ATRIAL FIBRILLATION

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ABSTRACT

The primary treatment of atrial fibrillation involves rate control, prevention of thromboembolic events and restoring the heart to sinus rhythm. Control of atrial fibrillation is achieved through pharmacological treatment and cardioversion. Refractory atrial fibrillation may benefit from surgical approaches to treatment. Diagnosis of atrial fibrillation requires investigation into possible reversible causes as a first approach to therapy. New guidelines influencing how atrial fibrillation is understood and treated, including newer pharmacological agents, are discussed.
**Policy Statement**

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**Continuing Education Credit Designation**

This educational activity is credited for 3 hours. Nurses may only claim credit commensurate with the credit awarded for completion of this course activity.

Pharmacology content is .5 hours (30 minutes).

**Statement of Learning Need**

Ongoing education is needed on the public health burden of atrial fibrillation. Current medical, surgical and pharmacological guidelines for the treatment of atrial fibrillation support clinicians caring for patients with an acute or chronic atrial fibrillation condition.

**Course Purpose**

To provide clinicians with current knowledge of the symptoms, diagnosis and treatment of patients with atrial fibrillation.
Target Audience

Advanced Practice Registered Nurses and Registered Nurses
(Interdisciplinary Health Team Members, including Vocational Nurses and Medical Assistants may obtain a Certificate of Completion)

Course Author & Planning Team Conflict of Interest Disclosures

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Acknowledgement of Commercial Support

There is no commercial support for this course.

Please take time to complete a self-assessment of knowledge, on page 4, sample questions before reading the article.
Opportunity to complete a self-assessment of knowledge learned will be provided at the end of the course.

1. The incidence of atrial fibrillation:
   a. increases with age
   b. decrease with age
   c. is not affected by age
   d. increases with age but only for women

2. Which of the following are established risk factors for atrial fibrillation?
   a. African American ethnicity, chronic kidney disease
   b. Hypertension, heart failure
   c. Coronary artery disease, COPD
   d. Cirrhosis of the liver, stroke

3. The diagnosis of atrial fibrillation can only be made by:
   a. signs and symptoms
   b. identification of risk factors
   c. blood tests
   d. a 12-lead ECG

4. Cardioversion is used if the patient with atrial fibrillation:
   a. is > age 80
   b. has a history of COPD
   c. is hemodynamically unstable
   d. is < age 40

5. Rhythm control in patients who have atrial fibrillation can be attained by using:
   a. beta blockers, diltiazem, or digoxin
   b. electrical or pharmacological cardioversion
   c. supportive care and lifestyle changes
   d. the Cox maze procedure
Introduction

Atrial fibrillation is the most common arrhythmia. It is characterized by ectopic atrial activity and an irregular, often rapid ventricular response. Atrial fibrillation is typically seen in patients who have common cardiovascular diseases such as hypertension and heart failure or who have lifestyle risk factors such as alcohol and or obesity. It is a significant cause of morbidity and mortality, the presence of atrial fibrillation increases the risk of developing heart failure and thromboembolism, and it is a common cause of stroke. Treatment of atrial fibrillation is rate and/or rhythm control, anticoagulant medication to reduce the risk of thromboembolism and stroke, and lifestyle modifications. These interventions can reduce morbidity and mortality and improve quality of life but despite the therapeutic options in use, atrial fibrillation is still widespread and it is a significant public health problem. Current information on the public health burden of atrial fibrillation, pathogenesis of the arrhythmia, risk factors that cause atrial fibrillation or are thought to be associated with its development, the complications of atrial fibrillation (especially stroke), the classification of atrial fibrillation and how atrial fibrillation is diagnosed, (i.e., electrocardiographic changes, signs and symptoms), and current medical, pharmacological, and surgical treatments for atrial fibrillation are reviewed in this course.

Atrial Fibrillation: Statistics

Atrial fibrillation is the most common sustained arrhythmia.\(^1,2\) The true prevalence of atrial fibrillation is not known as 10%-40% of patients who have this arrhythmia are asymptomatic and not diagnosed,\(^1\) but prevalence estimates of 2%-3.4% are typical\(^3,4\) and the life time risk of developing atrial fibrillation has been estimated to be 1 in 4.\(^4,5\) The prevalence of atrial fibrillation increases with age;\(^5\) more men than women have atrial
fibrillation,\textsuperscript{6,7} it is more common in Caucasians than in African Americans,\textsuperscript{5} and it is a rare arrhythmia in children and teenagers.\textsuperscript{8,9} The prevalence of atrial fibrillation has been slowly increasing\textsuperscript{1,2} (caused in part by the aging of the population), and people who have atrial fibrillation are approximately twice as likely to be hospitalized as those who do not.\textsuperscript{10}

Clearly, atrial fibrillation is a significant public health problem. It is associated with a greatly increased risk of the comorbidities of heart failure and stroke. Given how common this arrhythmia is and the projected increase in prevalence, atrial fibrillation is a serious health concern.

**Pathogenesis Of Atrial Fibrillation**

The pathogenesis of atrial fibrillation is complex and not completely understood, but there is substantial evidence for two pathogenic mechanisms by which this arrhythmia is initiated and sustained: a triggering focus and atrial remodeling, respectively.\textsuperscript{5,11} Atrial fibrillation may also be caused in part by changes in the autonomic nervous system, abnormal myocardial metabolic processes, atrial dilation, fibrosis caused by cardiac disease, re-entry mechanisms (rotors), or a pro-thrombotic state.\textsuperscript{2,11,12} It is also possible that in any single patient there are multiple ways that atrial fibrillation begins and is sustained and these pathologic processes probably interact and influence each other.

**Triggering Focus**

The pulmonary veins have been confirmed as a common site of the triggering focus - or foci - that initiates atrial fibrillation\textsuperscript{11,13} but other areas of the myocardium have been identified that can initiate atrial fibrillation and there may be multiple sites that act as triggering foci.\textsuperscript{11}
Atrial Remodeling

Electrophysiologic studies and examination of the myocardium have shown that there are electrical and structural changes in the heart, aka remodeling, that sustain atrial fibrillation once a triggering focus has begun to initiate the arrhythmia.\(^1\)\(^{11,14}\) These changes in the electrical and structural part of the heart can include a decreased refractory period, altered functioning of ion channels, autonomic dysfunction, development of re-entry circuits, sinus node dysfunction, fibrosis of the myocardium, and other damage that allows abnormal atrial impulses to be both conducted and sustained.\(^1\)\(^{11,14-18}\) How these changes occur is not entirely clear, but it may be that the triggering focus/foci induce them in a vulnerable heart.

The triggering focus or foci in atrial fibrillation depolarize the atria between 300-600 times per minute. This extremely rapid rate of atrial depolarization has two important effects: 1) the rate of atrial fibrillation is much faster than the rhythmic depolarization rate of the sinoatrial (SA) node so ventricular depolarization and contraction is determined by the ectopic foci in the atria, and 2) transmission of every impulse through the atrioventricular (AV) node and the other parts of the cardiac conduction system is not possible because of the refractory period of these structures, and the number of atrial impulses that reaches the ventricles is variable and always changing.

The rapid atrial depolarization of atrial fibrillation that supersedes the SA node as the cardiac pacemaker and the inability of the AV to transmit to the ventricles the hundreds of atrial impulses that are firing each minute cause
the characteristic electrocardiographic changes of atrial fibrillation. Atrial fibrillation involves an absence of P waves and an irregularly, irregular ventricular rate. These cardiac abnormalities also cause loss of atrial systole and several other changes in cardiac performance that will be discussed later in this study.

**Risk Factors Of Atrial Fibrillation**

There are many factors that can or may increase the risk of developing atrial fibrillation. Some are proven risk factors while others are considered risks by association. A complete discussion of the established, emerging, and potential risk factors would be quite lengthy; readers are referred to Andrade, et al. (2014) for detailed information. Established risk factors will be covered here, and the emerging and potential risk factors will simply be listed.

There are also several medical conditions that are direct causes of atrial fibrillation. These are listed in Table 3 shown later in the course. It’s important to understand that many patients who have atrial fibrillation have specific cardiovascular diseases, chronic diseases, and/or life style issues that are risk factors for atrial fibrillation or are thought to contribute to its development. However, Huxley, et al. (2011) studied almost 15,000 adults and found that 43% of the cases of atrial fibrillation were not explained by common risk factors for the disease.

