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. Atrial fibrillation is a disease that affects individuals in later years, however, rarely may affect children. A new diagnosis of atrial fibrillation requires investigation into possible reversible causes as a first approach to therapy. Prevention of atrial fibrillation is highlighted as a way to lower the risks associated with complications of atrial fibrillation and stroke, such as substance abuse and obesity. New guidelines influencing how atrial fibrillation is understood and treated, including newer pharmacological agents, are discussed.
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Statement of Need:
Ongoing education is needed on the public health burden of atrial fibrillation. Current medical, pharmacological, and surgical treatments for atrial fibrillation support nurses caring for patients with atrial fibrillation.

Course Purpose:
To provide nurses with current knowledge of the care needs of patients with a diagnosis of atrial fibrillation.
Learning Objectives:
1. Identify two pathogenic mechanisms of atrial fibrillation.
2. Identify risk factors that cause atrial fibrillation.
3. Identify surgical techniques that can be used to treat atrial fibrillation.
4. Identify medications and therapies used to attain rate or rhythm control.
5. Identify the medications used to prevent stroke in patients who have atrial fibrillation.

Target Audience:
Advanced Practice Registered Nurses, Registered Nurses, Licensed Vocational Nurses, and Associates

Course Author & Director Disclosures:
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Please take time to complete the self-assessment Knowledge 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 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 unstable
   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 thrombo-embolism in atrial fibrillation is achieved by:
    a. aspirin and warfarin
    b. warfarin, apixaban, dabigatran, or rivaroxaban
    c. ACE inhibitors and statins
    d. emergency or elective cardioversion
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.

The treatment of atrial fibrillation is rate and/or rhythm control, anti-coagulant medication to reduce the risk of 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 is a significant public health problem.

This study module will provide professional nurses with 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.

ATRIAL FIBRILLATION: STATISTICS

Atrial fibrillation is the most common arrhythmia.\(^1\)\(^2\) There is evidence that the incidence, prevalence, and mortality associated with atrial fibrillation
have increased and will continue to do so.\textsuperscript{3-5} The prevalence of atrial fibrillation in the general population has been noted to be one percent.\textsuperscript{6} Atrial fibrillation is more common in men (1.1\%) than in women (0.8\%).\textsuperscript{6} The arrhythmia is rare in children; the incidence increases with age, and atrial fibrillation is relatively common in adults 65 years of age or older.\textsuperscript{6}

The American Heart Association (AHA) reported in 2013 that there were 479,000 hospital admissions for atrial fibrillation.\textsuperscript{7} Data extrapolated from the Framingham Study reported a lifetime risk of developing atrial fibrillation of 26\% for men and 23\% for women.\textsuperscript{8} More than 2.7 million Americans have atrial fibrillation.\textsuperscript{7} The ATRIA study and other sources predict that by the year 2050 the prevalence of atrial fibrillation will more than double.\textsuperscript{6,9}

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{10-12} Atrial fibrillation may also be caused in part by changes in the autonomic nervous system, fibrosis caused by cardiac disease, pre-existing re-entry mechanisms, and changes in the renin-angiotensin aldosterone system. It is also possible that in any single patient there are multiple ways that atrial fibrillation begins and sustains.
**Triggering Focus**

The pulmonary veins have been confirmed as a common site of the triggering focus - or foci - that initiates atrial fibrillation\textsuperscript{10-12} 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{11,12}

**Atrial Remodeling**

Electrophysiologic studies and examination of the myocardium have shown that there are electrical and structural changes in the heart that sustain atrial fibrillation once a triggering focus has begun to initiate the arrhythmia.\textsuperscript{10-12} These changes can include a decreased refractory period, 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.\textsuperscript{10-12} How these changes occur is not entirely clear, but it may be that the triggering focus/foci induce them in a vulnerable heart; in simpler terms, atrial fibrillation may at times be the cause of atrial fibrillation. 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 sino-atrial (SA) node. Hence, ventricular depolarization and contraction is determined by the ectopic foci in the atria.
2. Transmission of all of these impulses through the atrio-ventricular (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 all of 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 the study module.

