Women and Heart Disease
Pharmacology

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Abstract

Heart disease is the leading cause of death in woman over the age of 20 in the United States, and is responsible for approximately 450,000 deaths each year. Women are five-times more likely to die from a heart attack than from breast cancer. Furthermore, research indicates that more women than men die from cardiovascular disease each year. This course aims to provide healthcare professionals with important information about the incidence of heart disease in women, its etiology and risk factors. Healthy lifestyle choices are essential to the prevention of heart disease, and topics related to diet, exercise, weight control, and smoking cessation will be highlighted. The latest diagnosis and treatment of heart disease is discussed.
Continuing Nursing Education Course Planners

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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 3 hours.

Statement of Learning Need
Heart disease is the leading cause of death in women within the United States. While awareness among nurses has increased in recent years, there still exists a need for continued education that will lead to prevention, earlier detection, and better treatment outcomes of heart disease in women.

Course Purpose
To provide nursing professionals with knowledge to increase their efforts to improve prevention and treatment of heart disease in women.
**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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Susan DePasquale, MSN, FPMHNP-BC – all have no disclosures

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There is no commercial support for this course.

**Activity Review Information**

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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. Drugs categorized as Class II are known as those that
   a. block calcium entry into cells.
   b. decrease cardiac automaticity, reduce contractility, and slow conduction velocity.
   c. include diltiazem and verapamil.
   d. block fast sodium channels.

2. Digoxin may be administered for control of cardiac arrhythmias and
   a. is not classified in one of the four main categories of the Vaughan-Williams classification system.
   b. is classified in one of the four main categories of the Vaughan-Williams classification system.
   c. is not considered an antiarrhythmic agent.
   d. works by increasing the rate of cardiac conduction.

3. True or False: Chemical cardioversion is done through administration of medication as an emergency procedure, particularly when the patient’s symptoms are causing hemodynamic instability.
   a. True.
   b. False.

4. Isosorbide dinitrate combined with hydralazine
   a. may be beneficial in the management of heart failure.
   b. may be particularly useful within high-risk groups.
   c. is recommended by the American College of Cardiology/American Heart Association guidelines for treatment of heart failure.
   d. All of the above.

5. Infectious endocarditis occurs when
   a. a virus attacks the lining of the heart.
   b. there is less surface area for microorganisms to grow.
   c. bacteria or fungi collect on the interior layers of the heart.
   d. Both a and b above.
Introduction

Research in women’s heart health and pharmacology are evolving to include a range of potential medical approaches. Health professionals are encouraged to recognize that women’s heart health and medical treatment needs are uniquely different than those of men. Of high significance is the unique difference in pharmacological approaches and potential adverse reactions to medication management that are unique to women. Gender differences in heart health and disease progression tend to be divided in the literature due to the evolution of research and the disparity of research related to women’s heart health as compared to men. It is widely accepted that health treatment teams need to be informed and fully aware of the differences in women’s heart health with conventional treatment options. Additionally, health providers need to be aware of how perimenopause and menopause affect women’s heart health.

Medications for Cardiac Arrhythmias

The specific medications used for the treatment of cardiac arrhythmias are determined by the causes of each condition, as arrhythmias can present as heart rates where the heart beats too fast, too slow, or even in a rhythm that is life threatening. The Vaughan-Williams classification system is often utilized to categorize the different drugs used for the management of cardiac arrhythmias. According to this classification, the drugs are categorized as is outlined below.

Class I: Sodium Channel Blockers

Class I drugs reduce the rate of action potential within the heart muscle cells by blocking fast sodium channels, and by slowing the rate of conduction
within certain cells, including within the Bundle of His and the Purkinje fiber system. Class I drugs are further categorized into three subclasses:

- **Class IA**: Quinidine, procainamide
- **Class IB**: Lidocaine, phenytoin
- **Class IC**: Flecainide, propafenone

**Class II: Beta Blockers**

Drugs categorized within this class decrease automaticity, reduce contractility, and slow the conduction velocity of the heart by reducing the rate of calcium entry and slowing the rate of depolarization in phase 4 of the cardiac cycle. Examples include propranolol, esmolol, or acebutolol.

**Class III Potassium Channel Blockers**

These drugs work by decreasing the rate of action potential of the myocardial cells and decreasing the refractory period. The ultimate effect is the delay in repolarization of the cells during the cardiac cycle. Examples of potassium channel blockers that are part of class III drugs include amiodarone and dronedarone.

**Class IV: Calcium Channel Blockers**

Drugs in this class work in a manner similar to those in class II, in that they depress action potentials by blocking calcium entry into cells. They act to decrease the rate of automaticity, slow conduction velocity, and reduce the refractory period. Examples of these types of agents include diltiazem and verapamil.
Other Drugs: Not in the Classification System

Other drugs that do not fit within the classification system but that are still used as antiarrhythmic agents include adenosine and digoxin. These drugs work by decreasing the rate of conduction in the atrioventricular (AV) node and by reducing automaticity in the sinoatrial (SA) node. They are technically not included in the Vaughan-Williams classification system, but are included here because they are commonly added as medications for arrhythmia management.\textsuperscript{1,2}

The type of medication used to treat the cardiac dysrhythmia is selected based on the type of arrhythmia present. The condition is usually treated when the cardiac rhythm affects the person’s quality of life and causes complications. Treatment often consists of initial management of the arrhythmia, followed by ongoing treatment with daily medication use. For example, a patient who presents for care with new-onset atrial fibrillation might be given medication to manage the acute phase of the condition, followed by a prescription for daily medication for long-term management.

In general, cardiac arrhythmias occur as one of two types: supraventricular dysrhythmias, which occur in the SA or AV nodes or in the atria; and ventricular dysrhythmias, which occur in the ventricles.\textsuperscript{1} Despite these classifications, the prescription and administration of exact kinds of medications for each particular arrhythmia is not an exact science. Instead, successful control of cardiac dysrhythmias through medication often depends on the patient’s response to the drug and its effects in the body.

The sodium channel blocking agents are classified into three subcategories because of the rate of their effects in the body, known as kinetics. Drugs in class IA have fast kinetics, those in class IB have slow kinetics, and those in
class IC have intermediate kinetics.\textsuperscript{2} Thus, the types of arrhythmias present will determine which subcategory of sodium channel blockers may be used. For instance, because medications in class IA have fast kinetics, they are more likely to be used for treatment of cardiac dysrhythmias that demonstrate very fast heart rates, while those in class IB could be used to treat dysrhythmias with rapid or normal heart rates.

Quinidine and procainamide, which are both class IA sodium channel blocking agents, are often used to treat severe tachyarrhythmias because of their fast kinetic actions. These drugs slow the movement of sodium ions through fast sodium channels, which ultimately slows cardiac conduction and the heart rate. They also widen QRS complexes and prolong QT intervals, as seen on the electrocardiogram (ECG). Quinidine is administered orally at doses between 200 and 400 mg per dose. Alternatively, procainamide can be given intravenously or orally. As an intravenous dose, procainamide is administered at 10 to 15 mg/kg as an initial bolus to correct the dysrhythmia, followed by a maintenance intravenous (IV) dose. Oral doses may be given as maintenance therapy and are available as regular or extended-release formulations, in doses that range from 250 to 750 mg per dose, depending on formulation and patient weight.\textsuperscript{2}

Class IB sodium channel blockers consist of the drugs lidocaine, mexiletine, and phenytoin. These drugs are often used for the more serious ventricular dysrhythmias, including those caused by digoxin toxicity. They work by binding to both open and inactivated sodium channels to affect the movement of sodium ions and to shorten the repolarization phase.\textsuperscript{5} They often show little to no effects on the ECG during monitoring after their administration, such as with the potential widening of QRS complexes that may be seen with class IA agents. Lidocaine is only administered
intravenously; it often requires an initial bolus dose, followed by a maintenance infusion. A typical dose of Lidocaine is approximately 100 mg of a bolus dose, given rapidly over about 2 minutes, followed by a maintenance infusion of 4 mg/minute. A second bolus may be administered if the arrhythmia is not corrected a few minutes after the maintenance infusion has started.

