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Objectives

1. Summarize nursing assessments and interventions used for the treatment of hypertension.
2. State recommended lifestyle modifications for a diagnosis of hypertension.
3. Identify 10 classes of drugs used to treat hypertension.
4. Review Figure 23-3 to identify options and progression of treatment for hypertension.
5. Identify specific factors the hypertensive patient can use to assist in managing the disease.
6. Develop patient education objectives for individuals with hypertension.
7. Summarize the action of each drug class used to treat hypertension.

Key Terms

arterial blood pressure  cardiac output (CO)
systolic blood pressure  hypertension
diastolic blood pressure  primary hypertension
pulse pressure  secondary hypertension
mean arterial pressure  systolic hypertension
(MAP)

CHAPTER 23

Drugs Used to Treat Hypertension

Chapter Content

Outlines

Hypertension
Prevention and Management of Hypertension
Drug Therapy for Hypertension
  Drug Class: Diuretics
  Drug Class: Beta Adrenergic–Blocking Agents
  Drug Class: Angiotensin-Converting Enzyme Inhibitors
  Drug Class: Angiotensin II Receptor Blockers
  Drug Class: Aldosterone Receptor Antagonist
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  Drug Class: Peripheral-Acting Adrenergic Antagonists
  Drug Class: Direct Vasodilators

HYPERTENSION

(For an introduction to cardiovascular diseases, see Chapter 21.) A primary function of the heart is to circulate blood to the organs and tissues of the body. When the heart contracts (systole), blood is pumped out through the pulmonary artery to the lungs and out through the aorta to the other organs and peripheral tissues. The pressure with which the blood is pushed from the heart is referred to as the arterial blood pressure or systolic blood pressure. When the heart muscle relaxes between contractions (diastole), the blood pressure drops to a lower level, the diastolic blood pressure. When recorded in the patient’s chart, the systolic pressure is recorded first, followed by the diastolic pressure (e.g., 120/80 mm Hg). The difference between the systolic and diastolic pressure is called the pulse pressure, which is an indicator of the tone of the arterial blood vessel walls. The mean arterial pressure (MAP) is the average pressure throughout each cycle of the heartbeat and is significant because it is the pressure that actually pushes the blood through the circulatory system to perfuse tissue. It is calculated by adding one third of the pulse pressure to the diastolic pressure or by using the following equation:

$$\text{MAP} = \frac{\text{systolic pressure} - \text{diastolic pressure}}{3} + \text{diastolic pressure}$$

Under normal conditions, the arterial blood pressure stays within narrow limits. It reaches its peak during high physical or emotional activity and is usually at its lowest level during sleep.

Arterial blood pressure (BP) can be defined as the product of cardiac output (CO) and peripheral vascular resistance (PVR):

$$\text{BP} = \text{CO} \times \text{PVR}$$

CO is the primary determinant of systolic pressure; peripheral vascular resistance determines the diastolic pressure. CO is determined by the stroke volume (the volume of blood ejected in a single contraction of the left ventricle), heart rate (controlled by the autonomic nervous system), and venous capacitance (capability of veins to return blood to the heart). Systolic blood pressure is thus increased by factors that increase heart rate or stroke volume. Venous capacitance affects the volume of blood (or preload) that is returned to the heart through the central venous circulation. Venous constriction decreases venous capacitance, increasing preload and systolic pressure, and venous dilation increases venous capacitance and decreases preload and systolic pressure. Peripheral vascular resistance is regulated primarily by contraction and dilation of arteri-
Hypertension is a disease characterized by an elevation of the systolic blood pressure, the diastolic blood pressure, or both. Statistics in North America show that blood pressures above 140/90 mm Hg are associated with premature death, which results from accelerated vascular disease of the brain, heart, and kidneys. Primary hypertension accounts for 90% of all clinical cases of high blood pressure. The cause of primary hypertension is unknown. At present, it is incurable but controllable. It is estimated that more than 50 million people in the United States have hypertension. The prevalence increases steadily with advancing age such that people who are normotensive at age 55 have a 90% lifetime risk of developing hypertension. In every age group, the incidence of hypertension is higher for African Americans than whites of both sexes. Other major risk factors associated with high blood pressure are listed in Box 23-1. Secondary hypertension occurs after the development of another disorder within the body (Box 23-2).

The Seventh Report of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure 2003 (JNC 7) has classified blood pressure by stages that represent the degree of risk of nonfatal and fatal cardiovascular disease events and renal disease (Table 23-1). The category of “prehypertension” was added to the classification system in the 2003 report because of the very high likelihood of people with a blood pressure in this range of having a heart attack, heart failure, stroke, and/or kidney disease. People with blood pressure in this range are in need of increased education and lifestyle modification to gain control of their blood pressure to prevent cardiovascular disease.

The JNC 7 guidelines consider an elevation in both systolic and diastolic blood pressure readings when making a diagnosis of hypertension. The individual should be seated quietly for at least 5 minutes in a chair (rather than an examination table), with feet on the floor, and the arm supported at heart level. An appropriately sized cuff (cuff bladder encircling at least 80% of the arm) should be used for accuracy. A person must have two or more elevated readings on two or more separate occasions after initial screening to be classified as having hypertension. When systolic and diastolic readings fall into two different stages, the higher of the two stages is used to classify the degree of hypertension present. Table 23-2 lists follow-up recommendations based on the initial set of blood pressure measurements. Measurement of blood pressure in the standing position is indicated periodically, especially in those at risk for postural hypotension.

In 2000, the Coordinating Committee of the National High Blood Pressure Education Program updated the JNC-VI guidelines and urged health practitioners to use the systolic blood pressure as the major criterion for the diagnosis and management of hypertension in middle-aged and older Americans. Prior to this time, the diastolic blood pressure had been the major determinant for the control of blood pressure. Recent evidence indicates that systolic hypertension is the most common form of hypertension and is present in about two thirds of hypertensive individuals older than 60 years of age.

When a person has been diagnosed with hypertension, further evaluation through medical history, physical examination, and laboratory tests should be completed to (1) identify causes of the high blood pressure, (2) assess the presence or absence of target organ damage and cardiovascular disease (see Box 23-1), and (3) identify other cardiovascular risk factors that may guide treatment (see Table 23-1).

**Box 23-1 Major Risk Factors Associated with Hypertension and Target Organ Damage**

<table>
<thead>
<tr>
<th>Major Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension*</td>
</tr>
<tr>
<td>Cigarette smoking</td>
</tr>
<tr>
<td>Obesity* (body mass index ≥30 kg/m²)</td>
</tr>
<tr>
<td>Physical inactivity</td>
</tr>
<tr>
<td>Dyslipidemia*</td>
</tr>
<tr>
<td>Diabetes mellitus*</td>
</tr>
<tr>
<td>Microalbuminuria or estimated glomerular filtration rate (GFR) &lt;60 mL/min</td>
</tr>
<tr>
<td>Age (older than 55 for men, 65 for women)</td>
</tr>
<tr>
<td>Family history of premature cardiovascular disease (men younger than age 55; women, age 65)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Target Organ Damage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
</tr>
<tr>
<td>Angina or prior myocardial infarction</td>
</tr>
<tr>
<td>Prior coronary revascularization</td>
</tr>
<tr>
<td>Heart failure</td>
</tr>
<tr>
<td>Brain</td>
</tr>
<tr>
<td>Stroke or transient ischemic attack</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
</tr>
<tr>
<td>Peripheral arterial disease</td>
</tr>
<tr>
<td>Retinopathy</td>
</tr>
<tr>
<td>Glomerular filtration rate (GFR)</td>
</tr>
<tr>
<td>Components of the metabolic syndrome</td>
</tr>
</tbody>
</table>


**Box 23-2 Identifiable Causes of Hypertension**

<table>
<thead>
<tr>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep apnea</td>
</tr>
<tr>
<td>Drug-induced or related causes</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
</tr>
<tr>
<td>Primary aldosteronism</td>
</tr>
<tr>
<td>Renovascular disease</td>
</tr>
<tr>
<td>Chronic steroid therapy and Cushing’s syndrome</td>
</tr>
<tr>
<td>Pheochromocytoma</td>
</tr>
<tr>
<td>Coarctation of the aorta</td>
</tr>
<tr>
<td>Thyroid or parathyroid disease</td>
</tr>
</tbody>
</table>

TREATMENT OF HYPERTENSION

The primary purpose for controlling hypertension is to reduce the frequency of cardiovascular disease (angina, myocardial infarction, heart failure, stroke, renal failure, retinopathy). To accomplish this goal, the blood pressure must be reduced and maintained below 140/90 mm Hg, if possible. Patients who also have conditions such as diabetes mellitus, heart failure, or renal disease should have a goal of less than 130/80 mm Hg. Major lifestyle modifications shown to lower blood pressure include weight reduction in those who are overweight or obese, adoption of the Dietary Approaches to Stop Hypertension (DASH) diet, dietary sodium reduction, physical activity, and moderation of alcohol consumption (Table 23-3). Treatment schedules should interfere as little as possible with the patient’s lifestyle; however, nonpharmacologic therapy must include elimination of smoking, weight control, routine activity, restriction of alcohol intake, stress reduction, and sodium control. If this therapy is successful in controlling high blood pressure, drug therapy is not necessary. Even if lifestyle changes are not adequate to control hypertension, they may reduce the number and doses of antihypertensive medications needed to manage the condition.

Patient education is vitally important in treating hypertension. This education should be emphasized and reiterated frequently by the physician, pharmacist, and nurse.

DRUG THERAPY FOR HYPERTENSION

Drugs used in the treatment of hypertension can be subdivided into several categories of therapeutic agents based on site of action (Figure 23-1). Clinical studies classify antihypertensive agents into preferred agents (diuretics and beta adrenergic blockers), alternative agents (angiotensin-converting enzyme [ACE] inhibitors, angiotensin II receptor antagonists [ARBs], calcium ion antagonists, and alpha-1 adrenergic blockers), and adjunctive agents (central-acting alpha-2 agonists, periph-

### Table 23-1 Classification and Management of Blood Pressure for Adults*

<table>
<thead>
<tr>
<th>BP CLASSIFICATION</th>
<th>SYSTOLIC</th>
<th>DIASTOLIC</th>
<th>LIFESTYLE MODIFICATION</th>
<th>INITIAL DRUG THERAPY WITHOUT COMPELLING INDICATION</th>
<th>INITIAL DRUG THERAPY WITH COMPELLING INDICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>and &lt;80</td>
<td>Encourage</td>
<td>No antihypertensive drug indicated</td>
<td>Drug(s) for compelling indications</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>or 80-89</td>
<td>Yes</td>
<td>No antihypertensive drug indicated</td>
<td>Drug(s) for compelling indications†</td>
</tr>
<tr>
<td>Stage 1 hypertension</td>
<td>140-159</td>
<td>or 90-99</td>
<td>Yes</td>
<td>Thiazide-type diuretics for most; may consider ACEI, ARB, BB, CCB, or combination</td>
<td>Drug(s) for the compelling indications†</td>
</tr>
<tr>
<td>Stage 2 hypertension</td>
<td>≥160</td>
<td>or ≥100</td>
<td>Yes</td>
<td>Two-drug combination for most‡ (usually thiazide-type diuretic and ACEI or ARB, or BB, or CCB)</td>
<td>Drug(s) for the compelling indications</td>
</tr>
</tbody>
</table>


*Treatment determined by highest BP category.
†Initial combined therapy should be used cautiously in those at risk for orthostatic hypotension.
ACEI, Angiotensin-converting enzyme inhibitor; ARB, angiotensin-receptor blocker; BB, beta blocker; CCB, calcium channel blocker.