**CAUSES OF ATRIAL FIBRILLATION: RISK FACTORS**

There are many factors that can, or may increase the risk of developing atrial fibrillation. Some are proven risk factors while others are considered to be a risk 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 below. While an in-depth discussion of medical causes of atrial fibrillation is not possible here, it’s important to understand that many patients who have atrial fibrillation also have specific cardiovascular diseases, chronic diseases, and/or life style factors that cause
atrial fibrillation or are thought to contribute to its development. However, there is epidemiological evidence that suggests that many cases of atrial fibrillation - almost 44% - cannot be explained by the presence of these risk factors.²

**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 50.¹³ This increase is thought to be due to structural remodeling in the atria.¹⁴,¹⁵ Even with the adjustment of other risk factors, men have a higher risk of developing atrial fibrillation than do women but it is not clear why.¹³,¹⁶

*Hypertension*

The association between hypertension and atrial fibrillation has been clearly established.¹³,¹⁶ The relative risk for someone who has hypertension of developing atrial fibrillation is not high (1.2% to 1.5%).¹³ However, as hypertension is very common, it has been estimated that 14% of all cases of atrial fibrillation are caused by hypertension.¹⁷,¹⁸ Data from the Framingham Heart Study and the Manitoba Follow-Up Study indicated that in people who have hypertension there is a 1.4 to 1.9 times greater risk of developing atrial fibrillation than normotensive individuals.¹⁷,¹⁹ Whelton (1994) reported that hypertension increased the risk for atrial fibrillation by up to 42%²⁰ and Nieuwlaat et al. (2005) found that 60% of people who have atrial fibrillation have hypertension.²¹

The mechanisms by which hypertension may cause or contribute to atrial fibrillation include stretching of the atria that causes abnormal atrial
repolarization (hypertension acting as a trigger) and/or structural and electrical remodeling (hypertension acting to continue atrial fibrillation). Activation of the renin-angiotensin system may also play a role.

**Valvular heart disease**

Valvular heart disease has been reported to increase the risk of atrial fibrillation by 1.8 to 3.4-fold in men and women, respectively. Left heart valvular lesions, in particular those caused by rheumatic heart disease and resulting in stenosis, are associated with the highest prevalence of atrial fibrillation; and, the worse the lesion, the higher the risk. Patients who have mild to moderate stenosis can have a 9.1% prevalence of atrial fibrillation. The prevalence increases to almost 34% with a severe stenosis. Isolated mitral valve regurgitation, isolated mitral stenosis, coexisting mitral regurgitation and stenosis, and mixed mitral or tricuspid valve disease have been associated with increased prevalence of atrial fibrillation.

**Heart failure and cardiac myopathy**

Atrial fibrillation (AF) and heart failure (HF) often coexist. Data from the Framingham Heart Study showed that many patients who had atrial fibrillation also had heart failure and those with heart failure often had, or developed, atrial fibrillation. Chugh et al. (2014) reported that patients who had heart failure had a six-fold increase in risk for atrial fibrillation and Luong et al. (2014) noted: “AF and HF can directly and independently lead to the development of the other,” and also: “The presence of one condition increases the risk of development of the other and is associated with poorer prognosis.”
The association between these two diseases is complex. Both atrial fibrillation and heart failure can cause cellular, electrical, functional, molecular and structural changes in the heart that predispose the heart to developing atrial fibrillation or heart failure and when these changes occur - as a result of atrial fibrillation or from heart failure - then the myocardium is “set up” for one to follow from the other. The prevalence of atrial fibrillation in patients who have hypertrophic cardiomyopathy has been reported to be 10% - 28%, and the incidence has been reported to be 4 - 6 times that of the general population.\(^28,29\)

