Mission control: Man
An estimated 2.2 million Americans have atrial fibrillation (AF), the most common cardiac dysrhythmia seen in clinical practice today. AF is responsible for more hospital admissions than any other dysrhythmia and is associated with an increased risk of heart failure and stroke. Mortality for patients with AF is about twice that of people with normal sinus rhythm, and is linked to the severity of underlying heart disease.

Over the past 2 decades, hospital admissions for AF have increased 66% due to the aging population, an increased incidence of chronic heart disease, and more frequent diagnosing through the use of ambulatory monitoring devices.

This article discusses normal cardiac conduction, the pathophysiology of AF, your role in recognizing it, and appropriate nursing interventions.

About cardiac conduction
Normal cardiac conduction, resulting in normal sinus rhythm and synchronized, coordinated contraction of the atria and ventricles, starts in the sinoatrial (SA) node. Pacemaker cells in the SA node discharge regular impulses at a rate of 60 to 100 beats/minute. As the electrical impulse leaves the SA node, it’s conducted through the left atria by way of Bachmann’s bundle and through the right atria via the internodal tracts, resulting in synchronized atrial depolarization and atrial contraction. The electrical activity is recorded on the ECG tracing as a P wave.

Leaving the atria, the electrical impulse is then conducted to the atrioventricular (AV) node, which, in addition to relaying electrical impulses from the atria to the ventricles, has three main functions:
- Slowing conduction to let the atria contract and empty their contents into the ventricles (atrial kick) before the ventricles contract. On the ECG, you’ll see this delay in the AV node as the flat (isoelectric) segment of the PR interval known as the PR segment.
- Serving as a backup pacemaker at a rate of 40 to 60 beats/minute if the SA node fails or its impulses are blocked, or if the SA node rate is less than the AV node rate.
- Blocking impulses to the ventricles when the atrial rate is rapid to protect the ventricles from dangerously fast rates.

After the delay in the AV node, an electrical impulse moves rapidly through the bundle of His, left and right bundle branches, and the
Purkinje fibers. On ECG, this electrical activity is recorded as the QRS complex, which represents ventricular depolarization and, in the normal heart, ventricular contraction.

The ST segment represents the end of ventricular depolarization and the beginning of ventricular repolarization. The T wave represents the latter phase of ventricular repolarization, and the QT interval represents ventricular depolarization and repolarization.

**AF: Off the beat**

In AF, the electrical impulses no longer travel in an orderly fashion through the normal conduction pathway. Instead, multiple ectopic pacemakers (pacemakers outside the SA node) in the atria take over cardiac conduction. The ectopic atrial pacemakers responsible for AF fire 400 to 600 beats/minute; as these impulses spread through the atria, they compete for a chance to travel through the AV node. The rapid firing causes the atria to quiver instead of contracting in a synchronized, coordinated manner. Atrial activity is irregular, disorganized, chaotic, and very rapid. On ECG, you’ll see irregular, wavy deflections (fibrillatory or f waves) replacing the P wave, and you won’t be able to count the atrial rate.

Fortunately, the healthy AV node generally limits the impulses that get through to the ventricles (unless the patient has an accessory conduction pathway). Many patients with AF have a slower ventricular rate, often 110 to 180 beats/minute in an irregularly irregular rhythm.

**What causes AF?**

AF that’s temporary and reversible may be associated with emotional stress or excessive alcohol consumption (sometimes called holiday heart syndrome), surgery, electrocution, myocardial infarction, pericarditis, myocarditis, pulmonary embolism, or metabolic disorders such as hyperthyroidism. Treating the underlying causes often resolves the AF.

Underlying heart diseases related to the development of AF include hypertension, coronary artery disease, valvular heart disease, heart failure, and congenital heart disease. Obesity is also a risk factor for development of AF, apparently related to left atrial dilation.

Familial or genetic AF is a dysrhythmia without underlying cardiac disease running in a family. Patients whose parents had AF appear to be more likely to develop the dysrhythmia themselves, for reasons not yet fully understood.

Older patients are somewhat more likely to have AF than younger ones, and men are slightly more likely than women to develop AF. However, women diagnosed with AF carry a longer-term risk of premature death.

