Chapter 3: AN OVERVIEW OF CARDIOVASCULAR NURSING
12 CONTACT HOURS

Learning objectives

- Define and discuss the most common types of cardiovascular disease.
- Discuss how to screen for cardiovascular disease.
- Discuss the ways to prevent cardiovascular disease.
- Discuss the treatment modalities of the various cardiovascular diseases.
- Describe the two most common kinds of stroke and common symptoms for stroke.
- Identify risk factors for stroke.
- List the steps used to diagnose stroke.
- Explain why timing is crucial in the treatment of strokes.
- Identify tPA and its use in treatment of acute ischemic stroke, as well as contraindications for its use.
- Describe key components of a nurse’s role in treatment of stroke patients.
- List the consensus stroke performance measures for stroke treatment developed through the Coverdell Registry program.

PART I: CARDIOVASCULAR DISEASES

Introduction

Cardiovascular disease is a broad term that is used interchangeably to describe “heart disease.” Cardiovascular diseases encompass many heart conditions that may overlap, such as coronary artery disease (CAD), hypertension (HTN), acute myocardial infarction (AMI or MI), congestive heart failure (CHF) coronary artery disease and arrhythmias. According to the World Health Organization (WHO), Centers for Disease Control and Prevention (CDC) and the American Heart Association (AHA) (2009), cardiovascular disease is the leading cause of death worldwide and a major cause of disability. It has actually been the leading cause of death since 1900 in the United States.

In 2003, the WHO reported that cardiovascular disease made up 16.7 million, or 29.2 percent of all global deaths. Of these deaths, 7.6 million were due to heart attacks and 5.7 million were due to a subset of the problem, cerebrovascular disease and cerebrovascular accident (CVA), or stroke, which will be addressed in Part II of this course. If current trends are allowed to continue, by 2015, an estimated 20 million people will die from cardiovascular disease (mainly from heart attacks and strokes). Cardiovascular disease kills more people than the next-most common causes of death combined, including cancer, chronic lower respiratory disease, accidents, diabetes, influenza and pneumonia. According to the heart association, in 2006, more than 80,000,000 people in the U.S. had one or more forms of cardiovascular disease. The most common forms of cardiovascular disease include hypertension, 73,600,000; coronary heart disease, 16,800,000; acute myocardial infarction, 7,900,000; angina pectoris (chest pain or discomfort caused by reduced blood supply to the heart muscle), 9,800,000; cerebrovascular accident, 6,500,000; and congestive heart failure, 5,700,000.

In 2009, the CDC estimated that 785,000 Americans would have a new coronary heart attack, and about 470,000 would have a recurrent attack. To put it into perspective, approximately every 25 seconds an individual would have a coronary event, and one person would die every minute.

Although cardiovascular disease is the leading cause of death, most times it is preventable by avoiding unhealthy habits, such as a high-fat diet, physical inactivity and smoking. Every day, nurses are responsible for patients and their families who are living with some form of cardiovascular disease. However, nurses are not immune either, and the daily stressful grind of their work can itself create a risk for nurses of developing cardiovascular disease or picking up bad habits during the long shifts, such as smoking and a high-fat diet. Nurses can use the information in this course in their personal lives as well as in their work caring for others to reduce the overall risk of cardiovascular disease in our nation.
Pathophysiology of cardiovascular diseases

The heart is a hollow, muscular organ that is responsible for continuously pumping an adequate supply of blood throughout the body to vital organs for survival. To reduce our risk of developing cardiovascular disease, it is important that we nurture our bodies with healthy, nutritious foods; exercise; and avoid harmful substances.

Cardiovascular diseases are mainly caused by a buildup of plaque (atherosclerosis) inside the coronary arteries. Over time, the plaque diminishes blood flow and oxygen to the heart, brain and other vital organs secondary to the inflammatory process. The extensive inflammation further exacerbates the ability of oxygenated blood to flow freely in the bloodstream, leading to a further buildup of plaque and an accumulation of blood clots. Blood clots accumulate when a blood vessel is stenosed secondary to the limited blood flow; therefore, it backs up into the previous chamber behind the valve. The pressure in the previous chamber will increase because of the resistance in the stenosed blood vessel. Consequently, the heart is forced to work harder, which results in hypertrophy (enlargement), especially left ventricular hypertrophy, which further increases the heart’s workload because of the resulting increased oxygen demands.

Once blood vessels are blocked by plaque or clots, they cannot supply blood to the heart and brain, which then become damaged and weakened and can lead to a myocardial infarction, congestive heart failure or arrhythmias. Although there are several types of plaque that may result in serious coronary events, retrospective analyses have demonstrated that 70 percent of all fatal acute myocardial infarctions and sudden coronary deaths are attributable to plaque rupture or erosion.

Although atherosclerosis is the predominant predictor of cardiovascular disease, there are other potential pathological rationales for each of the major specific cardiac diagnosis and significant overlapping in each of the most common heart conditions.

Coronary artery disease (CAD)

The most common type of heart disease, coronary artery disease (also called coronary heart disease, or CHD) is the primary cause of acute myocardial infarction secondary to atherosclerosis, and the leading cause of death in the United States for both men and women. Over time, CAD can weaken the heart muscle and lead to heart failure and arrhythmias.

What causes coronary artery disease?

Research suggests that coronary artery disease (CAD) starts when certain factors damage the inner layers of the coronary arteries. These factors include:

- Smoking.
- High amounts of certain fats and cholesterol in the blood.
- High blood pressure.
- High amounts of sugar in the blood caused by insulin resistance or diabetes.

When damage occurs, excess fatty tissues release compounds to promote the healing process. But this in turn causes plaque to build up where the arteries are damaged. The buildup of plaque in the coronary arteries may start in childhood. Over time, plaque can narrow or completely block the coronary arteries, which reduces the flow of oxygen-rich blood to the heart muscle. Plaque also can crack, which causes blood cells called platelets to clump together and form blood clots at the site of the cracks. This narrows the arteries more and worsens angina or causes a heart attack.

Risk factors for coronary artery disease

The major risk factors for CAD, and many other heart diseases, are:

- **Unhealthy blood cholesterol levels.** This includes high LDL cholesterol (sometimes called bad cholesterol) and low HDL cholesterol (sometimes called good cholesterol).
- **High blood pressure.** Blood pressure is considered high if it stays at or above 140/90 mm/Hg over a period of time.
- **Smoking.** This can damage and tighten blood vessels, raise cholesterol levels and raise blood pressure. Smoking also doesn’t allow enough oxygen to reach the body’s tissues.
- **Insulin resistance.** This condition occurs when the body can’t use its own insulin properly. Insulin is a hormone that helps move blood sugar into cells where it’s used.
- **Diabetes.** This is a disease in which the body’s blood sugar level is high because the body doesn’t make enough insulin or doesn’t use its insulin properly.

- **Being overweight or obese.** Being overweight is having extra body weight from muscle, bone, fat or water. Obesity is having a high amount of extra body fat.
- **Metabolic syndrome.** Metabolic syndrome is the name for a group of risk factors linked to being overweight or obese that raises the chance for heart disease and other health problems, such as diabetes and stroke.
- **Lack of physical activity.** Lack of activity can worsen other risk factors for CAD.
- **Age.** As people get older, the risk for CAD increases. Genetic or lifestyle factors cause plaque to build in the arteries as people age. By the time people are middle-aged or older, enough plaque has built up to cause signs or symptoms.
  - In men, the risk for CAD increases after age 45.
  - In women, the risk for CAD increases after age 55.
• **Family history of early heart disease.** The risk increases if a person’s father or a brother was diagnosed with CAD before 55 years of age, or if a mother or a sister was diagnosed with CAD before 65 years of age.

Scientists continue to study other possible risk factors for CAD.
• **High levels of a protein called C-reactive protein (CRP)** in the blood may raise the risk for CAD and heart attack. High levels of CRP are proof of inflammation in the body.

### Other factors

Other factors also may contribute to CAD. These include:

- **Sleep apnea.** Sleep apnea is a disorder in which breathing stops or gets very shallow while a person sleeps. Untreated sleep apnea can raise the chances of having high blood pressure, diabetes and even a heart attack or stroke.

- **Stress.** Research shows that the most commonly reported “trigger” for a heart attack is an emotionally upsetting event – particularly one involving anger.

- **Alcohol.** Heavy drinking can damage the heart muscle and worsen other risk factors for heart disease. Men should have no more than two drinks containing alcohol a day. Women should have no more than one drink containing alcohol a day.

### Acute myocardial infarction (AMI)

Commonly known as a heart attack, acute myocardial infarction is caused by reduced blood flow through one or more of the coronary arteries, secondary to coronary heart disease or cardiomyopathy (disease of the heart muscle fibers).

Myocardial infarction includes ST-segment elevation MI (STEMI), non-ST-segment elevation MI (NSTEMI), and unstable angina as a group of clinical diseases called acute coronary syndrome (ACS). Rupture or erosion of the plaque initiates all ACS conditions. Three stages occur when there is occlusion of a vessel: ischemia, injury and infarct.

- **Ischemia** is the first stage, and it indicates that blood flow and oxygen demands are out of balance. The electrocardiogram (ECG) will reveal ST-segment depression or T-wave changes.

- **Injury** is the second stage, and it indicates the ischemia is prolonged enough to damage that area of the heart. The ECG will reveal ST-segment elevation in at least two different leads.

- **Infarct** is the third stage, and it indicates actual death of the myocardial cells and is irreversible. In the early stages of an MI, the ECG will reveal hyperacute (very tall) or narrow T-waves. Within hours, the T-waves become inverted and ST-segment elevation occurs in the leads facing the area of the damage. The last stage is the development of a pathologic Q-wave. Q-waves are permanent evidence of myocardial necrosis.

During a heart attack, if the blockage in the coronary artery isn’t treated quickly, the heart muscle will begin to die and be replaced by scar tissue. This heart damage may not be obvious, or it may cause severe or long-lasting problems.

Severe problems linked to heart attack can include heart failure and life-threatening arrhythmias (irregular heartbeats). Ventricular fibrillation is a serious arrhythmia that can cause death if not treated quickly. Acting fast at the first sign of heart attack symptoms can save a life and limit damage to the heart. Treatment is most effective when started within one hour of the beginning of symptoms.

The most common heart attack signs and symptoms are:

- Chest discomfort or pain – uncomfortable pressure, squeezing, fullness, or pain in the center of the chest that can be mild or strong. This discomfort or pain lasts more than a few minutes or goes away and comes back.

- Upper body discomfort in one or both arms, the back, neck, jaw or stomach.

- Shortness of breath may occur with or before chest discomfort.

- Other signs include nausea (feeling sick to the stomach), vomiting, lightheadedness or fainting, or breaking out in a cold sweat.

### What causes heart attacks?

Most heart attacks occur as a result of coronary artery disease. CAD is the buildup over time of a material called plaque on the inner walls of the coronary arteries. Eventually, a section of plaque can break open, causing a blood clot to form at the site. A heart attack occurs if the clot becomes large enough to cut off most or all of the blood flow through the artery. The blocked blood flow prevents oxygen-rich blood from reaching the part of the heart muscle fed by the artery. The lack of oxygen damages the heart muscle. If the blockage isn’t treated quickly, the damaged heart muscle begins to die.

Heart attacks also can occur due to problems with the very small, microscopic blood vessels of the heart. This condition is called microvascular disease. It’s believed to be more common in women than in men.
Another less common cause of heart attack is a severe spasm (tightening) of a coronary artery that cuts off blood flow through the artery. These spasms can occur in coronary arteries that don’t have CAD. It’s not always clear what causes a coronary artery spasm, but sometimes it can be related to:

- Taking certain drugs, such as cocaine.
- Emotional stress or pain.
- Exposure to extreme cold.
- Cigarette smoking.

Risk factors for acute myocardial infarction

- Coronary artery disease.
- Smoking.
- High blood pressure.
- High blood cholesterol.
- Being overweight and obese.
- Physical inactivity.
- Diabetes (high blood sugar).
- Age. Risk increases for men older than 45 years and for women older than 55 years (or after menopause).
- Family history of early CAD. The risk increases if a person’s father or a brother was diagnosed with CAD before 55 years of age, or mother or a sister was diagnosed before 65 years of age.

Certain CAD risk factors tend to occur together. When they do, it's called metabolic syndrome. In general, a person with metabolic syndrome is twice as likely to develop heart disease and five times as likely to develop diabetes as someone without metabolic syndrome.

Angina

Angina chest pain or discomfort occurs when an area of the heart muscle doesn’t get enough oxygen-rich blood. Angina may feel like pressure or squeezing in the chest and also may occur in the shoulders, arms, neck, jaw or back. Angina pain may even feel like indigestion.

Angina isn’t a disease; it’s a symptom of an underlying heart problem, usually coronary artery disease. It occurs when plaque buildup interrupts blood flow to the heart muscle, causing the chest pain.

Angina also can be a symptom of coronary microvascular disease (MVD). This is heart disease that affects the heart’s smallest coronary arteries. Unlike traditional CAD, coronary MVD doesn’t always create blockages in the arteries. Studies have shown that coronary MVD is more likely to affect women than men.

Coronary MVD also is called cardiac syndrome X and nonobstructive CHD.

Types of angina

The types of angina are stable, unstable, variant (Prinzmetal’s) and microvascular. Knowing how the types differ is important. This is because they have different symptoms and require different treatments.

- Stable angina
  - Stable angina is the most common type of angina. It occurs if the heart is working harder than usual. Stable angina has a regular pattern.
  - A person who knows he has stable angina can learn to recognize the pattern and predict when the pain will occur. The pain usually goes away a few minutes after the person rests or takes angina medicine.
  - Stable angina isn’t a heart attack, but it suggests that a heart attack is more likely in the future.

- Unstable angina
  - Unstable angina doesn’t follow a pattern. It can occur with or without physical exertion, and it may not be relieved by rest or medicine.
  - Unstable angina is very dangerous and requires emergency treatment. This type of angina is a sign that a heart attack may happen soon.

- Variant (Prinzmetal’s) angina
  - Variant angina is rare. It usually occurs while a person is at rest, and the pain can be severe. Variant angina usually happens between midnight and early morning. Medicine can relieve this type of angina.

- Microvascular angina
  - Microvascular angina can be more severe and last longer than other types of angina; medicine may not relieve it. This type of angina may be a symptom of coronary MVD.

What causes angina?

The underlying cause of angina is usually coronary heart disease. However, a number of things can trigger angina pain, depending on the type of angina the patient has:

- Stable angina
  - Physical exertion is the most common trigger of stable angina. Severely narrowed arteries may allow enough blood to reach the heart when the demand for oxygen is low, such as when a person is sitting. However, with physical exertion – like walking up a hill or climbing stairs – the heart works harder and needs more oxygen.

- Unstable angina
  - Blood clots that partly or totally block an artery cause unstable angina. If plaque in an artery ruptures, blood clots may form. This creates a larger blockage. A clot
may grow large enough to completely block the artery and cause a heart attack.
• Blood clots may form, partly dissolve and later form again. Angina can occur each time a clot blocks an artery.

- **Variant angina**
  - A spasm in a coronary artery causes variant angina. The spasm causes the walls of the artery to tighten and narrow. Blood flow to the heart slows or stops. Variant angina may occur in people who have CAD and in those who don’t.
  - Other factors that can cause the coronary arteries to spasm are:
    - Exposure to cold.

**Congestive heart failure**

Also known as “heart” or “pump failure,” congestive heart failure is a syndrome that occurs when the heart is unable to adequately pump to meet the body’s metabolic needs. Heart failure develops over time as the heart’s pumping action grows weaker. The condition can affect the right side of the heart only, or it can affect both sides of the heart. Most cases involve both sides of the heart.

The leading causes of heart failure are diseases that damage the heart. Over time, the heart weakens. It isn’t able to fill with and/or pump blood as well as it should. As the heart weakens, certain proteins and other substances may be released into the blood. These substances have a toxic effect on the heart and blood flow, and they worsen heart failure.

Right-side heart failure occurs if the heart can’t pump enough blood to the lungs to pick up oxygen. Left-side heart failure occurs if the heart can’t pump enough oxygen-rich blood to the rest of the body. Right-side heart failure may cause fluid to build up in the feet, ankles, legs, liver, abdomen and the veins in the neck. Right-side and left-side heart failure also may cause shortness of breath and fatigue (tiredness).

Left-sided heart failure is broken down into two subcategories, systolic and diastolic heart failure.

**Systolic heart failure** results from the heart’s inability to contract forcefully during systole to eject an adequate amount of blood into circulation. In systolic heart failure, the following things occur [30]:

- **Preload increases** (degree of myocardial stretch at the end of diastole and just before contraction).
- **Decreased contractility of the heart muscle** that affects the stroke volume (SV) and cardiac output (CO). SV is the amount of blood ejected by the left ventricle during each systole. CO is the volume of blood in liters ejected by the heart each minute. The normal CO in adults varies from four to seven liters/per minute.
- **Afterload increases** (pressure/resistance that the ventricles must overcome to eject blood through the semilunar valves).
- **All of these inadequate functions lead to an increased peripheral resistance (hypertension).** The ejection fraction (EF) is the percentage of blood ejected from the heart during systole; normal is 50 to 70 percent.

**Diastolic heart failure** results when the left ventricle is unable to relax adequately during diastole (rest). Over time, the ventricle will stiffen due to the inability to relax completely, leading to insufficient blood filling, resulting in a decreased cardiac output (CO). The ejection fraction may be within the normal range. Diastolic heart failure occurs in 20 to 40 percent of all heart failure cases, especially in older adults and women after an MI.

- **Right-sided heart failure** occurs over time due to left ventricular failure, with myocardial infarction in the right ventricle or pulmonary hypertension occurring. In right-sided heart failure, the right ventricle is unable to empty completely, leading to increased volume and pressure in the systemic veins.

**Common causes of heart failure**
The most common causes of heart failure are coronary artery disease, high blood pressure and diabetes. Treating these problems can prevent or improve heart failure.

- **Coronary heart disease**
  - Plaque caused by CAD narrows the arteries and reduces blood flow to the heart muscle. It also makes it more likely that blood clots will form in the arteries. Blood clots can partially or completely block blood flow.
  - CAD can lead to chest pain or discomfort called angina, a heart attack, heart damage or even death.

- **High blood pressure**
  - Blood pressure is the force of blood pushing against the walls of the arteries. If this pressure rises and stays high over time, it can weaken the heart and lead to plaque buildup.
  - Blood pressure is considered high if it stays at or above 140/90 mm/Hg over time. (The mm/Hg is millimeters of mercury – the units used to measure blood pressure.) If you have diabetes or chronic kidney disease, high blood pressure is defined as 130/80 mm/Hg or higher.

- **Diabetes**
  - Diabetes is a disease in which the body’s blood glucose, or blood sugar, level is too high. Normally, the body breaks down food into glucose and then carries it to cells throughout the body. The cells use a hormone called insulin to turn the glucose into energy.
  - In diabetes, the body doesn’t make enough insulin or doesn’t use its insulin properly. Over time, high

blood sugar levels can damage and weaken the heart muscle and the blood vessels around the heart, leading to heart failure.

**Other causes**
- **Cardiomyopathy**, or heart muscle disease. Cardiomyopathy may be present at birth or due to injury or infection.
- **Heart valve disease**. Problems with the heart valves may be present at birth or due to infection, heart attack or damage from heart disease.
- **Arrhythmias**, or irregular heartbeats. These heart problems may be present at birth or due to heart disease or heart defects.
- **Congenital heart defects**. These heart problems are present at birth.

**Other factors**:
- Treatments for cancer, such as radiation and chemotherapy.
- Thyroid disorders (having either too much or too little thyroid hormone in the body).
- Alcohol abuse or cocaine and other illegal drug use.
- HIV/AIDS.
- Too much vitamin E.

Heart damage from obstructive sleep apnea may cause heart failure to worsen. Sleep apnea is a common disorder in which a person has one or more pauses in breathing or shallow breaths while sleeping. This can deprive the heart of oxygen and increase its workload. Treating this sleep problem may improve heart failure.

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**Hypertension**

Often called high blood pressure (HBP), hypertension is a serious condition that can lead to coronary heart disease, heart failure, stroke, kidney failure and other health problems. About one in three adults in the United States has high blood pressure, although the condition itself usually has no symptoms. A person can have hypertension for years without knowing it. During this time, though, it can damage the heart, blood vessels, kidneys and other parts of the body.

When HBP has no known cause, it may be called essential hypertension, primary hypertension or idiopathic hypertension. When another condition causes HBP, it’s sometimes called secondary high blood pressure or secondary hypertension. In some cases of HBP, only the systolic blood pressure number is high. This condition is called isolated systolic hypertension (ISH). Many older adults have this condition. ISH can cause as much harm as HBP in which both numbers are too high.

Over time, uncontrolled or prolonged elevation of the blood pressure (BP) can lead to a variety of changes in the myocardium (middle layer composed of striated muscle fibers), coronary vasculature and conduction system of the heart. The most significant changes can lead to the development of left ventricular hypertrophy (LVH), coronary artery disease, various conduction system diseases and systolic and diastolic dysfunction of the myocardium, which manifest clinically as angina or myocardial infarction, cardiac arrhythmias (especially atrial fibrillation, premature ventricular contractions and ventricular tachycardia), and congestive heart failure.

Hypertension is an established risk factor for the development of coronary artery disease, almost doubling the risk. It also increases the risk of sudden cardiac death.

- Fifteen to 20 percent of people will develop left ventricular hypertrophy, especially if the individual is obese.
- Left ventricular hypertrophy plays a significant role in cardiovascular disease. It occurs secondary to increased pressure demands on the left ventricle to become enlarged and thickened. As the left ventricle enlarges and becomes thick, it is unable to effectively pump out an adequate amount of oxygenated blood into the body. Therefore, blood backs up into the left atrium and then into the lungs, causing pulmonary congestion, dyspnea and activity intolerance.

**What causes high blood pressure?**

- Blood pressure tends to rise with age, unless steps are taken to prevent or control it.
- Certain medical problems, such as chronic kidney disease, thyroid disease and sleep apnea, may cause blood pressure to rise. Certain medicines, such as asthma medicines (for example, corticosteroids) and cold-relief products, also may raise blood pressure.
- In some women, blood pressure can go up if they use birth control pills, become pregnant or take hormone replacement therapy.
- Women taking birth control pills usually have a small rise in both systolic and diastolic blood pressures.
- Women who have high blood pressure and want to use birth control pills should discuss it with their doctor.
- Taking hormones to reduce the symptoms of menopause can cause a small rise in systolic blood pressure. Women who already have HBP and want to start using hormones should discuss the risks and benefits with their doctor.
- Children younger than 10 years who have HBP often have another condition that’s causing it (such as kidney disease). Treating the underlying condition may resolve the HBP.
- The older a child is when HBP is diagnosed, the more likely he or she is to have essential hypertension. This means that doctors don’t know what’s causing the HBP.
Risk factors for hypertension

- **Older age**
  - Blood pressure tends to rise with age. Males older than 45 and females older than 55 have a higher risk for HBP. Over half of all Americans aged 60 and older have HBP.
  - Isolated systolic hypertension (ISH) is the most common form of HBP in older adults. ISH occurs when only systolic blood pressure (the top number) is high. About two of three people over age 60 who have HBP have ISH.

