Drugs Affecting Blood Pressure

KEY TERMS
ACE inhibitor
angiotensin II receptors
baroreceptor
cardiovascular (vasomotor) centre
essential hypertension
hypotension
peripheral resistance
renin
renin–angiotensin system
shock
stroke volume

LEARNING OBJECTIVES
Upon completion of this chapter, you should be able to:
1. Outline the normal physiological controls of blood pressure and explain how the various drugs used to treat hypertension or hypotension affect these controls.
2. Describe the therapeutic actions, indications, pharmacokinetics and contraindications associated with the angiotensin-converting inhibitors, angiotensin II receptor antagonists, calcium channel antagonists and vasodilators.
3. Describe the most common adverse reactions and important drug–drug interactions associated with the angiotensin-converting inhibitors, angiotensin II receptor antagonists, calcium channel antagonists and vasodilators.
4. Discuss the use of drugs that affect blood pressure across the lifespan.
5. Compare and contrast the key drugs captopril, losartan, diltiazem and nitroprusside with other agents in their class and with other agents used to affect blood pressure.
6. Outline the nursing considerations, including important teaching points, for patients receiving drugs used to treat hypertension.

ANTIHYPERTENSIVE AGENTS

Angiotensin-Converting Enzyme Inhibitors
- captopril
- enalapril
- lisinopril
- moexipril
- perindopril
- quinapril
- ramipril
- trandolapril

Angiotensin II Receptor Antagonists
- candesartan
- eprosartan
- irbesartan
- losartan
- olmesartan
- telmisartan
- valsartan

Calcium Channel Antagonists
- amlodipine
- diltiazem

Vasodilators
- felodipine
- isradipine
- nicardipine
- nifedipine
- nisoldipine
- verapamil
- diazoxide
- hydralazine
- minoxidil
- nitroprusside
The cardiovascular system is a closed system of blood vessels responsible for delivering oxygenated blood to the tissues and removing waste products from the tissues. The blood in this system flows from areas of higher pressure to areas of lower pressure. The area of highest pressure in the system is always the left ventricle during systole. The pressure in this area propels the blood out of the aorta and into the system of arteries. The lowest pressure is in the right atrium, which collects all of the reduced oxygenated blood from the body. The maintenance of this pressure system is controlled by specific areas of the brain and various hormones. If the pressure becomes too high, the person is said to be hypertensive. If the pressure becomes too low and blood cannot be delivered effectively, the person is said to be hypotensive. Helping the patient to maintain their blood pressure within normal limits is the goal when drug therapy is introduced.

### Blood Pressure Control

The pressure in the cardiovascular system is determined by three elements:

- **Heart rate**, the frequency of the heartbeat.
- **Stroke volume** or the amount of blood that is pumped out of the ventricle with each heartbeat (primarily determined by the volume of blood in the system).
- **Total peripheral resistance** (TPR) or the resistance of the muscular arteries to the blood being pumped through.

The small arterioles are thought to be the most important sites in determining peripheral resistance. These vessels with muscular walls and small diameter lumens are able to almost stop blood flow into capillary beds when they constrict, building up tremendous pressure in the arteries behind them as they prevent the blood from flowing through. The arterioles are very responsive to stimulation from the sympathetic nervous system; they constrict when the sympathetic system is stimulated (via α1 adrenoreceptors), increasing TPR and blood pressure. The body uses this responsiveness to regulate blood pressure on a minute-to-minute basis to ensure that there is enough pressure in the system to deliver sufficient blood to the brain.

### Baroreceptors

As the blood leaves the left ventricle through the aorta, it influences specialized cells in the arch of the aorta called baroreceptors (pressure receptors which respond to stretch). Similar cells are located in the carotid arteries, which deliver blood to the brain. If there is sufficient pressure in these vessels, the baroreceptors are stimulated, sending that information to the brain. If the pressure falls, the stimulation of the baroreceptors falls off. That information is also sent to the brain.

The sensory input from the baroreceptors is received in the medulla oblongata, an area called the cardiovascular (vasomotor) centre. If the pressure is high, the medulla stimulates vasodilation and a decrease in cardiac rate and output, causing the pressure in the system to drop. If the pressure is low, the medulla directly stimulates an increase in cardiac rate and output and vasoconstriction; this increases TPR and raises the blood pressure. The medulla mediates these effects through the autonomic nervous system (see Chapter 28).

The baroreceptor reflex continually functions to maintain blood pressure within a predetermined range of normal. For example, if you have been lying down flat and suddenly stand up, the blood pools in your lower limbs (an effect of gravity), so venous return falls. You may even feel lightheaded or dizzy for a short time. When you stand and the blood flow drops, the baroreceptors are not stretched. The medulla oblongata senses this drop and stimulates a rise in heart rate, cardiac output and a generalized vasoconstriction, which increases TPR, and all these factors increase blood pressure. These increases should raise pressure in the system, which restores blood flow to the brain and stimulates the baroreceptors. The stimulation of the baroreceptors leads to a decrease in stimulatory impulses from the medulla and the blood pressure falls back within normal limits (Figure 42.1).