**Types of AF**

AF can fall into several overlapping classifications:

- **Paroxysmal AF** is self-resolving and lasts less than 7 days.
- **Persistent AF** lasts more than 7 days and doesn’t self-resolve.
- **Permanent AF** is defined as AF in which cardioversion has failed or hasn’t been attempted.
- **Recurrent AF** is defined as two or more episodes of AF. Both paroxysmal and persistent AF can also be recurrent.

**Recognizing AF**

Patients with AF may or may not be symptomatic. An embolic complication or exacerbation of heart failure may be the first sign of AF. The most common signs and symptoms include palpitations, dyspnea, chest pain, lightheadedness, fatigue, or syncope. An irregular pulse suggests AF.

Diagnosing AF can be challenging because of the sometimes unpredictable and intermittent nature of the dysrhythmia. After a thorough medical history and physical assessment, the following diagnostic tests may help identify AF:

- **12-lead ECG.** The irregularly irregular rhythm of AF is easy to recognize, provided AF occurs during the ECG. The patient’s atrial rate typically is 350 to 600 f waves/minute; the ventricular rate typically is 160 to 180 beats/minute if AF is untreated, and 60 to 80 beats/minute if AF is treated.
- **Ambulatory ECG.** Intermittent episodes of AF may not show up on a standard ECG, requiring a 24-hour recording of the heart’s rate and rhythm. If episodes of AF are infrequent, the ambulatory ECG recording
may not capture them. In this situation, the patient can wear a patient-activated event recorder for 1 to 4 weeks. After sensing irregular heartbeats or AF symptoms, the patient presses a button to start the recording. Ambulatory electrocardiography also can be used to evaluate rate control.1

- Electrophysiology (EP) studies. When ablative therapy to correct AF (or other dysrhythmias that trigger AF) is planned, an EP study is indicated.1 In this study, the electrophysiologist attempts to induce the patient’s dysrhythmia so it can be treated.

- Blood tests. Chemistries and thyroid studies may be ordered to rule out thyroid problems or blood chemistry abnormalities that may lead to AF. Hepatic and renal function studies also may be helpful, especially when the patient’s ventricular rate is hard to control. Toxicology testing may be done to rule out acute intoxication, and B-type natriuretic peptide (BNP) or pro-BNP levels may help rule out heart failure.

- Chest X-ray. This test may show enlarged cardiac chambers and heart failure, but mostly is used to check for pulmonary pathology and evaluate the pulmonary vasculature.1

- Echocardiography. All patients with AF should have a two-dimensional Doppler echocardiogram to assess the left atrium and ventricle, evaluate left ventricular wall thickness and function, and rule out occult valvular or pericardial disease and hypertrophic cardiomyopathy. This information can help guide antiarrhythmic and antithrombotic therapy.

**Clinical consequences of AF**

Because the atria are quivering and not emptying completely, blood pools in them, increasing the potential for thrombus formation. Thrombi from the right side of the heart can enter the pulmonic circulation and lead to pulmonic embolism; those from the left side of the heart can enter the systemic circulation and lead to ischemic stroke or an acute arterial occlusion of vessels supplying the lower extremities, intestines, or kidneys.

AF also causes a patient to lose atrial kick, the atrial component of the cardiac cycle. This loss, combined with the rapid irregular ventricular response, can reduce cardiac output by 20% to 25%. As cardiac output falls, patients may decompensate and become symptomatic. Fatigue and shortness of breath are common symptoms of inadequate cardiac output and heart failure.4

**Treating AF**

The goals of managing a patient with AF are to control the ventricular rate, restore normal sinus rhythm if possible, and prevent thromboembolic complications. Whether rate control or rhythm control is pursued first depends on many factors: the type and duration of AF; the severity and type of signs and symptoms; whether the patient has associated cardiovascular disease or other medical conditions; patient age, short- and long-term treatment goals, and treatment options. All patients in AF need antithrombotic therapy for thromboembolism prevention unless contraindicated.