- **Race/ethnicity**
  - HBP occurs more often in African American adults than in Caucasian or Hispanic American adults. In relation to these groups, African Americans:
    - Tend to get HBP earlier in life.
    - Often have more severe HBP.
    - Are more likely to be aware that they have HBP and to get treatment.
    - Are less likely than Caucasians and about as likely as Hispanic Americans to achieve target control levels with HBP treatment.
    - Have higher rates than Caucasians of premature death from HBP-related complications, such as coronary heart disease, stroke and kidney failure.
  - HBP risks vary among different groups of Hispanic American adults. For instance, Puerto Rican American adults have higher rates of HBP-related death than all other Hispanic groups and Caucasians. But Cuban Americans have lower rates than Caucasians.

- **Being overweight or obese**
  - People who are overweight or obese are more likely to develop prehypertension or HBP. Overweight is having extra body weight from muscle, bone, fat and/or water. Obesity is having a high amount of extra body fat.

- **Gender**
  - Fewer adult women than men have HBP. But younger women (aged 18–59) are more likely than men to be aware of and get treatment for HBP.
  - Women aged 60 and older are as likely as men to be aware of and treated for HBP. However, among treated women aged 60 and older, blood pressure control is lower than it is in men in the same age group.

- **Unhealthy lifestyle habits**
  - Eating too much sodium (salt).
  - Drinking too much alcohol.
  - Not getting enough potassium in the diet.
  - Not doing enough physical activity.
  - Smoking.

Other factors

- A family history of HBP raises the risk for the condition. Long-lasting stress also can put a person at risk for HBP.
- Prehypertension increases the likelihood of developing high blood pressure. Prehypertension is a normal blood pressure in the 120–139/80–89 mm/Hg range.
- Risk factors for children and teens:
  - Being overweight, which is on the rise in youth younger than 18 years. As a result, prehypertension and HBP also are becoming more common in this age group.
  - African American and Mexican American youth are more likely to have HBP and prehypertension than Caucasian youth. Boys are at higher risk for HBP than girls.

**Arrhythmias**

An arrhythmia is a problem with the rate or rhythm of the heartbeat. During an arrhythmia, the heart can beat too fast, too slow, or with an irregular rhythm.

A heartbeat that is too fast is called tachycardia. A heartbeat that is too slow is called bradycardia.

Most arrhythmias are harmless, but some can be serious or even life threatening. When the heart rate is too fast, too slow or irregular, the heart may not be able to pump enough blood to the body. Lack of blood flow can damage the brain, heart and other organs.

**The heart’s electrical system**

To understand arrhythmias, it helps to understand the heart’s internal electrical system. The heart’s electrical system controls the rate and rhythm of the heartbeat. With each heartbeat, an electrical signal spreads from the top of the heart to the bottom. As the signal travels, it causes the heart to contract and pump blood. The process repeats with each new heartbeat.

Each electrical signal begins in a group of cells called the sinus node or sinoatrial (SA) node. The SA node is located in the right atrium. In a healthy adult heart at rest, the SA node fires off an electrical signal to begin a new heartbeat 60 to 100 times a minute. From the SA node, the electrical signal travels through special pathways in the right and left atria. This causes the atria to contract and pump blood into the heart’s two lower chambers, the ventricles.

The electrical signal then moves down to a group of cells called the atrioventricular (AV) node, located between the atria and the ventricles. Here, the signal slows down just a little, allowing the ventricles time to finish filling with blood. The electrical signal then leaves the AV node and travels along a pathway called the bundle of His. This pathway divides into a right bundle branch and a left bundle branch. The signal goes down...
these branches to the ventricles, causing them to contract and pump blood out to the lungs and the rest of the body.

The ventricles then relax, and the heartbeat process starts all over again in the SA node.

Types of arrhythmia

The four main types of arrhythmia are premature (extra) beats, supraventricular arrhythmias, ventricular arrhythmias and bradyarrhythmia.

Premature (extra) beats

- Premature beats are the most common type of arrhythmia. They’re harmless most of the time and often don’t cause any symptoms. When symptoms do occur, they usually feel like fluttering in the chest or a feeling of a skipped beat. Most of the time, premature beats need no treatment, especially in healthy people.
- Premature beats that occur in the atria are called premature atrial contractions, or PACs. Premature beats that occur in the ventricles are called premature ventricular contractions, or PVCs.
- In most cases, premature beats occur naturally, not due to any heart disease. But certain heart diseases can cause premature beats. They also can happen because of stress, too much exercise, or too much caffeine or nicotine.

Supraventricular arrhythmias

- Supraventricular arrhythmias are tachycardias (fast heart rates) that start in the atria or the atrioventricular (AV) node. The AV node is a group of cells located between the atria and the ventricles.
- Types of supraventricular arrhythmias include atrial fibrillation (AF), atrial flutter, paroxysmal supraventricular tachycardia (PSVT) and Wolff-Parkinson-White (WPW) syndrome.
  - Atrial fibrillation
    - **AF is the most common type of serious arrhythmia. It’s a very fast and irregular contraction of the atria.** There are three types: Paroxysmal atrial fibrillation, in which the abnormal electrical signals and rapid heart rate begin suddenly and then stop on their own. Symptoms can be mild or severe and last for seconds, minutes, hours or days. Persistent atrial fibrillation, a condition in which the abnormal heart rhythm continues until it’s stopped with treatment. Permanent atrial fibrillation, a condition in which a normal heart rhythm can’t be restored with the usual treatments. Both paroxysmal and persistent AF may become more frequent and over time, result in permanent AF.
    - In AF, the heart’s electrical signal doesn’t begin in the SA node. Instead, the signal begins in another part of the atria or in the nearby pulmonary veins and is conducted abnormally. When this happens, the electrical signal doesn’t travel through the normal pathways in the atria. Instead, it spreads throughout the atria in a fast and disorganized manner. This causes the walls of the atria to quiver very fast (fibrillate) instead of beating normally. As a result, the atria aren’t able to pump blood into the ventricles the way they should.
    - In AF, electrical signals can travel through the atria at a rate of more than 300 per minute. Some of these abnormal electrical signals can travel to the ventricles, causing them to beat too fast and with an irregular rhythm. AF usually isn’t life-threatening, but it can be dangerous when it causes the ventricles to beat very fast.
    - The two most serious complications of chronic (long-term) AF are stroke and heart failure. Stroke can happen if a blood clot travels to an artery in the brain, blocking off blood flow.
    - In AF, blood clots can form because some of the blood “pools” in the fibrillating atria instead of flowing into the ventricles. If a piece of a blood clot in the left atrium breaks off, it can travel to the brain, causing a stroke. People who have AF often are treated with blood-thinning medicines to lower their risk for blood clots.
    - Heart failure is when the heart can’t pump enough blood to meet the body’s needs. AF can cause heart failure if the ventricles beat too fast and don’t have enough time to fill with blood to pump out to the body. Heart failure causes fatigue (tiredness), leg swelling and shortness of breath.
    - AF and other supraventricular arrhythmias can occur for no apparent reason. But most of the time, an underlying condition that damages the heart muscle and its ability to conduct electrical impulses causes AF. These conditions include high blood pressure, coronary artery disease, heart failure and rheumatic heart disease.
    - Other conditions also can lead to AF, including an overactive thyroid gland (too much thyroid hormone produced) and heavy alcohol use. AF also becomes more common as people get older.
  - Atrial flutter
    - Atrial flutter is similar to AF, but instead of the electrical signals spreading through the atria in a fast and irregular rhythm, they travel in a fast and regular rhythm.
    - Atrial flutter is much less common than AF, but it has similar symptoms and complications.
  - Paroxysmal supraventricular tachycardia
    - PSVT is a very fast heart rate that begins and ends suddenly. PSVT occurs due to problems
with the electrical connection between the atria and the ventricles.

- In PSVT, electrical signals that begin in the atria and travel to the ventricles can reenter the atria, causing extra heartbeats. This type of arrhythmia usually isn’t dangerous and tends to occur in young people. It can happen during vigorous exercise.

- A special type of PSVT is called Wolff-Parkinson-White syndrome. WPW syndrome is a condition in which the heart’s electrical signals travel along an extra pathway from the atria to the ventricles.

  This extra pathway disrupts the timing of the heart’s electrical signals and can cause the ventricles to beat very fast. This type of arrhythmia can be life-threatening.

**Ventricular arrhythmias**

- These arrhythmias start in the ventricles. They can be very dangerous and usually need medical attention right away. Ventricular arrhythmias include ventricular tachycardia and ventricular fibrillation (v-fib). Coronary heart disease, heart attack, weakened heart muscle and other problems can cause ventricular arrhythmias.

  - Ventricular tachycardia

    - Ventricular tachycardia is a fast, regular beating of the ventricles that may last for only a few seconds or for much longer. A few beats of ventricular tachycardia often don’t cause problems. However, episodes that last for more than a few seconds can be dangerous. Ventricular tachycardia can turn into other, more dangerous arrhythmias, such as v-fib.

  - Ventricular fibrillation

    - V-fib occurs when disorganized electrical signals make the ventricles quiver instead of pump normally. Without the ventricles pumping blood out to the body, a person will lose consciousness within seconds and die within minutes if not treated. To prevent death, the condition must be treated right away with an electric shock to the heart called defibrillation. V-fib may happen during or after a heart attack or in someone whose heart is already weak because of another condition. Health experts think that most of the sudden cardiac deaths that occur every year (about 335,000) are due to v-fib.

    - Torsades de pointes (torsades) is a type of v-fib that causes a unique pattern on an EKG. Certain medicines or imbalanced amounts of potassium, calcium or magnesium in the bloodstream can cause this condition.

**What causes an arrhythmia?**

An arrhythmia can occur if the electrical signals that control the heartbeat are delayed or blocked. This can happen if the special nerve cells that produce electrical signals don’t work properly, or if electrical signals don’t travel normally through the heart.

An arrhythmia also can occur if another part of the heart starts to produce electrical signals. This adds to the signals from the special nerve cells and disrupts the normal heartbeat.

- People who have long QT syndrome are at higher risk for torsades. People who have this condition need to be careful about taking certain antibiotics, heart medicines and over-the-counter medicines.

**Bradyarrhythmias**

- Bradyarrhythmias are arrhythmias in which the heart rate is slower than normal. If the heart rate is too slow, not enough blood reaches the brain, which can cause loss of consciousness. In adults, a heart rate slower than 60 beats per minute is considered a bradyarrhythmia. Some people normally have slow heart rates, especially people who are very physically fit. For them, a heartbeat slower than 60 beats per minute isn’t dangerous and doesn’t cause symptoms. But in other people, bradyarrhythmia can be due to a serious disease or other condition.

  - Bradyarrhythmias can be caused by:

    ■ Heart attack.
    ■ Conditions that harm or change the heart’s electrical activity, such as an underactive thyroid gland or aging.
    ■ An imbalance of chemicals or other substances, such as potassium, in the blood.
    ■ Some medicines, such as beta blockers.

  - Bradyarrhythmias also can happen as a result of severe bundle branch block. Bundle branch block is a condition in which an electrical signal traveling down either or both of the bundle branches is delayed or blocked. When this happens, the ventricles don’t contract at exactly the same time, as they should. As a result, the heart has to work harder to pump blood to the body. The cause of bundle branch block often is an existing heart condition.

**Arrhythmias in children**

- A child’s heart rate normally decreases as he or she gets older. A newborn’s heart beats between 95 to 160 times a minute. A 1-year-old’s heart beats between 90 to 150 times a minute, and a 6- to 8-year-old’s heart beats between 60 to 110 times a minute.

  - A baby or child’s heart can beat faster or slower than normal for many reasons. Like adults, when children are active, their hearts will beat faster. When they’re sleeping, their hearts will beat slower. Their heart rates can speed up and slow down as they breathe in and out. All of these changes are normal.

  - Some children are born with heart defects that cause arrhythmias. In other children, arrhythmias can develop later in childhood. Doctors use the same tests to diagnose arrhythmias in children and adults.

  - Treatments for children who have arrhythmias include medicines, defibrillation (electric shock), surgically implanted devices that control the heartbeat and other procedures that fix abnormal electrical signals in the heart.
Smoking, heavy alcohol consumption, use of certain drugs (such as cocaine or amphetamines), use of certain prescription or over-the-counter medicines, or too much caffeine or nicotine can lead to arrhythmias in some people. Strong emotional stress or anger can make the heart work harder, raise blood pressure and release stress hormones. In some people, these reactions can lead to arrhythmias.

A heart attack or an underlying condition that damages the heart’s electrical system also can cause arrhythmias. Examples of such conditions include high blood pressure, coronary heart disease, heart failure, overactive or underactive thyroid gland, pericarditis and rheumatic heart disease.

In some arrhythmias, such as Wolff-Parkinson-White syndrome, the underlying heart defect that causes the arrhythmia is congenital. Sometimes the cause of an arrhythmia can’t be found.

Who is at risk for an arrhythmia?

Millions of Americans have arrhythmias. They’re very common in older adults. About 2.2 million Americans have atrial fibrillation. Most serious arrhythmias affect people older than 60. This is because older adults are more likely to have heart disease and other health problems that can lead to arrhythmias. Older adults also tend to be more sensitive to the side effects of medicines, some of which can cause arrhythmias. Some medicines used to treat arrhythmias can even cause arrhythmias as a side effect.

Some types of arrhythmia happen more often in children and young adults. Paroxysmal supraventricular tachycardias (PSVTs), including Wolff-Parkinson-White syndrome, are more common in young people. PSVT is a fast heart rate that begins and ends suddenly.

- **Major risk factors**: Arrhythmias are more common in people who have diseases or conditions that weaken the heart, such as:
  - Heart attack.
  - Heart failure or cardiomyopathy, which weakens the heart and changes the way electrical signals move around the heart.

- **Other conditions** also can increase the risk for arrhythmias, such as:
  - Heart tissue that’s too thick or stiff or that hasn’t formed normally.
  - Leaking or narrowed heart valves, which make the heart work too hard and can lead to heart failure.
  - Congenital heart defects (problems that are present at birth) that affect the heart’s structure or function.

Peripheral arterial disease

Peripheral arterial disease (PAD) occurs when plaque builds up in the arteries that carry blood to the head, organs and limbs, a condition known as atherosclerosis. Over time, plaque can harden and narrow the arteries, which limits the flow of oxygen-rich blood to the organs and other parts of the body. PAD usually affects the legs, but also can affect the arteries that carry blood from the head, arms, kidneys and stomach.

Overview

Blocked blood flow to the legs can cause pain and numbness. It also can raise the risk of getting an infection in the affected limbs. It may be hard for the body to fight the infection.

If severe enough, blocked blood flow can cause tissue death (gangrene). In very serious cases, this can lead to leg amputation.

A person who has leg pain when walking or climbing stairs should talk to a doctor. Sometimes older people think that leg pain is just a symptom of aging. However, the cause for the pain could be PAD, and the doctor may wish to test for it.

Smoking is the main risk factor for PAD. The risk for PAD increases four times for a person who smokes or has a history of smoking. Other factors, such as age and having certain diseases or conditions, also increase the risk.

The risk for coronary artery disease, heart attack, stroke and transient ischemic attack (“mini-stroke”) is six to seven times greater than the risk for people who don’t have PAD. A person with heart disease has a one-in-three chance of having blocked leg arteries.

Although PAD is serious, the underlying atherosclerosis can be treated. PAD treatment may slow or stop disease progress and reduce the risk of complications. Treatments include lifestyle changes, medicines, and surgery or procedures. Researchers continue to explore new therapies for PAD.
What causes peripheral arterial disease?

The most common cause of peripheral arterial disease is atherosclerosis. The exact cause of atherosclerosis isn’t known. The disease may start when certain factors damage the inner layers of the arteries. These factors include:

- Smiling.
- High amounts of certain fats and cholesterol in the blood.
- High blood pressure.
- High amounts of sugar in the blood caused by insulin resistance or diabetes.

Who is at risk for peripheral arterial disease?

Peripheral arterial disease (PAD) affects 8 to 12 million people in the United States. African Americans are more than twice as likely as Caucasians to have PAD. The major risk factors for PAD are smoking, age and having certain diseases or conditions:

- **Smoking**
  - Smoking is more closely related to getting PAD than any other risk factor. On average, smokers who develop PAD have symptoms 10 years earlier than nonsmokers who develop PAD.
  - Quitting smoking slows the progress of PAD. Smoking even one or two cigarettes a day can interfere with PAD treatments. Smokers and people who have diabetes are at highest risk for PAD complications, including gangrene (tissue death) in the leg from decreased blood flow.
- **Age**
- **Ethnicity**
  - African Americans are more than twice as likely as Caucasians to have PAD.
- **Diseases and conditions**
  - Diabetes. One in three people who has diabetes and is older than 50 is likely to have PAD.
  - High blood cholesterol or a family history of it.
  - High blood pressure or a family history of it.
  - Heart disease or a family history of it.
  - Stroke or a family history of it.
  - In 2002, age-adjusted death rates for heart disease were highest among women aged 45-64. Unfortunately, women who suffer an acute myocardial infarction (MI) typically tend to die more frequently than men.
- **Ethnicity**. According to the CDC in 2009, cardiovascular disease death rates per 100,000 population for the five largest U.S. racial/ethnic groups are as follows: blacks, 300; whites, 228; Hispanics, 173; American Indian/Alaskan natives, 160; and Asian and Pacific Islanders, 128.
  - Blacks suffer from hypertension more than Caucasians and have a higher risk of developing cardiovascular disease, especially coronary artery disease.
  - In 2002, age-adjusted death rates for heart disease were higher among black women (169.7 per 100,000) than among white women (131.2 per 100,000).
  - White women with abdominal obesity (greater waist circumference than hip circumference) are more likely to develop cardiovascular disease than white women with distributed fat in the buttocks, hips and thighs (greater hip circumference than waist circumference).
  - Asian and Pacific Islanders are more likely than Americans, American Indians, native Hawaiians and some Asian Americans, all linked to higher rates of coinciding obesity and diabetes.
  - According to the CDC (2008), heart disease is the leading cause of death and stroke is the sixth among American Indians and Alaska natives. The heart disease...
death rate was 20 percent greater and the stroke death rate 14 percent greater among American Indians and Alaska natives (1996–1998) than among all U.S. races (1997) after adjusting for misreporting of American Indian and Alaska native races on state death certificates. American Indians and Alaska natives die from heart diseases at younger ages than other racial and ethnic groups in the U.S. Thirty-six percent of those who die of heart disease die before age 65. Diabetes is an extremely important risk factor for cardiovascular disease among American Indians.

- **Genetics.** Any family history for coronary artery disease in a first-degree relative (parent, sibling or child) is the major risk factor for developing cardiovascular disease. It is a higher risk than hypertension, obesity, diabetes or sudden cardiac death. In addition, the younger the age of onset in a first-degree relative, the greater the risk for developing coronary artery disease.

**Modifiable risks** are personal habits and choices that an individual has the ability to modify or change. (see chart on following page)

- **Cigarette smoking** is a major risk factor for cardiovascular disease, especially coronary artery disease and peripheral arterial disease (PAD). Individuals who choose to smoke increase their risk of developing cardiovascular disease two to four times higher than that of a nonsmoker. Cigarette smoking is also a powerful independent risk factor for sudden cardiac death in patients with coronary heart disease; smokers have about twice the risk of nonsmokers. Cigarette smoking also acts with other risk factors to greatly increase the risk for coronary heart disease.
  - Other forms of smoking, such as cigars, pipes and secondhand smoke, also add to the risk of developing coronary artery disease and CVA (cerebrovascular accident), although the risk is not as high as with cigarette smoking.
  - Nurses should inquire about patients’ smoking history in terms of the pack per years, which is the number of packs per day multiplied by the number of years smoked.

- **Physical inactivity.** A sedentary, inactive lifestyle is a significant risk factor for the development of cardiovascular disease. Regular, moderate-to-vigorous physical activity helps prevent heart and blood vessel disease by controlling the blood pressure, blood lipids and clotting factors.

- **Being obese or overweight.** Obesity leads to diabetes, hypertension and high cholesterol (hypercholesterolemia/ hyperlipidemia). According to the WHO in 2009, there are more than 1 billion overweight adults; at least 300 million of them are considered obese. In addition, the WHO has estimated there are 155 million overweight or obese children worldwide, and 22 million are overweight under the age of 5. In 2000, 22 percent of American preschool-age children were overweight, and 10 percent were considered obese. In essence, one in four preschool age children is overweight or obese. Obesity and overweight conditions are assessed by using body mass index (BMI), defined as the weight in kilograms divided by the square of the height in meters (kg/m2).
  - A BMI over 25 kg/m2 is defined as overweight, and a BMI of over 30 kg/m2 as obese. These markers provide common benchmarks for assessment, but the risks of disease in all populations can increase progressively from lower BMI levels.
  - Being overweight or obese leads to adverse metabolic effects in numerous ways, but especially on a person’s cardiovascular system, resulting in hypertension, hyperlipidemia and insulin resistance.

- **Psychological factors** such as stress may contribute to the risk of developing cardiovascular disease. Research has demonstrated that people who are highly competitive, concerned about meeting deadlines and often angry are at a higher risk of developing cardiovascular disease. In addition, researchers have noted a significant correlation between coronary heart disease risk and stress in a person’s life, their health behaviors and socioeconomic status. These factors may affect established risk factors. For example, people under stress may overeat, start smoking or smoke more than they otherwise would.
  - Nurses can assess this risk by asking the patient, “Have you ever experienced road rage?” or “How do you respond when you have to wait for an appointment?”