### Renin–Angiotensin System

Another compensatory system is activated when the blood pressure within the kidneys falls. As the kidneys require a constant perfusion to function properly, they have a compensatory mechanism to help ensure that blood flow is maintained. This mechanism is called the renin–angiotensin system (it is sometimes referred to as the renin–angiotensin–aldosterone system).

Low blood pressure or poor oxygenation of the nephrons in the kidneys causes the release of renin from the juxtaglomerular cells, a group of cells that monitor blood pressure and blood flow into the glomerulus. Renin is released into the bloodstream and arrives in the liver to convert the compound angiotensinogen (produced in the liver) to angiotensin I. Angiotensin I travels in the bloodstream to the lungs, where the metabolic cells of the alveoli and bronchial mucosa use angiotensin-converting enzyme (ACE) to convert angiotensin I to angiotensin II. Angiotensin II reacts with specific angiotensin II receptor sites on blood vessels to cause intense vasoconstriction. This effect raises the TPR and raises the blood pressure, restoring blood flow to the kidneys and decreasing the release of renin.

Angiotensin II also stimulates the adrenal cortex to release aldosterone. Aldosterone acts on the nephrons to cause the retention of sodium and water. This effect increases blood volume, which should also contribute to increasing blood pressure. The sodium-rich blood stimulates the osmoreceptors in the hypothalamus to cause

the release of antidiuretic hormone, which in turn causes retention of water in the nephrons, further increasing the blood volume. This increase in blood volume increases the blood pressure, which should increase blood flow to the kidneys. This should lead to a decrease in the release of renin, thus causing the compensatory mechanisms to stop (Figure 42.2).

**Hypertension**

When a person’s blood pressure is above ‘normal’ limits (see Table 42.1) for a sustained period, a diagnosis of hypertension is made. It is estimated that at least 40% of adults in England and Wales have hypertension and many are unaware of it (BHF 2006).

Ninety percent of the people with hypertension have what is called **essential hypertension** or hypertension with no known cause. People with essential hypertension usually have elevated TPR due to atherosclerosis or persistent activation of the sympathetic nervous system. Their organs are perfused effectively and they usually display no symptoms. A few people develop secondary hypertension or high blood pressure resulting from a known cause, for instance, kidney problems or a tumour in the adrenal medulla, called a phaeochromocytoma; in this case, hypertension usually resolves after the tumour is removed.

The underlying danger of hypertension of any type is the prolonged force on the vessels of the vascular system. The muscles in the arterial system eventually thicken, leading to a loss of responsiveness in the system. The left ventricle thickens (hypertrophy) because the muscle must constantly work hard to expel blood at a greater force. The thickening of the heart muscle and the increased pressure that the muscle has to generate increases the workload of the heart and the risk of coronary artery disease (CAD). The hydrostatic force of the blood being forced through arteries damages the lining of endothelial cells, making these vessels susceptible to atherosclerosis and to narrowing of the lumen of the vessels (see Chapter 46). Tiny vessels can be damaged and destroyed, leading to loss of vision (if the vessels are in the retina), kidney function (if the vessels include the glomeruli in the nephrons) or cerebral function (if the vessels are small and fragile in the brain).

Untreated hypertension increases the risk for the following conditions: CAD and cardiac death, stroke, renal failure and loss of vision. As hypertension has no symptoms, it is difficult to diagnose and treat and it is often called the ‘silent killer’. Most of the drugs used to treat hypertension have adverse effects, many of which are seen as unacceptable by otherwise healthy people. Nurses face a difficult challenge trying to

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**Table 42.1 Categories Rating the Severity of Hypertension**

<table>
<thead>
<tr>
<th>Blood Pressure (BP) Classification</th>
<th>Systolic Blood Pressure (mmHg)</th>
<th>Diastolic Blood Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal BP</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Normal BP</td>
<td>&lt;130</td>
<td>&lt;85</td>
</tr>
<tr>
<td>High–normal BP</td>
<td>130–139</td>
<td>85–89</td>
</tr>
<tr>
<td>Grade 1 hypertension (mild)</td>
<td>140–159</td>
<td>90–99</td>
</tr>
<tr>
<td>Grade 2 hypertension (moderate)</td>
<td>160–179</td>
<td>100–109</td>
</tr>
<tr>
<td>Grade 3 hypertension (severe)</td>
<td>≥180</td>
<td>≥110</td>
</tr>
</tbody>
</table>

Source: Guidelines for management of hypertension: report of the fourth Working Party of the British Hypertension Society, 2004 BHS IV

convince patients to comply with their drug regimens when they experience adverse effects and do not see any positive effects of the drugs. Research into the cause of hypertension is ongoing and many theories have been proposed for the cause of the disorder. Factors that are known to increase blood pressure in some people include high levels of psychological stress, exposure to high-frequency noise, a high-salt diet, lack of rest and genetic predisposition (see Box 42.1).