- Ventricular rate control. Uncontrolled AF is defined as AF with a ventricular rate greater than 100 beats/minute. To maximize cardiac output, the ventricular rate needs to be reduced to 60 to 80 beats/minute when the patient is at rest.1 (AF with a ventricular rate of less than 100 beats/minute is called AF with a controlled ventricular response.)5 Calcium channel blockers, beta-blockers, and digoxin prolong the AV node refractory period and can be used to control the ventricular rate, unless contraindicated. In older adults, if rate control alleviates the patient’s symptoms, the healthcare provider may decide not to attempt to restore sinus rhythm. But if rate control offers inadequate symptomatic relief, restoring normal sinus rhythm becomes a clear long-term goal.5

- Restoring normal sinus rhythm. The decision to attempt restoration of normal sinus rhythm depends on the severity of the patient’s symptoms and hemodynamic status, and the potential risk of antiarrhythmic drugs. Immediate synchronized electrical cardioversion is recommended for patients with myocardial ischemia, symptomatic hypotension, angina, or heart failure, and a rapid ventricular response that doesn’t respond to drugs. Immediate electrical cardioversion also
is recommended for patients with AF and preexcitation syndromes that lead to very rapid tachycardia or hemodynamic instability. For hemodynamically stable patients, electrical cardioversion is recommended if signs and symptoms of AF are unacceptable to the patient. If AF recurs after cardioversion, another attempt at electrical cardioversion can be made after the patient is given antiarrhythmic medication.3

Pharmacologic cardioversion can be attempted with flecainide, dofetilide, propafenone, ibutilide, or amiodarone. Patients who’ve been in AF for more than 48 hours or for whom the time of AF onset is unknown should take oral anticoagulation unless contraindicated, for at least 3 weeks before and 4 weeks after cardioversion, regardless of the method.3 Transesophageal echocardiography (TEE) may be performed as an alternative to anticoagulation before cardioversion to rule out mural thrombi. The patient then can be given I.V. heparin and cardioverted within 24 hours, followed by oral anticoagulation for 4 weeks. If the TEE reveals a thrombus, the patient will need oral anticoagulation for 3 weeks before cardioversion and 4 weeks or longer afterward. Hemodynamically unstable patients with AF or more than 48 hours duration require immediate cardioversion and concurrent administration of I.V. heparin, followed by oral anticoagulation for 4 more weeks.3

Restoring and maintaining normal sinus rhythm is less likely in patients with persistent and prolonged AF—such as those patients with asymptomatic AF—because the increasing duration of the episodes leads to shorter refractory periods, a phenomenon known as EP remodeling.3

• Preventing stroke. Long-term anti-thrombotic therapy with either warfarin or aspirin is recommended to prevent stroke in patients with AF. The therapy chosen is based on assessment of the patient’s stroke risk, bleeding risk, patient preferences, and access to high-quality anticoagulation monitoring.

• The risk of stroke in patients with AF varies according to age and associated vascular disease. The CHADS2 scoring system helps determine stroke risk. (CHADS2 is an acronym for Cardiac failure, Hypertension, Age, Diabetes, Stroke [doubled] scoring system.) The CHADS2 risk index assigns 2 points for a history of stroke or transient ischemic attack (TIA) and 1 point each for age over 75, a history of hypertension, history of diabetes, or recent heart failure.

Anticoagulation therapy is prescribed based on risk assessment. The treatment recommendations are:

• low-risk patients (score of 0) should take 81 to 325 mg of aspirin daily
• low-moderate-risk patients (score of 1) should take oral warfarin to maintain an international normalized ratio (INR) between 2 and 3, or 75 to 325 mg of aspirin daily
• moderate-risk patients (score of 2) and high-risk patients (score of 3 or more) should be prescribed warfarin with a target INR between 2 and 3.6

Patients who have AF and a history of stroke or TIA but no other CHADS2 risk factors are considered high risk and should be treated with warfarin unless contraindicated. Patients with paroxysmal or persistent AF and valvular heart disease (such as mitral stenosis) also are at high risk for embolism and should also be treated with oral warfarin.6

Studies have found that only about half of patients with AF who are candidates for anticoagulation receive warfarin, and that anticoagulation is particularly underused in patients age 75 and older who have AF possibly because anticoagulation therapy poses a higher risk of serious bleeding in older patients than younger ones. However, if the patient’s risk of ischemic stroke without anticoagulation is greater than the risk of bleeding, anticoagulation is warranted.6

Intracerebral bleeding, the most devastating complication of anticoagulation in older adults, can be exacerbated by age, poorly controlled hypertension, and concomitant use of aspirin or a nonsteroidal anti-inflammatory drug.6 Researchers aren’t sure if sustained control of hypertension in patients with AF reduces cardiogenic embolism. But controlling hypertension in patients with AF is critically important in reducing both the risk of ischemic stroke and the risk of intracerebral hemorrhage.

