Teaching patients to tame their hypertension

Controlling hypertension can require a complex regimen of drugs, each with its own actions, adverse effects, and nursing considerations. Review what you need to know about the six classes of antihypertensives and what you need to teach your patients.

By Cheryl Dumont, PhD, RN, and Jennifer Hardware, MSN, RN

IF IT SEEMS AS THOUGH you spend a lot of time administering antihypertensives to your patients, it's probably because you do. About 30% of Americans and as many as 60% of hospitalized patients have hypertension.

And they aren't the only big numbers. At least a hundred antihypertensives from six drug classes are available, and many patients need some combination of these drugs to control their hypertension. That presents a real challenge for nurses who need to know the actions, adverse effects, nursing considerations, and patient teaching points for all these drugs.

Defining hypertension

Hypertension is called the *silent killer* because many people have it but don't know they do until complications develop. To aid early detection, the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure created a prehypertension category. (See *Prehypertension and hypertension: By the numbers.*)

The American Heart Association defines hypertension as any of the following:

- having an untreated systolic blood pressure of 140 mm Hg or higher
- having a diastolic blood pressure of 90 mm Hg or higher
- taking antihypertensives
- being told at least twice by a physician or other healthcare professional that you have hypertension.

Types of hypertension

The two major types of hypertension are primary (also called essential) and secondary. Primary hypertension, or hypertension without a known cause, accounts for more than 90% of hypertension diagnoses. Major risk factors for primary hypertension are related to lifestyle and family history or genetics. Fortunately, people can change their lifestyle, and an awareness of a family history or genetic tendency can help motivate them to do so. The condition is more common and more severe in non-Hispanic blacks, elderly people, and obese people. The goal of therapy for primary hypertension is control, not cure.

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LEARNING OBJECTIVES

1. Differentiate the classes of drugs used to treat hypertension.
2. Identify a unique adverse effect of each major class.
3. Describe the necessary patient education for each major class.
4. Explain the cardiovascular benefits of therapy with angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, and beta blockers.

Secondary hypertension results from conditions such as kidney disease, thyroid disease, pheochromocytoma, preeclampsia, and obstructive sleep disorder. If the condition is successfully treated, the hypertension resolves.

Other types of hypertension include isolated systolic hypertension (ISH), resistant hypertension (RH), and white-coat hypertension (WCH). More common in the elderly, ISH is a condition in which only the systolic blood pressure is elevated. Age and obesity are associated with RH, defined as high blood pressure that persists despite therapy with three antihypertensives from different classes including a diuretic. A person whose blood pressure rises when measured by a healthcare professional has WCH.

Regardless of the type, a hypertensive patient needs therapy. For every 20/10 mm Hg increase in blood pressure above 115/75 mm Hg, the risk of cardiovascular disease doubles.

Blood pressure regulation

Blood pressure regulation depends on these factors:

- fluid volume, or the pressure exerted by fluid on the vessel walls
- heart rate and contractility, or the force exerted when pushing blood out of the heart
- resistance of the peripheral blood vessels.

All hypertensive therapy focuses

Prehypertension and hypertension: By the numbers

The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure defines normal blood pressure (BP), prehypertension, and two stages of hypertension as shown below.

	Systolic BP (mm Hg)	Diastolic BP (mm Hg)
Normal BP	< 120	< 80
Prehypertension	120-139	80-89
Hypertension stage I	140-159	90-99
Hypertension stage II	≥ 160	≥ 100

If a patient's systolic BP and diastolic BP fall into different categories, use the higher category.

on one or more of these factors, whose physiology involves a complex interplay of the renin-angiotensin-aldosterone system (RAAS), hormones of the sympathetic nervous system, peripheral autoregulatory components, vascular endothelial mechanisms, and fluid and electrolytes.

Treatment begins with lifestyle changes, such as losing weight, exercising, stopping smoking, making dietary changes, and reducing stress. However, if these measures aren't enough to control hypertension, healthcare providers turn to antihypertensives.

These drugs fall into six classes: diuretics, adrenergic blockers, calcium channel blockers, drugs acting on the RAAS, direct vascular dilators, and central adrenergic agonists. Each of the six classes uses a distinct mechanism to achieve the same result, and each class has its own potential adverse effects.

Diuretics

Diuretics are the first-line therapy for hypertension. When a diuretic alone can't control the condition, a prescriber adds one or more other types of antihypertensives until the blood pressure is under control. Diuretics work by eliminating excess salt and water from the body, thus decreasing the pressure from fluid on the vessel walls.