- **Alcohol** use can raise the blood pressure, leading to heart failure and stroke. It may also affect the cardiovascular system by contributing to high triglycerides, obesity and irregular heartbeats.
  - The risk of heart disease in people who drink moderate amounts of alcohol (an average of one drink for women or two drinks for men per day) is lower than in nondrinkers. One drink is defined as 1 1/2 ounces of 80-proof spirits (such as bourbon, Scotch, vodka and gin), 1 ounce of 100-proof spirits, 4 ounces of wine or 12 ounces of beer.
<table>
<thead>
<tr>
<th>Risk factor</th>
<th>How much it affects stroke risk</th>
<th>Why it affects stroke risk</th>
<th>What you can do</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>Hypertension causes a two- to four-fold increase in the risk of stroke before age 80. After age 80, the impact of hypertension declines and other risk factors become more important.</td>
<td>Hypertension promotes atherosclerosis and causes mechanical damage to the walls of blood vessels.</td>
<td>Blood pressure medications, such as thiazide diuretics and angiotensin-converting enzyme (ACE) inhibitors, can reduce the risk of stroke by 30 to 40 percent. Early treatment is essential. Among older people with normal blood pressure, prior mid-life hypertension increases stroke risk up to 92 percent. Guidelines from the Centers for Disease Control and Prevention recommend a target blood pressure of less than 140/90 mm/Hg.</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>Smoking causes about a two-fold increase in the risk of ischemic stroke and up to a four-fold increase in the risk of hemorrhagic stroke.</td>
<td>Smoking promotes atherosclerosis and aneurysm formation, and stimulates blood clotting factors.</td>
<td>Stroke risk decreases significantly two years after quitting smoking and is at the level of nonsmokers by five years.</td>
</tr>
<tr>
<td>Diabetes</td>
<td>In terms of stroke and cardiovascular disease risk, having diabetes is the equivalent of aging 15 years.</td>
<td>In diabetes, glucose is not efficiently taken up by the body’s cells and accumulates in the blood instead, where it can damage the vascular system. Hypertension is common among diabetics and accounts for much of their increased stroke risk.</td>
<td>Blood pressure medications, dietary changes and weight loss can lower stroke risk. Controlling blood sugar appears to reduce the risk of recurrent stroke.</td>
</tr>
<tr>
<td>Physical inactivity and obesity</td>
<td>Waist-to-hip ratio equal to or above the median (mid-value for the population) increases the risk of ischemic stroke three-fold.</td>
<td>Obesity is associated with hypertension, diabetes and heart disease.</td>
<td>While no clinical studies have tested the effects of exercise or weight loss on stroke risk, both tend to reduce hypertension and boost cardiovascular health.</td>
</tr>
<tr>
<td>Atrial fibrillation (AF)</td>
<td>AF affects fewer than one percent of people under age 60, but is more prevalent in older people. It is responsible for one in four strokes after age 80, and is associated with high mortality and disability.</td>
<td>AF refers to irregular contraction of the atrium – the chamber where blood enters the heart. AF can lead to blood stagnation and increased clotting.</td>
<td>Warfarin, a blood-thinning medication, can reduce the risk of stroke in people with AF. People under age 60 with AF and no other stroke risk factors may benefit from aspirin. Importantly, pacemakers have no effect on the risk of stroke associated with AF.</td>
</tr>
<tr>
<td>Cholesterol imbalance</td>
<td>High-density lipoprotein (HDL) cholesterol is generally considered protective against ischemic stroke. Low-density lipoprotein (LDL) cholesterol, when present in excess, is considered harmful.</td>
<td>LDL and HDL are needed to carry cholesterol (a fatty substance) through the blood (made up mostly of water), and deliver it to cells. Because LDL delivers cholesterol to cells throughout the body, excess LDL can cause cholesterol to build up in blood vessels, leading to atherosclerosis. HDL sends cholesterol to the liver to be eliminated.</td>
<td>Clinical trials have shown that cholesterol-lowering drugs known as statins reduce the risk of stroke. However, some studies point to only a weak association between stroke and cholesterol, and there is speculation that statins reduce stroke risk by acting through some unknown mechanism.</td>
</tr>
</tbody>
</table>
Other potential risk factors: It is important to assess the patient for other chronic health problems that may exacerbate the cardiac symptoms and increase the risk of cardiovascular disease. The nurse should inquire about other co-morbidities by obtaining information regarding the onset, duration, frequency, location and associating symptoms.

- **Cerebrovascular accident (CVA or stroke)** is a rapid onset of neurological deficits due to a decreased flow of oxygenated blood to the brain. CVA coincides with cardiovascular disease because most of the modifiable risk factors for CVA include hypertension, cardiac disease, hyperlipidemia and smoking. Strokes will be discussed in detail in Part II of this course.

- **Diabetes mellitus** seriously increases an individual’s risk of developing cardiovascular disease. Even if the overall glucose (blood sugar) levels are under control, diabetes increases the risk of heart disease and stroke, but the risks are even greater if blood sugar is not well controlled. According to the American Diabetes Association (ADA), most of the cardiovascular complications related to diabetes have to do with the way the heart pumps blood through the body. Diabetes can change the chemical makeup of some of the substances found in the blood, and this can cause blood vessels to narrow or to clog up completely, which is known as atherosclerosis. Because of the compounding effects of diabetes and cardiovascular disease, it is estimated that about 65 percent of people living with diabetes will die from some form of heart or blood vessel disease.

- **Hyperlipidemia/dyslipidemia** is an elevation of lipids (fats) in the bloodstream measured by assessing the cholesterol, triglycerides, LDL, HDL cholesterol esters (compounds), phospholipids and triglycerides. A high lipid profile is typically known as a modifiable risk factor because it can be the result of choosing a lifestyle that consists of a high fatty diet and possibly drinking alcohol. However, hyperlipidemia may be due to genetic factors beyond an individual’s choice.
  - Over the past few decades, numerous research studies have demonstrated that elevated plasma cholesterol levels, especially low-density lipoprotein cholesterol (LDL-C), has an enormous impact on developing cardiovascular disease.
  - **Hypertension (HTN)** increases the heart’s workload, causing the heart to thicken and become stiffer. It increases the risk of coronary artery disease, cerebrovascular accident, heart attack/myocardial infarction, kidney failure and congestive heart failure. The risk of cardiovascular disease is exacerbated if hypertension coincides with obesity, smoking, high blood cholesterol levels or diabetes.

- **Metabolic syndrome (also known as insulin resistance)** is a syndrome that occurs prior to the development of diabetes. The majority of individuals living with metabolic syndrome are unaware of it because they are usually asymptomatic. However, people with a severe form of insulin resistance (metabolic X) may have a condition called acanthosis nigricans, in which they will notice dark patches of skin, usually on the back of the neck, elbows, knees, knuckles and the armpits, an early sign of pre-diabetes. According to the American Heart Association and the National Heart, Lung and Blood Institute, metabolic syndrome is diagnosed when a minimum of three of the following criteria are met:
  - **Elevated waist circumference (abdominal obesity).** Increased abdominal adiposity (waist greater than 40 inches in men and greater than 35 inches for woman). The excess fat in the intra-abdominal area is a huge component of the metabolic syndrome. The majority of experts concur that the combination of obesity, obesity-related cytokines called adipokines, excess nutrients and inflammatory cytokines are the main contributors to beta cell death and insulin resistance in type 2 diabetes. Regardless of which event occurred, the mechanisms that are responsible for insulin receptor binding or post receptor can be reversed by weight loss.
  - **Elevated triglycerides (TG) greater than 150 mg/dl.**
  - **Reduced HDL cholesterol** (less than 40 mg/dl in men and less than 50 mg/dl for women).
  - **Fasting blood glucose** (hyperglycemia) greater than 100 mg/dl.
  - **Increased blood pressures** (130/85 mm/Hg or greater).

### Signs and symptoms of cardiovascular disease

Over time, many experienced health care professionals can speculate a potential diagnosis based upon their gut feeling and the symptoms of the patient. However, the best way to truly assess and diagnose the nature of the symptoms is by collecting a thorough history and performing an explicit assessment, because the severity of these symptoms varies. The symptoms may get more severe as the buildup of plaque continues to narrow the coronary arteries. In other instances, some people do not demonstrate any signs and symptoms of an inevitable myocardial infarction, congestive heart failure or an arrhythmia, which is called silent coronary artery disease.

- **Chest pain** is one of the most common complaints in adults and may indicate a cardiovascular disease [43]. Although cardiovascular disease is not always the primary cause of the chest pain, nurses and doctors need to be able to assess the symptomology and nature of the chest pain. Chest pain may be associated with cardiovascular problems, pulmonary, gastrointestinal (GI), musculoskeletal, neurologic, psychogenic or idiopathic causes [24]. Therefore, each of the most common cardiovascular natures of chest pain will be explored and elaborated upon in detail to help differentiate the nature of the pain [43]:
  - **Angina pectoris** chest pain or discomfort occurs when the heart muscle is not getting enough blood [4]. The most common symptom of coronary artery disease is angina, although in some individuals the first sign of CAD is a myocardial infarction. The angina pain is paroxysmal pain in the substernal area that may radiate to the precordium, upper extremities, neck or jaw. Angina pain typically lasts 30 seconds to a few minutes, and patients describe it as dull, pressing, squeezing or aching pain. Angina pain is precipitated by exertion, emotional
stress, sexual activity, exposure to cold and occasionally by eating. However, unstable angina may occur while at rest. Angina pain is alleviated by rest within 10 minutes and/or administration of a nitroglycerin (NTG) sublingual (S/L) tablet within two to four minutes. Typically, angina symptoms last less than 20 minutes (versus greater than 20 minutes with an MI) [13].

○ Myocardial infarction chest pain may be sudden and intense, similar to the “movie heart attack,” where no one doubts what is happening. However, the majority of myocardial infarctions start slowly, with mild pain or discomfort. Often people affected are not significantly sure what is going on. Although they may be aware of the symptoms of an MI, they typically will assume other irrational things are causing their symptoms.

Other common symptoms associated with MI chest pain include any of the following:

■ Chest discomfort. Most heart attacks involve discomfort in the center of the chest that lasts more than a few minutes, or that goes away and comes back. It can feel like uncomfortable pressure, squeezing, fullness or pain.

■ Discomfort in other areas of the upper body. Symptoms can include pain or discomfort in one or both arms, the back, neck, jaw or stomach.

■ Shortness of breath with or without chest discomfort.

■ Other signs may include breaking out in a cold sweat, nausea or light-headedness.

It is important to understand that women enduring an MI may present with subtle or other symptoms. As with men, women’s most common heart attack symptom is chest pain or discomfort. However, women are somewhat more likely than men to experience some of the other common symptoms, particularly shortness of breath, nausea/vomiting and back or jaw pain.

○ Mitral valve prolapse (MVP) chest pain typically occurs during rest and may last for a few minutes to several hours. Patients typically describe MVP as “sticking,” and it may be associated with tachyarrhythmia and light-headedness.

○ Palpitations are typically reported by patients who are at risk of developing cardiovascular disease. Palpitations are defined by the patient reporting the heart beating or skipping beats at times. It is important to distinguish the timing of the palpitations. For instance, if it occurs after exercise, it is probably a normal physiological response due to the increased release of catecholamines. However, more serious pathological causes may be contemplated if the patient verbalizes one of the following comments:

■ Heart “stopped momentarily” may imply an atrial or ventricular ectopic beat.

■ Significant skipping may imply a potential arrhythmia, such as atrial fibrillation.

The nurse should also inquire about other potential symptoms that coincide with the palpitations, such as anxiety, weakness, dizziness and light-headedness; fainting or nearly fainting; sweating; dyspnea; and chest pain.

○ Peripheral arterial disease causes signs or symptoms of or almost half of those who have it, although others may have a number of them.

People who have PAD may have symptoms when walking or climbing stairs. These may include pain, numbness, aching or heaviness in the leg muscles. Symptoms also may include cramping in affected legs and in the buttocks, thighs, calves and feet. Symptoms may ease after resting. These symptoms are called intermittent claudication. During physical activity, muscles need increased blood flow. If blood vessels are narrowed or blocked, they won’t get enough blood. When resting, the muscles need less blood flow, so the pain goes away.

○ About 10 percent of people who have PAD have claudication. This symptom is more likely in people who also have atherosclerosis in other arteries.

■ Weak or absent pulses in the legs or feet.

■ Sores or wounds on the toes, feet or legs that heal slowly, poorly or not at all.

■ A pale or bluish color to the skin.

■ A lower temperature in one leg compared to the other leg.

■ Poor nail growth on the toes and decreased hair growth on the legs.

■ Erectile dysfunction, especially among men who have diabetes.

Health authorities recommend patients discuss the need for screening with their doctor if they are:

■ Aged 70 or older.

■ Aged 50 or older and have a history of smoking or diabetes.

■ Younger than 50 and have diabetes and one or more risk factors for atherosclerosis.

No symptoms?

The nurse should never assume the heart of a patient without symptoms is functioning at the optimal level because some may be asymptomatic initially. The majority of patients with coronary artery disease may be unaware of the existence for
years due to the lack of symptoms, especially the elderly because the body compensates for the atherosclerosis by developing collateral circulation. Research has demonstrated that some patients with coronary artery disease may not develop any symptoms until 75 percent of the coronary artery is narrowed (stenosed).

Once the coronary artery has developed enough stenosis, the primary initial symptom is angina, which should lead the health care professional to immediately speculate coronary artery disease and/or myocardial infarction. Always inquire about other symptoms, such as radiation of chest pain, especially the left arm and jaw; nausea; dyspnea; and light-headedness. Early screening and prevention measures are imperative for early detection.

Screening for and diagnosing cardiovascular disease

Ideally, nurses and physicians should screen patients at risk for cardiovascular disease to try to prevent the development and damage to the patient’s heart muscle and left ventricle. Because of the prevalence of cardiovascular disease globally in all cultures, ages, races and socioeconomic statuses, there are stringent guidelines set aside by professional organizations to screen for cardiovascular disease as follows:

- **Blood pressure** recommendations are guided by the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. The JNC VII recommends accurately assessing the baseline blood pressure (BP) by making sure that the patient is sitting for a minimum of five minutes in a chair (rather than on an exam table), with feet on the floor and arms supported at heart level, and to be sure to use an appropriate sized cuff. At least two measurements should be made on two different occasions before confirming a hypertensive diagnosis.
  - Normal blood pressure is less than 120/80.
    - Prehypertension is 120-139 (systolic) or 80-89 (diastolic).
    - Stage 1 hypertension is 140-159 (systolic) or 90-99 (diastolic).
    - Stage 2 hypertension is greater than 160 (systolic) or 100 (diastolic).
  - For persons older than 50, systolic blood pressure greater than 140 mm/Hg is a much more important cardiovascular disease risk factor than diastolic blood pressure.
    - The risk of cardiovascular disease beginning at 115/75 mm/Hg doubles with each increment of 20/10 mm/Hg; individuals who are normotensive at age 55 have a 90 percent lifetime risk for developing hypertension. Individuals with a systolic blood pressure of 120-139 mm/Hg or a diastolic blood pressure of 80–89 mm/Hg should be considered as prehypertensive and require health-promoting lifestyle modifications to prevent cardiovascular disease.

- **Cholesterol and lipid profile**: The United States Preventive Services Task Force (USPSTF) (2001) and the American Academy of Family Physicians (AAFP) (2006) recommends routine screening of a fasting lipoprotein panel for men aged 20 to 35 and females age 20 to 45 if the patient is at risk for coronary artery disease (hypertension, smoking, DM, family history of coronary artery disease before age 50 in male relatives and age 60 in female relatives and/or family history suggestive of familial hyperlipidemia). The USPSTF and AAFP strongly recommend routine screening for all men at age 35 and women at 45. The National Cholesterol Education Program (NCEP III) (2004) recommends that all men and women aged 20 and over should have a fasting lipoprotein panel completed, then repeat every five years if the results are normal. Desirable or optimal levels for persons with or without existing heart disease are as follows:
  - **Total cholesterol**: Less than 200 milligrams (mg) per deciliter (dL).
  - **Low density lipoprotein (LDL) cholesterol** (“bad” cholesterol): Less than 100 mg/dL.
  - **High density lipoprotein (HDL) cholesterol** (“good” cholesterol): 40 mg/dL or higher. However, the AHA has recommended that men maintain a level above 50 mg/dL and women 60 mg/dL to reduce their individual risk of cardiovascular disease. Higher levels of HDL (“good” cholesterol) provide extra protection against heart disease. Smoking, being overweight and living a sedentary lifestyle can all result in a lower HDL cholesterol level. To raise their HDL levels, individuals should avoid tobacco smoke, maintain a healthy weight and exercise for at least 30-60 minutes more days than not.

- **Triglycerides**: Less than 150 mg/dL. Triglycerides are a form of fat. Many people have high triglyceride levels because of being overweight or obese, physical inactivity, cigarette smoking, excess alcohol consumption or a diet very high in carbohydrates (60 percent or more of calories). High triglycerides are a lifestyle-related risk factor; however, underlying diseases or genetic disorders can be the cause.

- **Coronary artery disease**: The AAFP, AHA and USPSTF all recommend against routine screening for heart disease with routine electrocardiograms (ECG), exercise tolerance test (ETT) or electrobeam computed tomography (CT) for coronary calcium because they aren’t cost-effective. According to an editorial submitted by the AAFP, cardiac CT is expensive ($400 to $500 per scan) and thus far has not been proven to affect patient-oriented outcomes. At this time, research has not demonstrated that the cost is beneficial for asymptomatic patients.
  - However, the AHA does recommend screening for patients with an intermediate risk based upon the Framingham score. The Framingham risk score assesses an individual’s risk of developing cardiovascular disease over a 10-year time frame based upon their age, diabetes, smoking, JNC-7 blood pressure category, NCEP cholesterol levels and total LDL. Each category correlates to a percentage range that is added up at the end of the seven categories to determine an individual’s risk level.
  - **Diabetes screening**: Since 2003, the USPSTF has recommended that adults with high blood pressure
or high cholesterol be screened for type 2 diabetes (insulin-resistant diabetes) as part of an integrated approach to reduce their risk of cardiovascular disease, but concluded that further research is needed to determine whether widespread screening of the general population would improve health outcomes. The ADA guidelines are partially congruent with the previous recommendations that screening should begin with a FBG or OGTT every three years, beginning at the age of 45, especially if BMI is greater than 25. According to the ADA, diabetes is diagnosed if:

- A fasting blood glucose greater than 126 mg/dl (no caloric intake for over eight hours) on two or more occasions (a normal fasting blood glucose is less than 100 mg/dl).
- A random plasma glucose concentration greater than 200mg/dl taken at any time, regardless of the last meal.
- A two-hour plasma oral glucose tolerance test (OGTT) greater than 200 mg/dl (two hours after ingesting 75 g of a glucose load). A normal fasting blood glucose level for non-diabetics is 70-110 mg/dl.

When cardiovascular disease is speculated, the physician or nurse practitioner will order specific tests to confirm a potential cardiac diagnosis. If the patient presents with potential life-threatening emergency cardiac symptoms, the patient will be immediately transferred via ambulance to the closest emergency room (ER). Other specific tests that may be ordered are:

- **B-type natriuretic peptide (BNP)**, which is a hormone produced and released by the ventricles secondary to volume and pressure overload, thus decreasing preload [16, 46]. BNP increases sodium and water excretion and signifies congestive heart failure. Therefore, it is a useful test to distinguish between congestive heart failure and other causes of shortness of breath. A normal level of BNP is less than 100 picograms per milliliter (pg/ml).

- **C-reactive protein (CRP)**, which is a marker of inflammatory process and may aide in the prediction of coronary events and signifies atherosclerosis. CRP is a protein produced by the liver and the smooth muscle cells within the atherosclerotic coronary arteries [24]. Although the CRP is a good predictor, it should not be relied on solely because it is only one piece to the puzzle of predicting coronary artery disease, myocardial infarction and cerebrovascular accident.

- **Cardiac catheterization** is the most definitive, invasive diagnostic test to aide in the diagnosis of cardiovascular disease. During the procedure, a thin, flexible tube is passed through an artery in the groin or arm to reach the coronary arteries to assess for any blockage, coronary artery disease and myocardial and valvular function.

- **Chest radiograph (CXR)** provides a picture of the organs and structures inside the chest to determine the size, silhouette and position of the heart.

- **Creatine kinase (CK)** is an enzyme specific to cells of the brain, myocardium and skeletal muscle. If a patient tests positive for CK, it indicates tissue necrosis or injury somewhere in the body. However, in order to definitely assess for cardiac necrosis or injury, the nurse needs to be familiar with the three types of isoenzymes:
  - CK-MM is the predominant isoenzyme of skeletal muscle.
  - CK-MB is found in the myocardial muscle and is most specific for demonstrating an MI.
  - CK-BB is found in the brain.

- **Echocardiogram (ECHO)** is a noninvasive test that allows cardiologists to assess the function of the valves and structure of the heart by visually observing and taking measurements via an ultrasound device.

- **Electrocardiogram (ECG or EKG)** measures the electrical function, rate and regularity of the patient’s heart rhythm. The ECG is a useful tool to distinguish between different types of arrhythmias and myocardial infarction, and demonstrates left ventricular hypertrophy and coronary artery disease.

- Any patient who presents with chest pain, is over 40 or has a history of the major heart ailments should have an ECG to assess and differentiate cardiac ischemia, injury, infarct, myocardial infarction or left ventricular hypertrophy:
  - Myocardial ischemia will result in the inversion of the T-wave.
  - Injury to the myocardial cells is more severe than ischemia and is manifested by ST segment depression or ST elevation.
  - MI (infarction) implies necrosis or death of the myocardial cells secondary to atherosclerosis. In the majority of cases, the left ventricle (LV) is the major site for infarction; however, it may occur in the right ventricle (RV).
  - Q-wave MI is defined as an initial downward deflection of a duration of 40 msec or more in any lead except III and a VR.
  - Non-Q-wave MI may be diagnosed in the presence of ST depression and T-wave abnormalities.

- Regardless, the doctor will order cardiac enzymes to assess for MI. In the absence of enzyme elevation, ST and T-wave abnormalities are interpreted as due to injury or ischemia rather than infarction.

- Left ventricular hypertrophy is complex to assess and more applicable for the cardiologists to definitively speculate a diagnosis based upon the height of the R and S waves. It is definitely diagnosed based upon the thickness of the wall measured during an echocardiogram (ECHO).

- **Homocysteine** is an amino acid that is produced when proteins are broken down. An elevated level may be a risk factor for cardiovascular disease. However, the correlation is still controversial.

- **Myoglobin** is another early marker of an MI; however, it is not specific to the heart. Myoglobin is a low-molecular protein found in the cardiac and skeletal muscle.

- **Stress test** is useful in assessing for ischemia caused by fixed coronary lesions, but it may provide inaccurate readings in patients at high risk or with coronary artery disease. Research studies have demonstrated that patients with coronary artery disease may have a false-positive, and patients in a high-risk category for developing it may have false-negatives. At that time, the patient’s cardiologists...
would need to evaluate the risk and history of the individual to decide whether further testing is warranted.

Peripheral arterial disease diagnosis is based on medical and family histories, a physical exam and results from tests. Medical and family histories may include questions about:
- Whether the patient has any risk factors for PAD.
- Symptoms, including any that occur when walking, exercising, sitting, standing or climbing.
- Diet.
- Medications the person takes, including prescription and over-the-counter medicines.
- Whether there is a family history of cardiovascular disease.

During the physical exam, the doctor will look for signs and symptoms of PAD. He or she may check the blood flow in the patient’s legs or feet to see whether there are weak or absent pulses. The doctor also may check the pulses in leg arteries for an abnormal whooshing sound called a bruit, which can be heard with a stethoscope. A bruit may be a warning sign of a narrowed or blocked section of artery. The physician also may compare blood pressure between the limbs to see whether the pressure is lower in the affected limb or check for poor wound healing or any changes in hair, skin or nails that may be signs of PAD.