**FIGURE 42.2** The renin-angiotensin system.

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**BOX 42.1** FOCUS ON THE EVIDENCE

**‘White Coat’ Hypertension**

The diagnosis of hypertension is accompanied by the impact of serious ramifications such as increased risk for numerous diseases and cardiovascular death, the potential need for significant lifestyle changes and the potential need for drug therapy, which may include many unpleasant adverse effects. Consequently, it is important that a patient be correctly diagnosed before being labelled hypertensive.

Researchers in the 1990s discovered that some patients were hypertensive only when they were in their doctor’s clinic having their blood pressure measured. This was correlated to a sympathetic stress reaction (which elevates systolic blood pressure) and a tendency to tighten the muscles (isometric exercise, which elevates diastolic blood pressure) while waiting to be seen and during the blood pressure measurement. The researchers labelled this phenomenon ‘white coat’ hypertension.

The British Heart Foundation (BHF) has put forward guidelines for the diagnosis and treatment of hypertension. A patient should have three consecutive blood pressure readings above normal, when taken by a clinician, over a period of 2 to 3 weeks. These guidelines point out the importance of using the correct technique when taking a patient’s blood pressure, especially because the results can have such a tremendous impact on a patient. It is good practice to periodically review the process for performing this routine task. For example, the nurse should

- Select a cuff that is the correct size for the patient’s arm (a cuff that is too small may give a high reading; a cuff that is too large may give a lower reading).
- Try to put the patient at ease, make them sit in a comfortable position and reassure them.
- Ensure that the arm that will be used is supported.
- Palpate the brachial artery before beginning.
- Identify the radial or radial artery and note pulse.
- Place the cuff over the brachial artery directly onto the patient’s skin instead of on top of clothing and palpating the radial pulse, inflate the cuff until the pulse can be no longer palpated. Continue to increase the pressure by 30 mmHg then deflate the cuff noting when the pulse could be felt again, this is an estimation of the systolic blood pressure.
- Deflate the cuff, place the stethoscope over the brachial artery and reinflate the cuff again, 30 mmHg above the point where the pulse reappeared.
- Listen carefully and record the first sound heard (systolic) and the absence of sound (the diastolic).

Nurses are the health care providers most likely to be taking and recording blood pressure, so it is important to always use the proper technique and to make accurate recordings.
Hypotension

If blood pressure becomes too low, the vital centres in the brain as well as the rest of the tissues of the body may not receive enough oxygenated blood to continue functioning. Hypotension can progress to shock, when waste products accumulate and cells begin to die from lack of oxygen. Hypotensive states can occur in the following situations:

- When the heart muscle is damaged and unable to pump effectively.
- With severe blood loss, when volume drops dramatically.
- When there is extreme stress and the body’s levels of adrenaline are depleted, leaving the body unable to respond to stimuli to raise blood pressure.

Antihypertensive Agents

As an underlying cause of hypertension is usually unknown, altering the body’s regulatory mechanisms is the best treatment currently available. Drugs used to treat hypertension work to alter the normal reflexes that control blood pressure. Treatment for essential hypertension does not cure the disease but is aimed at maintaining the blood pressure within normal (accepted) limits to prevent the damage that hypertension can cause. Not all patients respond the same way to antihypertensive drugs because different factors may contribute to each person’s hypertension. Patients may have complicating conditions such as diabetes or acute myocardial infarction (MI) that makes it unwise to use certain drugs (see Box 42.2).

Several different types of drugs, which affect different areas of blood pressure control, may need to be used in combination to actually maintain a patient’s blood pressure within normal limits. Trials of drugs and combinations of drugs are often needed to develop an individual regimen that is effective without producing adverse effects that are unacceptable to the patient. For current NICE guidelines on drug treatment for patients newly diagnosed with hypertension (see Figure 42.4).

Research is ongoing into the treatment of more specific hypertensions (e.g. pulmonary hypertension). The development of drugs that target specific blood vessel sites and chemicals could lead to a new approach to the treatment of essential hypertension in the future. For antihypertensive drug use across the life span (see Box 42.4).

Step1 Lifestyle modifications are instituted. These include weight reduction, smoking cessation, reduction in the use of alcohol and salt in the diet (all of these conditions have been shown to increase blood pressure) and an increase in physical exercise (which has been shown to decrease blood pressure and improve cardiovascular tone and reserve).