Nonpharmacologic therapy for AF

Many patients with AF need long-term treatment with medication for the prevention and control of AF. Unfortunately, medications are effective only 50% to 75% of the time and many patients can’t tolerate the adverse reactions, such as nausea, dizziness, and fatigue. As a result, several procedures have been developed to treat, prevent, and even cure AF7

• Percutaneous radiofrequency catheter ablation, a minimally invasive procedure, uses a catheter inserted into the groin and advanced into the heart to ablate the sources of abnormal conduction in the atria or pulmonary veins.8

Before the ablation, EP studies and advanced cardiac imaging and mapping techniques are used to identify these areas of abnormal conduction signals. A recent review found that catheter ablation is effective for up to
1 year when used as a second-line therapy in younger patients with little or no left atrial enlargement.9
• AV node ablation therapy also uses radiofrequency energy delivered via catheter to destroy AV node tissue and permanently sever the electrical connection between the fibrillating atria and the ventricles. This procedure effectively controls ventricular rate. However, the patient will need a permanent pacemaker to supply ventricular stimulation and, because the atria continue to fibrillate, the patient will continue to need anticoagulation therapy. Because of its irreversible nature and the need for a permanent pacemaker, this option is typically limited to highly symptomatic patients.
• An implantable atrial defibrillator can detect and convert AF to a normal rhythm through internal synchronous shocks, and may prevent AF recurrences over the long term. But cardioversion requires about 3 joules, and most patients find discharge energies over 1 joule uncomfortable without sedation, which requires a medical setting. However, atrial pacing is a painless therapy that successfully terminates AF about 20% of the time, making it useful for patients who tolerate AF poorly.
• The surgical maze procedure can be used to create a pattern or maze of scar tissue in the atria that interfere with stray electrical impulses causing AF. (Scarf tissue doesn’t conduct electrical impulses.) This procedure has a high success rate, but because it’s an open chest procedure requiring general anesthesia and cardiopulmonary bypass, it’s generally reserved for people who don’t respond to other treatments or is done during other necessary heart surgery.

Less-invasive variations of the surgical maze technique include thoracoscopic and catheter-based epicardial techniques. In these newer maze procedures, small incisions are made in the chest to access the heart. Tiny video cameras and robotics are used to make the maze incisions. Percutaneous ablation is an alternative method of creating lesions in the atria.

What patients need to know
Reassure patients with AF that even permanent AF may not interfere with a normal, active life. Encourage them to keep all appointments with their healthcare provider and take medications as prescribed. Teach them about adverse drug reactions and when to call the healthcare provider. Also teach patients to take all medications to every healthcare provider and ED visit and warn them to avoid over-the-counter medicines (some contain stimulants that can trigger rapid heart rhythms).

For patients taking warfarin, explain the importance of regular blood testing and recommend a medical-alert bracelet. Teach them to monitor for overt and occult bleeding and make sure they know when to call the healthcare provider or seek emergency care.

Patients who’ve had infrequent and brief episodes of AF may need no further treatment than learning to avoid triggers, such as caffeine, alcohol, or overeating. Those with AF that’s difficult to control should be involved in deciding whether to undergo one of the available procedures.

All patients should take steps to improve their cardiac health, such as not smoking; maintaining a healthy weight; getting 30 minutes of regular physical exercise daily; choosing a low-fat diet with a variety of grains, fruits, and vegetables; controlling hypertension and dyslipidemia and limiting or avoiding alcohol.

By understanding AF and its management, you can help your patient get appropriate care, avoid complications, and resume a normal lifestyle. ◆

REFERENCES

RESOURCES
American Heart Association http://www.americanheart.org
American Stroke Association http://www.strokeaha.org
Cleveland Clinic Heart and Vascular Institute http://www.clevelandclinic.org/heartcenter/pub/atrial_fibrillation/atrial.htm

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