**Established Risk Factors**

*Age and Gender*

The risk of developing atrial fibrillation increases with age, and the prevalence of atrial fibrillation doubles with each decade starting at age 50.
Although age is undoubtedly a risk factor for atrial fibrillation the reasons for this (aside from age increasing the incidence of other risk factors) are not completely understood.\textsuperscript{20-23}

Men have a higher risk of symptomatic and asymptomatic fibrillation than do women.\textsuperscript{15,21,24,25} Women who have atrial fibrillation: 1) have a higher mortality rate and are more likely to have a stroke than men; 2) they are equally as likely as men to progress from paroxysmal atrial fibrillation to sustained atrial fibrillation; 3) the clinical picture of symptomatic atrial fibrillation is slightly different for women; and, 4) women have been reported to have longer and more symptomatic episodes of atrial fibrillation and more episodes of paroxysmal atrial fibrillation.\textsuperscript{24-27}

\textit{Hypertension}

A consistent association between hypertension and atrial fibrillation has been clearly established.\textsuperscript{15,21,28} The relative risk for someone who has hypertension of developing atrial fibrillation has been estimated to be 1.2\% to 1.5\%.\textsuperscript{15} Most cases of atrial fibrillation occur in people who have hypertension,\textsuperscript{29} and hypertension increases the risk of paroxysmal atrial fibrillation progressing to permanent atrial fibrillation.\textsuperscript{30} The mechanisms by which hypertension may cause or contribute to atrial fibrillation include atrial stretching, fibrosis, myocardial inflammation, renal dysfunction, changes in the renin-angiotensin system, and structural and electrical remodeling.\textsuperscript{31-33}
Valvular Heart Disease

Any valvular abnormality that causes regurgitation or stenosis can cause atrial fibrillation.\textsuperscript{21} Left heart valvular lesions, especially lesions caused by rheumatic heart disease and resulting in stenosis, are associated with the highest prevalence of atrial fibrillation\textsuperscript{15} and the worse the lesion, the higher the risk. In addition, atrial fibrillation associated with rheumatic heart disease may increase the risk of mortality and stroke.\textsuperscript{34}

Aortic stenosis increases the risk for developing atrial fibrillation.\textsuperscript{35,36} Patients who have mild to moderate aortic stenosis can have a 9.1% prevalence of atrial fibrillation. The prevalence increases to almost 27% with a severe stenosis,\textsuperscript{36} and aortic stenosis and atrial fibrillation together increase mortality risks and the risk for stroke.\textsuperscript{36}

Heart Failure and Cardiac Myopathy

Atrial fibrillation and heart failure often coexist. Studies have shown that one-third to almost two-thirds of patients with heart failure have atrial fibrillation,\textsuperscript{37,38} and Chugh, \textit{et al.} (2014) reported that patients who had heart failure had a six-fold increase in risk for atrial fibrillation.\textsuperscript{39} The close association between these two diseases is further underscored by cardiac and myocardial pathologies that are common to both, and many researchers have noted that heart failure may cause atrial fibrillation and, alternatively, atrial fibrillation may cause heart failure.\textsuperscript{37,40}
The prognosis for people who have atrial fibrillation and heart failure is very poor, with a significant increase in the risk of mortality. Depending on the time of diagnosis of atrial fibrillation, the prevalence of atrial fibrillation in patients who have hypertrophic cardiomyopathy has been reported to be 5%-28%.

**Alcohol Consumption**

Heavy alcohol use is a well-known and well-documented cause of atrial fibrillation; the risk of atrial fibrillation increases by up to 30% if more than two drinks a day are consumed; and, the risk is progressive, the more alcohol that is consumed the greater the risk. Acute alcohol intoxication or sporadic binge drinking can also cause atrial fibrillation, a phenomenon called holiday heart syndrome.

It is unclear how alcohol increases the risk for atrial fibrillation. Studies in animals and humans have identified issues such as conduction abnormalities, electrolyte abnormalities, and increased vagal tone as possible pathologic mechanisms. Current (2016) research suggests that the primary way by which alcohol causes atrial fibrillation is physical damage to the left atrium.

**Obesity**

The relationship between body weight and atrial fibrillation is complex and still being worked out. Obesity is a risk factor for atrial fibrillation but...
being underweight has been identified as a risk factor, as well.\textsuperscript{53} There is also research that has shown that in specific situations body weight can have a negative or positive effect vis-a-vis atrial fibrillation. Examples of this include: Phan, \textit{et al.} (2016) found that obese patients had a higher risk of developing atrial fibrillation in the post-operative period;\textsuperscript{53} and, Sandhu, \textit{et al.} (2016) found that obese patients who had atrial fibrillation and were treated with anticoagulants had a better prognosis than normal weight patients who received the same treatment.\textsuperscript{55}

Table 1: Emerging Risk Factors\textsuperscript{15}

<table>
<thead>
<tr>
<th>Prehypertension</th>
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<tbody>
<tr>
<td>Increased pulse pressure</td>
</tr>
<tr>
<td>Obstructive sleep apnea</td>
</tr>
<tr>
<td>Physical activity: Cumulative lifetime practice &gt;1500 h</td>
</tr>
<tr>
<td>Diastolic dysfunction</td>
</tr>
<tr>
<td>Familial and genetic</td>
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<tr>
<td>Hypertrophic cardiomyopathy</td>
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<tr>
<td>Congenital heart disease</td>
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Table 2: Potential Risk Factors\textsuperscript{15}

<table>
<thead>
<tr>
<th>Coronary artery disease</th>
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<tbody>
<tr>
<td>Chronic kidney disease</td>
</tr>
<tr>
<td>Inflammation</td>
</tr>
<tr>
<td>Pericardial fat</td>
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<tr>
<td>Tobacco use</td>
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Table 3: Medical Conditions As A Cause Of Atrial Fibrillation

<table>
<thead>
<tr>
<th>Hyperthyroidism</th>
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<tbody>
<tr>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>Pheochromcytoma</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
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<tr>
<td>Thyrotoxicosis</td>
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Classification Of Atrial Fibrillation

Atrial fibrillation is classified into five categories. These are described below as: 1) Paroxysmal atrial fibrillation, 2) Persistent atrial fibrillation, 3) Long-standing persistent atrial fibrillation, 4) Permanent atrial fibrillation, and; 5) Non-valvular atrial fibrillation.

**Paroxysmal Atrial Fibrillation**

Paroxysmal atrial fibrillation terminates spontaneously or with intervention within seven days of onset. Paroxysmal atrial fibrillation may recur and it frequently progresses to persistent and/or permanent atrial fibrillation.

**Persistent Atrial Fibrillation**

Persistent atrial fibrillation persists for longer than seven days.

**Longstanding, Persistent Atrial Fibrillation**

Longstanding, persistent atrial fibrillation persists for more than 12 months.
Permanent Atrial Fibrillation

The term permanent atrial fibrillation is used when the patient and the medical clinician have decided not to make further attempts to restore sinus rhythm.

Non-valvular Atrial Fibrillation

Non-valvular atrial fibrillation occurs in the absence of rheumatic mitral stenosis, a mechanical or bio-prosthetic heart valve, or mitral valve repair. Valvular atrial fibrillation occurs in patients found to have the arrhythmia and mitral stenosis or an artificial heart valve.

The term *lone atrial fibrillation* is still used in the literature to describe atrial fibrillation that occurs in young adults who do not have cardiac disease, diabetes mellitus, or hypertension. There is no standard, accepted definition of lone atrial fibrillation and use of the term is discouraged by the current (2014) guidelines from the American Heart Association (AHA) and the American College of Cardiology (ACC) and other authorities.56,57

Diagnosis Of Atrial Fibrillation

Risk factors can help clinicians consider a diagnosis of atrial fibrillation in certain patients, and there are signs and symptoms that can indicate its presence. But atrial fibrillation can only be diagnosed by examining a 12-lead electrocardiogram (ECG).