### Table 23-2 Recommended Follow-Up Schedule After Initial Blood Pressure Measurement

<table>
<thead>
<tr>
<th>INITIAL BLOOD PRESSURE (mm Hg)*</th>
<th>SYSTOLIC</th>
<th>DIASTOLIC</th>
<th>FOLLOW-UP RECOMMENDED‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;130</td>
<td>&lt;85</td>
<td>Recheck in 2 years</td>
<td></td>
</tr>
<tr>
<td>130-139</td>
<td>85-89</td>
<td>Recheck in 1 year‡</td>
<td></td>
</tr>
<tr>
<td>140-159</td>
<td>90-99</td>
<td>Confirm within 2 months‡</td>
<td></td>
</tr>
<tr>
<td>160-179</td>
<td>100-109</td>
<td>Evaluate or refer to source of care within 1 month</td>
<td></td>
</tr>
<tr>
<td>≥180</td>
<td>≥110</td>
<td>Evaluate or refer to source of care immediately or within 1 week, depending on clinical situation</td>
<td></td>
</tr>
</tbody>
</table>

*If systolic and diastolic categories are different, follow recommendations for shorter time follow-up (e.g., 160/86 mm Hg should be evaluated or referred to source of care within 1 month).
‡Modify the scheduling of follow-up according to reliable information about past blood pressure measurements, other cardiovascular risk factors, or target organ disease.
§Provide advice about lifestyle modifications.
<table>
<thead>
<tr>
<th>MODIFICATION</th>
<th>RECOMMENDATION</th>
<th>APPROXIMATE SYSTOLIC BLOOD PRESSURE REDUCTION (RANGE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction</td>
<td>Maintain normal body weight (body mass index 18.5-24.9 kg/m²)</td>
<td>5-20 mm Hg/10 kg weight loss 8-14 mm Hg</td>
</tr>
<tr>
<td>Adopt DASH eating plan</td>
<td>Consume a diet rich in fruits, vegetables, and low-fat dairy products with a reduced content of saturated and total fat</td>
<td>2-14 mm Hg</td>
</tr>
<tr>
<td>Dietary sodium reduction</td>
<td>Reduce dietary sodium intake to no more than 100 mmol per day (2.4 g sodium or 6 g sodium chloride)</td>
<td>2-8 mm Hg</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Engage in regular aerobic physical activity such as brisk walking (at least 30 minutes per day, most days of the week)</td>
<td>4-9 mm Hg</td>
</tr>
<tr>
<td>Moderation of alcohol consumption</td>
<td>Limit consumption to no more than two drinks (1 oz or 30 ml ethanol [e.g., 24 oz beer, 10 oz wine, or 3 oz 80-proof whiskey]) per day in most men and no more than one drink per day in women and lighter weight persons</td>
<td>2-4 mm Hg</td>
</tr>
</tbody>
</table>


*For overall cardiovascular risk reduction, stop smoking. The effects of implementing these modifications are dose and time dependent and could be greater for some individuals. DASH, Dietary Approaches to Stop Hypertension.

**FIGURE 23-1** Sites of action of antihypertensive agents. \( \beta \)-blockers, beta adrenergic blockers; CCB, calcium channel blockers; ACE, angiotensin-converting enzyme inhibitors; ARB, angiotensin II receptor blocker; \( \alpha_1 \) blockers, alpha-1 adrenergic–blocking agents; central \( \alpha_2 \) agonists, central-acting alpha-2 agonists.
general-acting adrenergic antagonists, and direct vasodilators). Both preferred agents and alternative agents can be used alone, or in combination, to treat hypertension, but adjunctive agents should be used only in combination with a preferred or an alternative agent.

The guidelines also provide recommendations for specific groups of patients. For example, older patients with isolated systolic hypertension should first be treated with diuretics. Patients with diabetes and high blood pressure should be treated with the ACE inhibitors. Patients who have hypertension and have suffered a myocardial infarction should be treated with a beta adrenergic–blocking agent, and in most cases, an ACE inhibitor. Other studies have demonstrated that if a patient has heart failure, a diuretic and an ACE inhibitor may be beneficial. If a patient has angina pectoris, dihydropyridine calcium ion antagonists (e.g., amloidpine, nifedipine) may be added to other therapy because they have been proven to relieve chest pain and reduce the incidence of stroke. Other combinations of therapy found to be particularly effective are an ACE inhibitor plus a diuretic or calcium ion antagonist, or an ARB plus a diuretic. See individual monographs for mechanisms of action of each class of antihypertensive agent.

**Uses**

A key to long-term success with antihypertensive therapy is to individualize therapy for a patient based on demographic characteristics (e.g., age, gender, race), coexisting diseases and risk factors (e.g., migraine headaches, dysrhythmias, angina, diabetes mellitus), previous therapy (what has or has not worked in the past), concurrent drug therapy for other illnesses, and cost. As outlined in Figure 23-2, the JNC 7 recommends that if lifestyle modifications do not lower blood pressure adequately for patients with stage 1 or 2 hypertension, a diuretic or an alternative agent should be the initial treatment of choice. A low dose should be selected to protect the patient from adverse effects, although it may not immediately control the blood pressure. It must be recognized that it may take months to control hypertension adequately while avoiding adverse effects of therapy. If, after 1 to 3 months, the first drug is not effective, the dosage may be increased, another agent from another class may be substituted, or a second drug from another class with a different mechanism of action may be added (Figure 23-3). The guidelines also recommend that if the first drug started was not a diuretic, a diuretic should be initiated as the second drug, if needed, because the majority of patients will respond to a two-drug regimen if it includes a diuretic. In general, most patients with hypertension will require two or more antihypertensive medications to achieve goal blood pressure (<140/90 mm Hg, or <130/80 mm Hg for patients who have diabetes or chronic kidney disease). After blood pressure is reduced to the goal level and maintenance doses of medicines are stabilized, it may be appropriate to change a patient’s medication to a combination antihypertensive product to simplify the regimen and enhance compliance. See Table 23-4 for a list of the ingredients of antihypertensive combination products.

Patients with stage 2 hypertension may require more aggressive therapy with a second or third agent added if control is not achieved by monotherapy in a relatively short time. Patients with an average diastolic blood pressure of greater than 120 mm Hg require immediate therapy and, if significant organ damage is present, may require hospitalization for initial control.

Patients who have modified their lifestyles with appropriate exercise, diet, weight reduction, and control of hypertension for at least 1 year may be candidates for “step-down” therapy. The dosage of antihypertensive medications may be gradually reduced in a slow, deliberate manner. Most patients may still require some therapy, but occasionally, the medicine can be discontinued. Patients whose drugs have been discontinued should have regular follow-up examination because blood pressure often rises again to hypertensive levels, sometimes months or years later, especially if lifestyle modifications are not continued.

**NURSING PROCESS for Hypertensive Therapy**

**Assessment**

**History of Risk Factors**

- Make note of patient’s gender, age, and race. People who are older, male, and of the African American race have a higher incidence of hypertension.
- Has the client been told previously about the elevated blood pressure readings? If so, under what circumstances were the blood pressure readings taken?
- Is there a family history of hypertension, coronary heart disease, stroke, diabetes mellitus, or dyslipidemia?

**Smoking.** Obtain a history of the number of cigarettes or cigars smoked daily. How long has the person smoked? Has the person ever tried to stop smoking? Ask if the person knows what effect smoking has on the vascular system. How does the individual feel about modifying the smoking habit?

**Dietary Habits.** Obtain a dietary history. Ask specific questions to obtain data relating to the amount of salt used in cooking and at the table, as well as foods eaten that are high in fat, cholesterol, refined carbohydrates, and sodium. Using a calorie counter, ask the person to estimate the number of calories eaten per day. How much meat, fish, and poultry are eaten daily (size and number of servings)? Estimate the percent of total daily calories provided by fats. Discuss food preparation (e.g., baked, broiled, fried foods). How many servings of fruits and vegetables are eaten daily? What types of oils/fats are used in food preparation? See a nutrition text for further dietary history questions. What is the frequency and volume of alcoholic beverages consumed?

**Elevated Serum Lipids.** Ask whether the patient is aware of having elevated lipids, triglycerides, or cho-
If elevated, what measures has the person tried for reduction and what effect have the interventions had on the blood levels at subsequent examinations? Review laboratory data available (e.g., cholesterol, triglycerides, low-density lipoprotein [LDL], very low-density lipoprotein [VLDL]).

Renal. Has the patient had any laboratory tests to evaluate renal function (e.g., urinalysis: microalbuminuria, proteinuria, microscopic hematuria) or blood analysis showing an elevated blood urea nitrogen (BUN) or serum creatinine? Does the patient have nocturia?

Obesity. Weigh and measure the patient. Measure the waist circumference 2 inches above the navel. Ask about any recent weight gains or losses and whether intentional or unintentional. Note abnormal waist-hip ratio.

Psychomotor Functions
- Determine type of lifestyle. Ask the patient to describe exercise level in terms of amount (walking 3 miles), intensity (walking 3 mph), and fre-
frequency (walking every other day). Is the patient’s job physically demanding or of a sedentary nature?

- Determine psychological stress. How much stress does the individual estimate having in life? How does the person cope with stressful situations at home and in the workplace?
- Has the client experienced any fatigue or reduction in activity level due to intolerance or palpitations, angina, or dyspnea? When walking, does the individual experience severe leg cramps (claudication) that force him/her to stop and rest or to severely limit ambulation?

**Medication History**
- Has the patient ever taken or is the patient currently taking any medications for the treatment of high blood pressure? If blood pressure medications have been prescribed but are not being taken, why was the medicine discontinued? Were any side effects noticed while receiving the medications, and how did the patient manage them?
- Obtain a listing of all medications being taken, including prescribed, over-the-counter, herbal preparations, and street drugs. Research these medications in the drug monographs to determine potential drug-to-drug interactions that may affect the individual’s blood pressure or the effectiveness of the medicines prescribed.

- If the patient is female, ask if she is now or has been taking oral contraceptives or is receiving hormone replacement therapy (HRT).

**Physical Assessments**

**Blood Pressure.** Obtain two or more blood pressure measurements.
- The individual should be seated quietly for at least 5 minutes in a chair with back supported (rather than an examination table), with feet on the floor, and arm supported at heart level.
- An appropriately sized cuff (cuff bladder encircling at least 80% of the arm) should be used for accuracy.
- When measuring blood pressure, the cuff should be inflated to 30 mm Hg above the point at which the radial pulse disappears. The sphygmomanometer pressure should then be reduced at 2 to 3 mm/second. Two readings should be performed at least 1 minute apart.
- Verify the readings in the opposite arm. A difference in blood pressure between the two arms can be expected in about 20% of patients. The higher value should be the one used in treatment decisions.
- People must have two or more elevated readings on two or more separate occasions after initial screening to be classified as having hypertension.
- Orthostatic hypotension is defined by a decrease in systolic blood pressure of 20 mm Hg...
or more, or diastolic blood pressure of 10 mm Hg or more after 3 minutes of quiet standing. Food ingestion, time of day, age, and hydration can affect this form of hypotension, as can a history of parkinsonism, diabetes, or multiple myeloma.

- Ensure that the patient has not ingested caffeine within the past 2 to 3 hours.

**Height and Weight.** Weigh and measure the patient. What has the person’s weight been? Ask about any recent weight gains or losses and whether intentional or unintentional. Calculate the body mass index (BMI) (see Chapter 21 for more discussion and classification of BMI):

\[
\text{BMI (kg/m}^2\text{)} = \frac{\text{Weight (in kilograms)}}{\text{Height (in square meters [m}^2\text{])}}
\]

or

\[
\text{BMI (lb/in}^2\text{)} = \frac{\text{Weight (in pounds)}}{\text{Height (in square inches [in}^2\text{])}} \times 703
\]

**Bruit.** Check neck, abdomen, and extremities for the presence of bruits.

**Peripheral Pulses.** Palpate and record femoral, popliteal, and pedal pulses bilaterally.

**Eyes.** As appropriate to the level of education, perform a funduscopic examination of interior eye, noting arteriovenous nicking, hemorrhages, exudates, or papilledema.

**Nursing Diagnoses**

- Knowledge, deficient, related to hypertension (indication)
- Noncompliance with drug therapy (indication, side effects)
- Sexual dysfunction (side effects)

**Planning**

**History of Risk Factors**

- Examine data to determine the individual’s extent of understanding of hypertension and its control.
• Using the patient’s history, analyze lifestyle elements to determine health teaching needs of the individual and significant others.

Medication History

Plan patient education needed to implement or reinforce prescribed medication therapy.

Physical Assessment

Schedule physical assessments at specific intervals as appropriate to the patient’s status and clinical site policies (e.g., vital signs taken every 4 hours or 8 hours; intake and output, daily weights).

Baseline and Diagnostic Studies. Review the chart and reports available that are used to build baseline information (e.g., electrocardiogram; urinalysis; blood glucose and hematocrit, serum potassium, creatinine and calcium levels; a lipid profile [total cholesterol, LDL cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides] after a 9- to 12-hour fast).

Implementation

• Perform nursing assessments on a scheduled basis.
• Make referrals as indicated for stress management, smoking cessation, and dietary counseling and for an exercise program appropriate for the individual’s needs.
• When initiating antihypertensive therapy in the hospitalized patient, protect from possible falls secondary to hypotension by assisting during ambulation and carefully assessing for faintness. Take blood pressure in supine, sitting, and standing positions to identify hypotensive responses.

