense yellow colors, halos), nausea, vomiting, and abdominal pain (consider the possibility of digitalis-mediated reduction in splanchnic blood flow and mesenteric ischemia). Decreased digitalis clearance may occur in patients taking quinidine, amiodarone, captopril, diltiazem, verapamil, or nifedipine. It is contraindicated in patients with hypertrophic cardiomyopathy and WPW syndrome.

Rate control can be improved with concomitant intravenous magnesium.[41,43-46] Magnesium may be administered to all patients with suspected magnesium deficiency, including alcoholics and patients taking loop diuretics, but not in those with renal insufficiency or renal failure. It is synergistic with calcium channel blockers, amiodarone, and beta-blockers in the control of ventricular rate in AF.[7] It is inexpensive and it slows ventricular rate. It is usually given in combination with digoxin. Recall that magnesium administration increases potassium loss and is contraindicated in renal failure. The suggested loading dose is 2 grams IV over 15 minutes,[47] followed by a maintenance infusion of 1 gm/hr for up to four hours. Watch for respiratory depression or hypotension. The serum magnesium level should be approximately 2.5 mmol/L. The serum potassium should be monitored and kept at no less than 4.0 mmol/L.

It is noteworthy that neither digitalis, calcium channel blockers, nor beta-blockers has any efficacy in converting AF to normal sinus rhythm.[42]

Blitzer et al have suggested the following choices for ventricular rate control, given the following clinical scenarios.[19] *Please note: Arrows indicate suggested preference of use. (Targeted strategies for rate control of AF in different clinical scenarios are shown in Table 4.)*

1. No structural heart disease: Calcium blockers → beta-blockers → digoxin;
2. Hypertension: Calcium blockers (if LVH) → beta-blockers → digoxin;
3. Ischemic heart disease: beta-blockers → calcium channel blockers/digoxin → ablation;
4. Sick sinus syndrome: Pindolol → digoxin → pacemaker plus drug;
5. CHF/dilated cardiomyopathy: Digoxin → beta-blocker → Amiodarone vs. ablation and pacing;
6. Hypertensive cardiomyopathy: beta-blocker → verapamil → diltiazem/amiodarone vs. ablation with pacemaker;
7. COPD: Verapamil → diltiazem → digoxin;
8. Peripheral vascular disease: Diltiazem → verapamil → digoxin.

Other agents that have been used for rate control in AF include clonidine, an alpha-2 antagonist, which has been shown to slow ventricular response in AF by 29%, and amiodarone, which will be discussed further in part two of this series.[48]

## Conclusion

AF is extremely common and results from a wide range of both cardiac and noncardiac etiologies. As our population ages, an increase in patients with AF can be expected. Appropriate care of the patient with AF requires a keen sense of familiarity with the various etiologies and multiple strategies for ventricular rate control. Part II of this issue will discuss reducing the risk of thromboembolism and long-term management of AF.

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## Physician CME Questions

10. Atrial fibrillation is extremely common. Which of the following is true?
    A. The prevalence in the general population is approximately 10%.
    B. AF is the most common sustained arrhythmia, affecting an estimated 2 million Americans.
    C. One-half of all patients with AF are older than 85 years of age.
    D. The prevalence increases with age, peaks at 70 years of age, then declines.

11. Which of the following statements regarding chronic AF is correct?
    A. The risk of stroke is 2-3 times greater than in the general population, with an incidence of 3-4% per year.
    B. AF results in no significant change in all-cause mortality, except in patients with increased age.
    C. AF complicates 20-30% of myocardial infarctions.
    D. It is responsible for 45% of embolic strokes and 85% of all thromboembolic events originating from the heart.

12. AF has both cardiac and noncardiac etiologies. A common environmental cause of AF is:
    A. hyperthermia.
    B. hypothermia.
    C. near drowning.
    D. hymenoptera stings.

13. Which of the following is true regarding the classification of AF?
    A. New onset AF usually is acute onset with a duration of less than than 96 hours.
    B. New onset AF will terminate spontaneously within 24 hours in ~ 50% of episodes.
    C. Lone AF usually is associated with an acute self-limited illness.
    D. Paroxysmal AF typically is not self-terminating, which makes it difficult to maintain normal sinus rhythm.

14. Regarding heart rate responses in AF, which of the following is correct?
    A. Ventricular response typically is rapid and greater than 200 beats/min.
    B. Atrial rates are typically 100-160 beats/min.
    C. Rapid ventricular rates are seen more commonly in older patients and those on digitalis.
    D. In the presence of a rapidly conducting accessory pathway, ventricular rates may exceed 300 beats/min and precipitate ventricular fibrillation.

15. Match the correct drug choices (listed in descending preference) for rate control, for the following clinical scenarios.
    A. Ischemic heart diseases: Digoxin → verapamil → metoprolol.
    B. Hypertensive: Verapamil → digoxin → pacemaker.
    C. COPD: Digoxin → propranolol → diltiazem
    D. No structural heart disease: Diltiazem → metoprolol → digoxin

16. Which of the following statements regarding drug selection for AF rate control is correct?
    A. Verapamil has rapid onset (< 5 min); can be used in patients with low ejection fractions; should be avoided in WPW.
    B. Digoxin has slower onset of action (4 hours or more); should be used carefully in patients with renal insufficiency; is useful in WPW.
    C. Diltiazem has rapid onset (< 5 min); blocks AV node; is safer to give to patients with borderline hypotension or mild-moderate CHF than verapamil.
    D. Esmolol is a non-selective beta-blocker for IV use; has a rapid onset; is moderately short acting (~ 60 minutes duration); rarely causes hypotension.

17. Which of the following statements regarding electrical cardioversion is true?
    A. It is the procedure of choice for hemodynamically unstable or digoxin toxic patients.
    B. Overall, it is usually is successful in 50-70% of patients, even in chronic AF.
    C. It should be accomplished with synchronized cardioversion at 200 J, following patient sedation.
    D. It should be accomplished by three rapid, synchronized shocks at 360 J to decrease transthoracic impedance.

**In Future Issues:**

Atrial Fibrillation in the Elderly: Part II