**Alcohol consumption**

Heavy alcohol use and binge drinking are well-known and well-documented causes of atrial fibrillation.\(^{30,31}\) Sporadic binge drinking is also well-documented as a cause of atrial fibrillation, a condition that is known as “holiday heart.”\(^{32}\) It was long thought that moderate alcohol consumption did not confer a risk for the development of atrial fibrillation. However, several recent studies have shown that a daily consumption that is considered moderate (> 2 drinks a day for women, > 3 drinks a day for men, > 2 drinks a week of hard liquor or wine, or between 15 - 21 drinks a week) does significantly increase the risk of developing atrial fibrillation.\(^{31,33}\)

It is unclear how alcohol increases the risk for atrial fibrillation. Studies in animals and humans indicate that the pathologic mechanisms involved may be: cardiac conduction abnormalities; structural changes; increased vagal
activity; electrolyte imbalances; hypertension; and, increased oxidative stress.\textsuperscript{33}

\textit{Obesity}

Obesity and being overweight are proven risk factors for atrial fibrillation,\textsuperscript{13,34,35} and each unit of increase of the body mass index increases the risk for atrial fibrillation by 3\%.\textsuperscript{36} Part of this increase in risk is associated with the increased incidence of diabetes and cardiovascular disease in people who are obese or overweight.\textsuperscript{36}

\textbf{Table 1: Emerging Risk Factors}\textsuperscript{13}

<table>
<thead>
<tr>
<th>Prehypertension</th>
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<tbody>
<tr>
<td>Increased pulse pressure</td>
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<tr>
<td>Obstructive sleep apnea</td>
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<tr>
<td>Physical activity: Cumulative lifetime practice &gt;1500 h</td>
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<tr>
<td>Diastolic dysfunction</td>
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<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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\textbf{Table 2: Potential Risk Factors}\textsuperscript{13}

<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>
</tr>
<tr>
<td>Tobacco use</td>
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</table>
Table 3: Medical Condition as a Cause of Atrial Fibrillation

<table>
<thead>
<tr>
<th>Medical Condition as a Cause of Atrial Fibrillation</th>
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<tbody>
<tr>
<td>Hyperthyroidism</td>
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<tr>
<td>Myocardial infarction</td>
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<tr>
<td>Pheochromocytoma</td>
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<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 categories are briefly outlined below.

**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**

Atrial fibrillation persists for more than 12 months.

**Permanent Atrial Fibrillation**

The term permanent atrial fibrillation is used when the patient and the physician 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.

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) guideline from the American Heart Association and the American College of Cardiology and other authorities.37,38

**DIAGNOSIS OF ATRIAL FIBRILLATION**

Risk factors can help practitioners 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 mentioned above, are: 1) absence of P waves; and, 2) an irregularly, irregular ventricular rate. The QRS complex is usually narrow. Small fibrillatory waves, 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.
Physical Exam and History

The clinical presentation of atrial fibrillation can be completely benign or the patient may have mild and non-specific 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 atrial systole. 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 dyspnea, palpitations, etc.

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 a patient who may have atrial fibrillation the provider or nurse should ask whether there is a history of alcohol and/or illicit drug abuse and determine the pattern of use of tobacco; additionally, the patient should be asked about symptoms of chest pain, dizziness, dyspnea, dyspnea on exertion, fatigue, palpitations, or syncope. If any of these symptoms have occurred the provider should also find out *when* and *what* the patient was doing when they occurred. During the physical examination the provider should pay particular attention to breath sounds, heart sounds, and peripheral circulation.

Detecting atrial fibrillation can be difficult. 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. Finding atrial fibrillation, however, is very important in order to prevent a stroke; since atrial fibrillation can easily go undetected this is a significant challenge.

Atrial fibrillation is responsible for approximately 20% of all cryptogenic strokes and 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.
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.

The risk for stroke is increased five-fold by atrial fibrillation. Atrial fibrillation accounts for ≥ 15% of all strokes in the United States and accounts for 36% of all strokes in people > age 80, and these risk levels are probably underestimated. The risk for stroke 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. 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 risk of hospitalization, and a higher risk for re-admission.