Electrocardiogram

The two identifying characteristics of atrial fibrillation as seen on a 12-lead ECG are: 1) Absence of P waves; and, 2) An irregularly, irregular ventricular rate. The QRS complex is usually narrow. Small fibrillatory waves called F
waves may be seen but their absence or presence is not needed for a
diagnosis of atrial fibrillation. The ventricular rate can be quite variable and
it is usually rapid, between 110-140 beats per minute but it may be much
faster or abnormally slow. Because atrial fibrillation may not be present
when the patient is examined or when a 12-lead ECG is done, continuous,
telemetric ECG monitoring may be needed to detect the arrhythmia.

Patient Physical Examination and History

The clinical presentation of atrial fibrillation can be completely benign or the
patient may have mild and nonspecific symptoms such as dizziness, fatigue,
or palpitations. Patients may also have significant signs and symptoms such
as chest pain, dyspnea, heart failure, hypotension, or syncope. Some cases
of atrial fibrillation may be detected only after a life-threatening event like
cardiogenic shock or a stroke.

Less serious presentations may be seen only during times of stress. This
phenomenon is due to loss of atrial systole, decreased coronary blood flow,
impaired ventricular filling, and irregular ventricular response.
Loss of Atrial Systole

In atrial fibrillation, the atria do not contract in response to an SA node impulse. Instead, the very rapid atrial ectopic impulses cause the atria to “quiver” and effectively eliminate atrial systole. Approximately 20% - 30% of stroke volume comes from the volume of blood delivered by atrial systole, and patients may tolerate this loss of stroke volume until the heart rate increases or there is a demand for a higher than normal cardiac output. When these situations occur so can the signs and symptoms of loss of cardiac output such as dyspnea and dizziness.

Decreased Coronary Blood Flow

Atrial fibrillation is also associated with decreased coronary blood flow. If the heart rate increases or there is a demand for a higher than normal myocardial perfusion, the myocardium may be deprived of blood flow and oxygen.

Impaired Ventricular Filling

Atrial fibrillation is associated with cardiomyopathy, hypertension, and mitral stenosis and these can all impair ventricular filling. This effect combined with loss of atrial systole can further decrease stroke volume.

Irregular Ventricular Response

The ventricular response to the atrial impulses is irregular and the filling time and stroke volume vary, as well. When interviewing someone who may have atrial fibrillation the clinician should ask about symptoms of chest pain, dizziness, dyspnea, dyspnea on exertion, fatigue, palpitations, or syncope. If any of these symptoms have occurred the clinician should also find out when they occurred and what the patient was doing when they occurred. During
the physical examination, attention should be paid to the patient’s breath sounds, heart sounds, and peripheral circulation.

Detecting atrial fibrillation can be difficult as episodes of atrial fibrillation can be brief. They may last for only a few seconds and the patient may be asymptomatic, especially if the patient has paroxysmal atrial fibrillation. The detection and diagnosis of atrial fibrillation, however, is very important to prevent stroke and as atrial fibrillation can easily go unrecognized this is a significant challenge.

Atrial Fibrillation And Stroke

Stroke is the most common and most serious complication of atrial fibrillation. A stroke caused by atrial fibrillation is almost always an ischemic stroke caused by a thromboembolism. The embolism is typically part of a mural thrombus that has formed in the atria, caused by inefficient and/or absence of atrial systole.

Atrial fibrillation is also responsible for approximately 20% of all cryptogenic (obscure or of unknown origin) strokes. A 2014 study by Kishore, et al. found that 11% of all the patients they studied who suffered a stroke had atrial fibrillation that had not been detected prior to the stroke.

The risk for stroke is increased five-fold by atrial fibrillation. Atrial fibrillation accounts for ≥15% of all strokes in the United States and for 36% of all strokes in people >age 80, and these numbers are probably underestimates. The risk for stroke from atrial fibrillation increases from 1.5% in patients aged 50-59 to 23.5% in patients aged 80-89, and atrial fibrillation is a significant cause of silent cerebral infarction and transient
ischemic attack (TIA). Additionally, there is a high rate of recurrence of stroke caused by atrial fibrillation.

Even brief episodes of atrial fibrillation are considered to increase the risk for stroke, and patients who have a stroke caused by atrial fibrillation are more likely to have significant physical impairment when compared to stroke from other causes.

A stroke caused by atrial fibrillation is associated with increased hospitalization costs, increased subsequent risk of hospitalization, and a higher risk for hospital readmission, and ischemic stroke caused by atrial fibrillation has been estimated to triple the mortality rate. Furthermore, this risk appears to persist well beyond the immediate post-stroke period.

Treatment Of New Onset Atrial Fibrillation

Treatment of new onset atrial fibrillation should focus on the issues highlighted here.

• Note the signs and symptoms of atrial fibrillation. Is the patient asymptomatic, symptomatic but clinically stable, or having evidence of cardiac, neurological, or pulmonary distress?
• Is there an identifiable and potentially correctable cause of the atrial fibrillation such as electrolyte imbalance, hyperthyroidism, intoxication, myocardial ischemia, or pulmonary embolism?
• Does the patient need emergency cardioversion and if so, how should this be done?
• Should rate control, rhythm control, or both be initiated?
• Prevention of systemic embolization.

**Signs and Symptoms**

Many patients who have undiagnosed atrial fibrillation will have signs and symptoms that are caused by decreased cardiac output and/or a rapid ventricular rate, *i.e.*, chest pain, dyspnea on exertion, fatigue, or palpitations. These often occur intermittently, the patient may be asymptomatic at the time of the examination, and not all patients who have atrial fibrillation have symptoms.\(^7\)

Serious cardiac, neurological, and/or pulmonary signs or symptoms caused by new onset atrial fibrillation are unusual.

**Identifiable and Correctable Causes**

Identifiable and correctable causes of atrial fibrillation represent a small percentage of all cases of new onset atrial fibrillation. The appropriate referrals should be made and treatments done in these cases.

**Emergency or Immediate Cardioversion**

Emergency or immediate cardioversion to restore sinus rhythm in a patient who has new onset atrial fibrillation and a rapid ventricular rate should be performed, or at least considered, if the patient has any of the following:\(^5\)\(^6\)\(^7\)

• Active myocardial ischemia
• Evidence of organ hypo-perfusion
• Hypotension
• Severe heart failure
• First episode of atrial fibrillation with duration of < 48 hours
• Wolf-Parkinson-White syndrome, atrial fibrillation, and hemodynamic compromise

Cardioversion to restore sinus rhythm can be done electrically or pharmacologically and it can be done as an outpatient procedure or when the patient is hospitalized. Both electrical cardioversion and pharmacological cardioversion have been proven to be effective; each has its own risk-benefit ratio, but there are no controlled trials that have directly compared the two.\textsuperscript{72} If the patient is young, has persistent symptoms that interfere with health or quality of life, or attempts at rate control have failed, rhythm control would be the best approach.\textsuperscript{71,73}

Direct current cardioversion involves placing electrodes on the patient’s chest and delivering a synchronized electrical shock at a specific time in the cardiac cycle. The risks of direct current cardioversion include brady-arrhythmias, burns, complications of sedation used during the procedure, thromboembolism, reprogramming/altering the functions of an implanted cardiac device, sinus arrest, and ventricular fibrillation or tachycardia.\textsuperscript{71,73} Sinus arrest may happen if the patient has sinus node dysfunction, and ventricular fibrillation or tachycardia are rare complications.\textsuperscript{73} Elective
cardioversion should not be done if the patient has digoxin toxicity, hypokalemia, or hypomagnesemia.\textsuperscript{71-74} The basics of the procedure are highlighted below:

- Serum electrolytes and oxygen saturation should be measured before the procedure is done.
- During the procedure, the patient should be sedated and blood pressure, heart rate, CO\textsubscript{2} capnography, oxygen saturation, and cardiac rhythm should be continuously monitored.
- Depending on the circumstances some patients may be pretreated with an antiarrhythmic drug, \textit{i.e.}, amiodarone, propafenone, flecainide ibutilide, or sotalol.
- An electrical current of 100-200 joules is delivered.
- It is reasonable to make several attempts at electrical cardioversion if the initial attempt is unsuccessful.