Mexiletine may be administered as an oral or as an intravenous drug. Orally, the drug is available as a regular medication or as an extended-release formulation. Typical doses range from 100 to 360 mg, given every 8 to 12 hours, depending on the patient’s condition. When given intravenously, mexiletine starts with an initial infusion of approximately 250 mg given IV over 1 hour, then slowed to 250 mg IV over 2 hours, then a maintenance dose at 0.5 mg/min.

Class IC antiarrhythmic drugs include flecainide and propafenone. They are some of the most potent forms of sodium channel blockers and therefore should be used very carefully with administration. As with other sodium channel blockers, flecainide impairs the movement of sodium ions through cell membranes, which impacts the cardiac conduction system. In most cases, flecainide is only given in emergency situations, such as when a patient experiences ventricular tachyarrhythmias that have become life threatening and that are otherwise unresponsive to other drugs. It may be given orally or intravenously; as an oral preparation, flecainide is given at a dose of approximately 100 mg every 8 to 12 hours. Intravenously, flecainide is administered at 1 to 2 mg/kg rapidly, over approximately 10 minutes as a bolus. It should be noted that in the U.S., flecainide is only available in oral form and is not available as an intravenous dose.
Propafenone is almost exclusively used for treatment of life-threatening ventricular arrhythmias, particularly in cases where other medications have failed to work. Propafenone, which is also a class IC sodium channel blocker, works in a manner similar to flecainide by altering the movement of sodium ions in the cardiac cells. It results in a widening of the QRS complex as seen on the ECG and it prolongs the P-R interval. It is not available in intravenous form in the U.S. and is thereby only administered as an oral preparation. A typical dose given is 150 mg, three times daily, which can be titrated up to 300 mg three times per day.\(^2\)

Class II agents are beta-blockers, which are also referred to as beta-adrenergic antagonists. These drugs affect the pacing areas of the heart. When the sympathetic nervous system is stimulated, norepinephrine is released, which binds to the beta-adrenergic receptors in the nodal tissues of the heart. This leads to an increase in the rate of firing from the node, as well as an increase in conduction velocity. When beta-antagonists are activated, they block the stimulation of beta-adrenergic receptors in the nodal tissues, thereby decreasing late-stage depolarization and increasing the length of repolarization.\(^5\) This ultimately slows and regulates the heart rate, which is why these agents are used for the management of both supraventricular and ventricular tachyarrhythmias.

Examples of class II beta-blockers used for the management of cardiac arrhythmias include propranolol, acebutolol, and esmolol. All of these drugs prolong the P-R interval and have the potential to cause bradycardia. Although these drugs are all characterized as being beta-blockers, there are technically differences in their properties that further classify them as being first-, second-, or third-generation drugs. It must be remembered that there are different types of beta-receptors, beta-1 and beta-2 adrenergic
receptors, which are located primarily in the heart and lung tissue. Beta-1 receptors are more commonly found in the heart and when stimulated, they impact heart muscle tissues. Beta-2 receptors are found in the smooth muscle tissue in the lungs; their stimulation results in dilation of the bronchioles, which can ease breathing. Propranolol is an example of a first-generation beta-blocker, in that it acts as an antagonist against beta-1 and beta-2 adrenergic receptors. Alternatively, second-generation beta-blockers, such as acebutolol, are more selective for beta-1 adrenergic receptors. Third-generation drugs are antagonists against beta-1 adrenergic receptors, but they also cause vasodilation.

Unfortunately, beta-blockers are prone to cause various side effects, which can range from bronchospasm, depression, and insomnia to heart block and bradycardia. These side effects often develop because of their antagonistic effects and their potential to impact the central nervous system. As a result, they should be used with caution when treating arrhythmias. Both propranolol and acebutolol are administered orally. Esmolol is only administered intravenously. Other drugs that may also be considered within this particular group of agents include metoprolol, betaxolol, atenolol, bisoprolol, carvedilol, nadolol, and timolol.

Class III potassium channel blocking agents affect the rate of repolarization of the cardiac cells during the conduction cycle. During the action potential phase, calcium ions act as depolarizing agents and enter the cells; potassium ions are then part of the repolarization process as they move out of the cells. Potassium channel blockers therefore prolong this process of resetting the system, so to speak, which returns the heart rate to a normal rhythm and typically, a slower rate. Potassium channel blockers can be prescribed for both supraventricular and ventricular arrhythmias, and they
may be administered in life-threatening situations. In cases of toxicity, though, their effectiveness can lead to changes on the ECG and cause *torsades de pointes*, which occurs as a consistently prolonged Q-T interval that could lead to sudden cardiac death.\(^5\)

The potassium channel blockers can prevent sudden cardiac death related to ventricular arrhythmias, such as ventricular tachycardia or ventricular fibrillation. Sotalol is one type of drug that may be administered in these cases. It is actually a combination agent that is technically classified as a class II/class III antiarrhythmic drug in that it possesses beta-adrenergic receptor blocking abilities in addition to potassium channel blocking properties. According to a review in the *Journal of Cardiovascular Pharmacology*, sotalol is also composed of two different types of isomers: the \(d\)-isomer and the \(l\)-isomer. The \(d\)-isomer is effective in blocking potassium channels, while the \(l\)-isomer is able to block both potassium channels and to produce beta-adrenergic blocking effects. In this manner, patients who cannot otherwise tolerate beta-blocking agents may benefit from sotalol; it has been found to be most useful in cases of ventricular arrhythmias among patients who do not have underlying heart failure or renal disease.\(^6\) Sotalol may also be used for supraventricular arrhythmias, including atrial flutter or atrial fibrillation.

Atrial fibrillation, which is a type of supraventricular dysrhythmia, occurs when the atria beat in an abnormal pattern compared to the ventricles. The electrical impulse that is generated from the SA node starts to fire quickly and abnormally and the atria tend to quiver instead of contracting and pumping blood normally. As a result, normal blood flow through the heart is compromised. When this occurs, the patient may develop signs and symptoms of poor blood flow, including anxiety, malaise, dyspnea, syncope,
chest pain, and altered mental status. Treatment requires cardioversion to convert the electrical rhythm back into a normal pattern. Chemical cardioversion is done through administration of medication as an emergency procedure, particularly when the patient’s symptoms are causing hemodynamic instability.

Some of the more common medications used for management of atrial fibrillation include potassium-channel blockers (class III agents) and calcium channel blockers (class IV drugs). Amiodarone, while not technically approved for the management of atrial fibrillation, is actually one of the more common drugs administered for chemical cardioversion of atrial fibrillation when it develops. Amiodarone, which is a potassium channel blocker, works by blocking a number of ion channels to establish a regular rhythm. While technically classified as a class III agent, amiodarone actually has the properties of class I, class II, and class IV agents as well. It can affect the sodium and calcium channels as well as beta receptors because of its ability to affect the lipid membranes where the ion channels are located. In this method, amiodarone has been more frequently used in a number of different situations where arrhythmias have developed because of its abilities to control various mechanisms of the cardiac conduction cycle.

Amiodarone may be administered as an intravenous dose until the patient is stabilized; it can then be followed with an oral maintenance dose or the patient can be given a long-term stabilizing dose of a similar medication. Typical dosages of amiodarone given for the management of atrial fibrillation are 150 to 450 mg over 1 to 6 hours IV as a loading dose, followed by up to 2 mg/minute IV maintenance; or 600 to 1200 mg/day orally for 7 to 10 days, followed by a decreased rate of a maintenance dose. Amiodarone
should be closely monitored when administered, as it could potentially cause an opposite of its intended effects and lead to bradycardia.