The most common types of diuretics are potassium-sparing, loop, and thiazide. Prescribers often order a thiazide diuretic such as hydrochlorothiazide because these older drugs are not only effective but also relatively inexpensive. The major adverse effects of diuretics are dehydration and electrolyte imbalance.

Nursing considerations

- Diuretics can increase serum glucose and cholesterol levels, so monitor patients with diabetes or high cholesterol levels. (See *Conditions that can complicate therapy.*)
- Teach patients to take diuretics in the morning to avoid nocturnal diuresis and frequent nocturnal urination.
- Caution patients to stand up slowly to minimize the risk of dizziness from orthostatic hypotension.
- If your patient is taking a thiazide diuretic or loop diuretic, monitor him for signs of hypokalemia, such as muscle weakness and changes in mental status, including confusion and irritability.
- Patients taking a potassium-sparing diuretic, such as triamterene or the aldosterone antago-

Conditions that can complicate therapy

For patients with hypertension and another major condition, the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure makes the recommendations below. The numbered sequence serves only as a guide to stepwise therapy.

Conditions	Recommended therapy
Heart failure	1. Thiazide diuretic and angiotensin-converting enzyme (ACE) inhibitor 2. Beta blocker 3. Angiotensin II receptor blocker (ARB) 4. Aldosterone antagonist
Post-myocardial infarction	1. Beta blocker and ACE inhibitor 2. Aldosterone antagonist
High risk of cardiovascular disease	1. Beta blocker 2. ACE inhibitor 3. Calcium channel blocker 4. Diuretic
Diabetes	1. ACE inhibitor or ARB 2. Diuretic 3. Beta blocker 4. Calcium channel blocker
Chronic kidney disease	1. ACE inhibitor or ARB
Recurrent-stroke prevention	1. Diuretic and ACE inhibitor

nist spironolactone, are at risk for hyperkalemia. The risk is especially high in patients also taking an angiotensin-converting enzyme (ACE) inhibitor.
- Weigh patients daily at the same time using the same scale. Report a significant weight gain, such as 3 pounds in 3 days.
- Remind your patient that even if he feels fine, he should keep appointments with the healthcare provider because renal function must be monitored. (See *Teaching patients about their antihypertensives.*)

Teaching patients about their antihypertensives

When patients start antihypertensive therapy, advise them not to take over-the-counter drugs or herbal supplements without consulting their healthcare provider. As appropriate, provide patients with these cautions:
- **Nonsteroidal anti-inflammatory drugs, such as Motrin and Advil, can decrease antihypertensive effects.**
- **Antacids can interfere with the absorption of some antihypertensives.**
- **St. John's wort can increase metabolism and thus decrease blood levels of some drugs.**
- **Herbal supplements, such as ginseng and ephedra, may worsen hypertension.**
- **Garlic can potentiate antihypertensive effects.**

Also, give patients the following advice regarding diet, lifestyle, and prescription drugs:
- **Read food labels to avoid processed food and salt substitutes.**
- **Follow a low-sodium diet and don't add extra salt to food.**
- **Participate in mild to moderate exercise, such as walking short distances.**
- **Carry a list of your medications and make sure all prescribers have a copy to avoid possible drug interactions.**

Adrenergic blockers

Adrenergic blockers interfere with the sympathetic nervous system hormones that produce the fight-or-flight response, a response that increases blood flow to the heart, lungs, skeletal muscles, and brain.

When stimulated, $beta_1$ receptors in the heart increase heart rate and contractility, thus increasing cardiac output. Beta blockers, such as atenolol and metoprolol, selectively affect the $beta_1$ receptors in the heart, preventing increases in heart rate and contractility. Because they decrease the work of the heart, beta blockers protect it from ischemia and the damaging effects of hypertrophy and remodeling.

Another type of adrenergic receptor, alpha receptors, when stimulated, cause vasoconstriction of peripheral blood vessels, thus shunting blood to the heart, lungs, skeletal muscles, and brain. Alpha blockers, such as doxazosin and prazosin, inhibit this effect, resulting in vasodilation of the peripheral vessels and reduced blood pressure. A combination of an alpha and beta blocker, such as carvedilol and labetalol, can be prescribed to decrease cardiac output and increase peripheral vasodilation.