Step2 In hypertensive patients below the age of 55 years, the first choice of initial therapy should be an ACE inhibitor. If this is not tolerated by the patient, then an angiotensin receptor blocker (ARB) should be used.

Step3 In hypertensive patients of 55 years or older or black patients (African or Caribbean decent) of any age, first choice initial therapy should be a calcium channel blocker or a thiazide (thiazide-like) diuretic.

Step4 If the initial therapy was using a calcium channel blocker or a thiazide diuretic and a further drug is required, an ACE inhibitor (or an ARB, if an ACE inhibitor cannot be tolerated) should be used. If an ACE inhibitor was used as an initial therapy then a calcium channel blocker or thiazide diuretic can be added to the regimen.

Step5 If three drugs are required then a combination of ACE inhibitor (or an ARB, if an ACE inhibitor cannot be tolerated), a thiazide-like diuretic and a calcium channel blocker is recommended.

The current decision of not to recommend β-blockers for first-line therapy is based on research evidence that they perform less well as antihypertensives and that carry an increased risk of patients developing type 2 diabetes.
Angiotensin-Converting Enzyme Inhibitors

The ACE inhibitors block the conversion of angiotensin I to angiotensin II in the lungs (see Figure 42.2), as angiotensin II is a powerful vasoconstrictor, and these drugs stop the renin–angiotensin system, preventing vasoconstriction and aldosterone release. The ACE inhibitors may be used as a monotherapy for hypertension management or they may be combined with diuretics. ACE inhibitors that are used include the following agents:

- **Captopril** is indicated for use in hypertension and in treating congestive heart failure (CHF), diabetic nephropathy and left ventricular dysfunction after MI. It has been associated with a sometimes fatal pancytopenia (abnormal depression of all the cellular elements of the blood), a persistent dry cough and unpleasant gastrointestinal (GI) distress.

- **Enalapril** is used for the treatment of hypertension, CHF and left ventricular dysfunction; it has the advantage of parenteral use (if oral use is not feasible or rapid onset is desirable).

- **Lisinopril** is an oral drug used in treating hypertension and CHF and is used in stable patients within 24 hours after acute MI to improve the likelihood of survival.

- **Moexipril** is a less well-tolerated oral drug used in the treatment of hypertension; it is associated with many unpleasant GI and skin effects, cough and cardiac arrhythmias. Fatal MI and pancytopenia have sometimes been associated with this drug.

- **Perindopril** is an oral drug that is used alone or in combination with other antihypertensive agents to control blood pressure. It is associated with a sometimes fatal pancytopenia as well as a serious-to-fatal airway obstruction, mood and sleep disturbances.

- **Quinapril** is used orally for the treatment of hypertension and as an adjunct treatment of CHF; it is not associated with as many adverse effects as some of the other agents.

- **Ramipril** is used orally for the treatment of mild-to-moderate hypertension and as an adjunct treatment of CHF; it is not associated with as many adverse effects as some of the other agents.

- **Trandolapril** is used orally for the treatment of hypertension and for CHF after an acute MI and left ventricular dysfunction. It is fairly well tolerated.

**Therapeutic Actions and Indications**

The actions of ACE inhibitors include a decrease in blood pressure and in aldosterone secretion, with a resultant slight increase in serum potassium and a loss of serum sodium and fluid.

These drugs are indicated for the treatment of hypertension, alone or in combination with other drugs. They are also used in conjunction with digoxin and diuretics for the treatment of CHF and left ventricular dysfunction. Their therapeutic effect in these cases is thought to be related to a decrease in cardiac workload associated with the decrease in peripheral resistance and blood volume.

**Pharmacokinetics**

These drugs are well absorbed, widely distributed, metabolized in the liver and excreted in the urine and faeces. They are known to cross the placenta and have been associated with serious foetal abnormalities. These drugs should not be used during pregnancy. Several of these drugs have been detected in breast milk. As there is a potential for serious adverse effects in the neonate, another method of feeding the baby should be used during lactation or another antihypertensive should be chosen.

**Contraindications and Cautions**

ACE inhibitors reduce or abolish glomerular filtration and are likely to cause severe and progressive renal failure; therefore, ACE inhibitors are used with caution for patients with impaired renal function, as this could be exacerbated by the effects of this drug in decreasing renal blood flow, with pregnancy, because of the potential for adverse effects on the foetus and amniotic fluid production, and during lactation, because of potential decrease in milk production and effects on the neonate.