The mortality rate associated with stroke caused by atrial fibrillation is important for providers and nurses to understand. An 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 days and weeks.
TREATMENT OF NEW ONSET ATRIAL FIBRILLATION

Treatment of new onset atrial fibrillation is reviewed in this section. It involves the following issues.

- Signs and symptoms: Is the patient asymptomatic, symptomatic but clinically stable, or is he or she 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 and the patient may be asymptomatic at the time of the examination, and there is also a significant percentage of people who have undiagnosed atrial fibrillation who do not develop symptoms. Serious cardiac, neurological, and/or pulmonary signs or symptoms caused by 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 treatment 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 should be performed if the patient has the following:\(^{37,55}\)

- Active myocardial ischemia
- Evidence of organ hypoperfusion
- Severe heart failure
- A pre-excitation syndrome, i.e., Lown-Ganong-Levine syndrome and Wolff-Parkinson-White (WPW) syndrome that is associated with tachycardia and hemodynamic instability
- First episode of atrial fibrillation with duration of < 48 hours

Some physicians may prefer to try rate control methods instead of rhythm control. Rate control and rhythm control seem to be equal in terms of their effect on mortality.\(^{56}\) Rhythm control may offer a slight advantage in improvement in symptoms and quality of life,\(^ {57,58}\) but the evidence for this is inconclusive. A potential advantage of rhythm control is to prevent structural remodeling that sustains atrial fibrillation and makes effective treatment of the arrhythmia difficult.\(^ {38}\) However, at this time there is no conclusive evidence that rhythm control prevents or reverses atrial remodeling.\(^ {59}\)
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. Electrical cardioversion is usually more successful and is considered the first-line approach, but the choice of which one to use will depend on the clinical situation.

Electrical cardioversion is a relatively complicated procedure. The basic approach is as follows.

- Depending on the circumstances some patients may be pre-treated with an anti-arrhythmic drug, i.e., dofetilide, ibutilide, or sotalol.

- If the patient is at high risk for stroke, she or he should be anti-coagulated as soon as possible before or immediately after cardioversion. Intravenous heparin, a low molecular weight heparin, a factor Xa inhibitor, or a direct thrombin inhibitor should be used.

- If the patient is at low risk for stroke, anticoagulation with one of the drugs mentioned in #2 may be considered or anticoagulation therapy may be omitted.

- If the patient is hemodynamically unstable, cardioversion should not be delayed in order to start anticoagulation.

- Serum electrolytes and oxygen saturation should be measured before the procedure is done.
• During the cardioversion procedure the patient should be sedated and the blood pressure, heart rate, CO₂ capnography, oxygen saturation, and cardiac rhythm should be continuously monitored.

• An electrical current of 100-200 joules is delivered during cardioversion.

• It is reasonable to make several attempts at electrical cardioversion if the initial attempt is unsuccessful.

• Long-term anticoagulation should be initiated after cardioversion if the patient is at high risk for a thromboembolic event.

• Risks associated with cardioversion include: bradyarrhythmias, burns, complications from the sedation, muscle soreness, reprogramming of implanted cardiac devices, and ventricular fibrillation or tachycardia.

Patients who have new onset atrial fibrillation should not be cardioverted if: they are completely asymptomatic; > 80 years of age; the patient has a high risk for bleeding (risk determination for bleeding will be discussed later in the study module); and, the patient cannot be given anticoagulants before or after cardioversion.⁶⁰

**Pharmacologic Rhythm Control**

Amiodarone (oral or intravenous), dofetilide (oral), ibutilide (intravenous), flecainide (oral), and propafenone (oral) are used for pharmacological cardioversion of atrial fibrillation.³⁷ A complete discussion of the administration, doses, and the risks and benefits of each drug 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.