Nursing considerations

For beta-blocker therapy
- Teach patients that they shouldn't suddenly stop therapy. Because of the risk of rebound tachycardia and hypertension, a healthcare provider should monitor the cessation of beta-blocker therapy.
- Beta blockers can cause transient increases in serum lipid and glucose levels.
- Because beta blockers inhibit the sympathetic nervous system response, they also hide the symptoms of hypoglycemia and can be dangerous in patients with diabetes who use insulin.
- Some older beta blockers such as propranolol and high doses of $beta_1$ blockers can block the $beta_2$ receptors in the pulmonary vasculature, resulting in bronchoconstriction and asthma symptoms.
- Carefully assess patients with asthma or chronic lung disease for an exacerbation of their symptoms during beta-blocker therapy.
- Check for common adverse effects of beta blockers, such as dizziness, slowing of the pulse, fatigue, and hypotension.

For alpha-blocker therapy
- Warn patients about the risk of orthostatic hypotension, which can cause falls.
- Teach patients to take their first dose at bedtime and to move slowly from a sitting to a standing position.

Calcium channel blockers

The two types of calcium channel blockers are dihydropyridines and nondihydropyridines. Dihydropyridines, such as amlodipine and nifedipine, cause vasodilation of the peripheral blood vessels and the coronary arteries but no reduction in heart rate. These drugs may cause severe vasodilation, resulting in dizziness, and a reflex sympathetic discharge, causing tachycardia, flushing, and headache—reactions that can be dangerous in patients who have coronary artery disease and are prone to angina. To decrease the chances of sudden hypotension and a reflex tachycardia response, prescribers should order sustained-release formulations.

Nondihydropyridines, such as diltiazem and verapamil, block the slow calcium channels in the heart and reduce heart rate and cardiac output, thus reducing blood pressure. The key adverse effects are bradycardia and heart block. These drugs also have a negative inotropic effect and can precipitate heart failure in patients who have preexisting abnormalities.

Nursing considerations

- Tell patients to report dizziness and symptoms of an irregular heart rate.

- Teach your patients to avoid grapefruit juice because it inhibits the hepatic metabolism of calcium channel blockers and may lead to increased blood drug levels and increased pharmacologic effects.
- All calcium channel blockers should be used cautiously in patients with heart failure.
- Drugs that inhibit cytochrome P450 isoenzymes, such as erythromycin, inhibit the metabolism of amlodipine and may result in a stronger antihypertensive effect.

Drugs acting on the RAAS

The RAAS is the most powerful and complex hormonal component of blood pressure control. Three types of drugs lower blood pressure by inhibiting the RAAS: ACE inhibitors, angiotensin II receptor blockers (ARBs), and renin inhibitors. (See *Understanding the renin-angiotensin-aldosterone system.*)

ACE inhibitors interfere with the final step in the conversion of angiotensin I to angiotensin II, which works as a potent vasoconstrictor and stimulates aldosterone secretion from the adrenal cortex. Aldosterone, in turn, increases reabsorption of sodium and water by the kidneys. Angiotensin II increases myocardial and vascular endothelial remodeling, producing harmful cardiovascular effects. Studies show reduced morbidity and mortality for patients with heart failure who take ACE inhibitors. For patients with heart disease, therapy with ACE inhibitors or ARBs and beta blockers is considered best practice.

Angiotensin has 2 receptors, type 1 and type 2. ARBs block the receptor for type 1 and thus inhibit vasoconstriction, aldosterone and antidiuretic hormone secretion, sodium and water retention, sympathetic nervous system stimulation, and cellular growth. ARBs don't block receptors for type 2, which provides beneficial effects, including vasodilation, differentiation and development of tissues, and prevention of overgrowth and hypertrophy.

Renin inhibitors block the ability of renin to convert angiotensinogen to angiotensin I, thus stopping angiotensin II production early in the process.

Drugs that interfere with the RAAS not only lower blood pressure but also inhibit vascular hyperplasia and cardiac-muscle remodeling that occur in response to injury from chronic hypertension and myocardial infarction. These drugs are thought to be most beneficial for younger patients and white patients, who tend to have a more active renin system. Still, elderly patients and nonwhite patients receive the cardioprotective effects of these drugs.