Patient Education and Health Promotion

Smoking. Suggest that the patient stop smoking. Explain the increased risk of coronary artery disease if the habit is continued. It may be necessary to settle for a drastic decrease in smoking in some people, although abstinence should be the goal.

Nutritional Status. Dietary counseling is essential in the treatment of hypertension. Control of obesity alone may be sufficient to alter the hypertensive condition. Most patients are placed on a reduced-sodium diet (2.3 g sodium or <6 g table salt per day). The goal of dietary therapy is a reduction of cholesterol, lipids saturated fat, and alcohol consumption. Foods high in potassium and calcium are encouraged to decrease blood pressure. See the DASH diet for further information (see Table 23-3).

Dietary planning should always involve the patient in menu planning so that personal preferences, availability of food products, and costs are discussed. Include the person who purchases and prepares the meals in the dietary counseling.

Show the patient various food labels and explain what ingredients indicate a high sodium content (e.g., salt, sodium, sodium chloride, sodium bicarbonate, sodium aluminum sulfate). Suggest the use of a variety of spices as substitutes for sodium when cooking. Explain foods that should be avoided in large quantities (e.g., bacon, smoked meats, crab meat, tuna, crackers, processed cheeses, ham).

Teach the individual to record weights in the same clothing, at the same time daily, and using the same scale. Generally, a weight gain or loss of more than 2 pounds is reported to the health care provider; however, specific parameters may vary and should be discussed during initiation of therapy.

Medication Regimen

• Caution the patient that for the first 2 weeks of antihypertensive therapy drowsiness may occur. Patients should be told that this side effect is self-limiting. They should be cautious in operating...

Blood Pressure Monitoring. Demonstrate the correct procedure for taking blood pressure. It is best to have the patient or family bring in the blood pressure equipment that will be used at home to perform the blood pressure measurement. Validate the patient’s and family’s understanding by having them perform this task on several occasions under supervision. Monitor blood pressure, pulse, and respirations at least every shift while hospitalized and upon discharge in accordance with the health care provider’s orders, usually daily. The patient should be given some numerical guidelines, as established by the health care provider, for a desired goal of therapy and what to do if this is not being achieved. Normal home blood pressure should be at least lower than 137/85 mm Hg. Nighttime home blood pressure is usually lower than daytime pressure.

Stress Management

• Identify stress-producing situations in the patient’s life and seek means to significantly reduce these factors. In some cases, referral for training in stress management, relaxation techniques, meditation, or biofeedback may be necessary. If stress is produced in the work setting, it may be appropriate to involve the industrial nurse.
• Stress within the family is often significant and may require professional counseling for the family and patient.

Exercise and Activity. Develop a plan for moderate exercise to improve the patient’s general condition. Consult the health care provider for any individual modifications deemed appropriate. Suggest including activities that the patient finds helpful in reducing stress. Nurses can help an individual increase physical activity throughout the day by encouraging them to:

• Play active games with their children
• Engage in a sport
• Find a friend with whom to walk or jog
• Take a class in yoga or tai chi
• Walk a dog
• Garden on the weekends
• Walk or bicycle to school or work
• Take the stairs, never the elevator
• Park the car at the farthest point in the parking lot at work, school, or when shopping

Stress Management

• Identify stress-producing situations in the patient’s life and seek means to significantly reduce these factors. In some cases, referral for training in stress management, relaxation techniques, meditation, or biofeedback may be necessary. If stress is produced in the work setting, it may be appropriate to involve the industrial nurse.
• Stress within the family is often significant and may require professional counseling for the family and patient.

Exercise and Activity. Develop a plan for moderate exercise to improve the patient’s general condition. Consult the health care provider for any individual modifications deemed appropriate. Suggest including activities that the patient finds helpful in reducing stress. Nurses can help an individual increase physical activity throughout the day by encouraging them to:

• Play active games with their children
• Engage in a sport
• Find a friend with whom to walk or jog
• Take a class in yoga or tai chi
• Walk a dog
• Garden on the weekends
• Walk or bicycle to school or work
• Take the stairs, never the elevator
• Park the car at the farthest point in the parking lot at work, school, or when shopping

Blood Pressure Monitoring. Demonstrate the correct procedure for taking blood pressure. It is best to have the patient or family bring in the blood pressure equipment that will be used at home to perform the blood pressure measurement. Validate the patient’s and family’s understanding by having them perform this task on several occasions under supervision. Monitor blood pressure, pulse, and respirations at least every shift while hospitalized and upon discharge in accordance with the health care provider’s orders, usually daily. The patient should be given some numerical guidelines, as established by the health care provider, for a desired goal of therapy and what to do if this is not being achieved. Normal home blood pressure should be at least lower than 137/85 mm Hg. Nighttime home blood pressure is usually lower than daytime pressure.

Medication Regimen

• Caution the patient that for the first 2 weeks of antihypertensive therapy drowsiness may occur. Patients should be told that this side effect is self-limiting. They should be cautious in operating...
power equipment and motor vehicles while this symptom exists.

- A common side effect of antihypertensive medications is hypotension. Instruct the patient to rise slowly from a sitting or supine position. Tell the patient to avoid standing for long periods, especially within 2 hours of taking antihypertensive medication. Weakness, dizziness, or faintness can usually be relieved by increasing muscular activity or by sitting or lying down.

- Teach the person to perform exercises that prevent blood pooling in the extremities when sitting or standing for long periods. These exercises include flexing the calf muscles, wiggling the toes, rising on the toes, and then returning to the feet in a flat position.

- Teach the person and significant other how to take and record blood pressure at prescribed intervals.

- The patient should always report a lack of response to the medication prescribed and/or a blood pressure that continues to rise after medications have been taken. (Ask the health care provider to state specific parameters.)

**Fostering Health Maintenance**

- Throughout the course of treatment, discuss medication information and how it will benefit the patient.

- Drug therapy is one component in the management of hypertension. Lifestyle changes are equally important to drug therapy; therefore the need to maintain an exercise program and modify dietary habits to control obesity and serum cholesterol is crucial. Cessation of smoking and minimal alcoholic intake are strongly recommended.

- Provide the patient and significant others with the important information contained in the specific drug monograph for the drugs prescribed. Additional health teaching and nursing interventions for drug side effects to expect and report will be found in each drug monograph.

- Seek cooperation and understanding of the following points so that medication compliance is increased: name of medication, dosage, routes and times of administration, side effects to expect, and side effects to report.

  - The most effective therapy prescribed by the health care provider will control hypertension only if the patient is motivated. Motivation improves when the patient has a positive experience with and trust in the health care providers. Empathy builds trust and is an excellent motivator.

**Written Record.** Enlist the patient’s aid in developing and maintaining a written record of monitoring parameters (e.g., blood pressures, weight, exercise) (see p. 373 and Appendix I). Complete the Premedication Data column for use as a baseline to track response to drug therapy. Ensure that the patient understands how to use the form and instruct the patient to bring the completed form to follow-up visits. During follow-up visits, focus on issues that will foster adherence with the therapeutic interventions prescribed.

**DRUG CLASS: Diuretics**

**Actions**

The diuretics act as antihypertensive agents by causing volume depletion, sodium excretion, and vasodilation of peripheral arterioles. The mechanism of peripheral arteriolar vasodilation is unknown.

**Uses**

There are four classes of diuretic agents: carbonic anhydrase inhibitors, thiazide and thiazide-like agents, loop diuretics, and potassium-sparing diuretics (see Chapter 29). The carbonic anhydrase inhibitors are weak antihypertensive agents and therefore are not used for this purpose. The potassium-sparing diuretics are rarely used alone but are commonly used in combination with the thiazide and loop diuretics for added antihypertensive effect and to counteract the potassium-excreting effects of these more potent diuretic-antihypertensive agents.

The diuretics are the most commonly prescribed antihypertensive agents because they are one of the classes of agents that have been shown to reduce cardiovascular morbidity and mortality associated with hypertension. The thiazides are most effective if the renal creatinine clearance is greater than 30 mL per minute; however, as renal function deteriorates, the more potent loop diuretics are needed to continue excretion of sodium and water.

Diuretics are also commonly prescribed in combination therapy. They potentiate the hypotensive activity of the nondiuretic antihypertensive agents, have a low incidence of adverse effects, and are often the least expensive of the antihypertensive agents.

Diuretics are used (often with other classes of antihypertensive therapy) to treat all stages of hypertension. The agents are discussed more extensively in Chapter 29.

**Nursing Process for Diuretic Agents**

**Premedication Assessment**

1. Obtain baseline blood pressure readings in supine and standing positions.
2. Obtain baseline weight, blood pressure, and apical pulse.
3. Initiate laboratory studies requested by the health care provider (e.g., electrolytes).
4. Obtain baseline assessments of patient’s state of hydration.

**Planning**

**Availability.** See Chapter 29.

**Implementation**

**Dosage and Administration.** See Chapter 29.
Drugs Used to Treat Hypertension  CHAPTER 23

Evaluation
See Chapter 29.

DRUG CLASS: Beta Adrenergic–Blocking Agents

Actions
The beta adrenergic–blocking agents (beta blockers) (see Table 13-3) inhibit cardiac response to sympathetic nerve stimulation by blocking the beta receptors. As a result, the heart rate, cardiac output, and consequently, the blood pressure, are reduced. The beta blockers also inhibit renin release from the kidneys, diminishing the cascade of the renin-angiotensin-aldosterone system that would induce vasoconstriction and sodium reabsorption aggravating hypertension.

Uses
The beta adrenergic–blocking agents are agents of another class that have been shown to reduce morbidity and mortality associated with hypertension; therefore they are widely used as antihypertensive agents. The clinical advantages of the beta adrenergic–blocking agents in treating hypertension include minimal postural or exercise hypotension, minimal effect on sexual function, little or no slowing of the central nervous system (CNS).

The JNC 7 recommends beta blockers as initial therapy for stages 1 and 2 hypertension. However, beta blockers are not as effective in African American patients and should be avoided in patients with asthma, type 1 diabetes mellitus, heart failure caused by systolic dysfunction, and peripheral vascular disease.

Nursing Process for Beta Adrenergic–Blocking Agents

Premedication Assessment
1. Check history for respiratory conditions that could be aggravated by bronchoconstriction, type 1 diabetes mellitus, heart failure, or peripheral vascular disease.
   If any of these conditions are present, contact the health care provider to discuss the situation before initiation of beta adrenergic–blocking agent therapy.
2. Obtain baseline blood pressure readings and apical pulse.

Planning
   Availability. See Table 13-3.

Implementation
   Dosage and Administration. See Table 13-3.

   Individualization of Dosage. Although the onset of activity is rapid, it may often take several days to weeks for a patient to show optimal improvement and become stabilized on an adequate maintenance dosage. Patients must be periodically reevaluated to determine the lowest effective dosage necessary to control the disorder being treated.

   Sudden Discontinuation. Patients must be counseled against poor compliance or sudden discontinuation of therapy without a health care provider’s advice. Sudden discontinuation of therapy has resulted in an exacerbation of anginal symptoms followed in some cases by myocardial infarction. When discontinuing long-term treatment with beta blockers, the dosage should be gradually reduced over a period of 1 to 2 weeks with careful monitoring of the patient. If anginal symptoms develop or become more frequent, beta blocker therapy should be restarted at least temporarily.

Evaluation
Most of the adverse effects associated with beta adrenergic–blocking agents are dose related. Response by individual patients is highly variable. Many of these side effects may occur but may be transient. Strongly encourage patients to see their health care provider before discontinuing therapy. Minor dosage adjustment may be all that is required for most side effects.

   Side Effects to Expect and Report

   Bradycardia, Peripheral Vasoconstriction (Purple Mottled Skin). Withhold additional doses until the patient is evaluated by a health care provider.

   Bronchospasm, Wheezing. Withhold additional doses until the patient has been evaluated by a health care provider.

   Diabetic Patients. Monitor for hypoglycemia: headache, weakness, decreased coordination, general apprehension, diaphoresis, hunger, or blurred or double vision. Many of these symptoms may be masked by the beta adrenergic–blocking agents. Notify the health care provider if any of the above mentioned symptoms are appearing intermittently.

   Heart Failure. Monitor patients for an increase in edema, dyspnea, crackles, bradycardia, and orthopnea. Notify the health care provider if these symptoms develop.

Drug Interactions
   Antihypertensive Agents. All the beta blockers have hypotensive properties that are additive with antihypertensive agents (e.g., guanethidine, methyldopa, hydralazine, clonidine, prazosin, minoxidil, captropil, diltiazem, verapamil, reserpine).