**Rate Control**

If rhythm control is not needed or contraindicated, rate control should be tried. The drugs used for rate control in patients who have new onset atrial fibrillation are:\(^{37}\)

- Beta blockers: Esmolol, metoprolol, and propranolol (intravenous)
- Nondihydropyrodine calcium channel blockers: Diltiazem and verapamil (intravenous)
- Digoxin (intravenous)
- Amiodarone (intravenous)

The choice of a specific drug will depend on several factors. The beta-blockers appear to be the most effective and a combination of a beta-blocker and calcium channel blocker or a beta-blocker or calcium channel blocker and digoxin can be used. Digoxin can be used if the patient also has heart failure or left ventricular hypertrophy but, because the onset of action of digoxin is > 1 hour and its peak effect is in approximately six hours, digoxin is not considered first-line treatment for rate control in most cases of atrial fibrillation.\(^{37}\) Amiodarone is the last choice for attaining rate control in new onset atrial fibrillation.\(^{37}\) It can be effective at achieving rate control but there are many serious side effects associated with its use and it takes longer to achieve rate control, as well.\(^{37}\)
Prevention of Systemic Embolization

Phang and Olshansky (2013) note that unless the patient has risk factors for embolization, the risk of an embolic event in patients who have had atrial fibrillation < 48 hours is very low. The authors also noted that that there is little evidence for determining which patients with new onset atrial fibrillation should receive anticoagulation therapy and what drugs should be used. Phang and Olshansky highlight expert opinion on cardioversion in atrial fibrillation (AF) without anticoagulation or transesophageal echocardiography (TEE), discussed later in this study module, as below.

“... experts have recommended cardioversion without anticoagulation or TEE in patients with new onset AF of less than 48 hours duration.”

The American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society 2014 guideline for the management of patients with atrial fibrillation states that if the patient is at high risk for stroke, she or he should be anticoagulated as soon as possible before or immediately after cardioversion. Intravenous heparin, a low molecular weight heparin, a factor Xa inhibitor, or a direct thrombin inhibitor should be used.

If the patient is at low risk for stroke, anticoagulation with one of the aforementioned drugs may be considered or anticoagulation therapy may be omitted. Assessment for the risk of embolic stroke and the use of anticoagulants will be covered in detail in another section of the study module.
MANAGEMENT OF THE PATIENT WITH PERSISTENT OR PERMANENT ATRIAL FIBRILLATION

The treatment issues for this patient population are control or management of comorbidities and risk factors, rate and rhythm control, and prevention of embolization with anti-coagulant drugs. Discussion of the management of comorbidities and risk factors is beyond the scope of this study module. Rather, the focus will be on rate and rhythm control and prevention of systemic embolization. Anticoagulant medications will be discussed in a separate section.

Pharmacologic Rate and Rhythm Control

Drug therapy for rate control and rhythm control is an important treatment for persistent and permanent atrial fibrillation. Rate controlling agents and antiarrhythmics can reduce morbidity and improve quality of life, and these medications have been shown to produce a lasting effect, i.e., a heart rate consistently in the target range, maintenance of sinus rhythm, and prevention of the recurrence of atrial fibrillation. There is no evidence that either medication is superior and rate controlling agents and antiarrhythmics can be used alone or concurrently for the same patient.

Beta blockers, dihydropyridone calcium channel blockers, digoxin, and amiodarone are the drugs that are used to attain rate control for patients who have persistent or permanent atrial fibrillation. Antiarrhythmics that are used to maintain sinus rhythm after a patient has been cardioverted electrically or pharmacologically include amiodarone, disopyramide, dofetilide, dronedarone, flecainide, ibutilide, procainamide, propafenone, quinidine, and sotalol. The specific drugs used for rate and/or rhythm control will depend on the patient’s age, comorbidities, duration of the atrial
fibrillation, and symptoms profile. Readers are referred to Psotka and Lee (2014) for a complete discussion.