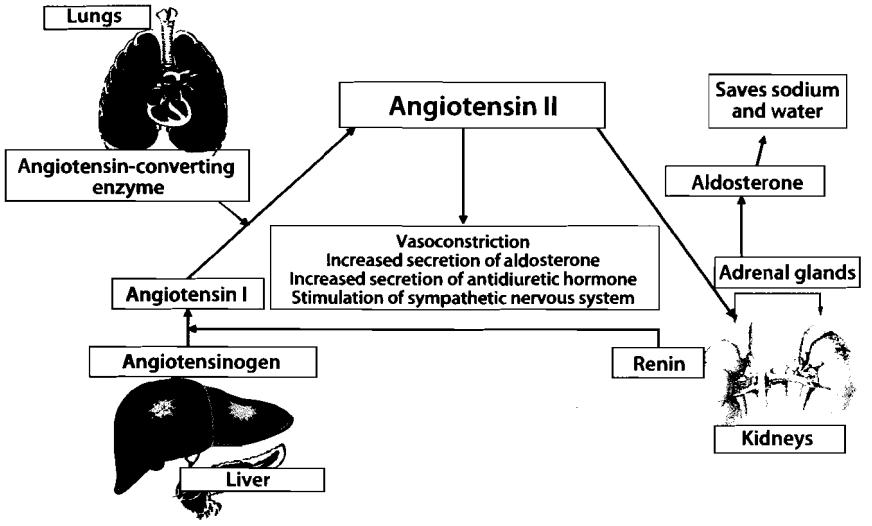
Nursing considerations

- Teach patients taking a drug that affects the RAAS system that they are at risk for high potassium levels, especially if they are taking a potassium supplement.
- Tell patients, especially those taking an ACE inhibitor, that they may experience a dry cough.
- Infrequent but dangerous adverse effects of drugs that act on the RAAS include agranulocytosis, proteinuria, glomerulonephritis, acute kidney failure, and angioedema.
- Angioedema, which occurs more often in blacks and smokers, can lead to airway swelling and requires emergent treatment.
- A history of angioedema contraindicates the use of ACE inhibitors. Prescribing ARBs to patients with a history of angioedema is controversial.

Direct vascular dilators

Direct vascular dilators, such as hydralazine and minoxidil, relax the smooth muscle in the arterial walls.

Understanding the renin-angiotensin-aldosterone system



The renin-angiotensin-aldosterone system involves a cascade of interactions and feedback mechanisms between hormones and neurotransmitters from the sympathetic nervous system, kidneys, adrenal glands, liver, and lungs.

Angiotensinogen is produced in the liver. In response to low blood flow, the kidneys produce renin, which acts on angiotensinogen to produce angiotensin I. Angiotensin-converting enzyme (ACE), which is made mostly in the lungs, then converts angiotensin I to angiotensin II. Angiotensin II acts on the adrenal glands to increase aldosterone production, causing sodium and water retention. Angiotensin II also causes vasoconstriction, secretion of antidiuretic hormone, and stimulation of the sympathetic nervous system, thus increasing blood pressure.

Because they don't improve cardiovascular health and may produce certain adverse effects, they aren't recommended as first-line drugs. Usually, they are added to the regimen when patients are resistant to diuretics, RAAS blockers, calcium channel blockers, and beta blockers.

Because direct vascular dilators work exclusively by decreasing vascular resistance, they can cause a precipitous drop in blood pressure followed by baroreceptor-mediated rebound tachycardia and sodium and water retention. These effects may be more prominent with minoxidil. To prevent angina and rebound hypertension, the prescriber may add a beta blocker and a diuretic to the regimen.

Nursing considerations

- Hydralazine may cause a lupus-like syndrome, which is reversible when hydralazine therapy is discontinued.
- Minoxidil can produce hair growth on the face, arms, back, and chest, which may cause patients, especially women, distress. Assure patients that hair growth reverses when minoxidil therapy stops.

Central adrenergic agonists

Central adrenergic agonists, such as clonidine and methyldopa, stimulate the alpha$_2$-adrenergic receptors in the central nervous system and decrease blood pressure by decreasing sympathetic activity. The drugs' effects include reduced heart rate and cardiac output and increased peripheral vasodilation. These drugs aren't usually recommended as first-line therapy, though methyldopa may be used as a first-line drug in pregnant women because of its safety profile.

Nursing considerations

- Central adrenergic agonists pose a higher risk of orthostatic hypotension, so tell patients to be careful when rising to a standing position.
- Explain that these drugs may cause depression, sedation, dry mouth, constipation, urine retention, and blurred vision.
- Tell patients not to stop taking these drugs on their own because an abrupt withdrawal can result in rebound hypertension.
- Teach patients using transdermal clonidine therapy to apply a new patch weekly to a clean, hairless area on the upper arm or chest; to change the patch site every week; to check for erythema or a rash; and to cover the patch with an adhesive to maintain good skin contact and promote absorption of the drug.

Controlling hypertension safely

To help patients tame hypertension, you must know which antihypertensives they are taking, how the drugs work, and which adverse effects they can cause. With this information, you can anticipate the plan of care and teach patients to manage their condition without adverse effects from the drugs or complications from uncontrolled hypertension. ★

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