   If it is decided to discontinue therapy in patients receiving beta blockers and clonidine concurrently, the beta blocker should be withdrawn gradually and discontinued several days before the gradual withdrawal of the clonidine. Severe rebound hypertension may occur if the beta blocker is not gradually discontinued first.

   Beta Adrenergic Agents. Depending on the doses used, the beta stimulants (e.g., isoproterenol, metaproterenol, terbutaline, albuterol [see Table 13-2]) may inhibit the action of the beta blockers, and vice versa.

   Lidocaine, Procainamide, Phenytoin, Disopyramide, Digoxin. Although these drugs are occasionally used concurrently, monitor patients carefully for additional dysrhythmias, bradycardia, and signs of heart failure.
Enzyme-Inducing Agents. Enzyme-inducing agents such as cimetidine, phenobarbital, pentobarbital, and phenytoin enhance the metabolism of propranolol, metoprolol, pindolol, and timolol. This reaction probably does not occur with nadolol or atenolol because they are not metabolized but excreted unchanged. The dosage of the beta blocker may have to be increased to provide therapeutic activity. If the enzyme-inducing agent is discontinued, the dosage of the beta blocking agent will also require reduction.

Nonsteroidal Antiinflammatory Drugs (NSAIDs). Indomethacin, and possibly other prostaglandin inhibitors, inhibit the antihypertensive activity of the beta blockers, resulting in loss of hypertensive control.

The dosage of the beta blocker may have to be increased to compensate for the antihypertensive inhibitory effect of NSAIDs.

**DRUG CLASS: Angiotensin-Converting Enzyme Inhibitors**

**Actions**

Angiotensin-converting enzyme (ACE) inhibitors represent a major breakthrough in the treatment of hypertension. The renin-angiotensin-aldosterone system plays a major role in the regulation of blood pressure. When there is a reduction in blood pressure, sodium concentration, or renal blood flow, renin is secreted by the kidneys. The renin converts angiotensinogen, which is secreted by the liver, to angiotensin I. Angiotensin I is then converted by angiotensin I–converting enzyme to angiotensin II.

Angiotensin II produces potent vasoconstriction by acting on receptors within blood vessels. It also promotes aldosterone secretion, which causes sodium retention by stimulation of mineralocorticoid receptors in the adrenal cortex. These actions result in increased blood pressure secondary to the vasoconstriction and enhanced cardiac output secondary to sodium retention. The ACE inhibitors inhibit angiotensin I–converting enzyme, the enzyme responsible for the conversion of angiotensin I to angiotensin II, thus reducing serum levels of this potent vasoconstrictor and aldosterone stimulant.

**Uses**

The ACE inhibitors reduce blood pressure, preserve cardiac output, and increase renal blood flow. They are effective as single therapy for stage 1 or 2 hypertension, severe accelerated hypertension, and renal hypertension. The JNC 7 considers them an alternative to diuretic or beta blocker therapy. Although they may be used alone, they tend to be more effective when combined with diuretic therapy. They are not as effective in lowering blood pressure in African Americans unless used with a diuretic. Advantages of ACE inhibitors are the infrequency of orthostatic hypotension; lack of CNS depression and sexual dysfunction side effects; lack of aggravation of asthma, obstructive pulmonary disease, gout, cholesterol levels, or diabetes; and an additive effect with diuretics. The ACE inhibitors are also effective in the treatment of heart failure and post-myocardial infarction, and routinely used to slow the progression of diabetic nephropathy.

**Therapeutic Outcomes**

The primary therapeutic outcome expected from the ACE inhibitors is reduction in blood pressure.

**Nursing Process for Angiotensin-Converting Enzyme Inhibitors**

**Premedication Assessment**

1. Obtain baseline blood pressure readings in supine and standing positions and apical pulse.
2. Obtain a history of bowel elimination patterns.
3. Initiate laboratory studies as requested by the health care provider (e.g., renal function tests such as blood urea nitrogen [BUN] and serum creatinine, electrolytes, and complete blood count [CBC] to serve as a baseline for future comparison).
4. Ask whether the patient is pregnant or likely to become pregnant. If so, discuss with the health care provider before initiating ACE inhibitor therapy.
5. Ask if the patient has a persistent cough.

**Planning**

**Availability.** See Table 23-5.

**Implementation**

**Dosage and Administration.** See Table 23-5. Captopril should be administered without food and requires twice-daily dosing. All of the other agents are administered once daily.

NOTE: The initial doses of ACE inhibitors may cause hypotension with dizziness, tachycardia, and fainting; these adverse effects occur more commonly in patients also receiving diuretics. Symptoms occur within 3 hours after the first several doses. This effect may be minimized by discontinuing the diuretic 1 week before initiating ACE inhibitor therapy. Patients should be warned that this side effect may occur, that it is transient, and that they should lie down immediately if symptoms develop.

**Evaluation**

**Side Effects to Expect**

Nausea, Fatigue, Headache, Diarrhea. These side effects are usually mild and tend to resolve with continued therapy. Encourage the patient not to discontinue therapy without first consulting a health care provider.

Orthostatic Hypotension (Dizziness, Weakness, Fainting). Although these side effects are infrequent and usually mild, certain patients, particularly those also receiving diuretics, may suffer some degree of orthostatic hypotension, particularly when therapy is initiated. Observe the patient closely for at least 2 hours after the initial dose and for at least an additional hour until blood pressure has stabilized.
Monitor the blood pressure in both the supine and standing positions. Anticipate the development of positional hypotension and take measures to prevent an occurrence. Teach the patient to rise slowly from a supine position and to sit or lie down if feeling faint.

**Side Effects to Report**

Swelling of the Face, Eyes, Lips, Tongue; Difficulty Breathing. Angioedema has been reported to occur in a small number of patients, especially after the first dose. Patients should be cautioned to discontinue further therapy and seek medical attention immediately.

**Neutropenia.** Neutropenia (300 neutrophils/mm³) and agranulocytosis (drug-induced bone marrow suppression) have rarely been observed in patients receiving ACE inhibitors. The neutropenia appears within the first 3 to 12 weeks of therapy and develops slowly; the white count falls to its nadir in 10 to 30 days. The white count returns to normal about 2 weeks after discontinuation of ACE inhibitor therapy.
The patients most susceptible are those receiving captopril who also have impaired renal function or serious autoimmune diseases, such as lupus erythematosus, or who are exposed to drugs known to affect the white cells or immune response, such as corticosteroids.

Patients at risk should have differential and total white cell counts before initiation of therapy and then every 2 weeks thereafter for the first 3 months of therapy. Stress the importance of returning for this laboratory work. Patients should be told to notify their health care provider promptly if any evidence of infection such as sore throat or fever, which may be an indicator of neutropenia, should develop.

**Nephrotoxicity.** A small number of hypertensive patients who are receiving ACE inhibitors, particularly those with preexisting renal impairment and those also taking NSAIDs, have developed increases in BUN and serum creatinine. These elevations have usually been minor and transient, especially when the ACE inhibitor was administered concomitantly with a diuretic. Renal function should be monitored during the first few weeks of therapy. Report an increasing BUN and creatinine level. Dosage reduction of the ACE inhibitor or possible discontinuation of the NSAID or diuretic may be required.

**Hyperkalemia.** Because ACE inhibitors inhibit aldosterone, patients may develop slight increases in serum potassium. Approximately 1% of patients may develop hyperkalemia (greater than 5.7 mEq/L). Most cases resolve without discontinuation of therapy. Patients most susceptible to the development of hyperkalemia are those with renal impairment or diabetes mellitus and those already receiving a potassium supplement or a potassium-sparing diuretic. Many symptoms associated with altered fluid and electrolyte balance are subtle and interspersed with general symptoms of drug toxicity or the disease process itself.

Gather data relative to changes in the patient’s mental status (e.g., alertness, orientation, and confusion), muscle strength, muscle cramps, tremors, nausea, and general appearance (e.g., drowsy, anxious, or lethargic).

Always check the electrolyte reports for early indications of electrolyte imbalance. Keep accurate records of intake and output, daily weights, and vital signs.

**Chronic Cough.** As many as one third of patients receiving ACE inhibitors may develop a chronic, dry, nonproductive, persistent cough. This is thought to be due to an accumulation of bradykinin. It may appear from 1 week to 6 months after initiation of ACE inhibitor therapy. Women appear to be more susceptible than men. Patients should be told to contact their health care provider if the cough becomes troublesome. The cough resolves within 1 to 30 days after discontinuation of therapy. An angiotensin II receptor–blocking (ARB) agent may be substituted for the ACE inhibitor if the frequency of cough is excessive.

**Pregnancy.** Medicines that act directly on the renin-angiotensin system can cause fetal and neonatal harm. There is concern about the potential for birth defects in neonates whose mothers received ACE inhibitors, especially during the second and third trimesters of pregnancy. Women who wish to become pregnant or become pregnant while receiving ACE inhibitors should discuss alternative therapies with their health care provider as soon as possible.

**Drug Interactions**

**Drugs That Enhance Therapeutic and Toxic Effects.** Diuretics, phenothiazines, alcohol, beta adrenergic–blocking agents (e.g., propranolol, atenolol, pindolol), and other antihypertensive agents. Probenecid blocks the excretion of captopril, causing an increased antihypertensive effect. Monitor the blood pressure response to the cumulative effects of antihypertensive agents. Take the blood pressure in supine and standing positions.

**Drugs That Reduce Therapeutic Effects.** Antacids may diminish absorption of ACE inhibitors. Separate the administration times by 2 hours. NSAIDs may reduce the antihypertensive effects of the ACE inhibitors. Rifampin may decrease the antihypertensive effects of enalapril. Monitor carefully for poor blood pressure control or a gradually increasing blood pressure.

**Digoxin.** ACE inhibitors may increase the serum levels of digoxin. Monitor the patient for symptoms of anorexia, nausea, vomiting, headaches, blurred or colored vision, and bradycardia. A digoxin serum level may be ordered by the health care provider.

**Lithium.** ACE inhibitors may induce lithium toxicity. Monitor for lithium toxicity manifested by nausea, anorexia, fine tremors, persistent vomiting, profuse diarrhea, hyperreflexia, lethargy, and weakness.

**Hyperkalemia.** ACE inhibitors may cause small increases in potassium levels by inhibiting aldosterone secretion. Patients should not take dietary supplements of potassium or potassium-sparing diuretics (e.g., triamterene, spironolactone, amiloride) without specific approval from the health care provider. If a patient has received spironolactone or eplerenone up to several months before ACE inhibitor therapy, the serum potassium level should be monitored closely, because the potassium-sparing effect of spironolactone or eplerenone may persist.

**Capsaicin.** Capsaicin may cause or aggravate coughing associated with ACE inhibitor therapy. Monitor for increased frequency of dry, persistent cough. Report to the health care provider.

**DRUG CLASS: Angiotensin II Receptor Blockers**

**Actions**

The angiotensin II receptor blockers (also known as ARBs) are a class of antihypertensive agents that act by binding to angiotensin II receptor sites, blocking the very potent vasoconstrictor from binding to the receptor (also called AT₁ receptor) sites in the vascular smooth muscle, brain, heart, kidneys, and adrenal glands. The blood pressure–elevating and sodium-retaining effects of angiotensin II are thus blocked. The angiotensin II recep-
tor antagonists have no effect on renal function, prostaglandin levels, triglycerides, cholesterol, or blood glucose levels. These agents do not affect bradykinin and therefore do not cause a dry cough.

Uses
The angiotensin II receptor antagonists have been found to be as effective in lowering blood pressure as the ACE inhibitors and beta blockers. Men and women usually have similar responses; however, African American patients do not respond as well to monotherapy. The angiotensin II receptor antagonists are indicated for the treatment of hypertension and may be used alone or in combination with other antihypertensive agents. The blood pressure–lowering effect is seen within 1 week, but may take 3 to 6 weeks for full therapeutic effect. If the antihypertensive effect is not controlled by angiotensin II receptor antagonists alone, a low dose of a diuretic, usually hydrochlorothiazide, may be added.

Therapeutic Outcomes
The primary therapeutic outcome expected from angiotensin II receptor antagonists is reduction in blood pressure.

Nursing Process for Angiotensin II Receptor Antagonists

Premedication Assessment
1. Obtain baseline blood pressure readings in supine and standing positions and apical pulse.

2. Initiate laboratory studies requested by the health care provider (e.g., renal function tests such as BUN, serum creatinine, electrolytes, and CBC) to serve as a baseline for future comparison.
3. Ask whether the patient is pregnant or likely to become pregnant. If so, discuss with the health care provider before initiating angiotensin II receptor antagonist therapy.
4. Obtain a history of bowel elimination patterns and any gastrointestinal (GI) symptoms.