**Catheter Ablation for Rhythm Control**

Catheter ablation is a procedure in which an electrode is threaded into the atria and thermal energy transmitted through the electrode destroys the ectopic atrial focus and allows sinus rhythm to be restored by the SA node. It has been shown to be an effective method for restoring sinus rhythm; and, it is considered to be:

“... *reasonable for selected patients with symptomatic persistent AF refractory or intolerant to at least 1 class I or III anti-arrhythmic medication.*”\(^{37}\)

Catheter ablation can reduce the severity of symptoms, improve left ventricular function, and improve functional capacity.\(^{63,64}\)

Several studies that compared catheter ablation to antiarrhythmic drugs reported that ablation was superior for preventing recurrence of atrial fibrillation,\(^{65,66}\) and the success rate for maintaining sinus rhythm at the 1 - 2 year follow-up mark has been reported to be 60% - 80%.\(^{10}\) However, recurrence of atrial fibrillation, immediately after the procedure or several years later is relatively common.\(^{37,65}\)

Patients must be able to tolerate anticoagulant therapy during the procedure and for several months post-procedure. The risk of major complications has been reported to be between 1.7% - 4.5%.\(^{67,68}\)
Table 4: Complications Associated with Catheter Ablation

- Air embolism
- Atrial-esophageal fistula
- Cardiac tamponade/perforation
- Gastric motility disorder
- Iatrogenic atrial flutter
- Mitral valve injury
- Myocardial infarction
- Pericarditis
- Phrenic nerve injury resulting in diaphragmatic paralysis
- Pulmonary vein stenosis
- Radiation injury/burn
- Stroke or TIA
- Vascular access complications, i.e., hematoma, arterio-venous fistula

AV Node Ablation, Cox maze and Pacemakers

Atrio-ventricular (AV) node ablation is another surgical technique that can be used to treat persistent or permanent atrial fibrillation. This procedure is considered to be a useful “. . . to control heart rate when pharmacological therapy is inadequate and rhythm control is not achievable.” Rate control with other method should be tried before this procedure is done because AV node ablation is irreversible and requires implantation of a cardiac pacemaker. Patients who have cardiomyopathy and do not respond to rate control are most likely to benefit from AV node ablation.

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 in the atria - the maze - 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 Cox maze intravenous procedure that is now performed uses bipolar radiofrequency and cryoablation to create the same effect. This technique is simpler to perform and the success rate is still high: a recent article that reviewed Cox maze intravenous (IV) noted that the “...Cox maze IV procedure has the best results for the surgical treatment of atrial fibrillation.”

Cardiac pacemakers have a limited role in the treatment of persistent or permanent atrial fibrillation. The use of pacemakers is primarily for patients who have atrial fibrillation complicated by symptomatic bradycardia.

**Cardioversion**

Cardioversion was discussed previously. 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 (LA) thrombus. The 2014 guideline notes that “...it is reasonable to perform a TEE prior to cardioversion, and then cardiovert if no LA thrombus is identified, provided anticoagulation is achieved before TEE and maintained after cardioversion for at least 4 wk.”
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.\(^{37}\) 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 module).

**Prevention of Embolization**

Prevention of embolization in patients who have atrial fibrillation is critically important. Patients who are at risk for embolization and stroke must be identified and the risk for bleeding during anticoagulation therapy must be accurately assessed.

There are two assessment tools commonly used to determine the risk for stroke in patients who have atrial fibrillation, the CHADS2 and the CHA2DS2-VASc.\(^{41,70,71}\) The CHA2DS2-Vasc is better at detecting patients at low risk for stroke and it is the assessment tool recommended by the American College of Cardiology and the American Heart Association.\(^{37}\) In most cases case of atrial fibrillation, a CHA2DS2-Vasc score of \(\geq 2\) indicates the need for anticoagulation.\(^{37}\)

**Table 5: CHADS2 and CHA2DS2-Vasc**

<table>
<thead>
<tr>
<th>CHADS2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure (1 point)</td>
</tr>
<tr>
<td>Hypertension (1 point)</td>
</tr>
<tr>
<td>Age ≥75 years (1 point)</td>
</tr>
<tr>
<td>Diabetes (1 point)</td>
</tr>
<tr>
<td>Stroke or transient ischemic attack (2 points)</td>
</tr>
</tbody>
</table>
## CHA2DS2-VASc25