Given the high risk for embolic stroke associated with atrial fibrillation and also with cardioversion for atrial fibrillation,\textsuperscript{75} anticoagulant therapy is an important consideration when performing direct current cardioversion. The risk of a stroke is the highest in the first 72 hours after cardioversion and most strokes will happen within 10 days of the procedure.\textsuperscript{71,73} The American College of Cardiology/American Heart Association 2014 guidelines for the management of patients who have atrial fibrillation have the following recommendations for anticoagulation and cardioversion.\textsuperscript{71}

- Appropriate anticoagulation management around the time of a cardioversion is essential for reducing thromboembolic risk.
- In patients with atrial fibrillation clearly of <48 hours duration, it is common practice to perform a cardioversion without transesophageal echocardiogram (TEE) or antecedent anticoagulation.
• For patients with atrial fibrillation requiring emergency cardioversion because of hemodynamic instability, the initiation of anticoagulation should not delay interventions to stabilize the patient. In these cases, it is reasonable to administer heparin (an intravenous bolus of unfractionated heparin [UFH] followed by infusion, or low molecular weight heparin [LMWH]), or a newer anticoagulant and to continue coagulation therapy after cardioversion, unless contraindicated.

• For patients with atrial fibrillation of less than 48-hours duration and with high risk of stroke, intravenous heparin, LMWH, or administration of a factor Xa or direct thrombin inhibitor is recommended as soon as possible before or immediately after cardioversion, followed by long-term anticoagulation therapy.

• Decisions regarding whether to initiate long-term systemic anticoagulation at the time of cardioversion in a patient with atrial fibrillation of <48 hours duration should be based on the patient's long-term risk of stroke using the CHA\textsubscript{2}DS\textsubscript{2}-VASc risk score.

• For patients with atrial fibrillation of 48-hours duration or longer or of unknown duration who have not been anticoagulated for the preceding 3 weeks, it is reasonable to perform a transesophageal echocardiogram (TEE) prior to cardioversion and proceed with cardioversion if no left atrial thrombus is identified, including in the left atrial appendage, if anticoagulation is achieved before TEE and maintained after cardioversion for at least 4 weeks.

• For patients with atrial fibrillation of \( \geq 48 \) hours or uncertain duration, oral anticoagulation is recommended for \( \geq 4 \) weeks after emergency cardioversion (as with patients undergoing elective cardioversion). If warfarin is used, bridging with UFH or LMWH is indicated until the INR is therapeutic.
Pharmacological cardioversion can also be tried on an immediate basis using intravenous amiodarone, flecainide, or ibutilide, but this approach is less effective than electrical cardioversion.

**Treatment Of Persistent Or Permanent Atrial Fibrillation**

The treatment issues for patients with persistent or permanent atrial fibrillation are control or management of comorbidities and risk factors, rate and rhythm control, and prevention of embolization with anticoagulant drugs. General treatment of persistent or permanent atrial fibrillation is covered here; a discussion of control and management of comorbidities and risk factors is beyond the scope of this study.

**Rate Control or Rhythm Control**

Rate control or rhythm control (or the two together) can be used to treat atrial fibrillation. Comparisons between the two have shown that rhythm control is only moderately effective for abolishing atrial fibrillation, it does not have as strong an effect on mortality, and it is associated with a greater number of adverse effects. Neither rate control or rhythm control reverse the structural remodeling that helps to sustain atrial fibrillation.

**Pharmacologic Rhythm Control**

Amiodarone, disopyramide, dofetilide, dronedarone, ibutilide, flecainide, propafenone, and quinidine can be used for rhythm control and, hopefully, cardioversion of atrial fibrillation. Drug choice is guided by the patient’s clinical conditions and comorbidities and by safety concerns and contraindications. The administration, doses, and the risks and benefits of each drug used for rhythm control are not included here; however, the online 2014 Guideline for the Management of Patients with Atrial Fibrillation...
provides this information at
(http://circ.ahajournals.org/content/early/2014/03/27/CIR.0000000000000041.full.pdf).

**Rate Control**

Rate control improves quality of life, decreases the potential for developing tachycardia-induced cardiomyopathy, and reduces morbidity.\(^\text{71}\) There are four clinical situations that indicate the need for rate control.\(^\text{76}\)

- As a well-controlled heart rate is important for patient health, rate control should be the first treatment for patients who have new onset atrial fibrillation.
- Rate control is a sensible first-choice treatment for patients who do not need sinus rhythm, *i.e.*, patients >80 years of age and who have no symptoms or only minor symptoms.
- Rate control is the only option when rhythm control by cardioversion, medications, or ablation has failed.
- Rate control is the treatment of choice for patients for whom the risk of restoring sinus rhythm is greater than the benefits, *i.e.*, patients who have sick sinus syndrome or paroxysmal atrial fibrillation with a high ventricular response rate.

Pharmacological rate control can be done by using beta-blockers, nondihydropyridine calcium channel blockers (diltiazem or verapamil), digoxin, or amiodarone.\(^\text{71,76}\) Beta-blockers have been considered the first-line drug of choice for rate control in atrial fibrillation, followed by a nondihydropyridine calcium channel blockers (diltiazem or verapamil) digoxin, and amiodarone. Evidence comparing the effectiveness of the rate control drugs is lacking so the clinical situation and the patient’s comorbidities will determine which drug should be used.\(^\text{71,76,82}\)
A complete discussion of the administration, doses, and the risks and benefits of each drug used for rate control is beyond the scope of this study module. Readers are referred to the 2014 *Guideline for the Management of Patients with Atrial Fibrillation* for more information. This article is available online at the following web link:
http://circ.ahajournals.org/content/early/2014/03/27/CIR.0000000000000041.full.pdf.

The American College of Cardiology/American Heart Association Task Force has outlined some general principles of pharmacologic rate control.\(^\text{71}\)

- Heart rate control (resting heart rate <80 bpm) is reasonable for symptomatic management of atrial fibrillation.
- Control of the ventricular rate using a beta-blocker or nondihydropyridine calcium channel antagonist is recommended for patients with paroxysmal, persistent, or permanent atrial fibrillation.
- Intravenous administration of a beta-blocker or nondihydropyridine calcium channel blocker is recommended to slow the ventricular heart rate in the acute setting in patients without pre-excitation. Hemodynamically unstable patients should be electrically cardioverted.
- If the patient has atrial fibrillation-related symptoms during activity, rate control should be assessed during exertion and necessary adjustments made.
- Intravenous amiodarone can be useful for rate control in critically ill patients who do not have pre-excitation. Oral amiodarone may be useful for ventricular rate control when other measures are unsuccessful or contraindicated.
- AV nodal ablation with permanent ventricular pacing is reasonable to control the heart rate when pharmacological therapy is inadequate and rhythm control cannot be achieved.
• A lenient rate-control strategy (resting heart rate <110 bpm) may be reasonable if the patient remains asymptomatic and left ventricular systolic function is preserved.

• AV nodal ablation with permanent ventricular pacing should not be performed to improve rate control without prior attempts to achieve rate control with medications.

• Nondihydropyridine calcium channel antagonists should not be used in patients with decompensated heart failure; this may cause further hemodynamic compromise.

• In patients with pre-excitation and atrial fibrillation, digoxin, nondihydropyridine calcium channel antagonists, or intravenous amiodarone should not be administered as they may increase the ventricular response and may result in ventricular fibrillation.

• Dronedarone should not be used to control the ventricular rate in patients who have permanent atrial fibrillation; it increases the risk of the combined endpoints of stroke, MI, systemic embolism, or cardiovascular death.

The goals of rate control are to: 1) prevent bradycardia; 2) reduce the risk of tachycardia-induced cardiomyopathy and heart failure; and, 3) attain a heart rate that produces a cardiac output that meets the physiological demands of the patient. The last point has been somewhat controversial and requires consideration of the patient’s needs; should a heart rate of <80 bpm (the so-called strict approach) or a heart rate of <110 bpm (the so-called lenient approach) be the goal? The only large randomized trial that addressed this question found that lenient rate control was safe and effective and should be considered if the patient has few symptoms. But the ACC/AHA 2014 guidelines and recent (2016) expert commentary (which included the lead author of the aforementioned study) have both
suggested that the optimal heart rate for atrial fibrillation is not known and further study is needed. Using the strict approach or the lenient approach must be decided on a case-by-case basis.