Planning
Availability. See Table 23-6.

Implementation
Dosage and Administration. See Table 23-6.

Evaluation
Side Effects to Expect
Headache, Dyspepsia, Cramps, Diarrhea. These side effects are usually mild and tend to resolve with continued therapy. Encourage the patient not to discontinue therapy without first consulting the health care provider.

Orthostatic Hypotension (Dizziness, Weakness, Faintness). Although these side effects are infrequent and usually mild, certain patients, particularly those also receiving diuretics, may suffer some degree of orthostatic hypotension, particularly when therapy is initiated. Observe the patient closely for at least 2 hours after the initial dose and for at least 1 additional hour until blood pressure has stabilized.

Drug Table 23-6 ANGIOTENSIN II RECEPTOR BLOCKERS

<table>
<thead>
<tr>
<th>GENERIC NAME</th>
<th>BRAND NAME</th>
<th>AVAILABILITY</th>
<th>DOSAGE RANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>candesartan</td>
<td>Atacand</td>
<td>Tablets: 4, 8, 16, 32 mg</td>
<td>PO: Initial—16 mg once daily; adjust over 4-6 weeks with total daily dosage from 8-32 mg. Dose may be administered once or twice daily for optimal control.</td>
</tr>
<tr>
<td>eprosartan</td>
<td>Teveten</td>
<td>Tablets: 600 mg</td>
<td>PO: Initial—600 mg once daily; adjust over 2-3 weeks with total daily dose of 400-800 mg daily. Dose may be administered once or twice daily for optimal control.</td>
</tr>
<tr>
<td>irbesartan</td>
<td>Avapro</td>
<td>Tablets: 75, 150, 300 mg</td>
<td>PO: Initial—150 mg once daily; adjust over 3-4 weeks with a total daily dose of 300 mg daily.</td>
</tr>
<tr>
<td>losartan</td>
<td>Cozaar</td>
<td>Tablets: 25, 50, 100 mg</td>
<td>PO: 50 mg once daily; adjust over 4-6 weeks with a total daily dose from 25-100 mg administered once or twice daily for optimal control.</td>
</tr>
<tr>
<td>olmesartan</td>
<td>Benicar</td>
<td>Tablets: 5, 20, 40 mg</td>
<td>PO: Initial—20 mg once daily; adjust over 2 weeks with total daily dose of 20-40 mg daily. Twice daily dosing offers no benefit.</td>
</tr>
<tr>
<td>telmisartan</td>
<td>Micardis</td>
<td>Tablets: 20, 40, 80 mg</td>
<td>PO: Initial—40 mg once daily; adjust over 4-6 weeks with total daily dose from 20-80 mg.</td>
</tr>
<tr>
<td>valsartan</td>
<td>Diovan</td>
<td>Capsules: 40, 80, 160, 320 mg</td>
<td>PO: Initial—80 mg once daily; adjust over 4-6 weeks with total daily dose from 80-320 mg.</td>
</tr>
</tbody>
</table>
Monitor the blood pressure in both the supine and standing positions. Anticipate the development of postural hypotension and take measures to prevent an occurrence. Teach the patient to rise slowly from a supine or sitting position and to sit or lie down if feeling faint.

**Side Effects to Report**

**Pregnancy.** Medicines that act directly on the renin-angiotensin system can cause fetal and neonatal harm. There is potential for birth defects in neonates whose mothers received ACE inhibitors, especially during the second and third trimesters of pregnancy. Women who wish to become pregnant or who become pregnant while receiving angiotensin II receptor antagonists should discuss alternative therapies with their health care provider as soon as possible.

**Hyperkalemia.** Because angiotensin II receptor antagonists inhibit aldosterone secretion, patients may develop slight increases in serum potassium. Most cases resolve without discontinuation of therapy. Patients most susceptible to the development of hyperkalemia are those with renal impairment or diabetes mellitus and those already receiving a potassium supplement, eplerenone, or potassium-sparing diuretic. Many symptoms associated with altered fluid and electrolyte balance are subtle and interspersed with general symptoms of drug toxicity or the disease process itself.

Gather data relative to changes in the patient’s mental status (e.g., alertness, orientation, confusion), muscle strength, muscle cramps, tremors, nausea, and general appearance (e.g., being drowsy, anxious, lethargic).

Always check the electrolyte reports for early indications of electrolyte imbalance. Keep accurate records of intake and output, daily weights, and vital signs.

**Drug Interactions**

**Drugs That Enhance Therapeutic and Toxic Effects.** Diuretics, phenothiazines, alcohol, beta adrenergic–blocking agents (e.g., propranolol, atenolol, pindolol), and other antihypertensive agents. Cimetidine and fluconazole inhibit the metabolism of losartan, causing an increased antihypertensive effect. Monitor the blood pressure response to the cumulative effects of antihypertensive agents. Take the blood pressure readings in supine and standing positions.

**Drugs That Reduce Therapeutic Effects.** Rifampin increases the metabolism of losartan, reducing its antihypertensive effects. The dosage of losartan may need to be increased, or the patient may be switched to another angiotensin II receptor antagonist.

**Hyperkalemia.** Angiotensin II receptor antagonists may cause small increases in potassium levels by reducing aldosterone secretion. Patients should not take dietary supplements of potassium or potassium-sparing diuretics (e.g., triamterene, spironolactone, amiloride) without specific approval from the health care provider. If a patient has received spironolactone or eplerenone up to several months before angiotensin II receptor antagonist therapy, the serum potassium level should be monitored closely because the potassium-sparing effect of spironolactone and eplerenone may persist.

**DRUG CLASS: Aldosterone Receptor Antagonist**

**eplerenone** (ep lehr’ en own)

**INSpra** (in’ sprah)

**Actions**

Eplerenone represents the first of a new class of antihypertensive agents known as aldosterone receptor blocking agents. The renin-angiotensin-aldosterone system plays a major role in the regulation of blood pressure. When there is a reduction in blood pressure, sodium concentration or renal blood flow, renin is secreted by the kidneys. The renin converts angiotensinogen, which is secreted by the liver, to angiotensin I. Angiotensin I is converted by angiotensin I–converting enzyme to angiotensin II. Angiotensin II produces potent vasoconstriction by acting on receptors within blood vessels. It also promotes aldosterone secretion, which causes sodium retention by stimulation of mineralocorticoid receptors in the adrenal cortex, blood vessels, and brain. These actions result in increased blood pressure secondary to the vasoconstriction and enhanced cardiac output secondary to sodium retention. Eplerenone, the aldosterone receptor–blocking agent, blocks stimulation of the mineralocorticoid receptors by aldosterone, thus preventing sodium reabsorption.

**Uses**

Eplerenone is used in treating hypertension either alone or in combination with other antihypertensive agents.

**Therapeutic Outcomes**

The primary therapeutic outcome expected from eplerenone is reduction in blood pressure.

**Nursing Process for Eplerenone**

**Premedication Assessment**

1. Obtain baseline blood pressure readings in supine and standing positions.
2. Obtain a history of bowel elimination patterns.
3. Initiate laboratory studies as requested by the health care provider (e.g., renal function tests such as BUN and serum creatinine, electrolytes, triglycerides, cholesterol, liver function tests, [e.g., bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyltransferase (GGT), alkaline phosphatase, prothrombin time] and uric acid) to serve as a baseline for future comparison.
4. Ask whether the patient is pregnant or likely to become pregnant. If so, discuss with the health care provider before initiating eplerenone therapy.
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CHAPTER 23

Planning

Availability. PO: 25 and 50 mg tablets.

Implementation

Dosage and Administration. PO: Initial: 50 mg once daily with or without food. The full therapeutic effect should be apparent within 4 weeks. For patients with an inadequate blood pressure response, the dosage may be increased to 50 mg two times daily.

NOTE: For patients older than the age of 65, or patients with mild to moderate hepatic failure, dosages greater than 50 mg daily are not recommended. For patients taking metabolism inhibitors such as cimetidine, erythromycin, saquinavir, verapamil, diltiazem, or fluconazole, the starting dose should be reduced to 25 mg once daily.

NOTE: Eplerenone therapy is contraindicated in patients with:

• Serum potassium greater than 5.5 mEq/L
• Type 2 diabetes with microalbuminuria
• Serum creatinine greater than 2.0 mg/dL in males or 1.8 mg/dL in females
• Creatinine clearance less than 50 mL/min
• Patients taking potassium-sparing diuretics (amiloride, spironolactone, or triamterene)
• Patients taking strong metabolic inhibitors (e.g., ketoconazole, itraconazole)

Evaluation

Side Effects to Expect

Nausea, Fatigue, Headache, Diarrhea. These side effects are usually mild and tend to resolve with continued therapy. Encourage the patient not to discontinue therapy without first consulting a health care provider.

Orthostatic Hypotension (Dizziness, Weakness, Faintness). Although these side effects are infrequent and usually mild, certain patients, particularly those also receiving diuretics, may suffer some degree of orthostatic hypotension, particularly when therapy is initiated.

Monitor the blood pressure in both the supine and standing positions. Anticipate the development of postural hypotension and take measures to prevent an occurrence. Teach the patient to rise slowly from a supine or sitting position and to sit or lie down if feeling faint.

Side Effects to Report

Hyperkalemia. Because eplerenone inhibits aldosterone, patients may develop slight increases in serum potassium. Patients most susceptible to the development of hyperkalemia (greater than 5.5 mEq/L) are those with renal impairment or diabetes mellitus. Many symptoms associated with altered fluid and electrolyte balance are subtle and interspersed with general symptoms of drug toxicity or the disease process itself.

Gather data relative to changes in the patient’s mental status (e.g., alertness, orientation, confusion), muscle strength, muscle cramps, heart rate and rhythm, tremors, nausea, and general appearance (e.g., drowsy, anxious, lethargic).

Always check the electrolyte reports for early indications of electrolyte imbalance. Keep accurate records of intake and output, daily weights, and vital signs.

Nephrotoxicity. A small number of hypertensive patients who are receiving eplerenone, particularly those with preexisting renal impairment, have developed increases in BUN and serum creatinine. Renal function should be monitored during the first few weeks of therapy. Report an increasing BUN and creatinine level. Dosage reduction or discontinuation of eplerenone may be required. Eplerenone therapy is not recommended in patients with creatinine clearances less than 50 mL/min.

Hypertriglyceridemia, Hypercholesterolemia, Hyperuricemia. Increases in serum triglyceride, cholesterol, and uric acid levels have been reported during eplerenone therapy. Report rising levels to the health care provider.

Hepatotoxicity. The symptoms of hepatotoxicity are anorexia, nausea, vomiting, jaundice, hepatomegaly, splenomegaly, and abnormal liver function tests (e.g., elevated bilirubin, AST, ALT, GGT, alkaline phosphatase, prothrombin time).

Gynecomastia, Vaginal Bleeding. A small number of men have developed gynecomastia and a small number of women have developed vaginal bleeding while receiving eplerenone therapy. Report these conditions to a health care provider.

Drug Interactions

Drugs That Enhance Therapeutic and Toxic Effects. Diuretics, phenothiazines, alcohol, beta adrenergic–blocking agents (e.g., propranolol, atenolol, pindolol), and other antihypertensive agents. Monitor the blood pressure response to the cumulative effects of antihypertensive agents. Take the blood pressures in supine and standing positions. Assess the patient for hypotension, lightheadedness, dizziness, and bradycardia. Provide for patient safety; prevent falls.

Drugs That May Induce Hyperkalemia. Concurrent use of eplerenone and the following agents may induce hyperkalemia: ACE inhibitors (e.g., lisinopril, captopril, enalapril, ramipril), angiotensin II receptor blockers (e.g., losartan, candesartan, valsartan), potassium-sparing diuretics (e.g., triamterene, amiloride, spironolactone), salt substitutes (often contain higher concentrations of potassium for flavoring), foods marketed as “low sodium” often have higher concentrations of potassium for flavoring.

Lithium. Eplerenone may induce lithium toxicity. Monitor for lithium toxicity manifested by nausea, anorexia, fine tremors, persistent vomiting, profuse diarrhea, hyperreflexia, lethargy, and weakness.

Grapefruit Juice, St. John’s Wort. Grapefruit juice and St. John’s wort slow the metabolism of eplerenone in a minor way. If the patient develops orthostatic hy-
potension, a dosage reduction in eplerenone may be required.