<table>
<thead>
<tr>
<th>Condition</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
</tr>
<tr>
<td>Age ≥75</td>
<td>2</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1</td>
</tr>
<tr>
<td>Stroke or transient ischemic attack</td>
<td>2</td>
</tr>
<tr>
<td>Vascular disease</td>
<td>1</td>
</tr>
<tr>
<td>Age 65-74</td>
<td>1</td>
</tr>
<tr>
<td>Sex category (female)</td>
<td>1</td>
</tr>
</tbody>
</table>

### Bleeding risk

An embolic stroke can be devastating but so can bleeding from an anticoagulant. As with any therapy or medication there are benefits and risks and anticoagulation therapy presents a distinct risk for serious bleeding. There are four assessment tools that can be used to determine the risk of bleeding and guide a clinician in deciding whether a patient who has atrial fibrillation can safely be prescribed an anticoagulant.

The 2014 guideline notes that although these assessment tools may be helpful in identifying patients who have a risk for bleeding “. . . their clinical utility is insufficient for use as evidence for recommendations in this guideline.”

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Table 6: Assessment Tools for Determining Bleeding Risk

| **Atria** |  
| (Anticoagulation and Risk factors in Atrial Fibrillation) |
| **HAS-BLED** |  
| (Hypertension, Abnormal renal/liver function, Stroke, Bleeding history or predisposition, Labile INR, Elderly, Drugs/alcohol concomitantly) |
| **HEMORRHAGES** |  
| (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) |
| **RIETE** |  
| Computerized Registry of Patients With Venous Thromboembolism |

**ANTICOAGULANT MEDICATIONS**

Anticoagulants have been proven to lower the risk for thromboembolic stroke in patients who have atrial fibrillation.\textsuperscript{72-74} Warfarin, apixaban (Eliquis), dabigatran (Pradaxa), and rivaroxaban (Xarelto) are the medications used to achieve anti-coagulation and prevent thromboembolic stroke in this patient population. It

Aspirin has not been shown to be effective for preventing stroke in patients who have atrial fibrillation.\textsuperscript{37}
should be mentioned that the latter three are commonly called the *new anticoagulants*. Warfarin acts by inhibiting vitamin K-dependent synthesis of the clotting factors II, VII, IX, and X. Apixiban and rivoxaban inhibit the clotting factor Xa; dabigatran inhibits the clotting factor IIa.

Choosing a drug must be done on a case-by-case basis. When warfarin and the new anticoagulants have been compared it has been found that: 37,75,76

- Warfarin and the new anticoagulants appear to be at least equally effective in terms of prevention of stroke in patients who have atrial fibrillation. The new anticoagulants are not recommended for use in patients who have atrial fibrillation caused by valvular disease.

- Compared to warfarin the new anticoagulants reduce total mortality, cardiovascular mortality, the risk of intracranial bleeding, and there is a trend toward less overall bleeding. However, dabigatran causes an increase in gastrointestinal bleeding.

- There are far more clinically important drug-drug interactions and drug-food interactions for warfarin.

- The time to onset and the half-life is much shorter for the new anticoagulants. This makes initiation and cessation of therapy simpler than it is for warfarin.

- The use of the new anticoagulants, unlike warfarin, does not require frequent laboratory testing. However, this also means that there is no simple way to monitor the effectiveness of these drugs.
• The new anticoagulants must be taken as prescribed. Missing one dose can remove their protective effect and put the patient at risk for a stroke.

• There is no reversal agent for the new anticoagulants.

• The new anticoagulants are more expensive.

• There are no clinical trials that have made a drug-to-drug comparison of one new anticoagulant to another.

• The new anticoagulant drugs should be avoided if the patient has severe renal insufficiency and an eGFR < 30 mL/min. Dose adjustments for people with chronic kidney are available.