Diagnostic tests may include:
- **Ankle-brachial index**
  - The ankle-brachial index (ABI) compares blood pressure in the ankle to blood pressure in the arm, which shows how well blood is flowing in the limbs. While the ABI can show whether PAD is affecting a person’s limbs, it won’t show which blood vessels are narrowed or blocked.
  - A normal ABI result is 1.0 or greater (with a range of 0.90 to 1.30). The test takes about 10 to 15 minutes to measure both arms and both ankles. This test may be done yearly to see whether PAD is getting worse.

- **Doppler ultrasound**
  - A Doppler ultrasound is a test that uses sound waves to show whether a blood vessel is blocked. This test uses a blood pressure cuff and special device to measure blood flow in the veins and arteries of the limbs. A Doppler ultrasound can help find out how severe PAD is.

- **Treadmill test**
  - A treadmill test can show how severe the symptoms are and what level of exercise brings them on. For this test, the patient walks on a treadmill to show whether there are any problems during normal walking.
  - An ABI test may be done before and after the treadmill test to compare blood flow in the arms and legs before and after exercise.

- **Magnetic resonance angiogram**
  - A magnetic resonance angiogram (MRA) uses magnetic and radio wave energy to take pictures of blood vessels inside the body. It can find the location of a blocked blood vessel and show how severe the blockage is.
  - Patients who have a pacemaker, man-made joint, stent, surgical clips, mechanical heart valve or other metallic devices in the body might not be able to have an MRA.

- **Arteriogram**
  - An arteriogram provides a “road map” of the arteries to find the exact location of a blocked artery.
  - For this test, dye is injected through a needle or catheter into an artery. The patient may feel mildly flushed. After the dye is injected, an x-ray is taken, which can show the location, type and extent of the blockage in the artery.
  - Some hospitals use a newer method of arteriogram that uses tiny ultrasound cameras that take pictures of the insides of the blood vessels. This method is called intravascular ultrasound.

- **Blood tests**
  - These may be done to check for PAD risk factors, for example, blood glucose levels to check for diabetes or lipid tests to check cholesterol levels.

Overall strategies for prevention of cardiovascular disease

Because of the significant prevalence of cardiovascular disease globally, it is imperative to consider primary prevention before cardiovascular diseases begin. In order to reduce the risk of developing cardiovascular disease, it is imperative to initiate healthy lifestyle choices. The Centers for Disease Control (CDC) and World Health Organization (WHO) recommend the best way to fight heart disease is a healthy diet and lifestyle. Healthy lifestyle choices for all people include exercising, not smoking and eating nutritious foods. Although it may appear difficult, it is all about making healthy choices to protect the heart and to be able to live life to the fullest.

There are a few dietary recommendations that overlap, yet all have one goal in mind: protecting the heart. Since 1998, then revised in 2006, the U.S. Department of Health and Human Services has encouraged Americans to adhere to a dietary approach to stop hypertension, called DASH. Another common diet that nurses may be familiar with in the hospital setting is the cardiac diet initiated by the American Heart Association and updated in 2006. The AHA, CDC and WHO have elaborated upon their diet recommendations by encouraging individuals to adhere to the following:
- **Consume a diet high in fruits, vegetables, fiber, nuts and whole grains, and low in refined grains.** Fruits and vegetables have folate, vitamin B6 and B12 to reduce the total homocysteine level, especially if the patient is at risk for developing cardiovascular disease.
- **Choose lean meats and poultry without skin and prepare them without added saturated and trans fat.** Limit energy intake from total fats and shift fat consumption away from saturated fats to unsaturated fats and towards the elimination of trans-fatty acids.
- **Select fat-free, 1 percent fat and low-fat dairy products.**
- **Increase consumption of omega-3 fatty acids from fish oil or plant sources to at least twice a week.** Recent research has demonstrated that eating oily fish containing omega-3 fatty acids (for example, salmon, trout and herring) may help limit an individual’s risk of death from coronary artery disease.
- Limit foods containing partially hydrogenated vegetable oils to reduce trans fat in the diet.
- Limit the total dietary cholesterol in a diet, with the goal to eat less than 300 mg of cholesterol every day.
- Limit the consumption of beverages and foods with added sugars.
- Limit the amount of alcohol consumed to no more than one drink per day for women and two drinks per day for men.
- Choose and prepare foods with little or no salt. Americans should aim to eat less than 2,300 mg of sodium per day (or less than 1,500 mg if there is a higher risk of being affected by hypertension for those already diagnosed).
  - The DASH diet reiterates the CDC and WHO recommendations by further elaborating that 2,300 mg is the highest level considered acceptable by the National High Blood Pressure Education Program. It is also the highest amount recommended for healthy Americans by the 2005 U.S. Dietary Guidelines for Americans. A 1,500 mg level can lower blood pressure further, and more recently is the amount recommended by the Institute of Medicine (IOM) as an adequate intake level and one that most people should try to achieve. In 2009, the CDC reiterated that patients at higher risk of developing cardiovascular disease and/or hypertension (including people 40 or older, African Americans or those currently hypertensive) should limit their total sodium intake to less than 1,500 mg/day. In 2009, a CDC report said two out of three (69 percent) adults in the U.S. fall into these three groups who are at especially high risk for health problems from consuming too much sodium.
  - Eating less sodium can help prevent, lower or even control blood pressure. Most of the sodium consumed comes from packaged, processed, store-bought and restaurant foods. Only about 5 percent comes from salt added during cooking, and about 6 percent comes from being added at the table. All nutritional information facts are available on food products and available at any restaurant.
  - Additional research has demonstrated that most Americans eat a lot more than the recommended sodium intake; men currently eat about 4,200 mg a day, and women consume 3,300 mg [45].

The recommended daily nutrient goals in the Third Report of the National Cholesterol Education Program (NCEP) and DASH to reduce the overall weight, blood pressure, lipid profile and to promote a healthy heart are very similar in the specific recommendations:
- Total fat, 27 percent of calories (DASH); 25-35 percent (NCEP).
- Saturated fat, 6 percent of calories (DASH); less than 7 percent of calories (NCEP).
- Polyunsaturated fat, up to 10 percent of calories (NCEP).
- Monounsaturated fat, up to 20 percent of calories (NCEP).
- Sodium, 2,300 mg; however 1,500 mg is ideal, especially to maintain a healthy blood pressure.
- Potassium, 4,700 mg.
- Protein, 18 percent of calories (DASH); 15 percent of calories (NCEP).
- Calcium, 1, 250 mg.
- Carbohydrates, 55 percent of calories (DASH); 50-60 percent of calories (NCEP).
- Magnesium, 500 mg.
- Cholesterol, 150 mg (DASH); less than 200 mg/day (NCEP).
- Fiber, 30 grams (g)/day (DASH); 20-30 g/day (NCEP).

NCEP is supported by the Detection, Evaluation and Treatment of High Blood Cholesterol in Adults; Adult Treatment Plan (ATP III) designated by the therapeutic lifestyle changes.

Although the majority of patients with cardiovascular disease may be considered overweight or obese based upon their BMI, it may occur in the thin patient as well. Therefore, nurses should never assume by the body size alone that an individual is “healthy.” The JNC VII recommends patients should maintain a normal body weight with a BMI of 18.5 to 24.9 kg/m2 to help maintain a normal blood pressure. In addition to eating healthy, all individuals should be encouraged to exercise for at least 30 minutes every day. The NCEP and the AHA have elaborated upon their recommendations based upon the following research:

Regular physical activity reduces very low density lipoprotein (VLDL) levels, raises HDL cholesterol and in some people, lowers the LDL levels. It also can lower blood pressure, reduce insulin resistance and improve the function of the heart. It should be important for the health care provider to find a level of activity that a patient can accomplish over the long term. The American Academy of Family Physicians (AAFP) recommends a combination of resistance and aerobic exercise, but any activity is better than none, and patients who have been sedentary need to start with walking and gradually increase duration and intensity.

Ideally, the AHA recommends regular aerobic physical activity to increase an individual’s overall fitness level and capacity for exercise. It also plays a role in both primary and secondary prevention of cardiovascular disease. Physical inactivity is a major risk factor for heart disease and stroke and is linked to cardiovascular mortality. Regular physical activity can help control blood lipid abnormalities, diabetes and obesity. Aerobic physical activity can also help reduce blood pressure. Therefore, in order to achieve health benefits to the heart, lungs and circulation, an individual must perform any moderate-to-vigorous-intensity aerobic activity for at least 30 minutes on most days of the week at 50-85 percent of his or her maximum heart rate. One can accumulate 30 minutes in 10- or 15-minute sessions. It is important to include physical activity as part of a regular routine.

- Ideal examples of aerobic activities that increase endurance include brisk walking, jumping rope, jogging, bicycling, rowing, swimming, cross-country skiing and dancing.

Ideally, nurses and physicians should elaborate upon recommended food choices, smoking cessation and choosing an active lifestyle to all their patients, regardless of their current risk of developing cardiovascular disease, because ultimately everyone is at risk at some point. Although we cannot change our risks due to our age, gender and genetics, we can choose a healthier life.
Treatment of cardiovascular diseases

Since 2001, the Joint Commission (JCAHO) has initiated core measures to be implemented by all hospitals in the U.S. to measure and improve patient care with certain diagnoses. JCAHO has specified guidelines for nurses and doctors to implement for patients with acute myocardial infarction and congestive heart failure upon admission to the hospital and discharge. In 2009, JCAHO aligned with the Centers for Medicare and Medicaid Services (CMS) to write a specific manual to treat both of these serious cardiovascular diseases. The mandatory 2009 guidelines and treatment modalities for patients presenting with an acute myocardial infarction are:

- If an acute myocardial infarction is speculated upon arrival at the hospital:
  - Administer the patient aspirin (ASA) to prevent and dissolve any potential clots circulating in the bloodstream.
  - Administer some form of reperfusion therapy to the patient, either with fibrinolytic therapy or percutaneous coronary intervention (PCI), especially if the patient has ST-segment elevation myocardial infarction (STEMI). Fibrinolysis should be provided within 30 minutes of first medical system contact, and primary PCI should be provided within 90 minutes for patients presenting with STEMI.
  - Contraindications include previous hemorrhagic cerebrovascular accident or other strokes, cerebrovascular events within the past year, known intracranial neoplasm, active internal bleeding (excluding menstruation) or suspected aortic disease.
  - Since 1993, thrombolytic agents have been available and work by converting plasminogen to the natural fibrinolytic agent plasmin. Plasmin lyases clots by breaking down the fibrinogen and fibrin contained in the clot. Fibrinolytics, sometimes referred to as plasminogen activators, are divided into two categories.
  - Fibrin-specific agents, including alteplase, reteplase and tenecteplase, produce limited plasminogen conversion in the absence of fibrin, whereas nonfibrin-specific agents such as streptokinase catalyze systemic fibrinolysis. Streptokinase is indicated for the treatment of an acute myocardial infarction. Streptokinase is not widely used in the U.S. because it is not as effective in opening occluded arteries and less effective in reducing mortality. However, it is used in other countries because of its cheaper cost. Alteplase is the only current lytic agent the U.S. Food and Drug Administration (FDA) has approved for acute myocardial infarction.
  - Tissue plasminogen activator (tPA) is a naturally occurring fibrinolytic agent found in vascular endothelial cells and is involved in the balance between thrombolysis and thrombogenesis. At the site of the thrombus, the binding of tPA and plasminogen to the fibrin surface induces a conformational change, facilitating the conversion of plasminogen to plasmin and dissolving the clot.

- During the hospitalization, the nurse needs to make sure of the following:
  - The function of the left ventricle has been documented in the medical record.
  - Initiate and administer an angiotensin converting enzyme (ACE) or angiotensin receptor blocker (ARB) for left-ventricle dysfunction that is less than 40 percent.
  - ACE/ARBs have demonstrated short and long-term improvements in surviving an MI, especially if a patient has a low EF. ACE inhibitors block the conversion of angiotensin I to the vasoconstrictor angiotensin II. Therefore, it lowers the blood pressure and prevents vasoconstriction, thus increasing blood flow, especially to the kidneys.
  - Educate and provide counseling on adult smoking cessation to prevent further damage to the heart muscle and to possibly prevent another MI.

- Upon discharge, the nurse needs to check that the patient is provided education and a prescription for ASA, ACE or ARB and a beta-blocker (BB). A lipid-lowering agent may be prescribed, depending on the physician’s recommendations and the lipid profile results.
  - ASA is beneficial because of its anti-platelet, blood thinner effect.
  - ACE/ARBs, as noted above, offer short- and long-term improvements.
  - Beta-blockers block the stimulation of beta 1 (myocardial) and beta 2 (pulmonary, vascular and uterine) receptor sites, thus lowering the overall blood pressure, suppressing arrhythmias and reducing the risk of a future MI because the overall contractility is reduced.

- The mandatory 2009 guidelines and treatment modalities for the patient presenting in heart failure are:
  - During the hospitalization, assess the function of the left ventricle and inform the patient, then initiate an ACE inhibitor or ARB for left ventricular dysfunction because the vasodilation effect reduces the risk of mortality by 20 percent (especially if the EF is less than 40 percent).
  - Provide smoking cessation education.
  - Upon discharge, the nurse needs to ensure patients are provided explicit details on managing their heart failure, such as activity, diet, medications, follow-up appointments, weight checks and options if their symptoms worsen. It is imperative that nurses stress the importance of the treatment and prevention of further attacks because the prognosis of congestive heart failure is poor. The five-year survival rate is less than 50 percent overall. Other treatment modalities not listed by JCAHO include:

  - Diuretic therapy. Research has demonstrated that diuretics are the most effective means of providing symptomatic relief in patients with congestive heart failure. Ideally, diuretics and ACE inhibitors block sodium reabsorption in the loop of Henle and in the proximal portion of the tubule. Initially, most patients are started on a thiazide diuretic and an ACE inhibitor. As the condition worsens, the diuretic will be changed to a loop diuretic due to the rapid
onset and shorter duration. One of the biggest side effects of the combination of an ACE and diuretic is hypotension, so patients normally are always started with a lower dose and slowly worked up to assess for this risk.

- Beta blockers may also be initiated because of a significant rise in the EF (averaging 10 percent) and reduction in the left ventricle size and mass.

- Other potential treatments include:
  - **Coronary artery disease.** It is imperative to reduce further progression of atherosclerosis, and in some patients, if properly treated, it may even regress. Therefore, if the patient clinically demonstrates an abnormal lipid profile implying atherosclerosis and risk of coronary artery disease, the following medications should be initiated [34]:
    - Hydroxymethylglutaryl coenzyme A (HMG-CoA), reductase inhibitors (statins) to prevent death, coronary events and CVA in patients with high cholesterol and/or high LDL and coinciding diabetes. Other cholesterol-lowering medications include bile acid sequestrants (but contraindicated if a patient has high triglycerides), fibrates to lower triglycerides and ezetimibe.
    - Niacin is beneficial for patients with abnormally low HDL or elevated lipoproteins.
    - Folic acid 1 milligram (mg)/day is beneficial in the treatment of elevated homocysteine levels to reduce the risk of vascular events. However, adding Vitamins B6 and B12 supplements demonstrate limited or no value in preventing vascular events.
    - Aspirin (ASA) 325 mg every other day, anti-platelet therapy in patients over the age of 50 is beneficial in reducing the risk of an MI. It is imperative to recommend a dose of 325 mg if the patient has no other risk and/or contraindicating co-morbidities because research has demonstrated that 100 mg every other day did not prevent MI in women 45 years and older.
    - Clopidogrel (Plavix) has been effective in preventing vascular episodes for nine to 12 months after acute coronary syndrome, yet it has not prevented vascular events.
  - **Hypertension.** According to the JNC VII, it is imperative to treat systolic (SBP) and diastolic blood pressure (DBP) to targets that are less than 140/90 mm/Hg, which is associated with a decrease in cardiovascular disease complications. However, in patients with hypertension and diabetes or renal disease, the BP goal is lower than 130/80 mm/Hg [39]. It is important to recognize and treat patients appropriately for hypertension to reduce further complications. Numerous research projects have demonstrated that medications should be initiated in patients based upon their blood pressure and overall cardiovascular risk:
    - Thiazide-type diuretics are the initial drug of choice in lowering the overall blood pressure by decreasing the plasma volume (by suppressing the tubular reabsorption of sodium, thus increasing the excretion of sodium and water) and CO. Diuretics lower the blood pressure in 50 percent of patients with mild to moderate hypertension. If dual therapy is required, diuretics should be used in combination with a potassium-sparing agent or an ACE/ARB. ACE inhibitors are ideal in mild to moderate elevations of blood pressure in white individuals. Research has demonstrated limited benefits in blacks and older patients with SBP. However, ACE inhibitors are beneficial in patients with type 1 diabetes with any renal dysfunction because they prevent further progression from the vasodilation effect.
  - The nurse should assess for the following:
    - Diuretics also increase the risk of uric acid, thus predisposing the patient to gout. Other potential side effects include but are not limited to hyperglycemia and elevated triglycerides, LDL and cholesterol.
    - ACE inhibitors may cause a new-onset diabetes, cough, hypotension, renal dysfunction, hyperkalemia, taste alteration and rash. ARBs are initiated if the patient develops a cough caused by the ACE inhibitor; no coughs or rashes are associated with ARBs.
    - Beta blockers also reduce the blood pressure in 50 percent of patients.
  - The nurse should assess for hypoglycemia because the symptoms are masked in type 1 diabetics.
  - **Arrhythmias:** Common arrhythmia treatments include medicines, medical procedures and surgery. Treatment is needed when an arrhythmia causes serious symptoms, such as dizziness, chest pain or fainting. Treatment also is needed if an arrhythmia increases the risk for complications, such as heart failure, stroke or sudden cardiac arrest.
    - **Medications:** Anti-arrhythmics can be used to speed up a heart that’s beating too slow or slow down a heart that’s beating too fast. They also can be used to convert an abnormal heart rhythm to a normal, steady rhythm. These include:
      - Beta blockers (such as metoprolol and atenolol), calcium channel blockers (such as diltiazem and verapamil), and digoxin (digitalis). These medicines often are used to slow the heart rate in people who have atrial fibrillation.
      - Medicines used to restore an abnormal heartbeat to a normal rhythm are amiodarone, sotalol, flecainide, propafenone, dofetilide, ibutilide, quinidine, procainamide and disopyramide. These medicines often have side effects, some of which can make an arrhythmia worse or even cause a different kind of arrhythmia.
      - People who have atrial fibrillation and some other arrhythmias often are treated with anticoagulants, or blood thinners, to reduce the risk of blood clots forming. Aspirin, warfarin (Coumadin®) and heparin are commonly used blood thinners.
      - Medicines also can control an underlying medical condition, such as heart disease or a thyroid condition, that might be causing an arrhythmia.
Medical procedures:
- Some arrhythmias are treated with a pacemaker, a small device that’s placed under the skin of the chest or abdomen to help control abnormal heart rhythms. This device uses electrical pulses to prompt the heart to beat at a normal rate. Most pacemakers contain a sensor that activates the device only when the heartbeat is abnormal.
- Some arrhythmias are treated with a jolt of electricity delivered to the heart. This type of treatment is called cardioversion or defibrillation, depending on which type of arrhythmia is being treated.
- Some people who are at risk for ventricular fibrillation are treated with a device called an implantable cardioverter defibrillator (ICD). Like a pacemaker, an ICD is a small device that’s placed under the skin in the chest. This device uses electrical pulses or shocks to help control life-threatening arrhythmias. An ICD continuously monitors the heartbeat. If it senses a dangerous ventricular arrhythmia, it sends an electric shock to the heart to restore a normal heartbeat.
- A procedure called catheter ablation is sometimes used to treat certain types of arrhythmia when medicines don’t work. During this procedure, a long, thin, flexible tube is put into a blood vessel in the arm, groin (upper thigh), or neck and guided to the heart through the blood vessel. A special machine sends energy through the tube to the heart. This energy finds and destroys small areas of heart tissue where abnormal heartbeats may cause an arrhythmia to start. Catheter ablation usually is done in a hospital as part of an electrophysiology study.

Surgery:
- Sometimes, an arrhythmia is treated with surgery. This often occurs when surgery is already being done for another reason, such as repair of a heart valve.
  - One type of surgery for atrial fibrillation is called “maze” surgery. In this operation, the surgeon makes small cuts or burns in the atria that prevent the spread of disorganized electrical signals.
  - If coronary heart disease is causing arrhythmias, coronary artery bypass grafting may be recommended. This surgery improves blood supply to the heart muscle.

Other treatments: Vagal maneuvers are another arrhythmia treatment. These simple exercises sometimes can stop or slow down certain types of supraventricular arrhythmias. They do this by affecting the vagus nerve, which helps control the heart rate. Some vagal maneuvers include:
- Gagging.
- Holding your breath and bearing down (Valsalva maneuver).
- Immersing the face in ice-cold water.
- Coughing.
- Putting fingers on the eyelids and pressing down gently. Vagal maneuvers aren’t an appropriate treatment for everyone. Before recommending, consult with the patient’s doctor.

Peripheral arterial disease
- Although PAD is serious, the underlying atherosclerosis can be treated. PAD treatment may slow or stop disease progress and reduce the risk of complications. Lifestyle changes, as noted above, can include to quit smoking, eat a healthy diet and exercise, and lower risk factors including blood pressure, cholesterol levels and blood glucose levels.
- Medications may include those listed above to lower blood cholesterol levels and for hypertension, blood thinners and pain medications to ease leg pain when walking or climbing steps.
- Surgical procedures may include:
  - Bypass grafting if the blood flow is blocked or nearly blocked. A blood vessel from another part of the patient’s body or man-made tubes may be used. The graft bypasses the blocked part of the artery. It doesn’t cure PAD but can increase blood flow and functioning to the affected limb.
  - Angioplasty may be utilized, in which a catheter with a balloon or other device is inserted into the artery. The balloon is inflated to push the plaque outward against the wall of the artery, which widens it and restores blood flow. A stent may be placed into the artery during angioplasty.

Conclusion
Because of the lifestyle of Americans and access to fast foods on every corner, heart disease will continue to rise unless people make better choices. Although we may have the ability to consume a high fat diet, smoke and remain inactive, heart disease will continue to compound and cause many deaths unless we take control of our lives. In addition to educating the public and the patients we care for as nurses, we need to also practice what we preach. Nurses are also potentially at risk while working long, stressful shifts and grabbing food quickly on the job. However, it is important to realize the potential consequences and complications of choosing to make unhealthy decisions in our personal lives and while working. Every one of us holds the key to make the appropriate changes to reduce our overall risk of being affected by cardiovascular disease.
PART II: STROKES AND CEREBROVASCULAR DISEASES

Introduction

More than 2,400 years ago, the father of medicine, Hippocrates, recognized and described stroke – the sudden onset of paralysis. Until recently, modern medicine has had very little power over this disease, but the world of stroke medicine is changing, and new and better therapies are being developed every day. Today, some people who have a stroke can walk away from the attack with no or few disabilities if they are treated promptly. Doctors can finally offer stroke patients and their families the one thing that until now has been so hard to give: hope.