**Adverse Effects**

The adverse effects associated with the ACE inhibitors are related to the effects of vasodilation and alterations in blood flow. Such effects include reflex tachycardia, chest pain, angina, CHF and cardiac arrhythmias; GI irritation, ulcers, constipation and liver injury; renal insufficiency, renal failure and proteinuria; and rash, alopecia, dermatitis and photosensitivity. Many of these drugs cause an unrelenting cough thought to be related to the accumulation of bradykinin in the bronchial mucosa, where the ACE is inhibited. This may lead patients to discontinue the drug treatment. Some of these drugs have been associated with fatal pancytopenia and MI.

*Always consult a current copy of the BNF for further guidance*

**Clinically Important Drug–Drug Interactions**

The risk of hypersensitivity reactions increases if these drugs are taken with allopurinol.
CHAPTER 42 — Drugs Affecting Blood Pressure

Key Drug Summary: Ramipril

**Indications:** Severe hypertension resistant to other therapy, CHF, diabetic nephropathy, left ventricular dysfunction after an MI

**Actions:** Blocks ACE from converting angiotensin I to angiotensin II, leading to a decrease in blood pressure, a decrease in aldosterone production and a small increase in serum potassium levels along with sodium and fluid loss

**Pharmacokinetics:**

<table>
<thead>
<tr>
<th>Route</th>
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<th>Peak</th>
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<tbody>
<tr>
<td>Oral</td>
<td>15 min</td>
<td>30–90 min</td>
</tr>
</tbody>
</table>

$T_{1/2}$: 2 hours; excreted in urine

**Adverse effects:** Cough, tachycardia, MI, rash, pruritus, gastric irritation, aphthous ulcers, peptic ulcers, proteinuria and bone marrow suppression.

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Clinically Important Drug–Food Interactions

Absorption of oral ACE inhibitors decreases if they are taken with food. They should be taken on an empty stomach, 1 hour before or 2 hours after meals.

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Nursing Considerations for Patients Receiving ACE Inhibitors

**Assessment:** History and Examination

Screen for the following conditions, which could be cautions or contraindications to use of the drug: any known allergies to these drugs, impaired kidney function, which could be exacerbated by these drugs, pregnancy or lactation, because of the potential adverse effects on the foetus or neonate, salt/volume depletion, which could be exacerbated by these drugs, and CHF. Include screening for baseline status before beginning therapy and for any potential adverse effects. Assess the following: body temperature and weight; skin colour, lesions and temperature; pulse, blood pressure, baseline ECG and perfusion; respirations and adventitious breath sounds; bowel sounds and abdominal examination; and renal function tests, complete blood count with differential and serum electrolytes.

Establish if patient is currently taking other medications or herbal therapies, which may potentially interact with the ACE inhibitors.

**Nursing Diagnoses**

The patient receiving an ACE inhibitor may have the following nursing diagnoses related to drug therapy:

- Ineffective tissue perfusion (total body) related to changes in cardiac output.
- Impaired skin integrity related to dermatological effects.
- Acute pain related to GI distress and cough.
- Deficient knowledge regarding drug therapy.

**Implementation With Rationale**

- Encourage the patient to implement lifestyle changes, including weight loss, smoking cessation, decreased alcohol and salt in the diet and increased exercise, to increase the effectiveness of antihypertensive therapy.
- Administer on an empty stomach, 1 hour before or 2 hours after meals, to ensure proper absorption of drug.
- Inform the surgeon of the patient’s medication if the patient is to undergo surgery, to alert medical personnel that the blockage of compensatory angiotensin II could result in hypotension after surgery that would need to be reversed with volume expansion.
- Give parenteral forms only if an oral form is not feasible and transfer to an oral form as soon as possible, to avert an increased risk of adverse effects.
- Consult with the prescriber to reduce dosage in patients with renal failure, to account for their decreased production of renin and lower-than-normal levels of angiotensin II.
- Monitor the patient carefully in any situation that might lead to a drop in fluid volume (e.g. excessive sweating, vomiting, diarrhoea and dehydration), to detect and treat excessive hypotension that may occur.
- Provide comfort measures to help the patient tolerate drug effects. These include small, frequent meals; environmental controls; safety precautions and appropriate skin care as needed.
- Provide thorough patient education, including the name of the drug, dosage prescribed, measures to avoid adverse effects, warning signs of problems.
and the need for periodic monitoring and evaluation, to enhance patient knowledge about drug therapy and to promote compliance.

- Offer support and encouragement, to help the patient deal with the diagnosis and the drug regimen.

**Evaluation**

- Arrange for regular review of the patient to allow monitoring of the patient response to the drug (maintenance of blood pressure within normal limits and stable urea and electrolytes (U&E’s)).
- Monitor for adverse effects (hypotension, cardiac arrhythmias, renal dysfunction, skin reactions, dry cough, pancytopenia and CHF).
- Evaluate the effectiveness of the teaching plan (patient can name drug, dosage, adverse effects to watch for, specific measures to avoid adverse effects and the importance of continued follow-up).
- Monitor the effectiveness of comfort measures and compliance with the treatment regimen.