Warfarin has been used since the 1950s and its adverse effects, contraindications, precautions, etc. are well known. There is comparatively much less clinical experience with the new anticoagulants. Clinicians should consult individual package inserts for specific information.

General concerns that are common to all three of the new anticoagulants include:77

• There are no laboratory tests that are used to monitor the effectiveness of these drugs and routine measurements of coagulation parameters are 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.
• 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. Whereas, Dabigatran and rivaroxaban should be taken with food.

• There are no known food restrictions for patients taking apixaban, dabigatran, or rivaroxaban.

• Non-steroidal anti-inflammatories should be avoided. Aspirin can be used in some patients.

• Patients who need dental work should consult with a physician prior to the work being done.

• If the patient needs surgery, she or he will need to be carefully assessed to determine when and if the drug should be discontinued before the surgery, when it should be resumed, the risk for bleeding, and what type of anesthesia can be safely used.

• Patients who have bleeding should notify a physician. If the bleeding is minor the drug may be continued and she or he may be managed with supportive care.
ATRIAL FIBRILLATION AND PREVENTION

An important question for clinicians to consider is: can atrial fibrillation be prevented? Atrial fibrillation is considered to be a heritable disease and having a family member who has atrial fibrillation has been associated with an increased risk of 40% for developing the arrhythmia. However, although genetic mutations associated with atrial fibrillation have been identified, genetic screening of the population is not recommended at this time.

The angiotensin converting enzyme (ACE) inhibitors and the angiotensin receptor blockers (ARBs), aldosterone inhibitors (i.e., spironolactone), and the statin drugs have all been investigated as therapies for the prevention of atrial fibrillation. These drugs can inhibit atrial remodeling and inflammation and, by that mechanism, there is some evidence that in certain patient populations they can prevent new onset atrial fibrillation. The 2014 guideline states that an ACE inhibitor or an ARB is reasonable for primary prevention of new onset atrial fibrillation in patients with heart failure, hypertension, or a reduced cardiac ejection fraction, but these drugs and the statins are not recommended as a preventive therapy if the patient does not have cardiovascular disease. The guideline also states that statin drugs may be considered for primary prevention of atrial fibrillation after coronary artery surgery.

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 all of these impulses through the atrio-ventricular (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 is a rapid, irregular heart and decreased cardiac output.

There are five categories of atrial fibrillation that have been discussed: 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 considered to be a heritable disease, as well.

Most patients who have atrial fibrillation will have non-specific 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. The ECG will be characterized by absence of P waves and an irregularly, irregular R-to-R interval. This 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. With every patient who has atrial fibrillation consideration should be given to the need for immediate cardioversion, rate control, rhythm control, and prevention of embolization. Cardoversion 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, procainamide, propafenone, quinidine, and sotalol.

Surgical procedures for treatment of atrial fibrillation include AV node ablation, catheter ablation of ectopic foci, 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, dabigatran, or rivaroxaban. Anticoagulation can significantly reduce the risk of stroke, but all of the anticoagulants can cause bleeding. The newer anticoagulants, apixaban, dabigatran, or rivaroxaban are at least as effective at preventing stroke as warfarin and they are easier to use. The
ACE inhibitors, ARBs, aldosterone inhibitors, and the statin drugs appear to be effective for the prevention of new onset atrial fibrillation in certain patient populations.

Health providers and nurses must continuously educate themselves on evolving professional guidelines that address the diagnosis and treatment of atrial fibrillation. Importantly, health professionals 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 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 the NURSECE4LESS.COM course planners evaluate nursing knowledge needs met following completion of this course by completing the self-assessment Knowledge Questions after reading the article.

Correct Answers, pg 41.
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 unstable
   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 thrombo-embolism in atrial fibrillation is achieved by:
    a. aspirin and warfarin
    b. warfarin, apixaban, dabigatran, or rivaroxaban
    c. ACE inhibitors and statins
    d. emergency or elective cardioversion
Correct Answers
1. A
2. C
3. B
4. D
5. A
6. C
7. B
8. D
9. C
10. B

Footnotes:


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