Ibutilide is another potassium-channel blocking agent that affects the rate of repolarization of the cardiac cells during the conduction cycle. It may be prescribed for cases of atrial fibrillation or atrial flutter and it is a good alternative to electrical cardioversion. Ibutilide should be used with caution in patients who have a history of long Q-T syndrome, as it may cause torsades de pointes. Nair, et al., in the Journal of the American Board of Family Medicine, described studies that have shown that ibutilide is more effective in the chemical cardioversion of atrial fibrillation or atrial flutter when compared to some other agents, including sotalol or procainamide, as well as amiodarone in some cases of recent onset. However, torsades de pointes has developed in up to 4 percent of patients who have been given ibutilide as the main form of treatment for cardiac arrhythmia without any other drug being administered. For this reason, administration of ibutilide should be done carefully and most often within the intensive care environment where close patient monitoring is essential. Additionally, it may not be the best choice for use as monotherapy and may need to be combined with other agents to treat serious cardiac arrhythmias to avoid significant side effects.

Dofetilide is yet another potassium-channel blocker used for the management of supraventricular arrhythmias, including atrial fibrillation and atrial flutter. Dofetilide is only available as an oral dose and it is given as a mechanism of chemical cardioversion for these types of supraventricular arrhythmias. As with ibutilide, dofetilide also has the potential to cause long Q-T intervals and torsades de pointes, so it is often only used in situations where the patient is extremely symptomatic from the arrhythmia and is
under close observation.\textsuperscript{5} It is contraindicated in cases where the patient has a history of long Q-T intervals. A typical oral dose of dofetilide is between 125 and 500 mcg; the actual amount is calculated for each patient according to creatinine clearance because of its potential adverse effects on renal function.\textsuperscript{2} There are a number of other class III antiarrhythmic agents in addition to the ones listed here. Examples include dronedarone and bretylium. Almost all drugs in this class also have an impact on the Q-T interval, so they require close monitoring with use, even when the drugs are administered orally.

The calcium channel blockers, which are class IV agents, are those that prevent the movement of calcium ions across cell membranes within the cardiac cells. Calcium channel blockers also affect calcium ion movement in smooth muscle cells, so they have an impact on vascular resistance and can cause vasodilation, particularly within the coronary arteries. As antiarrhythmic agents, calcium channel blockers decrease the rate of phase 4 repolarization and increase the length of repolarization in phases 1 and 2 of the conduction cycle. They are most commonly used for the management of supraventricular dysrhythmias. Currently, there are only two kinds of calcium channel blockers approved for use in cardiac arrhythmias, which are verapamil and diltiazem.

Verapamil is most often used for the management of atrial flutter or atrial fibrillation, as well as paroxysmal supraventricular tachycardia, which occurs as occasional periods of significant tachycardia that begin in the atria. In cases of paroxysmal supraventricular tachycardia (SVT), verapamil is often given as a preventive measure if the patient has suffered through more than one episode; giving verapamil may then prevent the condition from recurring. In cases of SVT, a patient who is currently experiencing the
Arrhythmia may require an IV dose of verapamil, while someone who is taking the drug as a preventive measure against future episodes would most likely benefit from an oral dosing regimen.

Because of the effects of verapamil on the movement of calcium ions through the cell membranes, it typically slows the rate of conduction through the AV node and it reduces cardiac output. Because of this response though, it should be used with caution in patients who have underlying hypotension, as the effects on heart rate and cardiac output could cause a further drop in blood pressure levels. An example of a dose of verapamil, given for supraventricular tachyarrhythmia is 5 to 15 mg, given over 10 minutes when a person presents in an acute state of arrhythmia; as a preventive dose, it may be administered from 40 to 120 mg orally, three times daily.$^2$

Diltiazem is another calcium channel blocker that has more than one use among cardiac conditions, including management of hypertension and angina, as well as some types of supraventricular tachyarrhythmias, including atrial fibrillation and atrial flutter. Diltiazem affects the movement of calcium ions through the cell membranes in a manner similar to that of verapamil. Its action controls the rate of ventricular contractions; additionally, diltiazem causes vasodilation, particularly in the coronary vessels, which can improve cardiac blood flow to control angina. Doses of diltiazem are 30 to 120 mg, three times per day when given orally. Alternatively, it may also be administered intravenously at 0.25 mg/kg initially, followed by a continuous infusion as needed.$^8$

Beyond the four general classes of antiarrhythmic drugs, as categorized by the Vaughan-Williams system, there are also some medications that do not fall into particular categories but that could still be used to control some
cases of cardiac arrhythmias. The drugs often included in this category are adenosine and digoxin.

Adenosine is actually produced, in part, within the body as a compound of a carbohydrate and adenine. It has the ability to bond with certain adenosine receptors in the body, typically the A1 receptors, which are found in the cardiac cells, and smooth muscle cells. Stimulation of these receptors causes inhibition of certain nerve cells as well as regulation of coronary oxygen consumption and blood flow. The stimulation of the A1 receptors also has an antiarrhythmic effect that causes a decrease in cardiac electrical conduction, suppression of the pacemaker activity in the SA node, and a decrease in overall heart rate.

As an antiarrhythmic drug, adenosine is most often used in the treatment of supraventricular tachyarrhythmias, including SVT, although it is less likely to be used for management of atrial fibrillation or atrial flutter. Adenosine has a very short half-life, so it is only administered intravenously and is given as rapid IV push. A typical dose is approximately 6 mg IV, given as a bolus, and then repeated at 12 mg IV push two more times, as needed. Adenosine has an approximate half-life of 10 seconds, so an IV push is often followed with a saline flush. Despite its short half-life, adenosine works rapidly in the body, often producing effects in less than one minute after administration.2,9

Digoxin has various uses in the management of cardiac abnormalities; it may be administered for control of cardiac arrhythmias but it is not classified in one of the four main categories of the Vaughan-Williams classification system. As an antiarrhythmic agent, digoxin is typically administered for the treatment of supraventricular arrhythmias, including atrial fibrillation and flutter, as well as SVT. It works by decreasing the rate of conduction and
increasing the refractory period in the AV node.\textsuperscript{10} It may be administered as an oral or an intravenous preparation; the doses vary greatly depending on the patient’s age. Because digoxin slows the heart rate after administration, prior to giving the drug, the nurse must check the patient’s apical pulse to assess that the rate is not below 60 beats/minute. There is also the potential for digitalis toxicity, in which there is a build up of the medication in the body, which causes serious effects, such as nausea, vomiting, visual disturbances, and altered mental status.

\textbf{Anticoagulants And Blood Clots}

In addition to the deleterious effects caused by cardiac arrhythmias, an abnormal heart rate can also lead to other problems and complications, including an increased risk of stroke, heart failure, or sudden cardiac death. For women at higher risk of these outcomes because of cardiac rhythm abnormalities, preventive medications may be required in addition to the drugs given to specifically treat the abnormal cardiac rhythm. Medications prescribed to prevent complications of cardiac arrhythmias often include anticoagulant drugs, antihypertensives, and diuretic medications to control symptoms of heart failure. Additionally, some of the drugs already mentioned that work as antiarrhythmic medications are also able to control heart rate, cardiac workload, and oxygen consumption of the heart, which places less stress and demand on the heart as well.

Because cardiac arrhythmias cause an alteration in the rate and rhythm of how the heart beats, the resulting blood flow may also be altered through the cardiovascular system. Instead of blood flowing smoothly through the chambers of the heart and into circulation, blood flow may become sluggish and it may pool in some areas, leading to an increased risk of thickening and blood clot formation. For example, in cases of atrial fibrillation, blood clots
can form in the chambers of the heart; if a clot moves through circulation as an embolus, it could eventually cause a stroke or heart attack if it lodges in a blood vessel and cuts off circulation. Those patients with atrial fibrillation who do not take any medication to prevent clot formation have an approximate 5 percent risk of stroke per year.11

The increased risk of blood clots with arrhythmia conditions often then requires administration of anticoagulant drugs to reduce risk. The main drugs prescribed in these cases are aspirin, clopidogrel, and warfarin. Aspirin, as previously discussed, is an antiplatelet agent that prevents clots from forming, particularly in cases of cardiac arrhythmias where a patient would be more likely to develop blood clots. Clopidogrel is also an antiplatelet drug that is sometimes called a “blood thinner.” This type of medication does not actually change the consistency of the blood, but it does prevent blood clots from forming and from their potentially negative consequences, such as stroke or heart attack.