In ancient times stroke was called “apoplexy,” a general term that physicians applied to anyone suddenly struck down with paralysis. Because many conditions can lead to sudden paralysis, the term apoplexy did not indicate a specific diagnosis or cause. Physicians knew very little about the cause of stroke, and the only established therapy was to feed and care for the patient until the attack ran its course.

The first person to investigate the pathological signs of apoplexy was Johann Jacob Wepfer. Born in Schaffhausen, Switzerland, in 1620, Wepfer studied medicine and was the first to identify postmortem signs of bleeding in the brains of patients who died of apoplexy. From autopsy studies, he gained knowledge of the carotid and vertebral arteries that supply the brain with blood. He also was the first person to suggest that apoplexy, in addition to being caused by bleeding in the brain, could be caused by a blockage of one of the main arteries supplying blood to the brain; thus stroke became known as a cerebrovascular disease (“cerebro” refers to a part of the brain; “vascular” refers to the blood vessels and arteries).

Medical science would eventually confirm Wepfer’s hypotheses, but until very recently, doctors could offer little in the area of therapy. Over the last two decades, basic and clinical investigators, including many sponsored and funded in part by the National Institute of Neurological Disorders and Stroke (NINDS), have learned a great deal about stroke. They have identified major risk factors for the disease and have developed surgical techniques and drug treatments for the prevention of stroke. But perhaps the most exciting new development in the field of stroke research is the approval of a drug treatment that can reverse the course of stroke if given during the first few hours after the onset of symptoms.

Studies with animals have shown that brain injury occurs within minutes of a stroke and can become irreversible within as little as an hour. In humans, brain damage begins from the moment the stroke starts and often continues for days afterward. Scientists now know that there is a very short window of opportunity for treatment of the most common form of stroke. Because of these and other advances in the field of cerebrovascular disease, stroke patients now have a chance for survival and recovery.

What is stroke?

A stroke occurs when the blood supply to part of the brain is suddenly interrupted or when a blood vessel in the brain bursts, spilling blood into the spaces surrounding brain cells. In the same way that a person suffering a loss of blood flow to the heart is said to be having a heart attack, a person with a loss of blood flow to the brain or sudden bleeding in the brain can be said to be having a “brain attack.”

Brain cells die when they no longer receive oxygen and nutrients from the blood or when they are damaged by sudden bleeding into or around the brain. Ischemia is the term used to describe the loss of oxygen and nutrients for brain cells when there is inadequate blood flow. Ischemia ultimately leads to infarction, the death of brain cells that are eventually replaced by a fluid-filled cavity (or infarct) in the injured brain.

When blood flow to the brain is interrupted, some brain cells die immediately, while others remain at risk for death. These damaged cells make up the ischemic penumbra and can linger in a compromised state for several hours. With timely treatment, these cells can be saved.

Even though a stroke occurs in the unseen reaches of the brain, the symptoms of a stroke are easy to spot. They include sudden numbness or weakness, especially on one side of the body; sudden confusion or trouble speaking or understanding speech; sudden trouble seeing in one or both eyes; sudden trouble walking, dizziness, or loss of balance or coordination; or sudden severe headache with no known cause. All of the symptoms of stroke appear suddenly, and often there is more than one symptom at the same time. Therefore, stroke can usually be distinguished from other causes of dizziness or headache. These symptoms may indicate that a stroke has occurred and that medical attention is needed immediately.

There are two forms of stroke:
- **Ischemic** – blockage of a blood vessel supplying the brain.
- **Hemorrhagic** – bleeding into or around the brain.

Ischemic stroke

An ischemic stroke occurs when an artery supplying the brain with blood becomes blocked, suddenly decreasing or stopping blood flow and ultimately causing a brain infarction. This type of stroke accounts for approximately 80 percent of all strokes. Blood clots are the most common cause of artery blockage and brain infarction. The process of clotting is necessary and
beneficial throughout the body because it stops bleeding and allows repair of damaged areas of arteries or veins. However, when blood clots develop in the wrong place within an artery they can cause devastating injury by interfering with the normal flow of blood. Problems with clotting become more frequent as people age.

Blood clots can cause ischemia and infarction in two ways. A clot that forms in a part of the body other than the brain can travel through blood vessels and become wedged in a brain artery. This free-roaming clot is called an embolus and often forms in the heart. A stroke caused by an embolus is called an embolic stroke. The second kind of ischemic stroke, called a thrombotic stroke, is caused by thrombosis, the formation of a blood clot in one of the cerebral arteries that stays attached to the artery wall until it grows large enough to block blood flow.

**Hemorrhagic stroke**

In a healthy, functioning brain, neurons do not come into direct contact with blood. The vital oxygen and nutrients the neurons need from the blood come to the neurons across the thin walls of the cerebral capillaries. The glia (nervous system cells that support and protect neurons) form a blood-brain barrier, an elaborate meshwork that surrounds blood vessels and capillaries and regulates which elements of the blood can pass through to the neurons.

When an artery in the brain bursts, blood spews out into the surrounding tissue and upsets not only the blood supply but also the delicate chemical balance neurons require to function. This is called a hemorrhagic stroke. Such strokes account for approximately 20 percent of all strokes.

Hemorrhage can occur in several ways. One common cause is a bleeding aneurysm, a weak or thin spot on an artery wall. Over time, these weak spots stretch or balloon out under high arterial pressure. The thin walls of these ballooning aneurysms can rupture and spill blood into the space surrounding brain cells.

Hemorrhage also occurs when arterial walls break open. Plaque-encrusted artery walls eventually lose their elasticity and become brittle and thin, prone to cracking. Hypertension increases the risk that a brittle artery wall will give way and release blood into the surrounding brain tissue.

A person with an arteriovenous malformation (AVM) also has an increased risk of hemorrhagic stroke. AVMs are a tangle of defective blood vessels and capillaries within the brain that have thin walls and can therefore rupture.

Bleeding from ruptured brain arteries can either go into the substance of the brain or into the various spaces surrounding the brain. Intracerebral hemorrhage occurs when a vessel within the brain bleeds into the brain itself. Subarachnoid hemorrhage is bleeding under the meninges, or outer membranes, of the brain into the thin fluid-filled space that surrounds the brain.

The subarachnoid space separates the arachnoid membrane from the underlying pia mater membrane. It contains a clear fluid (cerebrospinal fluid or CSF) as well as the small blood vessels that supply the outer surface of the brain. In a subarachnoid hemorrhage, one of the small arteries within the subarachnoid space bursts, flooding the area with blood and contaminating the cerebrospinal fluid. Since the CSF flows throughout the cranium, within the spaces of the brain, subarachnoid hemorrhage can lead to extensive damage throughout the brain. In fact, subarachnoid hemorrhage is the most deadly of all strokes.

**Transient ischemic attacks**

A transient ischemic attack (TIA), sometimes called a mini-stroke, starts just like a stroke but then resolves, leaving no noticeable symptoms or deficits. The occurrence of a TIA is a warning that the person is at risk for a more serious and debilitating stroke. Of the approximately 50,000 Americans who have a TIA each year, about one-third will have an acute stroke sometime in the future. The addition of other risk factors compounds a person’s risk for a recurrent stroke. The average duration of a TIA is a few minutes. For almost all TIAS, the symptoms go away within an hour. There is no way to tell whether symptoms will be just a TIA or persist and lead to death or disability. The patient should assume that all stroke symptoms signal an emergency and should not wait to see if they go away.

**Recurrent stroke**

Recurrent stroke is frequent; about 25 percent of people who recover from their first stroke will have another stroke within five years. Recurrent stroke is a major contributor to stroke disability and death, with the risk of severe disability or death
Physicians have several diagnostic techniques and imaging tools to help diagnose the cause of stroke quickly and accurately. The first step in diagnosis is a short neurological examination. When a possible stroke patient arrives at a hospital, a health care professional, usually a doctor or nurse, will ask the patient or a companion what happened and when the symptoms began. Blood tests, an electrocardiogram and a brain scan, such as CT or MRI, will often be done. One test that helps doctors judge the severity of a stroke is the standardized NIH Stroke Scale, developed by the NINDS. Health care professionals use the NIH Stroke Scale to measure a patient’s neurological deficits by asking the patient to answer questions and to perform several physical and mental tests. Other scales include the Glasgow Coma Scale, the Hunt and Hess Scale, the Modified Rankin Scale, and the Barthel Index. A copy of the NIH Stroke Scale can be found at the end of this chapter.

Health care professionals also use a variety of imaging devices to evaluate stroke patients. The most widely used imaging procedure is the computed tomography (CT) scan. Also known as a CAT scan or computed axial tomography, CT creates a series of cross-sectional images of the head and brain. Because it is readily available at all hours at most major hospitals and produces images quickly, CT is the most commonly used diagnostic technique for acute stroke. CT also has unique diagnostic benefits. It will quickly rule out a hemorrhage, can occasionally show a tumor that might mimic a stroke, and may even show evidence of early infarction. Infarctions generally show up on a CT scan about six to eight hours after the start of stroke symptoms.

If a stroke is caused by hemorrhage, a CT can show evidence of bleeding into the brain almost immediately after stroke symptoms appear. Hemorrhage is the primary reason for avoiding certain drug treatments for stroke, such as thrombolytic therapy, the only proven acute stroke therapy for ischemic stroke. Thrombolytic therapy cannot be used until the doctor can confidently diagnose the patient as suffering from an ischemic stroke because this treatment might increase bleeding and could make a hemorrhagic stroke worse.

Another imaging device used for stroke patients is the magnetic resonance imaging (MRI) scan. MRI uses magnetic fields to detect subtle changes in brain tissue content. One effect of stroke is the slowing of water movement, called diffusion, through the damaged brain tissue. MRI can show this type of damage within the first hour after the stroke symptoms start. The benefit of MRI over a CT scan is more accurate and earlier diagnosis of infarction, especially for smaller strokes, while showing equivalent accuracy in determining when hemorrhage is present. MRI is more sensitive than CT for other types of brain diseases, such as a brain tumor, that might mimic a stroke. MRI cannot be performed in patients with certain types of metallic or electronic implants, such as pacemakers for the heart.

Although increasingly used in the emergency diagnosis of stroke, MRI is not immediately available at all hours in most hospitals, where CT is used for acute stroke diagnosis. Also, MRI takes longer to perform than CT, and may not be performed if it would significantly delay treatment.

Other types of MRI scans, often used for the diagnosis of cerebrovascular disease and to predict the risk of stroke, are magnetic resonance angiography (MRA) and functional magnetic resonance imaging (fMRI). Neurosurgeons use MRA to detect stenosis (blockage) of the brain arteries inside the skull by mapping flowing blood. Functional MRI uses a magnet to pick up signals from oxygenated blood and can show brain activity through increases in local blood flow.

Duplex Doppler ultrasound and arteriography are two diagnostic imaging techniques used to decide whether an individual would benefit from a surgical procedure called carotid endarterectomy. This surgery is used to remove fatty deposits from the carotid arteries and can help prevent stroke.

Doppler ultrasound is a painless, noninvasive test in which sound waves above the range of human hearing are sent into the neck. Echoes bounce off the moving blood and the tissue in the artery and can be formed into an image. Ultrasound is fast, painless, risk-free and relatively inexpensive compared to MRA and arteriography, but it is not considered to be as accurate as arteriography. Arteriography is an X-ray of the carotid artery taken when a special dye is injected into the artery. The procedure carries its own small risk of causing a stroke and is costly to perform. The benefits of arteriography over MR techniques and ultrasound are that it is extremely reliable and still the best way to measure stenosis of the carotid arteries. Even so, significant advances are being made every day involving noninvasive imaging techniques such as MRI.
Who is at risk for stroke?

Some people are at a higher risk for stroke than others. Unmodifiable risk factors include age, gender, race/ethnicity and stroke family history. In contrast, other risk factors for stroke, like high blood pressure or cigarette smoking, can be changed or controlled by the person at risk.

Unmodifiable risk factors

It is a myth that stroke occurs only in elderly adults. In actuality, stroke strikes all age groups, from fetuses still in the womb to centenarians. It is true, however, that older people have a higher risk for stroke than the general population and that the risk for stroke increases with age. For every decade after the age of 55, the risk of stroke doubles, and two-thirds of all strokes occur in people over 65 years old. People over 65 also have a seven-fold greater risk of dying from stroke than the general population. And the incidence of stroke is increasing proportionately with the increase in the elderly population. As baby boomers move into the over-65 age group, stroke and other diseases will take on even greater significance in the health care field.

Gender also plays a role in risk for stroke. Men have a higher risk for stroke, but more women die from stroke. The stroke risk for men is 1.25 times that for women. But men do not live as long as women, so men are usually younger when they have their strokes and therefore have a higher rate of survival than women. In other words, even though women have fewer strokes than men, women are generally older when they have their strokes and are more likely to die from them.

Stroke seems to run in some families. Several factors might contribute to familial stroke risk. Members of a family might have a genetic tendency for stroke risk factors, such as an inherited predisposition for hypertension or diabetes. The influence of a common lifestyle among family members could also contribute to familial stroke.

The risk for stroke varies among different ethnic and racial groups. The incidence of stroke among African Americans is almost double that of white Americans, and twice as many African Americans who have a stroke die from the event compared to white Americans. African Americans between the ages of 45 and 55 have four to five times the stroke death rate of whites. After age 55, the stroke mortality rate for whites increases and is equal to that of African Americans.

Compared to white Americans, African Americans have a higher incidence of stroke risk factors, including high blood pressure and cigarette smoking. African Americans also have a higher incidence and prevalence of some genetic diseases, such as diabetes and sickle cell anemia, that predispose them to stroke.

Hispanics and Native Americans have stroke incidence and mortality rates more similar to those of white Americans. In Asian Americans, stroke incidence and mortality rates are also similar to those in white Americans, even though Asians in Japan, China and other countries of the Far East have significantly higher stroke incidence and mortality rates than white Americans. This suggests that environment and lifestyle factors play a large role in stroke risk.

The “Stroke Belt”

Several decades ago, scientists and statisticians noticed that people in the southeastern United States had the highest stroke mortality rate in the country. They named this region the stroke belt. For many years, researchers believed that the increased risk was due to the higher percentage of African Americans and an overall lower socioeconomic status (SES) in the Southern states. A low SES is associated with an overall lower standard of living, leading to a lower standard of health care and therefore an increased risk of stroke. But researchers now know that the higher percentage of African Americans and the overall lower SES in the Southern states does not adequately account for the higher incidence of, and mortality from, stroke in those states. This means that other factors must be contributing to the higher incidence of and mortality from stroke in this region.

Recent studies have also shown that there is a stroke buckle in the stroke belt. Three southeastern states, North Carolina, South Carolina and Georgia, have an extremely high stroke mortality rate, higher than the rate in other stroke belt states and up to two times the stroke mortality rate of the United States overall. The increased risk could be due to geographic or environmental factors or to regional differences in lifestyle, including higher rates of cigarette smoking and a regional preference for salty, high-fat foods.

Other risk factors

The most important risk factors for stroke are hypertension, heart disease, diabetes and cigarette smoking. Others include heavy alcohol consumption, high blood cholesterol levels, illicit drug use, and genetic or congenital conditions, particularly vascular abnormalities. People with more than one risk factor have what is called “amplification of risk.” This means that the multiple risk factors compound their destructive effects and create an overall risk greater than the simple cumulative effect of the individual risk factors.
Hypertension

Of all the risk factors that contribute to stroke, the most powerful is hypertension, or high blood pressure. People with hypertension have a risk for stroke that is four to six times higher than the risk for those without hypertension. One-third of the adult U.S. population, about 50 million people (including 40-70 percent of those over age 65) have high blood pressure. Forty to 90 percent of stroke patients have high blood pressure before their stroke event.

A systolic pressure of 120 mm/Hg over a diastolic pressure of 80 mm/Hg is generally considered normal. Persistently high blood pressure greater than 140 over 90 leads to the diagnosis of the disease called hypertension. The impact of hypertension on the total risk for stroke decreases with increasing age, suggesting that factors other than hypertension play a greater role in the overall stroke risk in elderly adults. For people without hypertension, the absolute risk of stroke increases over time until around the age of 90, when the absolute risk becomes the same as that for people with hypertension.

Like stroke, there is a gender difference in the prevalence of hypertension. In younger people, hypertension is more common among men than among women. With increasing age, however, more women than men have hypertension. This hypertension gender-age difference probably has an impact on the incidence and prevalence of stroke in these populations.

Anti-hypertensive medication can decrease a person’s risk for stroke. Recent studies suggest that treatment can decrease the stroke incidence rate by 38 percent and decrease the stroke fatality rate by 40 percent. Common hypertensive agents include adrenergic agents, beta-blockers, angiotensin converting enzyme inhibitors, calcium channel blockers, diuretics and vasodilators.

Heart disease

After hypertension, the second most powerful risk factor for stroke is heart disease, especially a condition known as atrial fibrillation. Atrial fibrillation is irregular beating of the left atrium, or left upper chamber, of the heart. In people with atrial fibrillation, the left atrium beats up to four times faster than the rest of the heart. This leads to an irregular flow of blood and the occasional formation of blood clots that can leave the heart and travel to the brain, causing a stroke.

Atrial fibrillation, which affects as many as 2.2 million Americans, increases an individual’s risk of stroke by 4 to 6 percent, and about 15 percent of stroke patients have atrial fibrillation before they experience a stroke. The condition is more prevalent in the upper age groups, which means that the prevalence of atrial fibrillation in the United States will increase proportionately with the growth of the elderly population. Unlike hypertension and other risk factors that have a lesser impact on the ever-rising absolute risk of stroke that comes with advancing age, the influence of atrial fibrillation on total risk for stroke increases powerfully with age. In people over 80 years old, atrial fibrillation is the direct cause of one in four strokes.

Other forms of heart disease that increase stroke risk include malformations of the heart valves or the heart muscle. Some valve diseases, like mitral valve stenosis or mitral annular calcification, can double the risk for stroke, independent of other risk factors.

Heart muscle malformations can also increase the risk for stroke. Patent foramen ovale (PFO) is a passage or a hole (sometimes called a “shunt”) in the heart wall separating the two atria, or upper chambers, of the heart. Clots in the blood are usually filtered out by the lungs, but PFO could allow emboli or blood clots to bypass the lungs and go directly through the arteries to the brain, potentially causing a stroke. Research is currently under way to determine how important PFO is as a cause for stroke. Atrial septal aneurysm (ASA), a congenital (present from birth) malformation of the heart tissue, is a bulging of the septum or heart wall into one of the atria of the heart. Researchers do not know why this malformation increases the risk for stroke. PFO and ASA frequently occur together and therefore amplify the risk for stroke. Two other heart malformations that seem to increase the risk for stroke for unknown reasons are left atrial enlargement and left ventricular hypertrophy. People with left atrial enlargement have a larger than normal left atrium of the heart; those with left ventricular hypertrophy have a thickening of the wall of the left ventricle.

Another risk factor for stroke is cardiac surgery to correct heart malformations or reverse the effects of heart disease. Strokes occurring in this situation are usually the result of surgically dislodged plaques from the aorta that travel through the bloodstream to the arteries in the neck and head, causing stroke. Cardiac surgery increases a person’s risk of stroke by about 1 percent. Other types of surgery can also increase the risk of stroke.

Blood cholesterol levels

Most people know that high cholesterol levels contribute to heart disease. But many don’t realize that a high cholesterol level also contributes to stroke risk. Cholesterol, a waxy substance produced by the liver, is a vital body product. It contributes to the production of hormones and vitamin D and is an integral component of cell membranes. The liver makes enough cholesterol to fuel the body’s needs, and this natural production of cholesterol alone is not a large contributing factor to atherosclerosis, heart disease and stroke. Research has shown that the danger from cholesterol comes from a dietary intake of foods that contain high levels of cholesterol. Foods high in saturated fat and cholesterol, like meats, eggs and dairy products, can increase the amount of total cholesterol.
in the body to alarming levels, contributing to the risk of atherosclerosis and thickening of the arteries.

Cholesterol is classified as a lipid, meaning that it is fat-soluble rather than water-soluble. Other lipids include fatty acids, glycerides, alcohol, waxes, steroids, and fat-soluble vitamins A, D, and E. Lipids and water, like oil and water, do not mix. Blood is a water-based liquid, so cholesterol does not mix with blood. In order to travel through the blood without clumping together, cholesterol needs to be covered by a layer of protein. The cholesterol and protein together are called a lipoprotein.

There are two kinds of cholesterol, commonly called the “good” and the “bad.” Good cholesterol is high-density lipoprotein, or HDL; bad cholesterol is low-density lipoprotein, or LDL. Together, these two forms of cholesterol make up a person’s total serum cholesterol level. Most cholesterol tests measure the level of total cholesterol in the blood and don’t distinguish between good and bad cholesterol. For these total serum cholesterol tests, a level of less than 200 mg/dL is considered safe, while a level of more than 240 is considered dangerous and places a person at risk for heart disease and stroke.

Most cholesterol in the body is in the form of LDL. LDLs circulate through the bloodstream, picking up excess cholesterol and depositing cholesterol where it is needed (for example, for the production and maintenance of cell membranes). But when too much cholesterol starts circulating in the blood, the body cannot handle the excessive LDLs, which build up along the inside of the arterial walls. The build-up of LDL coating on the inside of the artery walls hardens and turns into arterial plaque, leading to stenosis and atherosclerosis. This plaque blocks blood vessels and contributes to the formation of blood clots. A person’s LDL level should be less than 130 mg/dL to be safe. LDL levels between 130 and 159 put a person at a slightly higher risk for atherosclerosis, heart disease and stroke. A score over 160 puts a person at great risk for a heart attack or stroke.

The other form of cholesterol, HDL, is beneficial and contributes to stroke prevention. HDL carries a small percentage of the cholesterol in the blood, but instead of depositing its cholesterol on the inside of artery walls, HDL returns to the liver to unload its cholesterol. The liver then eliminates the excess cholesterol by passing it along to the kidneys. Currently, any HDL score higher than 35 is considered desirable. Recent studies have shown that high levels of HDL are associated with a reduced risk for heart disease and stroke and that low levels (less than 35 mg/dL), even in people with normal levels of LDL, lead to an increased risk for heart disease and stroke.

A person may lower his risk for atherosclerosis and stroke by improving his cholesterol levels. A healthy diet and regular exercise are the best ways to lower total cholesterol levels. In some cases, physicians may prescribe cholesterol-lowering medication, and recent studies have shown that the newest types of these drugs, called reductase inhibitors or statin drugs, significantly reduce the risk for stroke in most patients with high cholesterol. Scientists believe that statins may work by reducing the amount of bad cholesterol the body produces and by reducing the body’s inflammatory immune reaction to cholesterol plaque associated with atherosclerosis and stroke.