Catheter ablation or pacemaker implantation can be used for rate control and will be discussed in the following section.

**Catheter Ablation**

The goal of catheter ablation is to destroy the focus or foci that initiate atrial fibrillation. Catheter ablation is typically done using cryoablation or radiofrequency ablation, and the ablation procedure can target the pulmonary veins, the AV node, or other structures.\(^{79,84,85}\) Indications for catheter ablation as a treatment for atrial fibrillation include, but are not limited to: 1) patients who have symptomatic paroxysmal atrial fibrillation that is refractory to at least one class I or III antiarrhythmic drug, or who are intolerant of these drugs; and, 2) patients who have symptomatic persistent atrial fibrillation that is refractory to at least one class I or class III antiarrhythmic drug, or who are intolerant of these drugs.

Key points to know about catheter ablation for atrial fibrillation are:

- The success for catheter ablation of paroxysmal atrial fibrillation is higher than for persistent atrial fibrillation.\(^{85,86}\)
- Catheter ablation is considered superior to antiarrhythmic drugs for treating atrial fibrillation and preventing recurrences.\(^{85,87}\)
- No single catheter ablation approach is effective for all patients. This point is underscored by differing opinions about the effectiveness of pulmonary vein ablation alone versus pulmonary vein ablation plus
ablation of other sites; some authors have concluded pulmonary vein ablation by itself is sufficient while others have not.\textsuperscript{84-87}

- The success rate for catheter ablation has been reported to be 75\%-93\% for paroxysmal atrial fibrillation and 63\%-74\% for non-paroxysmal atrial fibrillation.\textsuperscript{85} Many patients will require a repeat ablation, but early recurrences can be treated with medications or cardioversion.\textsuperscript{74} It is not clear if catheter ablation reduces the risk of stroke. There is some evidence that it reduces the risk of mortality.\textsuperscript{88}

- Cryoablation has some procedural advantages that radiofrequency ablation does not. The outcomes of each approach for paroxysmal atrial fibrillation and persistent atrial fibrillation are comparable.\textsuperscript{89-91}

- Anticoagulation should be started one month before the procedure and continued for three months after.\textsuperscript{85} If the patient has persistent atrial fibrillation, TEE should be done prior to ablation to rule out presence of a left atrial or left atrial appendage clot.\textsuperscript{85}

- Complications/side effects include (but are not limited to) adverse effects from sedation used during the procedure, arterio-venous fistula or pseudo-aneurysm at the access site, cardiac tamponade, fluid overload, hematoma at the access site, phrenic nerve palsy (cryoablation), stroke, and transient ischemic attack.\textsuperscript{85,86,88}

**AV Node Ablation**

AV node ablation and pacemaker insertion is used if: 1) rate and rhythm control medications strategies have failed or medications cannot be tolerated; 2) ablation did not work; 3) the patient has become symptomatic, \textit{i.e.}, have heart failure; and, 4) the patient develops a long-term rapid ventricular rate, a risk for cardiomyopathy.\textsuperscript{71,76,79,92} AV node ablation is also used for patients who have atrial fibrillation and require biventricular pacing for management of systolic heart failure.\textsuperscript{92}
The success rate of AV node ablation in abolishing atrial fibrillation has been reported to be up to 95%,\textsuperscript{79,92} the complication rate is low, and it can improve quality of life and long-term survival.\textsuperscript{79} However, the procedure is not reversible so AV node ablation with pacemaker implantation “… should be restricted until, and only if, absolutely necessary.”\textsuperscript{76}

**Cox Maze Procedure**

The Cox maze is a surgical procedure that can be used to abolish atrial fibrillation. The Cox maze procedure was first performed in the 1980s and it has gone through several iterations since it was developed. The original procedure involved cutting and sewing to create lesions (the maze) in the atria that would block ectopic electrical impulses from reaching the ventricles. The Cox maze procedure had a high rate of success at achieving rate control but it is a very complex and technically demanding operation to perform. The iteration that is currently used, the Cox maze IV, has replaced the cut and sew technique with radiofrequency ablation and cryoablation,\textsuperscript{93,94} and the success rate (absence of atrial fibrillation) at 12-24 months post-procedure has been reported to be 90%.\textsuperscript{94}

The Cox maze IV can also be combined with radiofrequency ablation that is done six to eight weeks after the surgery.\textsuperscript{93} Indications for surgical ablation (including Cox maze IV) are: 1) patients who have symptomatic atrial fibrillation and who have not responded to medical therapy and/or have had
one more unsuccessful catheter ablation, or patients who prefer surgery; and, 2) patients who are having cardiac surgery and have symptomatic atrial fibrillation, regardless of whether or not antiarrhythmic medications have been started.  

**Cardioversion**

Patients who have persistent or permanent atrial fibrillation can be cardioverted, but if the cardioversion is elective they should be treated with warfarin for at least three weeks before and four weeks after the procedure to avoid left atrial thrombus. The 2014 guidelines notes that “... it is reasonable to perform a TEE prior to cardioversion and proceed with cardioversion if no LA (left atrial) thrombus is identified, including in the LAA (left atrial appendage), provided that anticoagulation is achieved before TEE and maintained after cardioversion for at least 4 weeks.” If the cardioversion must be done immediately, anticoagulation should be started as soon as possible and continued for at least four weeks after the procedure. Continuing anticoagulation past four weeks post-cardioversion may be needed, depending on the patient’s risk for thromboembolism. (Risk assessment will be discussed later in the study).

**Prevention Of Embolization**

Prevention of embolization in patients who have atrial fibrillation is critically important. The primary preventive technique is anticoagulation using oral medications, and oral anticoagulation has been shown to be very effective.
at preventing thromboembolic events in patients who have atrial fibrillation. There are three decisions that must be made before initiating oral anticoagulation therapy: 1) assessment of stroke risk; 2) assessment of bleeding risk; and, 3) choosing an anticoagulant.

**Assessment of Stroke Risk**

Assessment of stroke risk was traditionally done using the CHADS$_2$ algorithm, but the currently accepted tool is the CHA$_2$DS$_2$-VASc algorithm. In most cases of atrial fibrillation, a CHA$_2$DS$_2$-Vasc score of $\geq 2$ indicates the need for anticoagulation.

<table>
<thead>
<tr>
<th>CHADS$_2$</th>
<th>CHA$_2$DS$_2$-VASc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure (1 point)</td>
<td>Congestive heart failure (1 point)</td>
</tr>
<tr>
<td>Hypertension (1 point)</td>
<td>Hypertension (1 point)</td>
</tr>
<tr>
<td>Age $\geq$75 years (1 point)</td>
<td>Age $\geq$75 (2 points)</td>
</tr>
<tr>
<td>Diabetes (1 point)</td>
<td>Diabetes (1 point)</td>
</tr>
<tr>
<td>Prior stroke, TIA or thromboembolism (2 points)</td>
<td>Stroke or transient ischemic attack (2 points)</td>
</tr>
</tbody>
</table>

Vascular disease (1 point)

Age 65-74 (1 point)

Sex category (1 point for female)
Assessment of Bleeding Risk

Oral anticoagulants increase the risk of bleeding, so assessment of a patient’s risk for bleeding must be done before starting anticoagulation therapy. Assessment of bleeding risk is not as straightforward as assessment of risk for stroke. Multiple assessments tools have been developed such as ATRIA, HAS-BLED, HEMORRHAGES, ORBIT, and REITE, and each of these has its advocates and critics.