Warfarin is one of the most common anticoagulant drugs administered to prevent blood clots due to cardiac arrhythmias. Warfarin can be taken orally and it can be prescribed on a long-term basis for affected patients to take at home. It is considered to be more effective in the prevention of blood clots when compared to using aspirin alone, however, warfarin also increases the potential for bleeding because of its stronger effects. A study by Gutierrez, et al., in the journal American Family Physician noted that warfarin can reduce the risk of stroke in high-risk patients by 68 percent, while aspirin reduces the risk of stroke by 21 percent.11

A patient who takes warfarin must have periodic laboratory testing done to confirm rates of blood clotting, which can be slowed because of the
medication. Blood laboratory testing must therefore be routinely performed to ensure that warfarin has reached therapeutic levels in the body. It also has the potential to cause other problems, despite its clotting prevention abilities. Some patients who routinely take warfarin must also monitor food intake because of the higher risk of drug-nutrient interactions, and any unintentional injury or even a planned surgery needs to be carefully monitored for an increased risk of serious bleeding in those who take the drug.

Nevertheless, warfarin is beneficial in protecting some patients with cardiac arrhythmias who may otherwise be at higher risk of blood clot formation. Warfarin reduces instances of blood clots by inhibiting the body’s ability to use vitamin K in the liver, which normally produces blood-clotting proteins. Most patients who take it must have testing of prothrombin time (PT) approximately once per week; the PT time measures certain clotting factors within the blood to determine the rate at which blood clots. Additionally, those who take warfarin often require further education about how to protect themselves from unintentional injury that could cause severe bleeding, what signs or symptoms to look for that could indicate hemorrhage, what foods interact with warfarin that could lead to increased bleeding, and how to notify others about warfarin use and the potential for bleeding tendencies.

Women with atrial fibrillation are at higher risk of having thromboembolic events when compared to men. Consequently, women should be screened for their risk factors and should be prescribed appropriate anticoagulant therapy as needed, whether it is in the form of aspirin or a stronger formulation, such as warfarin.
Hypertension and Cardiac Arrhythmia

Hypertension is also commonly associated with cardiac arrhythmias. Persons with high blood pressure are at higher risk of developing supraventricular and ventricular tachyarrhythmias. The reasons for the connection between hypertension and cardiac arrhythmias are numerous; in some cases, high blood pressure stretches the fibers of the cardiac chambers, which causes changes in the electrical conduction system. The effects of blood pressure on circulation can also lead to changes in electrolyte levels which could lead to cardiac arrhythmias as well, such as in cases of hyperkalemia. As a result, patients who have cardiac arrhythmias are more likely to suffer from comorbidities associated with hypertension and may need to take medications to control blood pressure levels.

There are numerous forms of antihypertensive drugs. Choosing the most appropriate drug for a woman with heart disease can be challenging, particularly because hypertension often does not cause any symptoms and in some cases, cardiac arrhythmias may be asymptomatic. Antihypertensive medications work by either reducing the amount of blood volume within the cardiovascular system, which decreases the amount of pressure against the interior lumen of the arteries, or by reducing systemic vascular resistance, which is the resistance in the vascular system that the heart must pump against to pump blood into circulation. Some drugs work to reduce vascular resistance by increasing the overall size of the arteries through vasodilation. These drugs are known as vasodilators.

There are a number of different drugs classified as vasodilators that can be used to treat hypertension, whether or not it is already associated with a cardiac arrhythmia. Some drugs, such as certain calcium channel blockers, are already used in the treatment of cardiac arrhythmias, including
verapamil and diltiazem, which have been previously discussed. Other types of calcium channel blockers that may be prescribed for management of hypertension include amlodipine, nicardipine, and nifedipine.\textsuperscript{14}

Other vasodilators that may be used to control hypertension can be classified into different categories, including angiotensin-converting enzyme (ACE) inhibitors, alpha-adrenoceptor antagonists, angiotensin receptor blockers, and nitrodilators. These drugs have various mechanisms of action but they all produce vasodilation. For instance, ACE inhibitors are designed to inhibit the vasoconstrictor angiotensin II. The kidneys secrete renin, which acts on the protein angiotensinogen, which ultimately forms angiotensin I. The angiotensin-converting enzyme converts angiotensin I to angiotensin II, which exhibits its vasoconstrictive effects. By blocking this enzyme, the body does not experience the response of the vasoconstriction and the blood vessels remain dilated, which reduces blood pressure levels. Examples of ACE inhibitors include captopril, lisinopril, and enalapril.

Alpha-adrenoceptor antagonists, also called alpha-receptor blockers, bind to alpha adrenoceptors in the smooth muscles, including those in the vascular system. By binding to these sites, alpha-blockers prevent the normal contraction of the muscle tissue that would otherwise lead to vasoconstriction. Their blocking action then prevents constriction, which keeps the vessels dilated and prevents high blood pressure. Some examples of these drugs include prazosin, terazosin, and doxazosin.

Angiotensin receptor blockers, including losartan, valsartan, and irbesartan, exert their effects by blocking type 1 angiotensin II receptors on the blood vessels. These receptors also impact contraction of the smooth muscles of the blood vessels, which would normally lead to an increase in blood
pressure. By inhibiting these receptors, the blood vessels are less likely to constrict and blood pressure may remain within a normal range.

Nitrodilators cause arterial and venous dilation, which has a number of effects on the cardiovascular system, including a decrease in blood pressure levels, a reduction in preload and afterload, a reduction in cardiac oxygen demands, and an increase in oxygen delivery to the coronary vessels. Examples of nitrodilator medications that may be used to manage hypertension include nitroglycerin, sodium nitroprusside, isosorbide dinitrate, and isosorbide mononitrate.

**Antihypertensive Medication and Heart Failure**

Many of the antihypertensive medications included as treatment of high blood pressure or to prevent complications of arrhythmia may also be used as preventive measures for other cardiac complications, such as heart failure. Cardiac arrhythmias and heart failure have a contrary relationship in that prolonged arrhythmias may lead to heart failure, but development of heart failure that is poorly managed could also lead to cardiac arrhythmias. It is therefore necessary to consider the potential for each condition when treating the other. Heart failure develops when the heart is unable to effectively pump enough blood to perfuse the organs and tissues in the body. At times, it may also involve inadequate filling of the heart’s chambers with blood, often because they have become damaged or stretched beyond their normal size.

More and more people with heart disease are being diagnosed with heart failure because the condition can develop as a complication of other cardiac conditions. When a woman with heart failure seeks care through medication, the clinician should consider the potential for development of cardiac
arrhythmias, some of which could become life threatening. Over time, heart failure affects the structure of the muscle fibers of the heart and they become stretched; additionally, there may be scar formation and the cardiac chambers can become enlarged and dilated, all of which contribute to changes in the rate and rhythm of the heart’s electrical conduction system. Patients with heart failure may develop many different types of cardiac arrhythmias, which can range from bradycardia to sustained ventricular tachycardia.\(^1\)

Alternatively, a patient with a prolonged cardiac arrhythmia may be at greater risk of developing heart failure. This is more commonly seen in conditions where an underlying arrhythmia goes for a period without being properly treated because it usually produces few symptoms. An example is with atrial fibrillation; a patient with this condition may develop symptoms on occasion that eventually resolve on their own so that adequate treatment may not be pursued. Over time, the heart can become damaged and blood flow disrupted; and, the patient could eventually develop heart failure without proper treatment. Fortunately, managing cardiac arrhythmias through medications is often enough to prevent the development of heart failure in many situations.