**DRUG CLASS: Calcium Ion Antagonists**

**Actions**

Calcium ion antagonists are known variously as calcium antagonists, calcium channel blockers, slow channel blockers, and calcium ion influx inhibitors. These agents inhibit the movement of calcium ions across a cell membrane. This results in fewer dysrhythmias, a slower rate of contraction of the heart, and relaxation of smooth muscle of blood vessels, resulting in vasodilation and reduced blood pressure. The calcium ion antagonists are classified by structure: benzothiazepines—diltiazem; d-methylpiperidinoalanine ether—bepridil; diphenylalkylamine—verapamil; and dihydropyridines—amlodipine, felodipine, isradipine, nicardipine, nifedipine, nimodipine, and nisoldipine.

**Uses**

Although each of these agents act by calcium ion inhibition, there are significant differences in clinical use because they act somewhat differently on coronary blood vessels, systemic blood vessels, the pacemaker cells of the heart, and the conducting tissue of the heart. Their clinical effects are also dependent on the type and severity of the patient’s disease. All of the available calcium channel blockers are effective antihypertensive agents, but clinicians tend to use the dihydropyridine group more often because they have better peripheral vasodilating effects, reducing afterload. Calcium channel blockers are more effective in patients with higher pretreatment blood pressures. They increase renal sodium excretion and are usually well tolerated. Calcium channel blockers are ideal as first- or second-line medicines in patients with hypertension and coexisting angina and are an alternative to the use of beta blockers in patients with asthma or diabetes mellitus. They are particularly effective in African Americans and elderly hypertensive patients, who are more likely to have low-renin hypertension. The calcium channel blockers also do not affect gout or peripheral vascular disease.

**Therapeutic Outcomes**

The primary therapeutic outcome expected from calcium ion antagonist therapy is reduction in blood pressure.

**Nursing Process for Calcium Ion Antagonist Therapy**

**Premedication Assessment**

1. Obtain baseline blood pressure readings in the supine and standing positions and apical pulse.
2. Obtain baseline weight.
3. If the patient is taking digoxin concurrently, initiate close monitoring for potential digitalis toxicity.

**Planning**

*Availability.* See Table 23-7.

**Implementation**

*Dosage and Administration.* See Table 23-7.

*Dosage Adjustments.* See individual drugs for dosage parameters. Adjustments are made based on the individual patient’s response to therapy.

**Evaluation**

*Side Effects to Report*

**Hypotension and Syncope.** Caution the patient that hypotension and syncope may occur during the first week. These side effects decline once the dosage is stabilized.

Take blood pressure readings every shift in the hospitalized patient and stress the need for the patient to monitor blood pressure after discharge.

Prevent hypotensive episodes by instructing the patient to rise slowly from a supine or sitting position and perform exercises to prevent blood pooling when standing or sitting in one position for prolonged periods. If faintness occurs, instruct the patient to sit or lie down.

**Edema.** Assess the patient for development of edema. Perform daily weights at the same time, in similar clothing, and on the same scale. Report increases in weight to the health care provider for further evaluation.

*Drug Interactions*

**Drugs That Enhance Therapeutic and Toxic Effects.** Diuretics, phenothiazines, alcohol, beta adrenergic–blocking agents (e.g., propranolol, atenolol, pindolol), histamine H₂ antagonists (e.g., cimetidine, ranitidine), and other antihypertensive agents. Monitor the blood pressure response to the cumulative effects of antihypertensive agents. Take the blood pressures in supine and standing positions. Assess the patient for hypotension, lightheadedness, dizziness, and bradycardia. Provide for patient safety; prevent falls.

**Digoxin.** Calcium ion antagonists may increase serum levels of digoxin. Monitor the patient for symptoms of anorexia, nausea, vomiting, headaches, blurred or colored vision, and bradycardia. The health care provider may order a digitalis serum level.

**Glucose Metabolism.** The dosage of oral hypoglycemic agents may require adjustment in patients with type 2 diabetes mellitus. Assess for signs of hyperglycemia. Perform blood glucose testing on a regular basis.

**Verapamil, Disopyramide.** DO NOT administer disopyramide 48 hours before or 24 hours after the administration of verapamil.

**DRUG CLASS: Alpha-1 Adrenergic–Blocking Agents**

**Actions**

The alpha-1 blockers—doxazosin, prazosin, and terazosin—act by blocking postsynaptic alpha-1 adrenergic receptors to produce arteriolar and venous vasodilation, reducing peripheral vascular resistance without reducing cardiac output or inducing a reflex tachycardia.
They produce a decrease in standing blood pressure slightly greater than in supine blood pressure. These agents also have a modest positive effect on serum lipids, increasing HDL cholesterol and reducing LDL cholesterol, total cholesterol, and triglyceride concentrations.

The alpha-1 blockers do not increase catecholamines; therefore there is no increase in heart rate or myocardial oxygen consumption. They also have no effect on uric acid concentrations.

Because of the presence of alpha-1 receptors on the prostate gland and certain areas of the bladder, terazosin and doxazosin are also able to reduce urinary outflow resistance in men with enlarged prostate glands.

Uses

These agents may be used alone or in combination with other antihypertensive agents in the treatment of stage 1 or 2 hypertension. They have additive effects with beta blockers and diuretics. The JNC 7 lists these agents as alternative drugs if beta blockers, diuretic therapy, or other alternative therapy is not successful or not tolerated. Blood pressure response with alpha-1 blockers appears to be similar in African American and white patients. They can be used safely in patients with angina, gout, and hyperlipidemia. The three alpha-1 blockers have similar antihypertensive effects and adverse effects. Doxazosin and terazosin have a longer...
duration of action and can be administered once daily. Prazosin is often used with diuretic therapy because of its tendency to cause sodium and water retention.

Doxazosin and terazosin are also used to reduce mild to moderate urinary obstruction manifestations (e.g., hesitancy, terminal dribbling of urine, interrupted stream, impaired size and force of stream, sensation of incomplete bladder emptying) in men with benign prostatic hyperplasia.

**Therapeutic Outcomes**
The primary therapeutic outcomes expected from alpha-1 adrenergic receptor blocker therapy are reduction of blood pressure and reduced symptoms and improvement in urine flow associated with prostatic enlargement.

**Nursing Process for Alpha-1 Adrenergic–Blocking Agents**

**Premedication Assessment**
1. Obtain baseline blood pressure readings in supine and standing positions and apical pulse.
2. Check if patient is pregnant or has a history of severe cerebral or coronary arteriosclerosis, gastritis, or peptic ulcer disease. (Reduction of blood pressure may diminish blood flow to these regions, which causes therapy to worsen the condition.)

**Planning**

**Availability.** See Table 23-8.

**Implementation**

**Dosage and Administration.** See Table 23-8.

NOTE: The initial doses of doxazosin, prazosin, and terazosin may cause hypotension with dizziness, tachycardia, and fainting; these adverse effects occur in less than 1% of patients starting therapy. Symptoms occur 15 to 90 minutes after initial doses and occur most often in patients who are already receiving propranolol (and presumably other beta adrenergic–blocking agents). This effect may be minimized by giving the first doses with food and limiting the initial dose to 1 mg. Patients should be warned that this side effect may occur, that it is transient, and that they should lie down immediately if symptoms develop.

**Evaluation**

**Side Effects to Expect**
Drowsiness, Headache, Dizziness, Weakness, Lethargy. Tell the patient that these side effects may occur but that they tend to be self-limiting. Tell the patient

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**Drug Table 23-8**

<table>
<thead>
<tr>
<th>GENERIC NAME</th>
<th>BRAND NAME</th>
<th>AVAILABILITY</th>
<th>DOSAGE RANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>doxazosin</td>
<td>Cardura</td>
<td>Tablets: 1, 2, 4, 8 mg</td>
<td>Hypertension: PO: Initial—1 mg daily AM or PM. Hypotensive effects are most likely within 2-6 hours. Monitor standing blood pressure. Maintenance—Increase to 2 mg, then, if needed, 4, 8, and 16 mg to achieve desired reduction in blood pressure. Benign prostatic hyperplasia: PO: Initial—as for hypertension. Increase dosage at weekly intervals to 2 mg, then 4 and 8 mg once daily. Maintenance—8 mg daily; monitor blood pressure.</td>
</tr>
<tr>
<td>prazosin</td>
<td>Minipress</td>
<td>Capsules: 1, 2, 5 mg</td>
<td>Hypertension: PO: Initial—1 mg two or three times daily with first dose at bedtime to reduce syncopal episodes. Maintenance—6-15 mg/day in two or three divided doses. Maximum dose—20-40 mg/day.</td>
</tr>
<tr>
<td>terazosin</td>
<td>Hytrin</td>
<td>Capsules: 1, 2, 5, 10 mg Tablets: 1, 2, 5, 10 mg</td>
<td>Hypertension: PO: Initial—1 mg at bedtime. Measure blood pressure 2-3 hours after dosing and evaluate for symptoms of dizziness or tachycardia; if response is substantially diminished at 24 hours, increase dosage. Maintenance—1-5 mg daily Maximum dose—20 mg/day. Benign prostatic hyperplasia: PO: Initial—as for hypertension; gradually increase dosage in stepwise fashion to 2, 5, or 10 mg daily for acceptable urinary output. Maintenance—10 mg daily for 4-6 weeks to assess urinary response. Maximum dose—20 mg/day.</td>
</tr>
</tbody>
</table>
Drugs Used to Treat Hypertension  

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not to stop taking the medication and to consult a health care provider if the problem becomes unacceptable.

**Dizziness, Tachycardia, Fainting.** These side effects occur in about 1% of patients when therapy is initiated. They develop 15 to 90 minutes after the first dose is taken. To decrease the incidence, administer the first dose with food and limit the initial dose to 1 mg.

Instruct the patient to lie down immediately if these symptoms start to occur, and provide for the patient's safety.

**Drug Interactions**

**Drugs That Enhance Therapeutic and Toxic Effects.** Diuretics, tranquilizers, alcohol, barbiturates, antihistamines, beta adrenergic–blocking agents (e.g., propranolol, atenolol, pindolol), and other antihypertensive agents. Monitor the blood pressure response to the cumulative effects of antihypertensive agents. Take the blood pressures in supine and standing positions.

Monitor for an increase in severity of side effects such as sedation, hypotension, and bradycardia or tachycardia.

**DRUG CLASS: Central-Acting Alpha-2 Agonists**

**Actions**

The central-acting alpha-2 agonists (e.g., clonidine, guanabenz, guanfacine, methyldopa) act by stimulating the alpha-adrenergic receptors in the brainstem, resulting in reduced sympathetic outflow from the CNS with a decrease in heart rate and peripheral vascular resistance, resulting in a drop in both systolic and diastolic blood pressure.

**Uses**

The alpha-2 agonists are considered to be adjunctive antihypertensive agents and are recommended for use only in combination with preferred or alternative antihypertensive agents. Clonidine is available as a transdermal therapeutic system (TTS) that is applied once weekly. These drugs cause more frequent side effects such as sedation, dizziness, dry mouth, fatigue, and sexual dysfunction. When used alone, methyldopa often causes fluid retention. They can safely be used in combination with other agents such as diuretics, vasodilators, and beta blockers.

**Therapeutic Outcomes**

The primary therapeutic outcome expected from the alpha-2 agonists is reduction in blood pressure.

**Nursing Process for Central-Acting Alpha-2 Agonists**

**Premedication Assessment**

1. Obtain baseline blood pressure readings in supine and standing positions and apical pulse.
2. Assess the patient’s mental status; affective and cognitive behaviors should be used as a baseline for subsequent comparison. If depression is suspected, report to the health care provider.
3. Obtain baseline data relating to usual sleep pattern.