Millimeters of mercury – or mm/Hg – is the standard means of expressing blood pressure, which is measured using a sphygmomanometer. Using a stethoscope and a cuff that is wrapped around the patient’s upper arm, a health professional listens to the sounds of blood rushing through an artery. The first sound registered on the instrument gauge (which measures the pressure of the blood in millimeters on a column of mercury) is called the systolic pressure. This is the maximum pressure produced as the left ventricle of the heart contracts and the blood begins to flow through the artery. The second sound is the diastolic pressure and is the lowest pressure in the artery when the left ventricle is relaxing.

**Diabetes**

Diabetes is another disease that increases a person’s risk for stroke. People with diabetes have three times the risk of stroke compared to people without diabetes. The relative risk of stroke from diabetes is highest in the fifth and sixth decades of life and decreases after that. Like hypertension, the relative risk of stroke from diabetes is highest for men at an earlier age and highest for women at an older age. People with diabetes may also have other contributing risk factors that can amplify the overall risk for stroke. For example, the prevalence of hypertension is 40 percent higher in the diabetic population compared to the general population.

**Modifiable lifestyle risk factors**

*Cigarette smoking* is the most powerful modifiable stroke risk factor. Smoking almost doubles a person’s risk for ischemic stroke, independent of other risk factors, and it increases a person’s risk for subarachnoid hemorrhage by up to 3.5 percent. Smoking is directly responsible for a greater percentage of the total number of strokes in young adults than in older adults. Risk factors other than smoking – like hypertension, heart disease and diabetes – account for more of the total number of strokes in older adults.

Heavy smokers are at greater risk for stroke than light smokers. The relative risk of stroke decreases immediately after quitting smoking, with a major reduction of risk seen after two to four years. Unfortunately, it may take several decades for a former smoker’s risk to drop to the level of someone who never smoked.

Smoking increases the risk of stroke by promoting atherosclerosis and increasing the levels of blood-clotting factors, such as fibrinogen. In addition to promoting conditions linked to stroke, smoking also increases the damage that
results from stroke by weakening the endothelial wall of the cerebrovascular system. This leads to greater damage to the brain from events that occur in the secondary stage of stroke.

**High alcohol consumption** is another modifiable risk factor for stroke. Generally, an increase in alcohol consumption leads to an increase in blood pressure. While scientists agree that heavy drinking is a risk for both hemorrhagic and ischemic stroke, in several research studies daily consumption of smaller amounts of alcohol has been found to provide a protective influence against ischemic stroke, perhaps because alcohol decreases the clotting ability of platelets in the blood. Moderate alcohol consumption may act in the same way as aspirin to decrease blood clotting and prevent ischemic stroke. Heavy alcohol consumption, though, may seriously deplete platelet numbers and compromise blood clotting and blood viscosity, leading to hemorrhage. In addition, heavy drinking or binge drinking can lead to a rebound effect after the alcohol is purged from the body. The consequences of this rebound effect are that blood viscosity (thickness) and platelet levels skyrocket after heavy drinking, increasing the risk for ischemic stroke.

**Head and neck injuries**

Injuries to the head or neck may damage the cerebrovascular system and cause a small number of strokes. Head injury or traumatic brain injury may cause bleeding within the brain leading to damage akin to that caused by a hemorrhagic stroke. Neck injury, when associated with spontaneous tearing of the vertebral or carotid arteries caused by sudden and severe extension of the neck, neck rotation or pressure on the artery, is a contributing cause of stroke, especially in young adults. This type of stroke is often called “beauty-parlor syndrome,” which refers to the practice of extending the neck backwards over a sink for hair-washing in beauty parlors. Neck calisthenics, “bottoms-up” drinking, and improperly performed chiropractic manipulation of the neck can also put strain on the vertebral and carotid arteries, possibly leading to ischemic stroke.

**Infections**

Recent viral and bacterial infections may act with other risk factors to add a small risk for stroke. The immune system responds to infection by increasing inflammation and increasing the infection-fighting properties of the blood. Unfortunately, this immune response increases the number of clotting factors in the blood, leading to an increased risk of embolic-ischemic stroke.

**Genetic risk factors**

Although there may not be a single genetic factor associated with stroke, genes do play a large role in the expression of stroke risk factors such as hypertension, heart disease, diabetes and vascular malformations. It is also possible that an increased risk for stroke within a family is due to environmental factors, such as a common sedentary lifestyle or poor eating habits, rather than hereditary factors.

Vascular malformations that cause stroke may have the strongest genetic link of all stroke risk factors. A vascular malformation is an abnormally formed blood vessel or group of blood vessels. One genetic vascular disease is called CADASIL, which stands for cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy. CADASIL is a rare, genetically inherited, congenital vascular disease of the brain that causes strokes, subcortical dementia, migraine-like headaches and psychiatric disturbances. CADASIL is very debilitating, and symptoms usually surface about the age of 45. Although CADASIL can be treated with surgery to repair the defective blood vessels, patients often die by the age of 65. The exact incidence of CADASIL in the United States is unknown.

**What is CADASIL?**

CADASIL is an inherited form of cerebrovascular disease that occurs when the thickening of blood vessel walls blocks the flow of blood to the brain. The disease primarily affects small blood vessels in the white matter of the brain. A mutation in the Notch3 gene alters the muscular walls in these small arteries. CADASIL is characterized by migraine headaches and multiple strokes progressing to dementia. Other symptoms include white matter lesions throughout the brain, cognitive deterioration,
seizures, vision problems and psychiatric problems such as severe depression and changes in behavior and personality. Individuals may also be at higher risk of heart attack. Symptoms and disease onset vary widely, for some people in the mid-30s while others may not show signs of the disease until later in life. CADASIL – formerly known by several names, including hereditary multi-infarct dementia – is one cause of multi-infarct dementia (dementia caused by lack of blood to several areas of the brain). It is an autosomal dominant inheritance disorder, meaning that one parent carries and passes on the defective gene. Most individuals with CADASIL have a family history of the disorder.

There is no treatment to halt this genetic disorder. Individuals are given supportive care. Migraine headaches may be treated by different drugs and a daily aspirin may reduce stroke and heart attack risk. Drug therapy for depression may be given. Affected individuals who smoke should quit because that can increase the risk of stroke in CADASIL.

Symptoms usually progress slowly. By age 65, the majority of persons with CADASIL have severe cognitive problems and dementia. Some people lose the ability to walk, and most become completely dependent because of multiple strokes.

The National Institute of Neurological Disorders and Stroke (NINDS) conducts stroke research and clinical trials at its laboratories and clinics at the National Institutes of Health (NIH) and through grants to major medical institutions across the country. Scientists are currently studying different drugs to reduce cognitive problems seen in patients with CADASIL. Researchers are also looking at ways to overcome an over-reaction to hormones that lead to high blood pressure and poor blood supply in patients with CADASIL.

**Treating stroke**

Timing is everything in treatment of stroke, particularly acute ischemic stroke, which comprises about 80 percent of cerebrovascular accidents. Health organizations have tried to get the word out to the public: When symptoms appear, don’t delay; call 911. These organizations have encouraged emergency responders to also take action, and to “load and go” once a patient is stable enough for transport. Nurses and physicians staffing emergency departments must be informed about stroke symptoms and interventions and also “ready to roll” when a patient arrives.

**Medications**

Medicines that lower blood pressure and cholesterol can protect against atherosclerosis and reduce a person’s risk of stroke. Aspirin and other blood-thinning medications have been used for years to reduce the risk of ischemic stroke in individuals with AF or prior stroke. Recent studies have helped refine the use of these drugs to maximize safety and efficacy. This section, however, begins with a discussion of what happens when prevention fails and a person requires emergency treatment for an acute ischemic stroke.

**Thrombolytic drugs**

In treating acute ischemic stroke (acute meaning that the stroke has occurred within the past few hours), the immediate goal is to break apart the offending clot, a process known as thrombolysis. The body produces its own thrombolytic proteins, and some of these have been engineered into drugs. One, called tissue plasminogen activator (tPA), has a proven track record for treating heart attacks. In the late 1980s, NINDS-funded investigators laid the plans for the first placebo-controlled trial of tPA to treat acute ischemic stroke. They knew from animal studies that irreversible brain injury is likely to occur if blood flow is not restored within the first few hours after ischemic stroke. Therefore, the NINDS tPA Study Group tested the drug within a three-hour time window. Compared to individuals given a placebo, those given intravenous tPA were more likely to have minimal or no disability three months after treatment – a finding that persuaded the U.S. Food and Drug Administration to approve tPA for use against acute stroke. Trials in Europe and the U.S. subsequently confirmed those results. Recent studies attempt to identify individuals who may benefit even after three hours of stroke onset. In any case, more brain tissue will be saved the earlier the treatment is delivered.

Platelets (magnified here thousands of times) go to damaged areas of blood vessels and contribute to the formation of clots. Anti-platelet drugs can help reduce the risk of ischemic stroke.

A 1998 follow-up analysis of the NINDS trial found that, after their initial hospitalization, people who received tPA were less likely to require inpatient rehabilitation or nursing home care. The authors estimated that this lower dependency on long-term care would translate into a savings to the health care system of more than $4 million for every 1,000 individuals treated with tPA.

Because treatment with tPA interferes with blood clotting and has also been shown to increase leaking along the blood-brain barrier, it carries a risk of intracerebral hemorrhage. Therefore,
it is not recommended for some people, such as those with a history of brain hemorrhage or significantly elevated blood pressure (greater than 185/110 mm/Hg). The risk of tPA-induced hemorrhage increases over time from stroke onset, which has limited its use to the first three hours after stroke (where benefit was most clearly established in the U.S. trials).

**Anti-platelet drugs and anticoagulants**

Blood-thinning medications fall into two classes: anti-platelet drugs and anticoagulants. Anti-platelet drugs inhibit the activity of cells called platelets, which stick to damaged areas inside blood vessels and lay the foundation for blood clots. The most common anti-platelet drug is aspirin. Anticoagulants, such as heparin (produced by inflammatory cells in the body) and warfarin (found in plants and also known by the trade name Coumadin©), inhibit proteins in the blood that stimulate clotting.

Anti-platelet drugs and anticoagulants can help prevent a variety of potentially life-threatening conditions for which individuals with stroke are at risk, such as myocardial infarction, pulmonary embolism and deep vein thrombosis, which are caused by clots in the heart, lungs and deep veins of the legs, respectively. In recent years, the value of these drugs in treating and preventing stroke itself has been more closely scrutinized.

One focus of this research has been to determine whether there is any benefit in giving anti-platelet drugs or anticoagulants during an acute ischemic stroke as an adjunct to tPA or as an alternative for people ineligible to receive tPA. In an international trial coordinated by researchers in the United Kingdom in the late 1990s, individuals received aspirin, subcutaneous heparin injections, or neither treatment within 48 hours of an ischemic stroke. Aspirin significantly reduced the risk of a recurrent ischemic stroke at two weeks. A similar benefit from heparin was offset by an increased risk of hemorrhagic stroke. Around the same time, NINDS-funded researchers tested whether acute stroke could be treated with intravenous Org 10172, a form of heparin considered less likely to cause bleeding. This study, Trial of Org 10172 in Acute Stroke Treatment (TOAST), found that Org 10172 produced no significant benefit. The study authors also developed the TOAST criteria, a set of guidelines for classifying different subtypes of ischemic stroke that are now widely used in other studies.

Two other NINDS-sponsored trials compared the effectiveness of daily warfarin and aspirin for individuals who did not have AF but who had experienced a prior stroke, and thus were at risk for another. The Warfarin vs. Aspirin Recurrent Stroke Study (WARSS) showed that aspirin was as effective as warfarin in preventing recurrent stroke in people with no history of AF or other cardioembolic causes of stroke. The Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) trial focused more narrowly on individuals with stenosis of arteries in the brain and was terminated early because of a high rate of adverse events in participants treated with warfarin. Both trials concluded that aspirin is equivalent to warfarin for reducing the risk of stroke in people without AF.

**Medication for subarachnoid hemorrhage**

The drug nimodipine is used to treat cerebral vasospasm, a complication that sometimes follows subarachnoid hemorrhage. This refers to a constriction of blood vessels in the brain that can significantly reduce blood flow, leading to ischemia and infarction. Although its precise origins are unclear, cerebral vasospasm is thought to be triggered in part by an influx of calcium into the smooth muscles that control blood vessel diameter. Nimodipine is a calcium antagonist, meaning that it works by blocking the entry of calcium into cells. Nimodipine has been shown to reduce infarction and improve outcome in individuals with subarachnoid hemorrhage.

**Surgeries and other procedures**

Surgery is sometimes used to clear the congested blood vessels that cause ischemic stroke or to repair the vascular abnormalities that contribute to hemorrhagic stroke.

A surgery called carotid endarterectomy involves removing plaque to widen the carotids, a pair of arteries that ascend each side of the neck and are the main suppliers of blood to the brain. Stenosis that narrows a carotid artery by more than 50 percent is considered clinically significant. In some cases, carotid stenosis is first detected after a person experiences a stroke or other symptoms, such as a TIA. It is also sometimes detected in the absence of symptoms, as when a physician presses a stethoscope to the neck and hears a bruit, a sound made by blood flowing past an obstruction. The presence of carotid stenosis can be confirmed by angiography or Doppler ultrasound.
Data from NINDS-funded research show that the risk of ischemic stroke from clinically significant asymptomatic carotid stenosis is about 2 to 3 percent per year (meaning that out of 100 individuals with this condition, two or three will have a stroke each year). The risk of ischemic stroke from clinically significant symptomatic carotid stenosis is much higher – about 25 percent during the first two years following the appearance of symptoms.

**Nurses’ role in stroke treatment and care**

Nurses play a key role in stroke treatment from the time a patient arrives at an ED until discharge. All emergency nurses and staff should be aware that NINDS recommends that treatment of a patient with acute ischemic stroke should begin within 60 minutes of the person’s arrival, and that time could be even shorter to meet the treatment window requirement of three hours after onset of symptoms.

They must be able to quickly assess the patient’s symptoms for triage purposes and may help with a neurological assessment (such as the NIH Stroke Scale) and gather history from the patient or his/her family to ascertain the time of symptom onset, a key determinant of whether the person is a candidate for thrombolytic therapy.

- They must be ready to quickly implement orders for brain imaging and other tests a doctor will order to ascertain the origin of the stroke. This may mean alerting the proper departments that a patient will arrive soon and tests must be conducted stat.
- When intravenous thrombolytic therapy is ordered, a nurse will likely administer it. (Physicians generally administer intra-arterial thrombolysis, which is sometimes used on patients with AIS secondary to occlusion of the MCA.) The nurse also will be part of the critically important team that monitors the patient during the therapy and afterward.
  - The American Heart Association in its Comprehensive Overview of Nursing and Interdisciplinary Care of the Acute Ischemic Stroke Patient report (http://stroke.ahajournals.org/cgi/content/full/8/2911) recommends:
    - TPA be infused 0.9 mg/kg (with a maximum dose of 90 mg) over 60 minutes with 10 percent of the dose given as a bolus over one minute.
    - Blood pressure monitoring is critical during initial treatment and afterward; an elevated BP that cannot be safely lowered to 185/110 mm/Hg will disqualify a patient for thrombolytic therapy; and patients who have received the therapy also must be continuously monitored to ensure a rise does not cause bleeding complications. During administration of tPA, BP should be taken every 15 minutes for the first two hours, then every 30 minutes for the next six hours, and then hourly until 24 hours after treatment. A nurse should increase the frequency of blood pressure measurements if a systolic BP is at or greater than 180 mm/Hg or if diastolic BP is at or greater than 105 mm/Hg.
    - Neurological assessments should be performed every 15 minutes during the infusion and every 30 minutes after for the next six hours, then hourly until 24 hours after treatment.
    - After the administration of tPA, patient monitoring continues. AHA says that in addition to blood pressure and neurological assessments, nurses caring for all stroke patients should:
      - Oversee the patient’s transition from bed rest to mobilization, usually as soon the patient is considered stable. The report noted that some patients have neurological worsening with movement, and nurses must observe the transition from bed to chair carefully. Early mobilization is encouraged, however, because it lessens the likelihood of complications such as pneumonia, deep vein thrombosis, pulmonary embolism and pressure sores.
      - Pressure mattresses and close surveillance of the skin are recommended to help prevent pressure sores.
      - Monitor patients to avoid dehydration or malnutrition, which may slow recovery or cause additional problems, such as deep vein thrombosis, after a stroke. Problems swallowing are associated with a high risk of pneumonia and death.
      - Patients with infarctions of the brainstem, multiple strokes, major hemispheric lesions or mental impairments are at the greatest risk of aspiration.
      - Because pneumonia is a frequent problem in seriously affected immobile patients and an important cause of death after stroke, nurses must check for signs of fever and infection. Some measures to prevent aspiration and infection are:
        - Protection of the airway and suctioning.
        - Measures to treat nausea and vomiting.
        - Exercise and encouragement to take deep breaths.
        - Urinary tract infections are also common among patients with stroke. Bacteremia or sepsis can appear.
        - Screening of urine should occur whenever a patient develops a fever.
        - Indwelling catheters may be ordered to prevent incontinence and urinary retention, but their use comes with the risk of urinary infections.
        - Acidification of urine may reduce the risk of infection.
        - Anti-cholinergic agents may help recover bladder function.
    - Nurses must watch for serious complications that arise after a stroke, including:
      - Deep vein thrombosis and pulmonary embolism, which account for about 10 percent of deaths after stroke and generally arise from venous thrombi that developed in paralyzed lower limbs or pelvis. In addition to the potential to cause life-threatening events, deep vein thrombosis slows recovery and rehabilitation after stroke.
Early mobilization, anti-thrombotic agents and the use of external compression devices (such as stockings or alternating pressure devices) may lower the risk of the condition.

- **Ischemic brain swelling**, caused by a cytotoxic reaction mediated by multiple factors, including free radicals. It typically occurs about four days after onset, but some early swelling, called “malignant” swelling, that occurs within 24 hours has been seen. Very few clinical signs predict clinical deterioration from swelling. But researchers have found the following often are present in such cases:
  - A history of hypertension and heart failure.
  - Elevated white blood cell count.
  - Presence of more than 50 percent MCA hypodensity.

- Involvement of additional vascular territory.
- **Hemorrhagic transformation from the ischemic stroke**. Hematomas may be associated with neurological decline; small asymptomatic petechiae are usually less problematic.
- The use of all anti-thrombotics, but especially anticoagulants and thrombolytic agents, increases the likelihood of serious hemorrhagic transformation.
- **Seizures** usually occur within 24 hours of stroke and are usually partial. The risk of late seizures is higher in patients with pre-existing dementia.
- After a patient is stabilized, nurses may be involved in rehabilitation measures, including patient and family education and support.

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**POST-STROKE REHABILITATION FACT SHEET**

**National Institute of Neurological Disorders and Stroke**

In the United States, more than 700,000 people suffer a stroke each year, and approximately two-thirds of these individuals survive and require rehabilitation. The goals of rehabilitation are to help survivors become as independent as possible and to attain the best possible quality of life. Even though rehabilitation does not “cure” stroke in that it does not reverse brain damage, rehabilitation can substantially help people achieve the best possible long-term outcome.

**What is post-stroke rehabilitation?**

Rehabilitation helps stroke survivors relearn skills that are lost when part of the brain is damaged. For example, these skills can include coordinating leg movements in order to walk or carrying out the steps involved in any complex activity. Rehabilitation also teaches survivors new ways of performing tasks to circumvent or compensate for any residual disabilities. Patients may need to learn how to bathe and dress using only one hand, or how to communicate effectively when their ability to use language has been compromised. There is a strong consensus among rehabilitation experts that the most important element in any rehabilitation program is carefully directed, well-focused, repetitive practice – the same kind of practice used by all people when they learn a new skill, such as playing the piano or pitching a baseball.

Rehabilitative therapy begins in the acute-care hospital after the patient’s medical condition has been stabilized. The first steps involve promoting independent movement because many patients are paralyzed or seriously weakened. Patients are prompted to change positions frequently while lying in bed and to engage in passive or active range-of-motion exercises to strengthen their stroke-impaired limbs. (“Passive” range-of-motion exercises are those in which the therapist actively helps the patient move a limb repeatedly, whereas “active” exercises are performed by the patient with no physical assistance from the therapist.) Patients progress from sitting up and transferring between the bed and a chair to standing, bearing their own weight and walking, with or without assistance. Rehabilitation nurses and therapists help patients perform progressively more complex and demanding tasks, such as bathing, dressing and using a toilet, and they encourage patients to begin using their stroke-impaired limbs while engaging in those tasks. Beginning to reacquire the ability to carry out these basic activities of daily living represents the first stage in a stroke survivor’s return to functional independence.

For some stroke survivors, rehabilitation will be an ongoing process to maintain and refine skills and could involve working with specialists for months or years after the stroke.

**What disabilities can result from a stroke?**

The types and degrees of disability that follow a stroke depend upon which area of the brain is damaged. Generally, stroke can cause five types of disabilities: paralysis or problems controlling movement; sensory disturbances, including pain; problems using or understanding language; problems with thinking and memory; and emotional disturbances.

**Paralysis or problems controlling movement (motor control)**

Paralysis is one of the most common disabilities resulting from stroke. The paralysis is usually on the side of the body opposite the side of the brain damaged by stroke, and may affect the face, an arm, a leg or the entire side of the body. This one-sided...
paralysis is called hemiplegia (one-sided weakness is called hemiparesis). Stroke patients with hemiparesis or hemiplegia may have difficulty with everyday activities such as walking or grasping objects. Some stroke patients have problems with swallowing, called dysphagia, due to damage to the part of the brain that controls the muscles for swallowing. Damage to a lower part of the brain, the cerebellum, can affect the body’s ability to coordinate movement, a disability called ataxia, leading to problems with body posture, walking and balance.

Sensory disturbances including pain

Stroke patients may lose the ability to feel touch, pain, temperature or position. Sensory deficits may also hinder the ability to recognize objects that patients are holding and can even be severe enough to cause loss of recognition of one’s own limb. Some stroke patients experience pain, numbness or odd sensations of tingling or prickling in paralyzed or weakened limbs, a condition known as paresthesia.

Stroke survivors frequently have a variety of chronic pain syndromes resulting from stroke-induced damage to the nervous system (neuropathic pain). Patients who have a seriously weakened or paralyzed arm commonly experience moderate to severe pain that radiates outward from the shoulder. Most often, the pain results when a joint becomes immobilized because of lack of movement and the tendons and ligaments around the joint become fixed in one position. This is commonly called a “frozen” joint; “passive” movement at the joint in a paralyzed limb is essential to prevent painful freezing and to allow easy movement if and when voluntary motor strength returns. In some stroke patients, pathways for sensation in the brain are damaged, causing the transmission of false signals that result in the sensation of pain in a limb or side of the body that has the sensory deficit. The most common of these pain syndromes is called thalamic pain syndrome, which can be difficult to treat even with medications.