To prevent heart failure, or to manage the condition when it occurs so that it does not lead to further arrhythmia development, certain medications are necessary. Although these drugs do not undo the damage to the structure of the heart caused by heart failure, they can prevent many complications and they are often effective in improving the patient’s quality of life. As stated, many of the drugs used to control other cardiac conditions, such as hypertension, can be used to effectively control symptoms of heart failure as well.
Nitrodiiler medications, for example, are able to cause vasodilation to reduce the potential for high blood pressure, but their vasodilation also increases blood flow, which can improve cardiac output and can decrease potential complications of heart failure. Isosorbide dinitrate, when combined with hydralazine, may be beneficial in the management of heart failure, particularly within high-risk groups. A study in the *Journal of the American Heart Association* looked at the effects of combination hydralazine-isosorbide dinitrate therapy among African American patients who had low-ejection fraction states associated with heart failure. The study reviewed approximately 40,000 patients with heart failure and although the American College of Cardiology/American Heart Association guidelines recommend the use of isosorbide dinitrate in the treatment of heart failure, fewer than 15 percent of persons in the study were given a prescription for its use.\(^{15}\)

Although further research is needed regarding the benefits of nitrodiiler medication use in heart failure, particularly among women in high-risk groups, this type of medication has been shown to be beneficial in heart failure treatment and could be implemented even more as part of therapy.

Other drugs often included in the treatment of heart failure are ACE inhibitors, whose vasodilator properties improve blood flow to the heart and reduce the cardiac workload; and angiotensin-receptor blockers, whose antagonistic actions against angiotensin receptors also decrease blood vessel contraction and control blood pressure levels. Angiotensin-receptor blockers are sometimes used among people who otherwise cannot tolerate ACE inhibitors, such as when these drugs cause chronic cough or other side effects not associated with angiotensin-receptor blockers.

Beta-blockers, which are sometimes prescribed for the treatment of cardiac arrhythmias and hypertension, may also be included with the management
of heart failure. By blocking beta-adrenergic receptors in the heart’s tissues, these drugs can decrease overall heart rate and reduce blood pressure. Beta-blockers have the added advantage of improving the heart’s pumping ability, so they can improve cardiac output over time. The three types of beta-blockers currently approved by the FDA for the treatment of heart failure include bisoprolol, carvedilol, and metoprolol.  

Diuretics are medications that may be prescribed for different uses, including management of heart failure and hypertension. Diuretics differ slightly in their mechanisms of action, but they all result in the release of excess fluid from the body through the urine. It is for this reason that some patients refer to them as “water pills.” In cases of heart failure, the heart is not able to effectively pump blood to meet the rest of the body’s demands; consequently, blood and excess fluid may back up with the chambers of the heart and in the circulatory system. The affected person may be more likely to have problems with hypertension and may also suffer from other manifestations such as edema in the extremities, fatigue, weight changes, and dyspnea. Diuretic medications are therefore important to remove some of the excess fluid within circulation, which can decrease the severity of symptoms.

Diuretics used to treat heart failure or hypertension is typically classified as one of three different types, known as loop diuretics, thiazide diuretics, and potassium-sparing diuretics. Some are more potent than others and can quickly remove excess water from the body, but they also may place the patient at higher risk of electrolyte imbalances if too many nutrients are excreted at one time. Loop diuretics work within the loop of Henle in the kidney to excrete excess sodium and water from the body; however, they
may also cause a decrease in other important nutrients as well. Examples include furosemide and bumetanide.

Thiazide diuretics prevent excess sodium from being absorbed back into circulation, consequently, extra sodium and water are then excreted through the urine. Some types of thiazide diuretics include metolazone and hydrochlorothiazide (HCTZ). These drugs are not as potent as some other diuretics, so they may be combined with other drugs such as loop diuretics to achieve the full effects. Potassium-sparing diuretics may also be prescribed in combination with other types of diuretics, particularly because they do not allow for the excretion of excess potassium from the body. Examples of potassium-sparing diuretics include spironolactone and eplerenone.¹⁸

When administering diuretic therapy for patients with heart failure or hypertension, it is important to consider the effects of excess fluid loss within the body. While too much fluid can lead to high blood pressure, edema, and other complications associated with these conditions, the body requires the right balance of fluid because loss of too much through diuretic use can lead to dehydration, electrolyte imbalances, fatigue, dizziness, and even cardiac arrhythmias.

Women who are pregnant and develop high blood pressure need to be especially careful with diuretic medications, as the increased fluid loss from their use could be detrimental. However, studies have shown that most diuretics are not teratogenic, which means they do not cause birth defects in the growing fetus. Because hypertension is a relatively common complication of pregnancy, some women may be prescribed diuretic medications to control their blood pressure during this time. A meta-analysis by Al-Balas, et
In the journal *Canadian Family Physician* showed that diuretics can be safely prescribed for use during pregnancy; although the drugs impact overall plasma volume, this outcome is otherwise not associated with negative effects on the fetus. Women who experience hypertension prior to becoming pregnant or those who develop high blood pressure as a consequence of pregnancy should be able to safely take diuretic medications for fluid volume management.

### Management Of Heart Defects

The type of medication prescribed for management of heart defects often depends on the type of defect present. If a heart defect causes few to no symptoms or does not impact quality of life, medication may not be needed. Alternatively, some drugs are administered to control symptoms or complications that occur because of the defect, such as problems with circulation, fatigue, decreased oxygenation of tissues, or fluid buildup and edema. Medications used for the management of heart defects include those that increase the strength of the heart’s contractions, drugs that control the rate and rhythm of the heart and that prevent cardiac arrhythmias, drugs used to prevent blood clots, medications to release excess fluid in the cardiovascular system, and medications that prevent infection.

Drugs that decrease the oxygen demands of the heart and the overall workload of the heart can include ACE inhibitors, beta-blockers, calcium channel blockers, and digitalis. As described, these drugs work in a variety of methods, whether by blocking the effects of certain receptors within the cardiac tissue to slow the heart rate and lower blood pressure, or by inhibiting vasoconstriction and reducing systemic vascular resistance so that the heart does not have to pump so forcefully. Drugs such as ACE inhibitors and beta-blockers that have vasodilator properties affect the size and
diameter of the coronary arteries in addition to other blood vessels in the body. By improving blood flow through the coronary arteries, these drugs also allow for more oxygen and blood to reach the heart, which helps it to beat more efficiently.

Digitalis may also be used as a drug that increases the force of the heart’s contractions. The drug is used to improve the heart’s pumping ability and to regulate the heart rate to improve blood flow. Depending on the type of heart defect present, a drug such as digitalis may be administered to improve cardiac function. Many heart defects that are seen in adulthood cause symptoms that are similar to heart failure. For example, if a woman was born with an atrial septal defect, which is a hole in the septum between the two atria, and the condition was never corrected as a child, she may have grown into an adult with the ongoing defect. Eventually, the mixing of blood between the two atria could cause problems with proper oxygenation in the blood and part of the heart could become enlarged, leading to symptoms of heart failure. Although the defect is most commonly treated through surgical intervention, if symptoms of heart failure have developed, the patient may then need to take drugs to prevent complications of poor cardiac output, such as through the drugs previously prescribed.