**Planning**

**Availability.** See Table 23-9.

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**Drug Table 23-9  CENTRAL-ACTING ALPHA-2 AGONISTS**

<table>
<thead>
<tr>
<th>GENERIC NAME</th>
<th>BRAND NAME</th>
<th>AVAILABILITY</th>
<th>DOSAGE RANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>clonidine</td>
<td>Catapres</td>
<td>Tablets: 0.1, 0.2, 0.3 mg</td>
<td>PO: Initial—0.1 mg twice daily; Maintenance—0.2-0.8 mg daily in divided doses; Maximum—2.4 mg daily</td>
</tr>
<tr>
<td></td>
<td>Catapres-TTS</td>
<td>Transdermal patch: 2.5, 5, 75 mg</td>
<td>Transdermal—Apply to a hairless area of intact skin on upper arm or torso once every 7 days; use a different site each week; Initial—Start with 2.5-mg patch; after second week, add another 2.5-mg patch or use a larger system; Maximum—two 75-mg patches per week</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PO: Initial—2.5 mg twice daily; increase 4-8 mg daily every 1-2 weeks; Maximum—32 mg twice daily</td>
<td></td>
</tr>
<tr>
<td>guanabenz</td>
<td>Wytensin</td>
<td>Tablets: 4, 8 mg</td>
<td>PO: Initial—1 mg daily at bedtime; Maintenance—1-2 mg; Maximum—3 mg daily</td>
</tr>
<tr>
<td>guanfacine</td>
<td>Tenex</td>
<td>Tablets: 1, 2 mg</td>
<td>PO: Initial—1 mg twice daily; increase 4-8 mg daily every 1-2 weeks; Maximum—32 mg twice daily</td>
</tr>
<tr>
<td>methyldopa</td>
<td>Aldomet</td>
<td>Tablets: 250, 500 mg IV: 250 mg/5 mL in 5- and 10-mL vials</td>
<td>PO: Initial—250 mg two or three times daily; Maintenance—500 mg to 2 g daily in 2-4 doses</td>
</tr>
</tbody>
</table>

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Implementation

Dosage and Administration. See Table 23-9.

Sudden Discontinuation. Never suddenly discontinue clonidine or guanabenz because it may cause a rebound effect with a rapid increase in blood pressure, manifested by nervousness, agitation, restlessness, tremors, headache, nausea, and increased salivation.

Rebound symptoms are most pronounced after 1 to 2 months of therapy and may begin to appear within a few hours of a missed dose. Within 8 to 24 hours, severe symptoms may develop.

When therapy is to be discontinued, a gradual reduction in dosage is necessary over 2 to 4 days, during which blood pressure must be carefully monitored.

If the clonidine transdermal patch becomes loose, the adhesive overlay should be applied directly over the patch to ensure good adhesion.

Evaluation

Side Effects to Expect

Drowsiness, Dry Mouth, Dizziness. Tell the patient that these symptoms may occur but that they tend to be self-limiting. Tell the patient not to discontinue the medication and to consult a health care provider if the side effects become an unacceptable problem.

Altered Urine Color. Methyldopa or its metabolites may discolor the urine, causing it to darken on exposure to air. It is to be expected and is not harmful.

Altered Test Reactions. A false-positive urine glucose test may occur when using Clinitest. Diastix is not affected by methyldopa.

Methyldopa may cause up to 20% of patients to develop a positive reaction to the direct Coombs’ test. Less than 0.2% of these patients will develop hemolytic anemia, however. Blood counts should be determined annually during therapy to detect hemolytic anemia.

Side Effects to Report

Depression. Assess the patient’s affect (e.g., loneliness, sadness, anxiety, anger), cognition (e.g., confusion, ambivalence, loss of interest), and other behavioral responses (e.g., agitation, irritability, altered activity level, withdrawal) before starting therapy. After starting therapy with clonidine, carefully monitor the patient for changes in usual response patterns. Assess otherwise normal emotions for an increase in duration or intensity.

Note the patient’s degree of socialization, response to stimulation, and changes in interactions with others. All individuals taking this drug should be monitored for development of depression, especially those with a history of depression.

Rash. About 10% to 15% of patients using the clonidine patch develop contact dermatitis. Patients who develop moderate or severe erythema or vesicle formation at the site of application of clonidine transdermal patches should consult a health care provider about the possible need to remove the patch and use alternative therapy.

Drug Interactions

Drugs That Enhance Therapeutic and Toxic Effects. Guanethidine, digoxin, barbiturates, tranquilizers, antihistamines, alcohol, beta adrenergic-blocking agents (e.g., propranolol, atenolol, pindolol), verapamil, and other antihypertensive agents. Monitor the blood pressure response to the cumulative effects of antihypertensive agents. Take the blood pressure in supine and standing positions.

Monitor for an increase in severity of side effects such as sedation, hypotension, and bradycardia or tachycardia.

Drugs That Reduce Therapeutic Effects. Tricyclic antidepressants (e.g., amitriptyline, imipramine, desipramine), trazodone, and prazosin may block the antihypertensive effects of clonidine and methyldopa. The beta adrenergic-blocking agents (e.g., propranolol, atenolol, pindolol) may cause potentially life-threatening increases in blood pressure when taken with clonidine. Monitor carefully for poor blood pressure control or a gradually increasing blood pressure.

Sedative Effects. Alcohol, barbiturates, phenothiazines, benzodiazepines, and antihistamines all potentiate the sedative effects of guanabenz. Patients should be warned that their tolerance to alcohol and other depressants may be diminished.

Haloperidol. Methyldopa used concurrently with haloperidol may produce irritability, aggressiveness, abusive behavior, and dementia. Concurrent use is usually not recommended.

DRUG CLASS: Peripheral-Acting Adrenergic Antagonists

guanadrel (gwan’a drel)

HYLOREL (hi lor’ el)

Actions

Guanadrel is similar to guanethidine as an antihypertensive agent in that it causes a release and subsequent depletion of norepinephrine from adrenergic nerve endings. This causes a relaxation of vascular smooth muscle, which decreases total peripheral resistance and venous return. A hypotensive effect results that is greater in the standing than in the supine position. Heart rate is slightly decreased, but there is no significant change in cardiac output. Fluid retention often occurs.

Uses

Guanadrel is recommended for use in refractory hypertension uncontrolled by other agents with fewer side effects. Guanadrel is used in combination with a thiazide diuretic.

Therapeutic Outcomes

The primary therapeutic outcome of guanadrel is reduction in blood pressure.
**Nursing Process for Guanadrel**

**Premedication Assessment**
1. Obtain baseline blood pressure readings in the supine and standing positions and apical pulse.
2. Obtain baseline weight.

**Planning**

**Availability.** PO: 10 mg tablets.

**Implementation**

**Dosage and Administration.** Adult: PO: Initially 10 mg daily in two divided doses. Adjust the dosages weekly to monthly until the therapeutic goal has been attained. The usual dosage range is 20 to 75 mg divided into two or three daily doses.

**Evaluation**

**Side Effects to Expect**

**Orthostatic Hypotension.** Orthostatic hypotension occurs often, especially with sudden changes in posture. Patients can usually avoid this complication by rising slowly from supine and sitting positions. Patients should also be cautioned not to stand in one position for prolonged periods. These orthostatic effects are increased with alcohol consumption or prolonged standing with little movement.

**Sedation.** Sedation and lethargy commonly occur when guanadrel therapy is initiated or during adjustment to higher doses. These effects are most notable during the first few days and tend to subside with time.

**Side Effects to Report**

**Edema.** Some patients will develop significant salt and water retention, causing edema and heart failure. Weigh patients daily, using the same scale, at the same time of day, in similar clothing. Report increases of 2 pounds or more per week. Report edema of the extremities and increase in dyspnea, pallor, tachycardia, wheezing, and frothy or blood-tinged sputum.

**Drug Interactions**

**Drugs That Enhance Therapeutic and Toxic Effects.** Guanethidine, barbiturates, disopyramide, quinidine, diuretics, tranquilizers, antihistamines, alcohol, and beta adrenergic–blocking agents (e.g., propranolol, atenolol, pindolol), diuretics, and other antihypertensive agents. Monitor the blood pressure response to the cumulative effects of antihypertensive agents. Take the blood pressures in supine and standing positions. Monitor for an increase in severity of side effects such as sedation, hypotension, and bradycardia or tachycardia.

**Drugs That Reduce Therapeutic Effects.** Tricyclic antidepressants (e.g., amitriptyline, imipramine), amphetamines, ephedrine, phenothiazines, monoamine oxidase inhibitors (MAOIs), haloperidol. Monitor carefully for poor blood pressure control or a gradually increasing blood pressure.

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**Nursing Process for Guanethidine**

**Premedication Assessment**
1. Obtain baseline blood pressure readings in the supine and standing positions and apical pulse.
2. Obtain baseline weight.

**Planning**

**Availability.** PO: 10 and 25 mg tablets.

**Implementation**

**Dosage and Administration.** Adult: PO: Initially 10 mg daily. Increase the dose 10 mg every 5 to 7 days, if the blood pressure measurements so indicate and side effects are tolerable. Maintenance doses range between 25 and 50 mg daily; however, much higher doses are occasionally required.

**Evaluation**

**Side Effects to Expect**

**Lightheadedness, Weakness.** Guanethidine causes arteriolar and venous dilation that permits pools of blood to collect in the lower extremities, causing a reduction in cerebral blood flow. These symptoms often disappear during the day and can be lessened by rising slowly, sitting on the edge of the bed for a few minutes, and performing leg, foot, and toe exercises before standing. These orthostatic effects are increased with alcohol consumption or prolonged standing with little movement.

**Side Effects to Report**

**Edema.** Some patients will develop significant salt and water retention, causing edema and heart failure. Weigh patients daily, using the same scale, at the same
time of day, in similar clothing. Report increases of 2 pounds or more per week.

Report edema of the extremities, as well as increase in dyspnea, pallor, tachycardia, wheezing, and frothy or blood-tinged sputum.

**Drug Interactions**

**Drugs That Enhance Therapeutic and Toxic Effects.** Barbiturates, disopyramide, quinidine, diuretics, tranquilizers, antihistamines, alcohol, beta adrenergic–blocking agents (e.g., propranolol, atenolol, pindolol), and other antihypertensive agents. Monitor the blood pressure response to the cumulative effects of antihypertensive agents. Take the blood pressure in supine and standing positions. Monitor for an increase in severity of side effects, such as sedation, hypotension, and bradycardia or tachycardia.

**Drugs That Reduce Therapeutic Effects.** Tricyclic antidepressants (e.g., amitriptyline, imipramine), amphetamines, ephedrine, phenothiazines, and haloperidol. Monitor carefully for poor blood pressure control or a gradually increasing blood pressure.

**Insulin and Oral Hypoglycemic Agents.** Guanethidine may increase the hypoglycemic effects of insulin and oral hypoglycemic agents. Monitor patients with diabetes mellitus for headache, weakness, decreasing muscle coordination, and diaphoresis. (Onset of hypoglycemic symptoms may be rapid.) Give orange juice with 2 teaspoons of sugar if the patient is still alert and responsive.

**Diarrhea.** Diarrhea and stomach cramps may be associated with depressed sympathetic activity. These side effects tend to be self-limiting, but tell the patient to consult the health care provider if it becomes a serious problem.

**Nasal Stuffiness.** Encourage the patient not to treat this symptom with over-the-counter nasal decongestants because they aggravate the hypertension. Fortunately, this side effect tends to be self-limiting, but tell the patient to consult the health care provider if it becomes a serious problem.

**Depression.** Depression caused by this medication may progress to the point of the patient becoming suicidal.

**Evaluation**

**Side Effects to Expect**

**Nasal Stuffiness.** Encourage the patient not to treat this symptom with over-the-counter nasal decongestants because they aggravate the hypertension. Fortunately, this side effect tends to be self-limiting, but tell the patient to consult the health care provider if it becomes a serious problem.

**Diarrhea.** Diarrhea and stomach cramps may be associated with depressed sympathetic activity. These side effects tend to be self-limiting, but if they persist or if there is an increase in abdominal pain, the health care provider should be notified.

**Depression.** Depression caused by this medication may progress to the point of the patient becoming suicidal.

Assess the patient’s affect (e.g., loneliness, sadness, anxiety, anger), cognition (e.g., confusion, ambivalence, loss of interest), and other behavioral responses (e.g., agitation, irritability, altered activity level, withdrawal) before starting therapy. After starting medication therapy with reserpine, carefully monitor the patient for changes in usual response patterns. Assess otherwise normal emotions for an increase in duration or intensity.

Note the patient’s degree of socialization, responses to stimulation, and changes in interactions with others. All individuals taking this drug should be monitored for development of depression, especially those with a history of depression.

**Reserpine**

**Actions**

Reserpine is an alkaloid obtained from the root of a species of Rauwolfia. It is one of the oldest antihypertensive agents available. Reserpine acts as an antihypertensive agent by reducing norepinephrine levels in peripheral nerve endings, which slows heart rate and reduces peripheral vascular resistance. It also stimulates the vagus nerve, causing a further reduction in heart rate. Reserpine also depletes norepinephrine from various other organs, including the brain. Depletion of norepinephrine and serotonin in the brain may be the cause of the sedative and depressant actions of reserpine. Reserpine’s strong inhibition of sympathetic activity allows increased parasympathetic activity to occur, which is responsible for some of its side effects, including nasal stuffiness, increased gastric acid secretion, diarrhea, and bradycardia.