The loss of urinary continence is fairly common immediately after a stroke and often results from a combination of sensory and motor deficits. Stroke survivors may lose the ability to sense the need to urinate or the ability to control muscles of the bladder. Some may lack enough mobility to reach a toilet in time. Loss of bowel control or constipation may also occur. Permanent incontinence after a stroke is uncommon. But even a temporary loss of bowel or bladder control can be emotionally difficult for stroke survivors.

Problems using or understanding language (aphasia)

At least one-fourth of all stroke survivors experience language impairments, involving the ability to speak, write and understand spoken and written language. A stroke-induced injury to any of the brain’s language-control centers can severely impair verbal communication. Damage to a language center located on the dominant side of the brain, known as Broca’s area, causes expressive aphasia. People with this type of aphasia have difficulty conveying their thoughts through words or writing. They lose the ability to speak the words they are thinking and to put words together in coherent, grammatically correct sentences.

In contrast, damage to a language center located in a rear portion of the brain, called Wernicke’s area, results in receptive aphasia. People with this condition have difficulty understanding spoken or written language and often have incoherent speech. Although they can form grammatically correct sentences, their utterances are often devoid of meaning.

The most severe form of aphasia, global aphasia, is caused by extensive damage to several areas involved in language function. People with global aphasia lose nearly all their linguistic abilities; they can neither understand language nor use it to convey thought.

A less severe form of aphasia, called anomic or amnesic aphasia, occurs when there is only a minimal amount of brain damage; its effects are often quite subtle. People with anomic aphasia may simply selectively forget interrelated groups of words, such as the names of people or particular kinds of objects.

Problems with thinking and memory

Stroke can cause damage to parts of the brain responsible for memory, learning and awareness. Stroke survivors may have dramatically shortened attention spans or may experience deficits in short-term memory. Individuals also may lose their ability to make plans, comprehend meaning, learn new tasks or engage in other complex mental activities. Two fairly common deficits resulting from stroke are anosognosia, an inability to acknowledge the reality of the physical impairments resulting from stroke, and neglect, the loss of the ability to respond to objects or sensory stimuli located on one side of the body, usually the stroke-impaired side. Stroke survivors who develop apraxia lose their ability to plan the steps involved in a complex task and to carry the steps out in the proper sequence. Stroke survivors with apraxia may also have problems following a set of instructions. Apraxia appears to be caused by a disruption of the subtle connections that exist between thought and action.
Emotional disturbances

Many people who survive a stroke feel fear, anxiety, frustration, anger, sadness and a sense of grief for their physical and mental losses. These feelings are a natural response to the psychological trauma of stroke. Some emotional disturbances and personality changes are caused by the physical effects of brain damage. Clinical depression, which is a sense of hopelessness that disrupts an individual’s ability to function, appears to be the emotional disorder most commonly experienced by stroke survivors. Signs of clinical depression include sleep disturbances, a radical change in eating patterns that may lead to sudden weight loss or gain, lethargy, social withdrawal, irritability, fatigue, self-loathing and suicidal thoughts. Post-stroke depression can be treated with antidepressant medications and psychological counseling.

What medical professionals specialize in post-stroke rehabilitation?

Post-stroke rehabilitation involves physicians; rehabilitation nurses; physical, occupational, recreational, speech-language and vocational therapists; and mental health professionals.

Physicians

Physicians have the primary responsibility for managing and coordinating the long-term care of stroke survivors, including recommending which rehabilitation programs will best address individual needs. Physicians are also responsible for caring for the stroke survivor’s general health and providing guidance aimed at preventing a second stroke, such as controlling high blood pressure or diabetes and eliminating risk factors such as cigarette smoking, excessive weight, a high-cholesterol diet and high alcohol consumption.

Neurologists usually lead acute-care stroke teams and direct patient care during hospitalization. They sometimes remain in charge of long-term rehabilitation. However, physicians trained in other specialties often assume responsibility after the acute stage has passed, including physiatrists, who specialize in physical medicine and rehabilitation.

Rehabilitation nurses

Nurses specializing in rehabilitation help survivors relearn how to carry out the basic activities of daily living. They also educate survivors about routine health care, such as how to follow a medication schedule, how to care for the skin, how to manage transfers between a bed and a wheelchair, and special needs for people with diabetes. Rehabilitation nurses also work with survivors to reduce risk factors that may lead to a second stroke, and provide training for caregivers.

Nurses are closely involved in helping stroke survivors manage personal care issues, such as bathing and controlling incontinence. Most stroke survivors regain their ability to maintain continence, often with the help of strategies learned during rehabilitation. These strategies include strengthening pelvic muscles through special exercises and following a timed voiding schedule. If problems with incontinence continue, nurses can help caregivers learn to insert and manage catheters and to take special hygienic measures to prevent other incontinence-related health problems from developing.

Physical therapists

Physical therapists specialize in treating disabilities related to motor and sensory impairments. They are trained in all aspects of anatomy and physiology related to normal function, with an emphasis on movement. They assess the stroke survivor’s strength, endurance, range of motion, gait abnormalities and sensory deficits to design individualized rehabilitation programs aimed at regaining control over motor functions.

Physical therapists help survivors regain the use of stroke-impaired limbs, teach compensatory strategies to reduce the effect of remaining deficits and establish ongoing exercise programs to help people retain their newly learned skills. Disabled people tend to avoid using impaired limbs, a behavior called learned non-use. However, the repetitive use of impaired limbs encourages brain plasticity and helps reduce disabilities.

Strategies used by physical therapists to encourage the use of impaired limbs include selective sensory stimulation such as tapping or stroking, active and passive range-of-motion exercises, and temporary restraint of healthy limbs while practicing motor tasks. Some physical therapists may use a new technology, transcutaneous electrical nerve stimulation (TENS), that encourages brain reorganization and recovery of function. TENS involves using a small probe that generates an electrical current to stimulate nerve activity in stroke-impaired limbs.

In general, physical therapy emphasizes practicing isolated movements, repeatedly changing from one kind of movement to another, and rehearsing complex movements that require a great deal of coordination and balance, such as walking up or down stairs or moving safely between obstacles. People too weak to bear their own weight can still practice
repetitive movements during hydrotherapy (in which water provides sensory stimulation as well as weight support) or while being partially supported by a harness. A recent trend in physical therapy emphasizes the effectiveness of engaging in goal-directed activities, such as playing games, to promote coordination. Physical therapists frequently employ selective sensory stimulation to encourage use of impaired limbs and to help survivors with neglect regain awareness of stimuli on the neglected side of the body.

**Occupational and recreational therapists**

Like physical therapists, occupational therapists are concerned with improving motor and sensory abilities. They help survivors relearn skills needed for performing self-directed activities—occupations, such as personal grooming, preparing meals and housecleaning. Therapists can teach some survivors how to adapt to driving and provide on-road training. They often teach people to divide a complex activity into its component parts, practice each part and then perform the whole sequence of actions. This strategy can improve coordination and may help people with apraxia relearn how to carry out planned actions.

Occupational therapists also teach people how to develop compensatory strategies and how to change elements of their environment that limit activities of daily living. For example, people with the use of only one hand can substitute Velcro closures for buttons on clothing. Occupational therapists also help people make changes in their homes to increase safety, remove barriers and facilitate physical functioning, such as installing grab bars in bathrooms.

Recreational therapists help people with a variety of disabilities to develop and use their leisure time to enhance their health, independence and quality of life.

**Speech-language pathologists**

Speech-language pathologists help stroke survivors with aphasia relearn how to use language or develop alternative means of communication. They also help people improve their ability to swallow, and they work with patients to develop problem-solving and social skills needed to cope with the aftereffects of a stroke.

Many specialized therapeutic techniques have been developed to assist people with aphasia. Some forms of short-term therapy can improve comprehension rapidly. Intensive exercises such as repeating the therapist’s words, practicing following directions and doing reading or writing exercises form the cornerstone of language rehabilitation. Conversational coaching and rehearsal as well as the development of prompts or cues to help people remember specific words are sometimes beneficial. Speech-language pathologists also help stroke survivors develop strategies for circumventing language disabilities. These strategies can include the use of symbol boards or sign language. Recent advances in computer technology have spurred the development of new types of equipment to enhance communication.

Speech-language pathologists use noninvasive imaging techniques to study swallowing patterns of stroke survivors and identify the exact source of their impairment. Difficulties with swallowing have many possible causes, including a delayed swallowing reflex, an inability to manipulate food with the tongue or an inability to detect food remaining lodged in the cheeks after swallowing. When the cause has been pinpointed, speech-language pathologists work with the individual to devise strategies to overcome or minimize the deficit. Sometimes, simply changing body position and improving posture during eating can bring about improvement. The texture of foods can be modified to make swallowing easier; for example, thin liquids, which often cause choking, can be thickened. Changing eating habits by taking small bites and chewing slowly can also help alleviate dysphagia.

**Vocational therapists**

Approximately one-fourth of all strokes occur in people between the ages of 45 and 65. For most people in this age group, returning to work is a major concern. Vocational therapists perform many of the same functions that ordinary career counselors do. They can help people with residual disabilities identify vocational strengths and develop resumes that highlight those strengths. They also can help identify potential employers, assist in specific job searches and provide referrals to stroke vocational rehabilitation agencies.

Most important, vocational therapists educate disabled individuals about their rights and protections as defined by the Americans with Disabilities Act of 1990. This law requires employers to make “reasonable accommodations” for disabled employees. Vocational therapists frequently act as mediators between employers and employees to negotiate the provision of reasonable accommodations in the workplace.
## Where can a stroke patient get rehabilitation?

Rehabilitation should begin as soon as a stroke patient is stable, often within 24 to 48 hours after a stroke. This first stage of rehabilitation usually occurs within an acute-care hospital. At the time of discharge from the hospital, the stroke patient and family coordinate with hospital social workers to locate a suitable living arrangement. Many stroke survivors return home, but some move into some type of medical facility.

### Inpatient rehabilitation units

Inpatient facilities may be freestanding or part of larger hospital complexes. Patients stay in the facility, usually for two to three weeks, and engage in a coordinated, intensive program of rehabilitation. Such programs often involve at least three hours of active therapy a day, five or six days a week. Inpatient facilities offer a comprehensive range of medical services, including full-time physician supervision and access to the full range of therapists specializing in post-stroke rehabilitation.

### Outpatient units

Outpatient facilities are often part of a larger hospital complex and provide access to physicians and the full range of therapists specializing in stroke rehabilitation. Patients typically spend several hours, often three days each week, at the facility taking part in coordinated therapy sessions and return home at night. Comprehensive outpatient facilities frequently offer treatment programs as intense as those of inpatient facilities, but they also can offer less demanding regimens, depending on the patient’s physical capacity.

### Nursing facilities

Rehabilitative services available at nursing facilities are more variable than are those at inpatient and outpatient units. Skilled nursing facilities usually place a greater emphasis on rehabilitation, whereas traditional nursing homes emphasize residential care. In addition, fewer hours of therapy are offered compared to outpatient and inpatient rehabilitation units.

### Home-based rehabilitation programs

Home rehabilitation allows for great flexibility so that patients can tailor their program of rehabilitation and follow individual schedules. Stroke survivors may participate in an intensive level of therapy several hours per week or follow a less demanding regimen. These arrangements are often best suited for people who lack transportation or require treatment by only one type of rehabilitation therapist. Patients dependent on Medicare coverage for their rehabilitation must meet Medicare’s “homebound” requirements to qualify for such services; at this time, lack of transportation is not a valid reason for home therapy. The major disadvantage of home-based rehabilitation services is the lack of specialized equipment. However, undergoing treatment at home gives people the advantage of practicing skills and developing compensatory strategies in the context of their own living environment.

### What research is being done?

The National Institute of Neurological Disorders and Stroke (NINDS), a component of the federal government’s National Institutes of Health (NIH), has primary responsibility for sponsoring research on disorders of the brain and nervous system, including the acute phase of stroke and the restoration of function after stroke. The NINDS also supports research on ways to enhance repair and regeneration of the central nervous system. Scientists funded by the NINDS are studying how the brain responds to experience or adapts to injury by reorganizing its functions (plasticity) by using noninvasive imaging technologies to map patterns of biological activity inside the brain. Other NINDS-sponsored scientists are looking at brain reorganization after stroke and determining whether specific rehabilitative techniques, such as constraint-induced movement therapy and transcranial magnetic stimulation, can stimulate brain plasticity, thereby improving motor function and decreasing disability. Other scientists are experimenting with implantation of neural stem cells, to see whether these cells may be able to replace the cells that died as a result of a stroke.

### Where can I get more information?

For more information on neurological disorders or research programs funded by the National Institute of Neurological Disorders and Stroke, contact the Institute’s Brain Resources and Information Network (BRAIN) at:

**BRAIN**

P.O. Box 5801
Bethesda, MD 20824
(800) 352-9424

National initiatives

Health care agencies and authorities have made public education about stroke a priority with national ad campaigns urging people to understand the urgent need to call 911 at the first signs of a stroke in themselves or others. They want people to understand that there is something that can be done to contain or even reverse damage from the dreaded event if they act quickly. Many research studies are under way to try new treatment strategies, including some to determine whether the window for thrombolytic treatment may be longer than three hours. New procedures and medication therapies are being tested.

Emergency responders are being trained in the procedures for handling suspected stroke patients, and physicians, nurses and other health care professionals are sharpening their skills to provide faster and better treatment for patients presenting with stroke symptoms.

The Paul Coverdale Stroke Registry

In 2001, Congress charged the Centers for Disease Control (CDC) with implementing state-based registries that measure and track acute stroke care and to use data from the registries in efforts to improve the quality of that care. Congress further directed that this project be named the Paul Coverdell National Acute Stroke Registry to memorialize the late U.S. Sen. Paul Coverdell of Georgia, who suffered a fatal stroke in 2000 while serving in Congress.

CDC, in consultation with stroke experts and organizations, piloted eight prototype registry projects led by academic and medical institutions across the country to test models for measuring the quality of care delivered to stroke patients. “Wave I” projects, funded in 2001, were located in Georgia, Massachusetts, Michigan and Ohio. “Wave II” projects, funded in 2002, were located in California, Illinois, North Carolina and Oregon. These prototype projects gathered data concerning each step of emergency and hospital care for stroke patients, from emergency response to the patients’ eventual discharge from a hospital. At the end of the three-year pilot period, the results showed that large gaps existed between generally recommended guidelines for treating stroke patients and actual hospital practices. Intensive quality improvement efforts are needed to close those gaps.

In June 2004, CDC provided funds to the state health departments of Georgia, Illinois, Massachusetts and North Carolina to establish statewide Coverdell stroke registries for acute care hospitals in their states. The purpose of these registries was to develop and implement systems for collecting data on acute stroke care provided to patients, analyze the collected data, and use the results of those analyses to guide quality improvement interventions at the hospital level through partnerships with hospital doctors, stroke-care teams and administrators. All acute care hospitals serving the general population in participating states were eligible for the program.

In the first year of program activities, states established partnerships with leading medical experts, various hospital associations, local affiliates of the American Hospital
Association and other groups interested in improving health care for stroke patients; developed strategies for identifying and recruiting eligible hospitals; selected and implemented customized Web-based data-collection systems for hospital use; and recruited hospitals to participate in the registry. In the second and third years, states reviewed collected data to identify specific areas of need for quality improvement, worked with hospitals to implement quality improvement interventions to improve care, and evaluated progress toward improving statewide acute stroke care and promoting long-term systemic changes in how that care is provided. By the end of the 2004-2007 project period, more than 180 hospitals were participating in a stroke registry, and the percentages of total statewide stroke admissions treated by participating hospitals ranged from 40 percent to 79 percent among the four states.

In June 2007, CDC expanded funding to six state health departments in Georgia, Massachusetts, Michigan, Minnesota, Ohio and North Carolina for the Coverdell Registry for a new five-year funding period. Illinois will continue to participate in stroke quality improvement activities and provide information to CDC on its progress. In 2007, CDC also came to an agreement with The Joint Commission’s Primary Stroke Center Certification program and with the American Heart Association/ American Stroke Association’s Get With The Guidelines – Stroke program to jointly release a set of standardized stroke performance measures and clinical practice guidelines for use by all three programs. This effort will reduce duplication, increase collaboration and encourage hospitals to participate in one or more of the programs. The National Quality Forum endorsed eight of these performance measures in 2008.

Consensus Stroke Performance Measures

Hospital performance measures for acute stroke care have been developed based on evidence from multiple clinical trials and in the peer-reviewed stroke literature. The Coverdale registry endorses these measures as a foundation of its work to improve quality of care:

For hemorrhagic and ischemic stroke patients
- Deep vein thrombosis prophylaxis by end of the second hospital day.
- Dysphagia screening.
- Assessment for rehabilitation.
- Smoking cessation counseling (TIA patients also).
- Stroke education (TIA patients also).

For ischemic stroke and TIA patients
- Anti-thrombotic therapy by end of day two and prescribed at discharge.
- Lipid-lowering therapy for patients with strokes of atherosclerotic origin with LDL 100.
- Anticoagulation for atrial fibrillation.
- Use of tPA intravenous thrombolytic therapy – a clot-busting medicine (ischemic stroke patients only).
- Staten medication prescribed on discharge.

The near-term goals of the Paul Coverdell National Acute Stroke Registry program are to:
- Increase the number of states with Coverdell stroke registries.
- Develop and disseminate best practices in hospital recruitment and training, data collection and quality improvement based on lessons learned.
- Encourage the development of statewide systems of care for stroke patients through coordination with emergency medical services and collaboration among statewide partners.
- Communicate with major stakeholders in stroke care to ensure ongoing improvement in the quality of that care.

The long-term goal of this program is to ensure that all Americans receive the highest quality of acute stroke care currently available and to reduce the number of untimely deaths attributable to stroke, prevent stroke-related disability and prevent stroke patients from suffering recurrent strokes.

In addition, the Joint Commission on Accreditation of Healthcare Organization, a nonprofit, independent organization whose primary purpose is to provide voluntary accreditation for health care facilities, in 2003 began a program to provide primary stroke care certification to hospitals. The program was based upon recommendations for primary stroke centers and the American Stroke Center’s statements/guidelines for stroke care. A list of facilities that have earned the primary stroke center accreditation can be accessed at the Joint Commission’s website (http://www.qualitycheck.org/consumer/searchQCR.aspx). Use the “advanced” button to check by city or Zip Code for health care facilities with specialty programs and designations.

Conclusion

Strokes have long devastated lives, and they still do. But finally, there is some hope for those who suffer its effects. An effective treatment is now available for some people, and heightened awareness of that and the need to quickly seek aid when a stroke occurs should mean more people will not only survive a stroke, but also see many of its effects curtailed or reversed. The work will continue as researchers seek more and better treatments for stroke. Health care officials will continue to push the message that healthier choices make for a healthier person. And perhaps fewer and fewer people will hear the dreaded news that a loved one has suffered a stroke – or at least the news won't seem quite so hopeless.
 Apoptosis — a form of cell death involving shrinking of the cell and eventual disposal of the internal elements of the cell by the body’s immune system. Apoptosis is an active, non-toxic form of cell suicide that does not induce an inflammatory response. It is often called programmed cell death because it is triggered by a genetic signal, involves specific cell mechanisms and is irreversible once initiated.

 Arteriography — an X-ray of the carotid artery taken when a special dye is injected into the artery.

 Arteriovenous malformation (AVM) — a congenital disorder characterized by a complex tangled web of arteries and veins.

 Atherosclerosis — a blood vessel disease characterized by deposits of lipid material on the inside of the walls of large to medium-sized arteries that make the artery walls thick, hard, brittle, and prone to breaking.

 Atrial fibrillation — irregular beating of the left atrium, or left upper chamber, of the heart.

 Blood-brain barrier — an elaborate network of supportive brain cells, called glia, that surrounds blood vessels and protects neurons from the toxic effects of direct exposure to blood.

 Carotid artery — an artery located on either side of the neck that supplies the brain with blood.

 Carotid endarterectomy — surgery used to remove fatty deposits from the carotid arteries.

 Central stroke pain (central pain syndrome) — pain caused by damage to an area in the thalamus. The pain is a mixture of sensations, including heat and cold, burning, tingling, numbness, and sharp stabbing and underlying aching pain.

 Cerebrovascular disease — a reduction in the supply of blood to the brain either by narrowing of the arteries through the buildup of plaque on the inside walls of the arteries, called stenosis, or through blockage of an artery due to a blood clot.

 Cholesterol — a waxy substance, produced naturally by the liver and also found in foods, that circulates in the blood and helps maintain tissues and cell membranes. Excess cholesterol in the body can contribute to atherosclerosis and high blood pressure.

 “Clipping” — surgical procedure for treatment of brain aneurysms, involving clamping an aneurysm from a blood vessel, surgically removing this ballooned part of the blood vessel, and closing the opening in the artery wall.

 Computed tomography (CT) scan — a series of cross-sectional X-rays of the brain and head; also called computerized axial tomography or CAT scan.

 Coumadin® — a commonly used anticoagulant, also known as warfarin.

 Cytokines — small, hormone-like proteins released by leukocytes, endothelial cells and other cells to promote an inflammatory immune response to an injury.

 Cytotoxic edema — a state of cell compromise involving influx of fluids and toxic chemicals into a cell, causing subsequent swelling of the cell.

 Detachable coil — a platinum coil that is inserted into an artery in the thigh and strung through the arteries to the site of an aneurysm. The coil is released into the aneurysm creating an immune response from the body. The body produces a blood
clot inside the aneurysm, strengthening the artery walls and reducing the risk of rupture.

**Duplex Doppler ultrasound** – a diagnostic imaging technique in which an image of an artery can be formed by bouncing sound waves off the moving blood in the artery and measuring the frequency changes of the echoes.

**Dysarthria** – a disorder characterized by slurred speech due to weakness or incoordination of the muscles involved in speaking.

**Dysphagia** – trouble swallowing.

**Edema** – the swelling of a cell that results from the influx of large amounts of water or fluid into the cell.

**Embolic stroke** – a stroke caused by an embolus.

**Embolus** – a free-roaming clot that usually forms in the heart.

**Endothelial wall** – a flat layer of cells that make up the innermost lining of a blood vessel.

**Excitatory amino acids** – a subset of neurotransmitters; proteins released by one neuron into the space between two neurons to promote an excitatory state in the other neuron.

**Extracranial/intracranial (EC/IC) bypass** – a type of surgery that restores blood flow to a blood-deprived area of brain tissue by rerouting a healthy artery in the scalp to the area of brain tissue affected by a blocked artery.

**Functional magnetic resonance imaging (FMRI)** – a type of imaging that measures increases in blood flow within the brain.

**Glutamate** – also known as glutamic acid, an amino acid that acts as an excitatory neurotransmitter in the brain.

**Hemiparesis** – weakness on one side of the body.

**Hemiplegia** – complete paralysis on one side of the body.

**Hemorrhagic stroke** – sudden bleeding into or around the brain.

**Heparin** – a type of anticoagulant.

**High-density lipoprotein (HDL)** – also known as the good cholesterol; a compound consisting of a lipid and a protein that carries a small percentage of the total cholesterol in the blood and deposits it in the liver.