Table 6: Assessment Tools For Assessing Bleeding Risk

<table>
<thead>
<tr>
<th>Assessment Tool</th>
<th>Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atria</td>
<td>(Anticoagulation and Risk factors in Atrial Fibrillation)</td>
</tr>
<tr>
<td>HAS-BLED</td>
<td>(Hypertension, Abnormal renal/liver function, Stroke, Bleeding history or predisposition, Labile INR, Elderly, Drugs/alcohol concomitantly)</td>
</tr>
<tr>
<td>HEMORRHAGES</td>
<td>(Hepatic or renal disease, Ethanol abuse, Malignancy, Older age, Reduced Platelet Count Reduced platelet count or function, Re-bleeding, Hypertension, Anemia, Genetic Factors, Excessive Fall Risk and Stroke)</td>
</tr>
<tr>
<td>ORBIT</td>
<td>(Older than 74, Reduced hemoglobin, abnormal hemoglobin, or anemia, Bleeding history, Insufficient kidney function, Treatment with any antiplatelet drug</td>
</tr>
<tr>
<td>RIEITE</td>
<td>Computerized Registry of Patients with Venous Thromboembolism</td>
</tr>
</tbody>
</table>

The question of course, is which one is the best and should be used. The ACC/AHA 2014 guidelines note that “… these scores may be helpful in defining patients at elevated bleeding risk, their clinical utility is insufficient
for use as evidence for the recommendations in this guideline.” Steinberg (2016) writes that: 1) There have been no randomized clinical trials that have shown that withholding anticoagulation because of a high risk for bleeding is beneficial; and, 2) Large observational studies suggest that there is a preventative benefit to oral anticoagulation, even if there is a high risk for bleeding as per one of these assessment tools. Steinberg also points out that these assessment tools “... help remind clinicians of bleed risk factors, and particularly to highlight management of reversible risk factors for bleeding.”

**Choosing an Anticoagulant**

Anticoagulants have been proven to lower the risk for thromboembolic stroke in patients who have atrial fibrillation. The anticoagulants that are used for this purpose are warfarin, and the new oral anticoagulants apixaban, dabigatran, edoxaban, and rivaroxaban. Warfarin inhibits vitamin K-dependent synthesis of the clotting factors II, VII, IX, and X. Apixaban, deoxidant, and rivaroxaban inhibit the clotting factor Xa; dabigatran is a direct thrombin inhibitor.

Choosing between warfarin and a new anticoagulant - and if the latter is chosen, which of the four to use - is challenging. The decision should be made on a case-by-case basis but unless a newer anticoagulant is contraindicated, these drugs are the preferred choice. However, if a patient is well managed on warfarin there is no reason to stop using it.

Points to consider when choosing an anticoagulant for a patient who has atrial fibrillation are highlighted below.
• The new anticoagulants are as effective as warfarin (and in some cases, more so), and they have a smaller risk of intracranial bleeding than warfarin.\textsuperscript{96,97} Patients who have valvular atrial fibrillation should be treated with warfarin.\textsuperscript{71}

• The new anticoagulants have fewer drug-drug interactions than warfarin and fewer dietary effects, as well, but a higher risk for gastrointestinal bleeding than warfarin.\textsuperscript{71} The onset of effect is faster and more predictable than it is for warfarin.\textsuperscript{71}

• There is no laboratory test that can be used to evaluate the effectiveness of the new oral anticoagulants; there is no reversal agent for bleeding caused by apixaban, edoxaban, or rivaroxaban, and these drugs are more expensive than warfarin.\textsuperscript{71,96} In addition, these drugs have short half-lives so missing even one dose can put the patient at risk.\textsuperscript{96}

• There are no head-to-head clinical trials that compared any new anticoagulant to another. Choosing which one to use must be done on a case-by-case basis by considering the patient’s clinical characteristics and the risks and benefits of each drug.

• The new anticoagulants should not be used for patients who have valvular atrial fibrillation or significant liver disease.\textsuperscript{96} These drugs are excreted by the kidneys so they must be used cautiously in patients who have renal impairment, or not at all.\textsuperscript{96}

General concerns that are common to all the new anticoagulants include:\textsuperscript{98}

• There are no laboratory tests that are used to monitor the effectiveness of these drugs and routine measurements of coagulation parameters are \textit{not} recommended. If the patient is having a stroke, or needs surgery on an urgent basis it might be
useful to measure specific coagulation tests such as direct thrombin inhibitor or anti-factor Xa levels.

- Idarucizumab is an FDA approved reversal agent for bleeding caused by dabigatran. There are no reversal agents for the other new anticoagulants.
- One month after drug therapy has been initiated the patient should be assessed for adherence to the medication regimen and for adverse effects. Periodic assessments should be made to check for adverse effects and compliance.
- Apixaban can be taken with or without food. Dabigatran and rivaroxaban should be taken with food.
- Non-steroidal anti-inflammatories should be avoided. Aspirin can be used in some patients.

**Antiplatelet Therapy**

Antiplatelet therapy with aspirin or aspirin and clopidogrel has limited use as a preventive therapy in patients with atrial fibrillation. Aspirin alone or aspirin and clopidogrel have been shown to be inferior to warfarin for preventing thromboembolic events and stroke and the risk for bleeding is higher.

**Prevention of Atrial Fibrillation**

Atrial fibrillation is considered in part a heritable disease and having a family member who has atrial fibrillation has been associated with an increased risk for developing atrial fibrillation and some of its complications. Genetic variation mutations that increase the risk for developing atrial fibrillation and that may influence individual response to treatment have been identified. Widespread practical use of this
information is not yet possible and genetic screening of the population is not recommended at this time.\textsuperscript{71,105}

The angiotensin converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), aldosterone inhibitors (\textit{i.e.}, spironolactone), beta-blockers, corticosteroids, and the statin drugs have all been investigated - and some have been used - as therapies for the prevention of atrial fibrillation.\textsuperscript{106-108} These medications hopefully provide upstream protection against pathologic mechanisms such as atrial remodeling, inflammation, and oxidative stress that contribute to the development of atrial fibrillation. The evidence for the effectiveness of this approach is mixed and conflicting and more research is needed.

The ACC/AHA guidelines comments on upstream therapies are shown below.\textsuperscript{71}

- An ACE inhibitor or an ARB is reasonable for primary prevention of new-onset atrial fibrillation in patients with heart failure and reduced left ventricular ejection fraction.
- Therapy with an ACE inhibitor or an ARB may be considered for primary prevention of new-onset atrial fibrillation in the setting of hypertension.
- Statin therapy may be reasonable for primary prevention of new-onset atrial fibrillation after coronary artery surgery.
- Therapy with an ACE inhibitor, ARB, or statin is not beneficial for primary prevention of atrial fibrillation in patients without cardiovascular disease.
Summary

Atrial fibrillation is the most common arrhythmia. It affects millions of people, the incidence increases with each decade after age 50, and it is a major cause of morbidity and mortality, especially embolic stroke. The pathogenesis of atrial fibrillation is thought to involve an initial triggering focus and atrial remodeling that sustains the arrhythmia. The triggering focus or foci in atrial fibrillation depolarize the atria between 300-600 times a minute, and this extremely rapid rate of atrial depolarization has several important effects.

Firstly, ventricular depolarization and contraction is determined by the ectopic foci in the atria resulting in rapid and irregular ventricular contraction. Secondly, transmission of these impulses through the AV node and the other parts of the cardiac conduction system is not possible because of the refractory period of these structures, and the number of atrial impulse that reaches the ventricles is variable and always changing. Finally, loss of atrial systole decreases stroke volume. The result of these three changes in impulse formation and conduction is a rapid, irregular heart rate and decreased cardiac output.

The five categories of atrial fibrillation were reviewed as paroxysmal atrial fibrillation, persistent atrial fibrillation, longstanding, persistent atrial fibrillation, permanent atrial fibrillation, and non-valvular atrial fibrillation. Hypertension, alcohol consumption, obesity, valvular heart disease, heart failure, increasing age and male gender significantly increase the risk for developing atrial fibrillation. Many other risk factors have been associated with atrial fibrillation such as coronary artery disease and smoking, and atrial fibrillation is a heritable disease, as well.
Most patients who have atrial fibrillation will have nonspecific signs and symptoms such as dyspnea on exertion, fatigue, or palpitations and these usually occur during exercise or times of stress. However, some patients will be asymptomatic and a small number will present with hemodynamic instability, myocardial ischemia, or a stroke. Often a stroke will be the first sign of atrial fibrillation. A clinical examination and checking for precipitating causes are important, but a 12-lead ECG is needed to make the diagnosis of atrial fibrillation; characterized by the absence of P waves and an irregularly, irregular R-to-R interval. The heart rate is usually rapid, up to 140 beats a minute or faster, but it may be abnormally slow, as well.