**Angiotensin II Receptor Antagonists**

The ARBs selectively bind to angiotensin II receptors in blood vessels to prevent vasoconstriction and in the adrenal cortex to prevent the release of aldosterone. These actions block the blood pressure-raising effects of the renin-angiotensin system and lower blood pressure. They are indicated to be used alone or in combination therapy for the treatment of hypertension and for the treatment of CHF in patients who are intolerant to ACE inhibitors. Recently, they were also found to slow the progression of renal disease in patients with hypertension and type 2 diabetes. This action is thought to be related to the effects of blocking angiotensin receptors in the vascular endothelium. Unlike ACE inhibitors, they do not inhibit the breakdown of bradykinin and other kinins, and are thus unlikely to cause the persistent dry cough, which complicates ACE inhibitor therapy. They are, therefore, a useful alternative for patients who have to discontinue an ACE inhibitor because of a persistent cough.

**Pharmacokinetics**

These agents are well absorbed and undergo metabolism in the liver by the cytochrome P450 system. They are excreted in faeces and in urine. Known to cross the placenta, the ARBs have been associated with serious foetal abnormalities and even stillbirth when given in the second or third trimester. Women of childbearing age should be advised to use barrier contraceptives to avoid pregnancy; if a pregnancy does occur, the ARB should be discontinued immediately. Candesartan, eprosartan, irbesartan, olmesartan and telmisartan should not be used during the second or third trimester of pregnancy because of associated foetal abnormalities and death. Losartan and valsartan should not be used at any time during pregnancy. It is not known whether the ARBs enter breast milk during lactation; however, because of the potential for serious adverse effects in the neonate, these drugs should not be used in lactating women.

**Contraindications and Cautions**

The ARBs are contraindicated in the presence of allergy to any of these drugs, during pregnancy, because of associated foetal death and severe abnormalities, and during lactation, because of potential adverse effects on the neonate. Caution should be used in the presence of hepatic or renal dysfunction, which could alter the metabolism.
and excretion of these drugs; and with hypovolaemia, because of the blocking of potentially life-saving compensatory mechanisms.

These drugs should not be prescribed to patients with renal disease as they reduce renal blood flow and may reduce GFR in patients with already compromised renal function.

**Adverse Effects**

The adverse effects most commonly associated with ARBs include the following: headache, dizziness, syncope and weakness, which could be associated with drops in blood pressure; hypotension; GI complaints including diarrhoea, abdominal pain, nausea, dry mouth and tooth pain; symptoms of upper respiratory tract infections; rash, dry skin and alopecia. Hyperkalaemia occurs occasionally; angioedema has also been reported with some angiotensin II receptor antagonists.

**Clinically Important Drug–Drug Interactions**

The risk of decreased serum levels and loss of effectiveness increases if the ARB is taken in combination with phenobarbital. If this combination is used, the patient should be closely monitored and dosage adjustments made.

*Always consult the most recent edition of the BNF.*

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**Key Drug Summary:** **Losartan**

*Indications:* Alone or as part of combination therapy for the treatment of hypertension; treatment of diabetic nephropathy with an elevated serum creatinine and proteinuria in patients with type 2 diabetes and hypertension

*Actions:* Selectively blocks the binding of angiotensin II to specific tissue receptors found in the vascular smooth muscle and adrenal glands and blocks the vasoconstriction and release of aldosterone associated with the renin–angiotensin system

*Pharmacokinetics:*

<table>
<thead>
<tr>
<th>Route</th>
<th>Onset</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral</td>
<td>Varies</td>
<td>1–3 h</td>
<td>24 h</td>
</tr>
</tbody>
</table>

$T_{1/2}$: 2 hours, then for the metabolites 6 to 9 hours; metabolized in the liver and excreted in urine and faeces

*Adverse effects:* Dizziness, headache, diarrhoea, abdominal pain, symptoms of upper respiratory tract infection, cough, back pain, fever, muscle weakness and hypotension

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**Calcium Channel Antagonists**

The calcium channel antagonists prevent the movement of calcium into the cardiac and smooth muscle cells when the cells are stimulated. This blocking of calcium interferes with the muscle cell’s ability to contract, leading to a loss of smooth muscle tone, vasodilation and a decrease in peripheral resistance. These effects decrease blood pressure, cardiac workload and myocardial oxygen consumption. Calcium channel antagonists are very effective in the treatment of angina (see Chapter 45) because they decrease the cardiac workload (see Critical Thinking Scenario 42-1).