Some heart defects can impact the heart’s rate and rhythm and can lead to cardiac arrhythmias; this may be more likely to occur in cases in which the patient has a defect that causes symptoms that are similar to congestive heart failure, as heart failure is more likely to cause complications of arrhythmias. The patient may need medication to prevent dysrhythmia from developing in the first place or she may need to take the drugs in addition to other medications for a heart defect if an arrhythmia is already present.
Medications administered for the management of cardiac arrhythmias are classified as described according to the Vaughan-Williams classification system (described above), which basically delineates the drugs into sodium channel blockers, beta blockers, potassium channel blockers, and calcium channel blockers, in addition to some other types of drugs that also possess antiarrhythmic effects but that do not necessarily fall into these categories. The type of medication prescribed depends on the type of arrhythmia the patient is experiencing, such as whether she has symptoms of a supraventricular or ventricular arrhythmia. Additionally, some drugs may not be prescribed unless the patient experiences ongoing symptoms of an arrhythmia, the arrhythmia is otherwise not well controlled with other types of therapeutic interventions, the cardiac condition is causing other complications for the patient, or the patient has a decreased quality of life because of the arrhythmia. As an example, a patient who has lived with a heart defect may have developed signs and symptoms similar to heart failure because of the effects of the defect over time. The damage to the heart also may cause electrical disturbances in the cardiac conduction system, leading to changes in the firing of the SA node and eventual atrial fibrillation. In this case, the patient may need medications such as amiodarone or ibutilide to control the arrhythmia, particularly if it causes unpleasant symptoms and increases the risk of blood clots.

As described, anticoagulants are also drugs that may be prescribed for prevention of blood clots. These drugs may be necessary for some patients with heart defects who are at increased risk of blood clot formation. This most commonly occurs in situations in which a patient has a defect that increases the risk for an arrhythmia, which further increases the chances of clot formation. Anticoagulant drugs may also be prescribed in situations where a heart defect causes malformations of the cardiac valves. When
heart valves are not functioning properly, there is a higher risk of blood clot formation on the valves, which further increases the patient’s risk of stroke or heart attack. Persons who undergo valve replacement as part of treatment for congenital heart disease or another form of valve disorder also often require prophylactic anticoagulant drugs, such as with warfarin or aspirin.\textsuperscript{20}

When excess fluid develops because of an increase in circulatory volume, the patient may need diuretic medications to excrete extra fluid through the urine. When the heart does not pump properly, the patient can experience an excess of fluid volume in the circulatory system. When this happens, the patient is more likely to develop symptoms similar to heart failure, including edema, weakness, fatigue, and shortness of breath. As an example, when a septal defect is present, there is abnormal blood flow through the chambers of the heart and some areas may become enlarged, causing the heart to pump blood abnormally. Eventually, symptoms of heart failure may develop and excess fluid remains within circulation, which could cause swelling in the extremities or fluid buildup in the lungs, making it more difficult for the patient to breathe or causing a wet cough. Administration of diuretic medications, such as loop, thiazide, or potassium-sparing diuretics can help to rid the body of excess fluid through the urine, which eventually can help to decrease overall fluid volume and can relieve some symptoms. This decrease in fluid volume also decreases the workload of the heart, so that it does not need to pump as forcefully to move blood through circulation.

**Cardiomyopathy Treatment**

When an individual develops cardiomyopathy, medication may be needed as part of treatment, depending on the specific symptoms involved and the extent to which symptoms affect the patient’s quality of life. As the heart
becomes more enlarged with cardiomyopathy, its overall function diminishes. Therefore, drugs used as part of treatment are often aimed at increasing the pumping action of the heart, assisting the heart with pumping enough blood to adequately perfuse the tissues and organs, and preventing complications of cardiomyopathy. The type of cardiomyopathy present also impacts the type of medication prescribed; for example, a person with restrictive cardiomyopathy may suffer from symptoms of heart failure and may need medications to control excess circulatory volume, while another person with dilated cardiomyopathy may be more likely to suffer from cardiac arrhythmias and may need drugs to regulate the heart rate and to prevent blood clots.

Medications may also be prescribed depending on patient symptoms and how much they impact quality of life. A patient with alterations in left ventricular function because of cardiomyopathy may experience fatigue, shortness of breath, and excess fluid. Consequently, the drugs prescribed are often based on the pathophysiology of the disease process and how the condition is manifested.

Beta blockers are some of the more commonly prescribed drugs used for management of cardiomyopathy. Because they work to resolve a number of symptoms, including cardiac arrhythmias, as well as angina or dyspnea, beta-blockers are often a first choice of drug to improve heart rate and rhythm, to improve cardiac contractility, and to decrease cardiac excitability. In a review in the *European Heart Journal* that looked at the use of beta blockers in patients with hypertrophic cardiomyopathy, sotalol and nadolol were two of the more commonly used agents for treatment of this condition, particular when left ventricular outlet obstruction was present. Current
guidelines recommend beta-blockers as a first-line choice of medication for treatment of hypertrophic cardiomyopathy.\textsuperscript{21}

Other drugs that may also be included in cardiomyopathy management include calcium channel blockers, such as verapamil and diltiazem. These drugs may decrease the heart rate, which allows for a greater period of filling time in the ventricles, which can lead to improved cardiac output. Angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers, diuretics, and digoxin may also be prescribed for management of cardiomyopathy symptoms. The type and amount of each of these drugs varies depending on the type of cardiomyopathy present and the patient’s overall condition and symptoms.

\textbf{Treatment Of Heart Infections}

The mainstay of prevention or treatment of cardiac infections, particularly endocarditis, is administration of antimicrobial therapy to control microorganism levels and to prevent further complications of the infective process. Infectious endocarditis occurs when bacteria or fungi collect on the interior layers of the heart; the risk of infection is increased in areas where there is more surface area for the microorganisms to grow, such as in cases of prosthetic heart valves or areas of coagulation.

Valve replacement, through surgical intervention for the management of heart valve disease, is one of the most common causes of endocarditis, but the condition is also a risk among people who have congenital or structural heart disease, those who are intravenous drug users, people with a history of rheumatic heart disease, or people who have recently undergone a high-risk, invasive procedure.\textsuperscript{22}
The type of antimicrobials administered for treatment of infective endocarditis depends on the infectious organism and whether the patient has a prosthetic heart valve. The American Heart Association has provided a summary of recommended antibiotics to be given for patients with specific strains of organisms causing their infections. The patient requires blood cultures and testing to identify the type of microorganism causing the infection and its sensitivity to certain antibiotics in order to be able to provide effective treatment.

Pierce, et al., in American Family Physician offers a listing of recommended treatments for specific infections according to the American Heart Association guidelines. The results are listed in Table 1 below.22

<table>
<thead>
<tr>
<th>MICROORGANISM</th>
<th>PARENTERAL ANTIBIOTIC REGIMEN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin-susceptible viridans <em>Streptococcus</em> or <em>Streptococcus bovis</em></td>
<td>Penicillin G or ceftriaxone (Rocephin) for four weeks</td>
</tr>
<tr>
<td></td>
<td>Or</td>
</tr>
<tr>
<td></td>
<td>Penicillin G plus gentamicin for two weeks</td>
</tr>
<tr>
<td></td>
<td>Or</td>
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<tr>
<td></td>
<td>Ceftriaxone plus gentamicin for two weeks</td>
</tr>
<tr>
<td></td>
<td>Or</td>
</tr>
<tr>
<td></td>
<td>Vancomycin for four weeks</td>
</tr>
<tr>
<td>Relatively penicillin-resistant viridans <em>Streptococcus</em> or <em>S. bovis</em></td>
<td>Penicillin G or ceftriaxone for four weeks, plus gentamicin for two weeks</td>
</tr>
<tr>
<td></td>
<td>Or</td>
</tr>
<tr>
<td></td>
<td>Vancomycin for four weeks</td>
</tr>
<tr>
<td>Penicillin-resistant viridans <em>Streptococcus</em> or <em>S. bovis</em></td>
<td>Ampicillin plus gentamicin for four to six weeks</td>
</tr>
<tr>
<td><strong>Oxacillin-susceptible staphylococci</strong></td>
<td><strong>Oxacillin-resistant staphylococci</strong></td>
</tr>
<tr>
<td>----------------------------------------</td>
<td>-------------------------------------</td>
</tr>
<tr>
<td>Or Penicillin G plus gentamicin for four to six weeks</td>
<td>Or Vancomycin for six weeks</td>
</tr>
<tr>
<td>Or Nafcillin or oxacillin for six weeks, plus gentamicin for three to five days (optional)</td>
<td>Or Cefazolin for six weeks, plus gentamicin for three to five days (optional)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Enterococcus strains susceptible to penicillin, gentamicin, and vancomycin</strong></th>
<th><strong>Enterococcus strains resistant to penicillin, but susceptible to aminoglycosides and vancomycin</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin plus gentamicin for four to six weeks</td>
<td>Ampicillin/sulbactam (Unasyn) plus gentamicin for a minimum of six weeks</td>
</tr>
<tr>
<td>Or Penicillin plus gentamicin for four to six weeks</td>
<td>Or Vancomycin plus gentamicin for six weeks</td>
</tr>
<tr>
<td>Or Vancomycin and gentamicin for six weeks</td>
<td>Or Vancomycin plus gentamicin for six weeks</td>
</tr>
</tbody>
</table>
Antimicrobial therapy should start as soon as a blood culture determines the presence of an infection. Once therapy has been initiated, the patient should have repeated cultures checked every 24 to 48 hours until there is no further growth and the infection has cleared.\(^{22}\)