The treatment of atrial fibrillation will depend on patient characteristics, the type of atrial fibrillation, the duration of the arrhythmia, and the presenting signs and symptoms. For every patient with atrial fibrillation, consideration should be given to the need for immediate cardioversion, rate control, rhythm control, and prevention of embolization. Cardioversion can be done emergently or electively, and it can be done using direct current electrical cardioversion, cardioversion using rhythm control drugs, or a combination of the two. Rate control can be accomplished using beta-blockers, digoxin, diltiazem or verapamil, or amiodarone. Rhythm control can be accomplished by using amiodarone, disopyramide, dofetilide, dronedarone, flecainide, ibutilide, propafenone, or quinidine.

Surgical procedures for treatment of atrial fibrillation include AV node ablation, catheter ablation, and the Cox maze procedure. Pacemakers have a limited role for the treatment of atrial fibrillation. Anticoagulation to prevent embolization is accomplished by using warfarin, apixaban, edoxaban, dabigatran, or rivaroxaban. Anticoagulation can significantly reduce the risk of stroke, but the anticoagulants can cause bleeding. The newer
anticoagulants, apixaban, dabigatran, edoxaban or rivaroxaban are at least as effective at preventing stroke as warfarin, they are easier to use, and for most patients they are the preferred anticoagulant. Upstream therapies such as ACE inhibitors, ARBs, aldosterone inhibitors, and the statin drugs may be effective for the prevention of new onset atrial fibrillation in certain patient populations.

Health clinicians must continuously educate themselves on evolving professional guidelines that address the diagnosis and treatment of atrial fibrillation. Importantly, medical and nursing clinicians have a key role in guiding patients to understand risk factors associated with atrial fibrillation and corresponding complications, such as stroke. Prevention strategies have been specifically highlighted here to guide clinicians to help patients learn lifestyle choices that increase their risk for atrial fibrillation, such as use of alcohol and drugs, and obesity.

Please take time to help NurseCe4Less.com course planners evaluate the nursing knowledge needs met by completing the self-assessment of Knowledge Questions after reading the article, and providing feedback in the online course evaluation.

Completing the study questions is optional and is NOT a course requirement.
1. The incidence of atrial fibrillation:
   a. increases with age
   b. decrease with age
   c. is not affected by age
   d. increases with age but only for women

2. Which of the following are considered pathogenic mechanisms of atrial fibrillation?
   a. Infarcts in the atria and scarring of the SA node
   b. Abnormal conduction pathways and ventricular hypertrophy
   c. An initiating focus and atrial remodeling
   d. Decreased myocardial oxygen delivery and abnormal AV pathways

3. Which of the following are established risk factors for atrial fibrillation?
   a. African American ethnicity, chronic kidney disease
   b. Hypertension, heart failure
   c. Coronary artery disease, COPD
   d. Cirrhosis of the liver, stroke

4. The diagnosis of atrial fibrillation can only be made by:
   a. signs and symptoms
   b. identification of risk factors
   c. blood tests
   d. a 12-lead ECG

5. The heart rate of someone who has atrial fibrillation is typically:
   a. very rapid and irregularly irregular
   b. very slow and irregular
   c. very rapid and regular
   d. very rapid and regular
6. Cardioversion is used if the patient with atrial fibrillation:
   a. is > age 80
   b. has a history of COPD
   c. is hemodynamically compromised
   d. is < age 40

7. Rhythm control in patients who have atrial fibrillation can be attained by using:
   a. beta blockers, diltiazem, or digoxin
   b. electrical or pharmacological cardioversion
   c. supportive care and life style changes
   d. the Cox maze procedure

8. Rate control in patients who have atrial fibrillation is typically attained by using:
   a. AV node ablation
   b. sotalol or amiodarone
   c. a pacemaker
   d. beta blockers or certain calcium channel blockers

9. The most common and most serious complication of atrial fibrillation is:
   a. hypertension
   b. chronic kidney disease
   c. stroke
   d. myocardial infarction

10. Prevention of thromboembolism in atrial fibrillation is achieved by:
    a. aspirin and warfarin
    b. warfarin, apixaban, dabigatran, edoxaban, or rivaroxaban
    c. ACE inhibitors and statins
    d. emergency or elective cardioversion
11. The risk for stroke is increased _______ by atrial fibrillation.
   a. two-fold
   b. three-fold
   c. five-fold
   d. ten-fold

12. True or False. The new anticoagulants have fewer drug-drug interactions than warfarin and fewer dietary effects, as well, as well as lower risk for gastrointestinal bleeding than warfarin.
   a. True.
   b. False.

13. Aspirin alone or aspirin and clopidogrel have been shown to be ________________ to warfarin for preventing thromboembolic events and stroke and the risk for bleeding is higher.
   a. inferior
   b. superior
   c. equal
   d. None of the above.

14. The Cox maze IV can also be combined with ________________ that is done six to eight weeks after the surgery.
   a. Cardioversion
   b. Inderal
   c. Radiofrequency ablation
   d. Plavix

15. In patients with pre-excitation and atrial fibrillation should not be administered as they may increase the ventricular response and may result in ventricular fibrillation.
   a. inderal, alpha blocking agents, or IV digoxin
   b. digoxin, nondihydropyridine calcium channel antagonists, or intravenous amiodarone
   c. procainamide, clonidine, or dopamine
   d. amiodarone, dopamine, or lidocaine
16. True or False. Nondihydropyridine calcium channel antagonists should not be used in patients with decompensated heart failure; this may cause further hemodynamic compromise.
   a. True.
   b. False.

17. The risk of atrial fibrillation increases by up to ______% if more than two drinks a day are consumed.
   a. 10%
   b. 20%
   c. 30%
   d. 40%

18. Warfarin inhibits vitamin K-dependent synthesis of the clotting factors II, VII, IX, and X.
   a. Vitamin A
   b. Vitamin K
   c. Vitamin D
   d. Vitamin B

19. Rhythm control can be accomplished by all of the following medication, EXCEPT:
   a. Lasix.
   b. Amiodarone.
   c. Dofetilide.
   d. Quinidine.

20. Surgical procedures for treatment of atrial fibrillation include
   a. AV node ablation.
   b. catheter ablation.
   c. Cox maze procedure.
   d. All of the above.
21. Cardioversion to restore sinus rhythm can be done electrically or pharmacologically and it can be done as an outpatient procedure or when the patient is hospitalized.
   a. True.
   b. False.

22. In most cases of atrial fibrillation, a CHA2DS2-Vasc score of ______ indicates the need for anticoagulation.
   a. <
   b. ≥2
   c. ≥5
   d. ≤5

23. Comparisons between rate control and rhythm control have shown that __________________________ are/is only moderately effective for abolishing atrial fibrillation, it does not have as strong an effect on mortality, and it is associated with a greater number of adverse effects.
   a. Rhythm control
   b. Rate control
   c. Both rate and rhythm control
   d. None of the above.

24. The risks of direct current cardioversion include all EXCEPT
   a. burns.
   b. thromboembolism.
   c. hemorrhage.
   d. sinus arrest.
25. Indications for catheter ablation as a treatment for atrial fibrillation include, but are not limited to:

a. patients who have symptomatic paroxysmal atrial fibrillation that is refractory to at least one class I or III antiarrhythmic drug, or who are intolerant of these drugs
b. patients who have symptomatic persistent atrial fibrillation that is refractory to at least one class I or class III antiarrhythmic drug, or who are intolerant of these drugs.
c. patients who have symptomatic persistent atrial fibrillation that is refractory to at least one class I or class III antiarrhythmic drug, or who have developed tolerance of these drugs.
d. Both a. and b. above.

CORRECT ANSWERS:

1. The incidence of atrial fibrillation:
   a. increases with age

   p. 8: “The risk of developing atrial fibrillation increases with age, and the prevalence of atrial fibrillation doubles with each decade starting at age 50."

2. Which of the following are considered pathogenic mechanisms of atrial fibrillation?
   c. An initiating focus and atrial remodeling

   p. 6: “The pathogenesis of atrial fibrillation is complex and not completely understood, but there is substantial evidence for two pathogenic mechanisms by which this arrhythmia is initiated and sustained: a triggering focus and atrial remodeling, respectively.”