Not all calcium channel antagonists are used to treat hypertension. Some are considered safe and effective in treating hypertension only if they are given as sustained-release or extended-release preparations. The calcium channel antagonists used in treating hypertension include the following:

- **Amlodipine** an oral drug that may be used alone or in combination with other agents to treat hypertension. It is also used for angina.
- **Diltiazem** is a sustained-release preparation recommended for the treatment of hypertension.
- **Felodipine** is indicated alone or in combination with other agents for the treatment of hypertension. This drug may be used as a prophylaxis for angina.
- **Isradipine** is not used for angina but is indicated alone or in combination with thiazide diuretics for the treatment of hypertension.
- **Nicardipine** is used alone or in combination with other agents to treat hypertension and as a prophylaxis for angina. It is also available in intravenous form for short-term use when oral administration is not feasible.
- **Nifedipine** is indicated for the treatment of hypertension, prophylaxis of angina and Raynaud’s phenomenon.
- **Nisoldipine** comes in extended-release tablets and is indicated for the treatment of hypertension as monotherapy or as part of combination therapy and for the prophylaxis of angina.
- **Verapamil** comes in extended-release tablets and is indicated for the treatment of essential hypertension; other preparations are used for angina and treating various arrhythmias. This drug should not be given by injection to patients on β-blockers due to the risk of hypotension and asystole.

**Therapeutic Actions and Indications**

Calcium channel antagonists inhibit the movement of calcium ions across the membranes of myocardial and arterial muscle cells, altering the action potential and...
blocking muscle cell contraction. This effect depresses myocardial contractility, slows cardiac impulse formation in the conductive tissues, reduces vascular tone as it relaxes and dilates arteries, causing a fall in blood pressure and a decrease in venous return.

**Pharmacokinetics**

These drugs are generally well absorbed, metabolized in the liver and excreted in the urine. These drugs cross the placenta and enter breast milk. Foetal toxicity has been reported in animal studies, and, although there are no well-defined studies about effects during human pregnancy, they should not be used during pregnancy unless the benefit to the mother clearly outweighs any potential risk to the foetus.

**Contraindications and Cautions**

These drugs are contraindicated in patients with heart block or sick sinus syndrome, which could be exacerbated by the conduction-slowing effects of these drugs, with renal or hepatic dysfunction, which could alter the metabolism and excretion of these drugs, and with pregnancy or lactation, because of the potential for adverse effects on the foetus or neonate.

**Adverse Effects**

The adverse effects associated with these drugs relate to their effects on cardiac output and on smooth muscle. Central nervous system (CNS) effects include dizziness, light-headedness, headache and fatigue. GI problems include nausea and hepatic injury related to direct toxic effects on hepatic cells. Cardiovascular effects include hypotension, bradycardia, peripheral oedema and heart block. Skin flushing and rash may also occur.

*For further information and guidance, always consult the most recent edition of the BNF.*

**Clinically Important Drug-Drug Interactions**

Drug–drug interactions vary with each of the calcium channel antagonists used to treat hypertension. A potentially serious effect to note is an increase in serum levels and toxicity of cyclosporine if taken with diltiazem.

**Diuretics**

Diuretics are drugs that increase the excretion of sodium and hence water from the kidney (see Chapter 50 and Figure 42.3). These drugs are often the first agents tried in mild hypertension; they affect blood sodium levels and blood volume.

**Key Drug Summary: Diltiazem**


*Actions:* Inhibits the movement of calcium ions across the membranes of cardiac and arterial muscle cells, depressing the impulse and leading to slowed conduction, decreased myocardial contractility and dilation of arterioles, which lowers blood pressure and decreases myocardial oxygen consumption

*Pharmacokinetics:*

<table>
<thead>
<tr>
<th>Route</th>
<th>Onset</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral</td>
<td>30–60 min</td>
<td>6–11 h</td>
<td>12 h</td>
</tr>
</tbody>
</table>

$T_{1/2}$: 5 to 7 hours; metabolized in the liver and excreted in urine

*Adverse effects:* Dizziness, light-headedness, headache, peripheral oedema, bradycardia, atroventricular block, flushing and nausea

**Nursing Considerations for Patients Receiving Angiotensin II Receptor Antagonists, Vasodilators and Calcium Channel Antagonists**

Assessment: History and Examination

Screen for the following conditions, which could be cautions or contraindications to use of the drug: any known allergies to these drugs, impaired kidney or liver function, which could be exacerbated by these drugs, pregnancy and lactation, because of the potential adverse effects on the foetus and neonate, and hypovolaemia, which could potentiate the blood pressure-lowering effects.

Include screening for baseline status before beginning therapy and for any potential adverse effects. Assess the following: body temperature and weight; skin colour, lesions and temperature; pulse, blood pressure, baseline...
electrocardiogram (ECG) and perfusion; respirations and adventitious breath sounds; bowel sounds and abdominal examination; and renal and liver function tests.