**Uses**

Reserpine has an extremely long duration of action; it may take 2 to 6 weeks before the maximum effect of the drug is seen. It is used to treat stage 1 hypertension. It is relatively inexpensive when compared with other antihypertensive agents and is thus preferred by fund-
Nightmares, Insomnia. If these symptoms occur, report them to the health care provider for evaluation. Drug therapy may need to be changed.

Gastric Symptoms. Patients experiencing gastric symptoms such as burning, pain, nausea, or vomiting should report them immediately because this medication can cause formation of new ulcers or exacerbation of old ulcers.

**Drug Interactions**

**Drugs That Enhance Therapeutic and Toxic Effects.** Phenothiazines, procainamide, disopyramide, thiothixene, quinidine, diuretics, tranquilizers, antihistamines, alcohol, beta adrenergic–blocking agents (e.g., propranolol, atenolol, pindolol), and other antihypertensive agents. Monitor the blood pressure response to cumulative effects of antihypertensive agents. Take blood pressure readings in supine and standing positions. Monitor for an increase in severity of side effects such as sedation, hypotension, and bradycardia or tachycardia.

**Drugs That Reduce Therapeutic Effects.** Tricyclic antidepressants (e.g., amitriptyline, imipramine, doxepin). Monitor carefully for poor blood pressure control or a gradually increasing blood pressure.

**DRUG CLASS: Direct Vasodilators**

- **Hydralazine (hy dral’ ah been)**
  - **Apresoline (ah pres’ o been)**

**Actions**

Hydralazine causes direct arteriolar smooth muscle relaxation, resulting in reduced peripheral vascular resistance. The reduction in peripheral resistance causes a reflex increase in heart rate, cardiac output, and renin release with sodium and water retention. Consequently, the hypotensive effectiveness is reduced unless the patient is also taking a sympathetic inhibitor (e.g., beta blockers) and a diuretic.

**Uses**

This antihypertensive agent is used to treat stage 2 hypertension and hypertension associated with renal disease and toxemia of pregnancy. It may also be used to provide symptomatic relief in patients with heart failure by reducing resistance (afterload) to left ventricular output. Because of the reflex increase in cardiac rate, hydralazine is often used in combination with a drug that inhibits tachycardia (e.g., beta blockers, clonidine, methyldopa).

A combination product (BiDil) containing hydralazine and isosorbide dinitrate has recently been approved by the U.S. Food and Drug Administration (FDA). This combination has been shown to reduce hospitalizations, improve quality of life, and reduce mortality among African Americans with hypertension and heart failure.

**Therapeutic Outcomes**

The primary therapeutic outcome of hydralazine is reduction in blood pressure.

**Nursing Process for Hydralazine**

**Premedication Assessment**

Obtain baseline blood pressure readings in the supine and standing positions and apical pulse.

**Planning**

**Availability.** PO: 10, 25, 50, and 100 mg tablets; intramuscular (IM), intravenous (IV): 20 mg/mL in 1-mL ampules.

**Implementation**

**Dosage and Administration.**

- **Adult:** PO: Initially, 10 mg four times daily for the first 2 to 4 days, then 25 mg four times daily. The second week, increase the dosage to 50 mg four times daily as the patient tolerates the dosage and the blood pressure is brought under control. IM, IV: 20 to 40 mg repeated as necessary. Monitor blood pressure often. Results usually become evident within 10 to 20 minutes.

**Evaluation**

**Side Effects to Expect**

- Nausea, Dizziness, Palpitations, Tachycardia, Numbness and Tingling in the Legs, Nasal Congestion. Although these symptoms may be anticipated, they require monitoring. If severe, they should be reported so that the dosage can be adjusted appropriately. Nasal congestion can be treated with an antihistamine, such as chlorpheniramine.

- **Orthostatic Hypotension.** This may occur particularly during initiation of therapy. Patients can usually avoid this complication by rising slowly from supine and sitting positions.

**Side Effects to Report**

- Fever, Chills, Joint and Muscle Pain, Skin Eruptions. Tell patients to report the development of these symptoms. Monitor laboratory reports for leukocyte counts and the antinuclear antibody (ANA) titer.

**Drug Interactions**

**Drugs That Enhance Therapeutic and Toxic Effects.** Diuretics, alcohol, beta adrenergic–blocking agents (e.g., propranolol, atenolol, pindolol), and other antihypertensive agents. Monitor the blood pressure response to the cumulative effects of antihypertensive agents. Take the blood pressure in supine and standing positions. Monitor for an increase in severity of side effects, such as sedation, hypotension, and bradycardia or tachycardia.

- **Minoxidil (min ox’ i dil)**
  - **Loniten (lon’ i ten)**

**Actions**

Minoxidil acts by direct relaxation of the smooth muscle of arterioles, reducing peripheral vascular resistance. Because of the decrease in peripheral vascular...
resistance, there is a compensatory increase in heart rate and sodium and water retention. For this reason, minoxidil is usually administered in conjunction with a beta adrenergic-blocking agent and a potent diuretic such as furosemide or bumetanide.

**Uses**
Minoxidil is used only for severely hypertensive patients who do not respond adequately to maximum therapeutic doses of a diuretic and two other antihypertensive agents.

**Therapeutic Outcomes**
The primary therapeutic outcome of minoxidil is reduction in blood pressure.

**Nursing Process for Minoxidil**

**Premedication Assessment**
1. Obtain baseline blood pressure reading in the supine and standing positions and apical pulse.
2. Obtain baseline weight.
3. Obtain resting pulse rate to serve as a baseline for subsequent comparisons.

**Planning**

**Availability.** PO: 2.5 and 10 mg tablets.

**Implementation**

**Dosage and Administration.** Adult: PO: Initially 5 mg daily. Dosage may be gradually increased after at least 3-day intervals to 10 mg, 20 mg, and then 40 mg daily in one or two doses. Maintenance: 10 to 40 mg daily. Maximum dosage is 100 mg daily.

**Evaluation**

**Side Effects to Expect**

**Hair Growth.** Within 3 to 6 weeks after starting therapy, about 80% of patients will start developing hypertrichosis, an elongation, thickening, and increased pigmentation of fine body hair. It is usually noticed first on the face and later extends to the back, arms, legs, and scalp. Growth may be controlled by shaving or by depilatory creams. After discontinuation, new hair growth stops, but it may take up to 6 months for a complete return to pretreatment appearance.

**Side Effects to Report**

**Gynecomastia.** Swelling or tenderness of the breasts may develop in men.

**Salt and Water Retention.** This drug is usually administered with a diuretic and a beta adrenergic-blocking agent to reduce the incidence of fluid retention and for additive antihypertensive effects. Perform daily weights using the same scale, in similar clothing, and at approximately the same time of day. Report gains of more than 2 pounds per week and swelling or puffiness of the face, ankles, or hands to a health care provider.

**Increased Resting Pulse.** Instruct and validate the patient’s ability to take own pulse. A resting pulse that increases 20 or more beats per minute above normal should be reported.

**Lightheadedness, Fainting, Dizziness.** These symptoms should be reported to a health care provider. If possible, the patient’s blood pressure during these episodes should be taken and reported.

**Orthostatic Hypotension.** This may occur, particularly during initiation of therapy. Patients can usually avoid this complication by rising slowly from supine and sitting positions.

**Heart Failure.** Assess for development of dyspnea, orthopnea, edema, and weight gain.

**Drug Interactions**

**Drugs That Enhance Therapeutic and Toxic Effects.** Diuretics, alcohol, beta adrenergic-blocking agents (e.g., propranolol, atenolol, pindolol), guanethidine, guanadrel, and other antihypertensive agents. Monitor the blood pressure response caused by the cumulative effects of antihypertensive agents. Take the blood pressure in supine and standing positions. Monitor for increase in severity of side effects, such as sedation, hypotension, and bradycardia or tachycardia.

**Key Points**
- The public has made significant strides in the past two decades in recognizing the risk factors associated with cardiovascular disease. This awareness has led to reduction in the incidence of heart attacks and strokes. Hypertension, however, is still a national health problem. Nurses can play a significant role in public education efforts, monitor for nonadherence, monitor blood pressure response to therapy, and en-
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- Encourage patients to make changes in lifestyle to reduce the severity of hypertension.
- Nonadherence to hypertensive therapy is a major problem, and nurses need to understand that educating the patient regarding the consequences of discontinuing therapy is not enough; rather the nurse must investigate the “whys” for discontinuing the therapy. Many of the reasons the patient gives may be overcome or reduced by proper interventions or a change in the type of antihypertensive agents prescribed.

Because hypertension is a “silent killer,” it is important that nurses and other health professionals extend public screening programs to the community.

Go to your Companion CD-ROM for Appendices, an Audio Glossary, animations, Drug Dosage Calculators, customizable Patient Self-Assessment forms, and Review Questions for the NCLEX® Examination.

Be sure to visit the companion Evolve site at http://evolve.elsevier.com/Clayton for WebLinks and additional online resources.

MATH REVIEW QUESTIONS

1. Order: Clonidine hydrochloride (Catapres) 0.6 mg PO daily in two divided doses.
   Available: Clonidine hydrochloride (Catapres) 0.1- and 0.2-mg tablets.
   Give: _____ tablets of ____ mg, and _____ tablets of ____ mg.
   (Catapres is also available in 0.3-mg tablets. What nursing action would be appropriate?)

2. Order: Enalapril (Vasotec) 10 mg bid
   Available: Vasotec 5-mg tablets
   Give: _____ tablets bid.

3. Order: Captopril (Capoten) 25 mg daily
   Available: Capoten 12.5 mg
   Give: _____ tablets daily

4. Order: Carvedilol (Coreg) 12.5 mg daily
   Available: Coreg 6.25 mg
   Give: _____ tablets daily.

5. Order: Propranolol (Inderal) 3 mg IV stat
   Available: Inderal 1 mg/mL in 1-mL ampules
   Give: _____ mL.
   (What must be monitored when administering this medication IV?)

CRITICAL THINKING QUESTIONS

1. A male patient is receiving methyldopa 3 g per day. Sexual dysfunction is a possible nursing diagnosis related to methyldopa therapy manifested by impotence or failure to ejaculate. Address the health teaching needed and how the nurse could approach this subject.

2. Discuss the essential patient education needed regarding the initiation of therapy with captopril.

3. State the nursing assessments needed to monitor therapeutic response and the development of side effects to expect or report from beta adrenergic–blocking agents and calcium ion antagonists.

4. Review beta adrenergic–blocking agent information in the monograph and develop patient education objectives for a patient receiving this class of drugs for treatment of hypertension.

5. A female patient is being started on a drug regimen for hypertension that includes the use of losartan. Initially her blood pressure (BP) is 160/100 mm Hg, pulse is 64, respirations are 20 per minute, and weight is 148 pounds. She seems quiet and introspective and contributes little information other than “yes” or “no” responses during an initial assessment. What further nursing actions would be appropriate?

6. Lifestyle modifications are extremely important for the treatment of hypertension, but habits can be difficult to change for patients. What should be the nurse’s approach?

Continued
With this type of antihypertensive agent, the premedication assessment should include checking for respiratory conditions that are present because this class of drug may cause a chronic cough:
1. angiotensin II receptor antagonists.
2. diuretics.
3. angiotensin-converting enzyme inhibitors.

The generic drug names of all but one drug in this classification end in “pril.”
1. Angiotensin II receptor antagonists
2. Diuretics
3. Angiotensin-converting enzyme inhibitors
4. Beta adrenergic–blocking agents

Angiotensin II receptor antagonists act by:
1. binding to angiotensin I receptor sites.
2. binding to angiotensin II receptor sites.
3. altering renal function.
4. altering calcium ion movement across the cells.

Initial therapy for hypertension is most often:
1. diuretics and alpha blockers.
2. beta blockers and calcium ion antagonists.
3. diuretics and angiotensin II receptor blockers.
4. diuretics or beta adrenergic–blocking agents.

A drug that reduces peripheral vascular resistance will:
1. decrease the heart rate.
2. decrease cardiac output.
3. reduce blood pressure.
4. increase blood pressure.

All antihypertensive agents in this class end in “sartan.”
1. Angiotensin II receptor antagonists
2. Diuretics
3. Angiotensin-converting enzyme inhibitors
4. Beta adrenergic–blocking agents

The aldosterone receptor blocker, eplerenone, acts by:
1. blocking conversion of angiotensin I to angiotensin II.
2. reducing sodium reabsorption.
3. volume depletion, sodium excretion, and vasodilation of peripheral arterioles.
4. blocking calcium ion movement across the cell membrane.