**Homeostasis** – a state of equilibrium or balance among various fluids and chemicals in a cell, in tissues, or in the body as a whole.

**Hypertension (high blood pressure)** – characterized by persistently high arterial blood pressure defined as a measurement greater than or equal to 140 mm/Hg systolic pressure over 90 mm/Hg diastolic pressure.

**Hypoxia** – a state of decreased oxygen delivery to a cell so that the oxygen falls below normal levels; see anoxia.

**Incidence** – the extent or frequency of an occurrence; the number of specific new events in a given period of time.

**Infarct** – an area of tissue that is dead or dying because of a loss of blood supply.

**Infarction** – a sudden loss of blood supply to tissue, causing the formation of an infarct.

**Interleukins** – a group of cytokine-related proteins secreted by leukocytes and involved in the inflammatory immune response of the ischemic cascade.

**Intracerebral hemorrhage** – occurs when a vessel within the brain leaks blood into the brain.

**Ischemia** – a loss of blood flow to tissue, caused by an obstruction of the blood vessel, usually in the form of plaque stenosis or a blood clot.

**Ischemic cascade** – a series of events lasting for several hours to several days following initial ischemia that results in extensive cell death and tissue damage beyond the area of tissue originally affected by the initial lack of blood flow.

**Ischemic penumbra** – areas of damaged, but still living, brain cells arranged in a patchwork pattern around areas of dead brain cells.

**Ischemic stroke** – ischemia in the tissues of the brain.

**Lacunar infarction** – occlusion of a small artery in the brain resulting in a small area of dead brain tissue, called a lacunar infarct; often caused by stenosis of the small arteries, called small vessel disease.

**Large vessel disease** – stenosis in large arteries of the cerebrovascular system.

**Leukocytes** – blood proteins involved in the inflammatory immune response of the ischemic cascade.

**Lipoprotein** – small globules of cholesterol covered by a layer of protein; produced by the liver.

**Low-density lipoprotein (LDL)** – also known as the bad cholesterol; a compound consisting of a lipid and a protein that carries the majority of the total cholesterol in the blood and deposits the excess along the inside of arterial walls.

**Magnetic resonance angiography (MRA)** – an imaging technique involving injection of a contrast dye into a blood vessel and using magnetic resonance techniques to create an image of the flowing blood through the vessel; often used to detect stenosis of the brain arteries inside the skull.

**Magnetic resonance imaging (MRI) scan** – a type of imaging involving the use of magnetic fields to detect subtle changes in the water content of tissues.

**Mitochondria** – the energy producing organelles of the cell.
Mitral annular calcification – a disease of the mitral valve of the heart.

Mitral valve stenosis – a disease of the mitral heart valve involving the buildup of plaque-like material on and around the valve.

Necrosis – a form of cell death resulting from anoxia, trauma or any other form of irreversible damage to the cell; involves the release of toxic cellular material into the intercellular space, poisoning surrounding cells.

Neuron – the main functional cell of the brain and nervous system, consisting of a cell body, an axon and dendrites.

Neuroprotective agents – medications that protect the brain from secondary injury caused by stroke.

Oxygen-free radicals – toxic chemicals released during the process of cellular respiration and released in excessive amounts during necrosis of a cell; involved in secondary cell death associated with the ischemic cascade.

Plaque – fatty cholesterol deposits found along the inside of artery walls that lead to atherosclerosis and stenosis of the arteries.

Plasticity – the ability to be formed or molded; in reference to the brain, the ability to adapt to deficits and injury.

Platelets – structures found in blood that are known primarily for their role in blood coagulation.

Prevalence – the number of cases of a disease in a population at any given point in time.

Recombinant tissue plasminogen activator (rtPA) – a genetically engineered form of tPA, a thrombolytic, anti-clotting substance made naturally by the body.

Small vessel disease – a cerebrovascular disease defined by stenosis in small arteries of the brain.

Stenosis – narrowing of an artery due to the buildup of plaque on the inside wall of the artery.

Stroke Belt – an area of the Southeastern United States with the highest stroke mortality rate in the country.

Stroke buckle – three Southeastern states, North Carolina, South Carolina and Georgia, that have an extremely high stroke mortality rate.

Subarachnoid hemorrhage – bleeding within the meninges, or outer membranes, of the brain into the clear fluid that surrounds the brain.

Thrombolytics – drugs used to treat an ongoing, acute ischemic stroke by dissolving the blood clot causing the stroke and thereby restoring blood flow through the artery.

Thrombosis – the formation of a blood clot in one of the cerebral arteries of the head or neck that stays attached to the artery wall until it grows large enough to block blood flow.

Thrombotic stroke – a stroke caused by thrombosis.

Tissue necrosis factors – chemicals released by leukocytes and other cells that cause secondary cell death during the inflammatory immune response associated with the ischemic cascade.

Total serum cholesterol – a combined measurement of a person’s high-density lipoprotein (HDL) and low-density lipoprotein (LDL).

tPA – see recombinant tissue plasminogen activator.

Transcranial magnetic stimulation (TMS) – a small magnetic current delivered to an area of the brain to promote plasticity and healing.

Transient ischemic attack (TIA) – a short-lived stroke that lasts from a few minutes up to 24 hours; often called a mini-stroke.

Vasodilators – medications that increase blood flow to the brain by expanding or dilating blood vessels.

Vasospasm – a dangerous side effect of subarachnoid hemorrhage in which the blood vessels in the subarachnoid space constrict erratically, cutting off blood flow.

Vertebral artery – an artery on either side of the neck; see carotid artery.

Warfarin – a commonly used anticoagulant, also known as Coumadin®.
APPENDIX

The ischemic cascade

The brain is the most complex organ in the human body. It contains hundreds of billions of cells that interconnect to form a complex network of communication. The brain has several different types of cells, the most important of which are neurons. The organization of neurons in the brain and the communication that occurs among them lead to thought, memory, cognition and awareness. Other types of brain cells are generally called glia (from the Greek word meaning “glue”). These supportive cells of the nervous system provide scaffolding and support for the vital neurons, protecting them from infection, toxins and trauma. Glia make up the blood-brain barrier between blood vessels and the substance of the brain.

Stroke is the sudden onset of paralysis caused by injury to brain cells from disruption in blood flow. The injury caused by a blocked blood vessel can occur within several minutes and progress for hours as the result of a chain of chemical reactions that is set off after the start of stroke symptoms. Physicians and researchers often call this chain of chemical reactions that lead to the permanent brain injury of stroke the ischemic cascade.

Primary cell death

In the first stage of the ischemic cascade, blood flow is cut off from a part of the brain (ischemia). This leads to a lack of oxygen (anoxia) and lack of nutrients in the cells of this core area. When the lack of oxygen becomes extreme, the mitochondria, the energy-producing structures within the cell, can no longer produce enough energy to keep the cell functioning. The mitochondria break down, releasing toxic chemicals called oxygen-free radicals into the cytoplasm of the cell. These toxins poison the cell from the inside-out, causing destruction of other cell structures, including the gated channels of the cell membrane that normally maintain homeostasis to open and allow toxic amounts of calcium, sodium and potassium ions to flow into the cell. At the same time, the injured ischemic cell releases excitatory amino acids, such as glutamate, into the space between neurons, leading to overexcitation and injury to nearby cells. With the loss of homeostasis, water rushes into the cell making it swell (called cytotoxic edema) until the cell membrane bursts under the internal pressure. At this point the nerve cell is essentially permanently injured and for all purposes dead (necrosis and infarction). After a stroke starts, the first cells that are going to die may die within four to five minutes. The response to the treatment that restores blood flow as late as 2 hours after stroke onset would suggest that, in most cases, the process is not over for at least 2 to 3 hours. After that, with rare exceptions, most of the injury that has occurred is essentially permanent.

Secondary cell death

Because of exposure to excessive amounts of glutamate, nitric oxide, free radicals and excitatory amino acids released into the intercellular space by necrotic cells, nearby cells have a more difficult time surviving. They are receiving just enough oxygen from cerebral blood flow (CBF) to stay alive. A compromised cell can survive for several hours in a low-energy state. If blood flow is restored within this narrow window of opportunity, at present thought to be about two hours, then some of these cells can be salvaged and become functional again. Researchers have learned that restoring blood flow to these cells can be achieved by administrating the clot-dissolving thrombolytic agent tPA within three hours of the start of the stroke.

Inflammation and the immune response

While anoxic and necrotic brain cells are doing damage to still viable brain tissue, the immune system of the body is injuring the brain through an inflammatory reaction mediated by the vascular system. Damage to the blood vessel at the site of a blood clot or hemorrhage attracts inflammatory blood elements to that site. Among the first blood elements to arrive are leukocytes, white blood cells that are covered with immune system proteins that attach to the blood vessel wall at the site of the injury. After they attach, the leukocytes penetrate the endothelial wall, move through the blood-brain barrier and invade the substance of the brain, causing further injury and brain cell death. Leukocytes called monocytes and macrophages release inflammatory chemicals (cytokines, interleukins, and tissue necrosis factors) at the site of the injury. These chemicals make it harder for the body to naturally dissolve a clot that has caused a stroke by inactivating anti-clotting factors and inhibiting the release of natural tissue plasminogen activator. NINDS researchers are currently working to create interventional therapies that will inhibit the effects of cytokines and other chemicals in the inflammatory process during stroke.

These brain cells survive the loss of blood flow (ischemia) but are not able to function. These areas of still-viable brain cells exist in a patchwork pattern within and around the area of dead brain tissue (also called an infarct).

NIH stroke scale
(on the following pages)
Patient Identification _ _ _ _ - _ _ _ _ - _ _ _ _ - _ _ _ _  
Pt. Date of Birth _ _ / _ _ / _ _  
Hospital __________________ ( _ _ - _ _ )  
Date of Exam _ _ / _ _ / _ _  

Interval: [ ] Baseline  [ ] 2 hours post treatment  [ ] 24 hours post onset of symptoms +20 minutes  [ ] 7 - 10 days  
[ ] 3 months  [ ] Other ________________________________ ( _ _ )  

Time: _ _ : _ _  [ ] am  [ ] pm  

Person Administering Scale ________________________________  

Administer stroke scale items in the order listed. Record performance in each category after each subscale exam. Do not go back and change scores. Follow directions provided for each exam technique. Scores should reflect what the patient does, not what the clinician thinks the patient can do. The clinician should record answers while administering the exam and work quickly. Except where indicated, the patient should not be coached (i.e., repeated requests to patient to make a special effort).

<table>
<thead>
<tr>
<th>Instructions</th>
<th>Scale Definition</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a. Level of Consciousness:</td>
<td>The investigator must choose a response if a full evaluation is prevented by such obstacles as an endotracheal tube, language barrier, orotracheal trauma/ bandages. A 3 is scored only if the patient makes no movement (other than reflexive posturing) in response to noxious stimulation.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0 = Alert; keenly responsive.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 = Not alert; but arousable by minor stimulation to obey, answer, or respond.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 = Not alert; requires repeated stimulation to attend, or is obtunded and requires strong or painful stimulation to make movements (not stereotyped).</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 = Responds only with reflex motor or autonomic effects or totally unresponsive, flaccid, and areflexic.</td>
<td></td>
</tr>
<tr>
<td>1b. LOC Questions: The patient is asked the month and his/her age. The answer must be correct - there is no partial credit for being close. Aphasics and stuporous patients who do not comprehend the questions will score 2. Patients unable to speak because of endotracheal intubation, orotracheal trauma, severe dysarthria from any cause, language barrier, or any other problem not secondary to aphasia are given a 1. It is important that only the initial answer be graded and that the examiner not “help” the patient with verbal or non-verbal cues.</td>
<td>0 = Answers both questions correctly.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 = Answers one question correctly.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 = Answers neither question correctly.</td>
<td></td>
</tr>
<tr>
<td>1c. LOC Commands: The patient is asked to open and close the eyes and then to grip and release the non-paretic hand. Substitute another one step command if the hands cannot be used. Credit is given if an unequivocal attempt is made but not completed due to weakness. If the patient does not respond to command, the task should be demonstrated to him or her (pantomime), and the result scored (i.e., follows none, one or two commands). Patients with trauma, amputation, or other physical impediments should be given suitable one-step commands. Only the first attempt is scored.</td>
<td>0 = Performs both tasks correctly.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 = Performs one task correctly.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 = Performs neither task correctly.</td>
<td></td>
</tr>
<tr>
<td>2. Best Gaze: Only horizontal eye movements will be tested. Voluntary or reflexive (oculocephalic) eye movements will be scored, but caloric testing is not done. If the patient has a conjugate deviation of the eyes that can be overcome by voluntary or reflexive activity, the score will be 1. If a patient has an isolated peripheral nerve paresis (CN III, IV or VI), score a 1. Gaze is testable in all aphasic patients. Patients with ocular trauma, bandages, pre-existing blindness, or other disorder of visual acuity or fields should be tested with reflexive movements, and a choice made by the investigator. Establishing eye contact and then moving about the patient from side to side will occasionally clarify the presence of a partial gaze palsy.</td>
<td>0 = Normal.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 = Partial gaze palsy; gaze is abnormal in one or both eyes, but forced deviation or total gaze paresis is not present.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 = Forced deviation, or total gaze paresis not overcome by the oculocephalic maneuver.</td>
<td></td>
</tr>
</tbody>
</table>

Rev 10/1/2003
### Strobe Scale

**Patient Identification**

| __ | __ | __ | __ |

**Pt. Date of Birth**

| __ | / | __ | __ |

**Hospital**

| __ | __ | __ | ( | __ | __ |

**Date of Exam**

| __ | / | __ | __ |

**Interval:**

- [ ] Baseline
- [ ] 2 hours post treatment
- [ ] 24 hours post onset of symptoms +20 minutes
- [ ] 3 months
- [ ] Other ___________________________ ( __ __ )
- [ ] 7 - 10 days

---

#### 3. Visual:

Visual fields (upper and lower quadrants) are tested by confrontation, using finger counting or visual threat, as appropriate. Patients may be encouraged, but if they look at the side of the moving fingers appropriately, this can be scored as normal. If there is unilateral blindness or enucleation, visual fields in the remaining eye are scored. Score 1 only if a clear-cut asymmetry, including quadrantanopia, is found. If patient is blind from any cause, score 3.

Double simultaneous stimulation is performed at this point. If there is extinction, patient receives a 1, and the results are used to respond to item 11.

| 0 = | No visual loss. |
| 1 = | Partial hemianopia. |
| 2 = | Complete hemianopia. |
| 3 = | Bilateral hemianopia (blind including cortical blindness). |

---

#### 4. Facial Palsy:

Ask – or use pantomime to encourage – the patient to show teeth or raise eyebrows and close eyes. Score symmetry of grimace in response to noxious stimuli in the poorly responsive or non-comprehending patient. If facial trauma/bandages, orotracheal tube, tape or other physical barriers obscure the face, these should be removed to the extent possible.

| 0 = | Normal symmetrical movements. |
| 1 = | Minor paralysis (flattened nasolabial fold, asymmetry on smiling). |
| 2 = | Partial paralysis (total or near-total paralysis of lower face). |
| 3 = | Complete paralysis of one or both sides (absence of facial movement in the upper and lower face). |

---

#### 5. Motor Arm:

The limb is placed in the appropriate position: extend the arms (palms down) 90 degrees (if sitting) or 45 degrees (if supine). Drift is scored if the arm falls before 10 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the non-paretic arm. Only in the case of amputation or joint fusion at the shoulder, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice.

| 0 = | No drift; limb holds 90 (or 45) degrees for full 10 seconds. |
| 1 = | Drift; limb holds 90 (or 45) degrees, but drifts down before full 10 seconds; does not hit bed or other support. |
| 2 = | Some effort against gravity; limb cannot get to or maintain (if cued) 90 (or 45) degrees, drifts down to bed, but has some effort against gravity. |
| 3 = | No effort against gravity; limb falls. |
| 4 = | No movement. |
| UN = | Amputation or joint fusion, explain: __________ |

---

#### 6. Motor Leg:

The limb is placed in the appropriate position: hold the leg at 30 degrees (always tested supine). Drift is scored if the leg falls before 5 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the non-paretic leg. Only in the case of amputation or joint fusion at the hip, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice.

| 0 = | No drift; leg holds 30-degree position for full 5 seconds. |
| 1 = | Drift; leg falls by the end of the 5-second period but does not hit bed. |
| 2 = | Some effort against gravity; leg falls to bed by 5 seconds, but has some effort against gravity. |
| 3 = | No effort against gravity; leg falls to bed immediately. |
| 4 = | No movement. |
| UN = | Amputation or joint fusion, explain: __________ |

---

**Rev 10/1/2003**
7. Limb Ataxia: This item is aimed at finding evidence of a unilateral cerebellar lesion. Test with eyes open. In case of visual defect, ensure testing is done in intact visual field. The finger-nose-finger and heel-shin tests are performed on both sides, and ataxia is scored only if present out of proportion to weakness. Ataxia is absent in the patient who cannot understand or is paralyzed. Only in the case of amputation or joint fusion, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice. In case of blindness, test by having the patient touch nose from extended arm position.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Absent</td>
</tr>
<tr>
<td>1</td>
<td>Present in one limb.</td>
</tr>
<tr>
<td>2</td>
<td>Present in two limbs.</td>
</tr>
<tr>
<td>UN</td>
<td>Amputation or joint fusion, explain:______________________________</td>
</tr>
</tbody>
</table>

8. Sensory: Sensation or grimace to pinprick when tested, or withdrawal from noxious stimulus in the obtunded or aphasic patient. Only sensory loss attributed to stroke is scored as abnormal and the examiner should test as many body areas (arms [not hands], legs, trunk, face) as needed to accurately check for hemisensory loss. A score of 2, “severe or total sensory loss,” should only be given when a severe or total loss of sensation can be clearly demonstrated. Stuporous and aphasic patients will, therefore, probably score 1 or 0. The patient with brainstem stroke who has bilateral loss of sensation is scored 2. If the patient does not respond and is quadriplegic, score 2. Patients in a coma (item 1a=3) are automatically given a 2 on this item.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal; no sensory loss.</td>
</tr>
<tr>
<td>1</td>
<td>Mild-to-moderate sensory loss; patient feels pinprick is less sharp or is dull on the affected side; or there is a loss of superficial pain with pinprick, but patient is aware of being touched.</td>
</tr>
<tr>
<td>2</td>
<td>Severe to total sensory loss; patient is not aware of being touched in the face, arm, and leg.</td>
</tr>
<tr>
<td>UN</td>
<td>Amputation or joint fusion, explain:______________________________</td>
</tr>
</tbody>
</table>

9. Best Language: A great deal of information about comprehension will be obtained during the preceding sections of the examination. For this scale item, the patient is asked to describe what is happening in the attached picture, to name the items on the attached naming sheet and to read from the attached list of sentences. Comprehension is judged from responses here, as well as to all of the commands in the preceding general neurological exam. If visual loss interferes with the tests, ask the patient to identify objects placed in the hand, repeat, and produce speech. The intubated patient should be asked to write. The patient in a coma (item 1a=3) will automatically score 3 on this item. The examiner must choose a score for the patient with stupor or limited cooperation, but a score of 3 should be used only if the patient is mute and follows no one-step commands.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No aphasia; normal.</td>
</tr>
<tr>
<td>1</td>
<td>Mild-to-moderate aphasia; some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression. Reduction of speech and/or comprehension, however, makes conversation about provided materials difficult or impossible. For example, in conversation about provided materials, examiner can identify picture or naming card content from patient’s response.</td>
</tr>
<tr>
<td>2</td>
<td>Severe aphasia; all communication is through fragmentary expression; great need for inference, questioning, and guessing by the listener. Range of information that can be exchanged is limited; listener carries burden of communication. Examiner cannot identify materials provided from patient response.</td>
</tr>
<tr>
<td>3</td>
<td>Mute, global aphasia; no usable speech or auditory comprehension.</td>
</tr>
</tbody>
</table>

10. Dysarthria: If patient is thought to be normal, an adequate sample of speech must be obtained by asking patient to read or repeat words from the attached list. If the patient has severe aphasia, the clarity of articulation of spontaneous speech can be rated. Only if the patient is intubated or has other physical barriers to producing speech, the examiner should record the score as untestable (UN), and clearly write an explanation for this choice. Do not tell the patient why he or she is being tested.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal.</td>
</tr>
<tr>
<td>1</td>
<td>Mild-to-moderate dysarthria; patient slurs at least some words and, at worst, can be understood with some difficulty.</td>
</tr>
<tr>
<td>2</td>
<td>Severe dysarthria; patient’s speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia, or is mute/anarthric.</td>
</tr>
<tr>
<td>UN</td>
<td>Intubated or other physical barrier, explain:______________________________</td>
</tr>
</tbody>
</table>

Rev 10/1/2003
11. Extinction and Inattention (formerly Neglect): Sufficient information to identify neglect may be obtained during the prior testing. If the patient has a severe visual loss preventing visual double simultaneous stimulation, and the cutaneous stimuli are normal, the score is normal. If the patient has aphasia but does appear to attend to both sides, the score is normal. The presence of visual spatial neglect or anosagnosia may also be taken as evidence of abnormality. Since the abnormality is scored only if present, the item is never untestable.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No abnormality.</td>
</tr>
<tr>
<td>1</td>
<td>Visual, tactile, auditory, spatial, or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities.</td>
</tr>
<tr>
<td>2</td>
<td>Profound hemi-inattention or extinction to more than one modality; does not recognize own hand or orients to only one side of space.</td>
</tr>
</tbody>
</table>

Rev 10/1/2003
AN OVERVIEW OF CARDIOVASCULAR NURSING

Final Examination Questions
Choose True or False for questions 21 through 30 and mark them on the answer sheet found on page 107
or complete your test online at Nursing.EliteCME.com

21. Cardiovascular disease is the leading cause of death worldwide and has been the leading cause of death since 1900 in the United States.
   - True
   - False

22. Cardiovascular diseases are mainly caused by a buildup of plaque (atherosclerosis) inside the coronary arteries.
   - True
   - False

23. Premature (extra) heart beats are dangerous and require immediate treatment.
   - True
   - False

24. For persons older than 50, a high systolic blood pressure is much less of an important cardiovascular disease factor than diastolic blood pressure.
   - True
   - False

25. The best way to prevent cardiovascular disease is to exercise, don’t smoke and eat a healthy diet.
   - True
   - False

26. Computed tomography (CT or CAT scan) is of little use in diagnosing strokes.
   - True
   - False

27. The thrombolytic drug tPA can be used for all strokes if administered quickly enough.
   - True
   - False

28. Of all the risk factors that contribute to stroke, the most powerful is hypertension.
   - True
   - False

29. Acute ischemic stroke comprises about 80 percent of cerebrovascular accidents.
   - True
   - False

30. Early mobilization of a patient after a stroke is discouraged.
   - True
   - False