3. Which of the following are established risk factors for atrial fibrillation?
   b. Hypertension, heart failure
Most cases of atrial fibrillation occur in people who have hypertension, and hypertension increases the risk of paroxysmal atrial fibrillation progressing to permanent atrial fibrillation… Atrial fibrillation and heart failure often coexist. Studies have shown that one-third to almost two-thirds of patients with heart failure have atrial fibrillation.”

4. The diagnosis of atrial fibrillation can only be made by:

d. a 12-lead ECG

Risk factors can help clinicians consider a diagnosis of atrial fibrillation in certain patients, and there are signs and symptoms that can indicate its presence. But atrial fibrillation can only be diagnosed by examining a 12-lead electrocardiogram (ECG).”

5. The heart rate of someone who has atrial fibrillation is typically:

a. very rapid and irregularly irregular

The rapid atrial depolarization of atrial fibrillation that supersedes the SA node as the cardiac pacemaker and the inability of the AV to transmit to the ventricles the hundreds of atrial impulses that are firing each minute cause the characteristic electrocardiographic changes of atrial fibrillation. Atrial fibrillation involves an absence of P waves and an irregularly, irregular ventricular rate.”

6. Cardioversion is used if the patient with atrial fibrillation:

c. is hemodynamically compromised

Emergency or immediate cardioversion to restore sinus rhythm in a patient who has new onset atrial fibrillation and a rapid ventricular rate should be performed, or at least considered, if the patient has any of the following.

- Active myocardial ischemia
- Evidence of organ hypo-perfusion
- Hypotension
- Severe heart failure
- First episode of atrial fibrillation with duration of < 48 hours
- Wolf-Parkinson-White syndrome, atrial fibrillation, and hemodynamic compromise”
7. **Rhythm control in patients who have atrial fibrillation can be attained by using:**

   b. electrical or pharmacological cardioversion

   p. 20: “Cardioversion to restore sinus rhythm can be done electrically or pharmacologically …”

8. **Rate control in patients who have atrial fibrillation is **typically **attained by using:**

   d. beta blockers or certain calcium channel blockers

   p. 24: “Pharmacological rate control can be done by using beta-blockers, nondihydropyridine calcium channel blockers (diltiazem or verapamil) …”

9. **The most common and most serious complication of atrial fibrillation is:**

   c. stroke

   p. 17: “Stroke is the most common and most serious complication of atrial fibrillation.”

10. **Prevention of thromboembolism in atrial fibrillation is achieved by:**

    b. warfarin, apixaban, dabigatran, edoxaban, and rivaroxaban

    p. 33: “Anticoagulants have been proven to lower the risk for thromboembolic stroke in patients who have atrial fibrillation. The anticoagulants that are used for this purpose are warfarin, and the new oral anticoagulants apixaban, dabigatran, edoxaban, and rivaroxaban.”

11. **The risk for stroke is increased ________ by atrial fibrillation.**

    c. five-fold

    p. 17: “The risk for stroke is increased five-fold by atrial fibrillation.”
12. True or False. The new anticoagulants have fewer drug-drug interactions than warfarin and fewer dietary effects, as well, as well as lower risk for gastrointestinal bleeding than warfarin.
   b. False.

   p. 34: “The new anticoagulants have fewer drug-drug interactions than warfarin and fewer dietary effects, as well, but a higher risk for gastrointestinal bleeding than warfarin.”

13. Aspirin alone or aspirin and clopidogrel have been shown to be ________________ to warfarin for preventing thromboembolic events and stroke and the risk for bleeding is higher.
   a. inferior

   p. 35: “Aspirin alone or aspirin and clopidogrel have been shown to be inferior to warfarin for preventing thromboembolic events and stroke and the risk for bleeding is higher.”

14. The Cox maze IV can also be combined with ________________ that is done six to eight weeks after the surgery.
   c. Radiofrequency ablation

   p. 29: “The Cox maze IV can also be combined with radiofrequency ablation that is done six to eight weeks after the surgery.”

15. Patients with pre-excitation and atrial fibrillation should not be administered the following medication as they may increase the ventricular response and may result in ventricular fibrillation.
   b. digoxin, nondihydropyridine calcium channel antagonists, or intravenous amiodarone

   p. 26: “In patients with pre-excitation and atrial fibrillation, digoxin, nondihydropyridine calcium channel antagonists, or intravenous amiodarone should not be administered as they may increase the ventricular response and may result in ventricular fibrillation.”

16. True or False. Nondihydropyridine calcium channel antagonists should not be used in patients with decompensated heart failure; this may cause further hemodynamic compromise.
   a. True.
p. 26: “Nondihydropyridine calcium channel antagonists should not be used in patients with decompensated heart failure; this may cause further hemodynamic compromise.”

17. **The risk of atrial fibrillation increases by up to ______% if more than two drinks a day are consumed.**

   c. 30%

   p. 11: “the risk of atrial fibrillation increases by up to 30% if more than two drinks a day are consumed…”

18. **Warfarin inhibits vitamin K-dependent synthesis of the clotting factors II, VII, IX, and X.**

   b. Vitamin K

   p. 33: “Warfarin inhibits vitamin K-dependent synthesis of the clotting factors II, VII, IX, and X.”

19. **Rhythm control can be accomplished by all of the following medication, EXCEPT:**

   a. Lasix.

   p. 38: “Rhythm control can be accomplished by using amiodarone, disopyramide, dofetilide, dronedarone, flecainide, ibutilide, propafenone, or quinidine.”

20. **Surgical procedures for treatment of atrial fibrillation include**

   a. AV node ablation.
   b. catheter ablation.
   c. Cox maze procedure.
   d. **All of the above.**

   p. 38: “Surgical procedures for treatment of atrial fibrillation include AV node ablation, catheter ablation, and the Cox maze procedure.”
21. Cardioversion to restore sinus rhythm can be done electrically or pharmacologically and it can be done as an outpatient procedure or when the patient is hospitalized.
   
   a. True.
   p. 20: “Cardioversion to restore sinus rhythm can be done electrically or pharmacologically and it can be done as an outpatient procedure or when the patient is hospitalized.”

22. In most cases of atrial fibrillation, a CHA2DS2-Vasc score of ______ indicates the need for anticoagulation.
   
   b. ≥ 2
   
   p. 31: “In most cases of atrial fibrillation, a CHA2DS2-Vasc score of ≥ 2 indicates the need for anticoagulation.”

23. Comparisons between rate control and rhythm control have shown that __________________ are/is only moderately effective for abolishing atrial fibrillation, it does not have as strong an effect on mortality, and it is associated with a greater number of adverse effects.
   
   a. Rhythm control
   
   p. 23: “Rate control or rhythm control (or the two together) can be used to treat atrial fibrillation. Comparisons between the two have shown that rhythm control is only moderately effective for abolishing atrial fibrillation, it does not have as strong an effect on mortality, and it is associated with a greater number of adverse effects.”

24. The risks of direct current cardioversion include all EXCEPT
   
   c. hemorrhage.
   
   p. 20: “The risks of direct current cardioversion include brady-arrhythmias, burns, complications of sedation used during the procedure, thromboembolism, reprogramming/altering the functions of an implanted cardiac device, sinus arrest, and ventricular fibrillation or tachycardia.”
25. **Indications for catheter ablation as a treatment for atrial fibrillation include, but are not limited to:**

   a. patients who have symptomatic paroxysmal atrial fibrillation that is refractory to at least one class I or III antiarrhythmic drug, or who are intolerant of these drugs
   
   b. patients who have symptomatic persistent atrial fibrillation that is refractory to at least one class I or class III antiarrhythmic drug, or who are intolerant of these drugs.
   
   c. patients who have symptomatic persistent atrial fibrillation that is refractory to at least one class I or class III antiarrhythmic drug, or who have developed tolerance of these drugs.
   
   d. **Both a. and b. above.**

   p. 27: “Indications for catheter ablation as a treatment for atrial fibrillation include, but are not limited to: 1) patients who have symptomatic paroxysmal atrial fibrillation that is refractory to at least one class I or III antiarrhythmic drug, or who are intolerant of these drugs; and, 2) patients who have symptomatic persistent atrial fibrillation that is refractory to at least one class I or class III antiarrhythmic drug, or who are intolerant of these drugs.”

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**References Section**

The References below include published works and in-text citations of published works that are intended as helpful material for your further reading.


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