In contrast to endocarditis, the inflammation associated with myocarditis is often caused by a viral infection or a post-viral immune mediated response.\(^{23}\) Because of this, treatment with antibiotics is typically not necessary, nor is it effective. Instead, treatment is aimed at controlling symptoms, which are often similar to those seen with heart failure. In some cases, the exact cause of the virus causing myocarditis may be identified, however most treatment for this condition focuses on management of heart failure symptoms and prevention of complications, including progression to dilated cardiomyopathy.

Treatment of most cases of myocarditis involves prescription ACE inhibitors or angiotensin receptor blockers to improve left ventricular function and to prevent progression of cardiomyopathy. Beta-blockers have also been used to improve cardiac function and they may play a role in reducing inflammation associated with the infection. Studies have shown that using beta blockers for the treatment of myocarditis can not only improve ventricular function but can also reduce hospitalizations for worsening symptoms of heart failure.\(^{23}\) Other medications and treatments used for management of myocarditis depend on the causative agent. For example, an autoimmune process causes some cases in which the body attacks its own tissues to cause the inflammation. When this occurs, the myocarditis might be treated with immunosuppressants such as corticosteroids or cyclosporine, in addition to therapies required for management of heart failure symptoms.
Pericarditis, which develops as inflammation of the pericardial sac surrounding the heart, can cause serious complications that can restrict the heart’s ability to pump effectively; the condition can lead to pericardial effusion or cardiac tamponade in some cases. In most cases, pericarditis occurs because of a viral infection, such as following a respiratory infection, although it can also be caused by bacterial or fungal infections as well, although these are less likely. The medications prescribed for management of pericarditis are then focused on treating the underlying cause.

In many cases of pericarditis, unless the patient has developed serious complications such as pericardial effusion or she has a condition that would cause immunosuppression, the exact identification of the causative virus is unknown and is not necessarily tested through specimen culture. Instead, treatment is aimed at reducing inflammation through use of non-steroidal anti-inflammatory agents (NSAIDs) or other anti-inflammatory drugs, including colchicine.

For those patients with myocarditis that has not led to other complications, standard treatment through NSAIDs can reduce inflammation and can control pain associated with the condition. NSAIDs work by inhibiting COX-1 and COX-2 enzymes that are responsible for production of prostaglandins that produce inflammation and pain. By blocking the COX enzymes, there is less inflammation and the patient is less likely to experience pain or fever associated with the condition. A patient with pericarditis may be prescribed NSAID therapy until symptoms of the condition have resolved and there are no complications. In some cases, continued laboratory analysis by checking for inflammation, such as through C-reactive protein levels, may be necessary throughout treatment.
The most common NSAIDs used in management of pericarditis include ibuprofen, aspirin, and indomethacin.\textsuperscript{24} Ibuprofen is effective in treating inflammation and pain; it is available over the counter, although larger doses may be administered through prescription. A typical regimen of ibuprofen to relieve inflammation and fever associated with myocarditis is approximately 600 to 800 mg, three times per day as oral doses. Alternatively, aspirin may be used for treatment because of its anti-inflammatory properties. Although aspirin is used as an anti-platelet regimen for some types of heart disease, it is also effective in treating inflammation. Aspirin is also available without a prescription and it may be given at doses of 650 to 1000 mg orally, up to three times daily.\textsuperscript{24}

Indomethacin, another type of anti-inflammatory drug, is sometimes used to treat pain and inflammation associated with arthritis or gout. It is available by prescription but should not be used with other drugs that have NSAID properties that can be purchased over the counter. Indomethacin doses of 50 mg orally, three times per day, have been shown to be beneficial in the management of pain and inflammation associated with pericarditis. Ibuprofen, aspirin, and indomethacin all can cause possible side effects associated with gastrointestinal bleeding, particularly if the patient is also taking anticoagulant therapy to protect against blood clots. When administering these drugs for pericarditis treatment, it is important to determine what other medications the patient is taking to best protect against gastrointestinal irritation and bleeding among those who are most susceptible.

Colchicine is a drug that may also be used as part of the treatment for pericarditis; it is a type of anti-inflammatory drug that is normally used in the treatment of gouty arthritis, although some studies have indicated that it
should be used as a first-line treatment for acute pericarditis as well, since it is safe to use and has relatively few side effects. Colchicine has been shown to reduce inflammation and pain associated with acute pericarditis and in cases of pericarditis that have otherwise been refractory to conventional forms of treatment with other NSAIDs. The exact dose may vary but an example dose of the drug that has been successful for treatment is 0.5 mg given orally twice per day. It should be noted that this drug, while effective, is only used in an off-label method for recurring cases of pericarditis, as it is not technically approved for use in those particular situations.

**Heart Valve Disease**

Heart valve disease can develop as a consequence of illness or infection or it may be the result of a congenital heart condition that is present at birth. Symptoms and complications of heart valve disease depend on which valves are affected as well as the presence of any other underlying conditions. Thus, treatment also is aimed at controlling symptoms associated with the affected heart valves and managing the effects that valve disease can have on circulation.

When valve stenosis is present, the affected heart valve becomes thickened and does not function properly; in many cases, surgery is the main option for treatment, in which the patient would receive a prosthetic valve to replace the stenotic one. However, there are many patients who cannot tolerate this type of surgical procedure and who otherwise require medical management of valve stenosis through pharmacologic therapy. In cases of aortic valve stenosis, there are no medications that will treat the disease or reverse the damage caused by the stenotic valves. However, other medications may be beneficial in management of symptoms caused by the stenosis, including vasodilators, statin therapy, and prophylactic antibiotics.
Use of statins was at one time recommended for the management of aortic stenosis, as it was thought that these drugs might be able to slow the progression of the disease. Statins, which are drugs taken for hypercholesterolemia because they reduce levels of cholesterol and triglycerides in the blood, can prevent heart attack or stroke when prescribed for certain patients who have high cholesterol. However, later studies have shown that statins are not necessarily effective in reducing the effects of aortic stenosis and their treatment is not recommended solely for this type of therapy, unless the patient already has hypercholesterolemia for which they would be used instead.26

Prophylactic antibiotics, such as those administered prior to dental procedures, were at one time used in patients with valve stenosis to prevent infections that could have come from through the procedure. However, studies have shown that many patients with valve disease were not necessarily benefitting from prophylactic antibiotics prior to dental procedures and, in fact, some people were at increased risk of infections with drug-resistant bacteria.27 Some patients with artificial heart valves placed because of stenotic disease still benefit from prophylactic antibiotics prior to dental procedures. Although, it is these patients, along with those who suffer from very serious congenital heart diseases, who should be treated with antibiotics for a brief period when a dental procedure is scheduled in order to reduce the risk of the spread of bacteria to the bloodstream where it can eventually infect the heart.

Heart valve regurgitation, also called valve insufficiency, is one of the more common heart valve disorders in which blood leaks backward through the valve, which ultimately reduces the amount of blood flow being pumped through the heart. In some cases, if the leak is small, it causes few
symptoms for the patient and does not require treatment. However, the condition can lead to cardiac arrhythmias, increased pulmonary artery pressures, and left ventricular failure. One of the more common types of regurgitation is associated with the mitral valve. This occurs when some blood leaks backward from the ventricle into the atrium as it is being pumped through. The management of mitral valve regurgitation is very similar to that for heart failure, as the continued symptoms of the condition can lead to much of the same damage that occurs with heart failure. When asymptomatic, mitral valve regurgitation often does not require medications for treatment. When symptoms of heart failure develop, the patient may then benefit from medications that decrease the amount of afterload, which is the pressure the heart must pump against. This puts less pressure on the heart and decreases the risk of further complications.

Medications often prescribed for symptomatic valve regurgitation include ACE inhibitors, angiotensin-receptor blockers, and beta blockers. As described, these drugs can help the heart to pump more efficiently and can reduce the stress and workload of the heart. In some cases, the patient may also have fluid build up because of poor blood flow; when this occurs, diuretic medications to rid the body of excess fluid and to control edema are also helpful. The disease process also increases the patient’s risk for atrial fibrillation due to damage to the atria. This may further increase the need for medication for this condition to include antiarrhythmia medications, as well as anticoagulant drugs to prevent blood clots that are often associated with atrial fibrillation.

Many patients can live comfortably with medications used to control valve insufficiency, although no medication will correct the defect and cause the valve to function properly again. In severe cases, surgery is often
warranted, although many people are unable to tolerate surgery, particularly if their symptoms have progressed. In these cases, further discussion is needed to determine the benefits of pharmacological management for heart disease or whether the patient needs to pursue other options for treatment, such as through lifestyle changes or surgical intervention.

**Summary**

Unique differences exist in the pharmacological approaches and potential adverse reactions to medication management in women. It is generally accepted that gender differences in heart health and disease progression exist, although there continues to be division in the literature as the research evolves and disparity related to women’s heart health as compared to men continues to be debated. Health treatment teams need to be informed and fully aware of the differences in women’s heart health with conventional treatment options, specifically as it related to how perimenopause and menopause affect women’s heart health. This article has reviewed the specific medications used for the treatment of cardiac conditions in women. Helpful tables of medication classifications and discussion related to guidelines published by the American Heart Association have been included with regard to recommended treatments for the specific conditions presented.

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1. **Drugs categorized as Class II are known as those that**
   a. block calcium entry into cells.
   b. decrease cardiac automaticity, reduce contractility, and slow conduction velocity.
   c. include diltiazem and verapamil.
   d. block fast sodium channels.

2. **Digoxin may be administered for control of cardiac arrhythmias and**
   a. is not classified in one of the four main categories of the Vaughan-Williams classification system.
   b. is classified in one of the four main categories of the Vaughan-Williams classification system.
   c. is not considered an antiarrhythmic agent.
   d. works by increasing the rate of cardiac conduction.

3. **True or False. Chemical cardioversion is done through administration of medication as an emergency procedure, particularly when the patient’s symptoms are causing hemodynamic instability.**
   a. True.
   b. False.

4. **Isosorbide dinitrate combined with hydralazine**
   a. may be beneficial in the management of heart failure.
   b. may be particularly useful within high-risk groups.
   c. is recommended by the American College of Cardiology/American Heart Association guidelines for treatment of heart failure.
   d. All of the above.

5. **Infectious endocarditis occurs when**
   a. a virus attacks the lining of the heart.
   b. there is less surface area for microorganisms to grow.
   c. bacteria or fungi collect on the interior layers of the heart.
   d. Both a and b above.
6. Choosing the most appropriate antihypertensive drug for a woman with heart disease can be challenging because

   a. hypertension often does not cause any symptoms.
   b. cardiac arrhythmias may also be asymptomatic.
   c. Both a., and b.
   d. None of the above.

7. ________________ are used as antiarrhythmic agents even though they do not fit within the Vaughan-Williams classification system.

   a. Quinidine and procainamide
   b. Diltiazem and verapamil
   c. Adenosine and digoxin
   d. Flecainide and propafenone

8. Cardiac arrhythmias that occurs in the sinoatrial (SA) or atrioventricular (AV) nodes or in the atria are called

   a. severe tachyarrhythmias.
   b. supraventricular dysrhythmias.
   c. ventricular dysrhythmias.
   d. None of the above.

9. ________________ is usually only given in emergency situations, such as with life-threatening ventricular tachyarrhythmias that are unresponsive to other drugs.

   a. Flecainide
   b. Esmolol
   c. Propranolol
   d. Acebutolol

10. True or False: In the United States, flecainide is only available in oral form and is not available as an intravenous dose.

    a. True.
    b. False.
11. Beta-2 receptors are found in the smooth muscle tissue in the
   a. heart.
   b. atria.
   c. coronary vessels.
   d. lungs.

12. ________________ are prone to cause various side effects, which can range from bronchospasm, depression, and insomnia to heart block and bradycardia.
   a. Potassium channel blocking agents
   b. Beta-blockers
   c. Sodium channel blockers
   d. Antiarrhythmic agents

13. Use of digoxin has the potential of leading to digitalis toxicity, which can cause
   a. torsades de pointes.
   b. bronchospasm, depression, insomnia, heart block and bradycardia.
   c. nausea, vomiting, visual disturbances, and altered mental status.
   d. vasodilation.

14. Potassium channel blockers can be prescribed for
   a. supraventricular arrhythmias.
   b. ventricular arrhythmias.
   c. life-threatening situations.
   d. All of the above.

15. Sotalol is a combination agent that is technically classified as a
   a. class III antiarrhythmic drug.
   b. class II antiarrhythmic drug.
   c. class II/class III antiarrhythmic drug.
   d. class I/class II antiarrhythmic drug.

16. True or False: Studies have shown that most diuretics are not teratogenic, in other words, they do not cause birth defects in the growing fetus.
   a. True.
   b. False.
17. The sotalol agent, l-isomer type, is able to
   a. block potassium channels.
   b. block potassium channels and effect beta-adrenergic blocking.
   c. produce beta-adrenergic blocking effects.
   d. None of the above.

18. Amiodarone may be administered as an intravenous dose under what circumstances?
   a. Once the patient is stabilized
   b. Until the patient is stabilized
   c. Never
   d. At any time for patients who do not have renal disease

19. __________ may be used to partly treat pericarditis.
   a. Flecainide
   b. Esmolol
   c. Colchicine
   d. Digoxin

20. In cases of aortic valve stenosis, where surgery is not an option,
   a. medications may be used to manage the symptoms.
   b. medications may be used to treat the disease.
   c. medications may be used to reverse the disease.
   d. statins are used to slow the progress of the disease.

21. Some studies have indicated that __________ should be used as a first-line treatment for acute pericarditis since it is safe to use and has relatively few side effects.
   a. colchicine
   b. esmolol
   c. quinidine
   d. procainamide
22. When administering diuretic therapy for patients with heart failure or hypertension, excess fluid loss within the body may cause

   a. edema.
   b. high blood pressure.
   c. vasodilation.
   d. electrolyte imbalances.

23. ACE inhibitors have _____________ properties improve blood flow to the heart and reduce the cardiac workload.

   a. vasodilator
   b. diuretic
   c. angiotensin-receptor blocking
   d. constrictive

24. True or False: Women with atrial fibrillation are at higher risk of having thromboembolic events when compared to men.

   a. True
   b. False

25. Over time, heart failure affects the structure of the muscle fibers of the heart and they

   a. blocked.
   b. harden.
   c. shrink.
   d. become stretched.

Correct Answers:

References Section

The reference section of in-text citations include published works intended as helpful material for further reading. Unpublished works and personal communications are not included in this section, although may appear within the study text.


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