Establish if patient is currently taking other medications or herbal therapies, which may potentially interact with the angiotensin II receptor antagonists, vasodilators and calcium channel antagonists.

**Nursing Diagnoses**

The patient receiving an ARB, vasodilator antagonists or calcium channel may have the following nursing diagnoses related to drug therapy:

- Ineffective tissue perfusion (total body) related to changes in cardiac output.
- Impaired skin integrity related to dermatological effects.
- Acute pain related to GI distress, cough, skin effects and headache.
- Deficient knowledge regarding drug therapy.

**Implementation With Rationale**

- Encourage the patient to implement lifestyle changes, where appropriate, for example, weight loss, smoking cessation, decreased alcohol and salt in the diet and increased exercise, to increase the effectiveness of antihypertensive therapy.
- Administer with food, to decrease GI distress if needed.
- Alert the surgeon and mark the patient’s chart prominently if the patient is to undergo surgery, to notify medical personnel that the blockage of compensatory angiotensin II could result in hypotension after surgery that would need to be reversed with volume expansion.
- Ensure that the female patient is not pregnant before beginning therapy and suggest the use of barrier contraceptives while she is taking this drug, to avert potential foetal abnormalities and foetal death, which have been associated with these drugs.
- Find an alternative method of feeding the baby if the patient is nursing, to prevent the potentially dangerous blockade of the renin–angiotensin system in the neonate.
- Monitor the patient carefully in any situation that might lead to a drop in fluid volume (e.g. excessive sweating, vomiting, diarrhoea and dehydration), to detect and treat excessive hypotension that may occur.
- Provide comfort measures, to help the patient tolerate drug effects, including small, frequent meals; access to bathroom facilities; safety precautions if CNS effects occur; environmental controls; appropriate skin care as needed and analgesics as needed.
- Provide thorough patient teaching (education), including the name of the drug, dosage prescribed, measures to avoid adverse effects, warning signs of problems and the need for periodic monitoring and evaluation, to enhance patient knowledge about drug therapy and to promote concordance.
- Offer support and encouragement, to help the patient deal with the diagnosis and the drug regimen.

**Evaluation**

- Arrange for regular review of the patient to allow monitoring of the patient and their response to the drug (maintenance of blood pressure within normal limits).
- Monitor for adverse effects (hypotension, GI distress, skin reactions, cough, headache and dizziness). Evaluate effectiveness of the teaching plan (patient can name drug, dosage, adverse effects to watch for, measures to avoid adverse effects and the importance of continued follow-up).
- Monitor the effectiveness of comfort measures and compliance to the regimen.

**Other Antihypertensive Agents**

**Sympathetic Nervous System Antagonists**

Drugs that block the effects of the sympathetic nervous system (see Chapter 30) are useful in blocking many of the compensatory effects of the sympathetic nervous system (see Figure 42.3).

- **β-adrenergic antagonists** block vasoconstriction, decrease heart rate, decrease cardiac muscle contraction and tend to increase blood flow to the kidneys, leading to a decrease in the release of renin. These drugs have many adverse effects and are not recommended for all people. They are often used as monotherapy in step 2 treatment, and in some patients, they control blood pressure adequately.
- **α and β-adrenergic antagonists** are useful in conjunction with other agents and tend to be somewhat more powerful, blocking all of the receptors in the sympathetic system. Patients often complain of fatigue, loss of libido, inability to sleep and GI and genitourinary disturbances, and they may be unwilling to continue taking these drugs.
- **α-adrenergic antagonists** inhibit the postsynaptic α₁-adrenergic receptors, decreasing sympathetic tone in the vasculature and causing vasodilation, which leads to a lowering of blood pressure. However, these drugs also block presynaptic α₂-receptors, preventing the feedback
control of adrenaline release. The result is an increase in the reflex tachycardia that occurs when blood pressure decreases. These drugs are used to diagnose and manage episodes of phaeochromocytoma, but they have limited usefulness in essential hypertension because of the associated adverse effects.

- **α₁-adrenergic antagonists** are used to treat hypertension because of their ability to block the postsynaptic α₁-receptor sites. This decreases vascular tone and promotes vasodilation, leading to a fall in blood pressure. These drugs do not block the presynaptic α₁-receptor sites and therefore the reflex tachycardia that accompanies a fall in blood pressure does not occur.

- **α₂-adrenergic agonists** stimulate the α₂-receptors in the CNS and inhibit the cardiovascular centres, leading to a decrease in sympathetic outflow from the CNS and a resultant drop in blood pressure. These drugs are associated with many adverse CNS and GI effects as well as cardiac dysrhythmias.

## Vasodilators

If other drug therapies do not achieve the desired reduction in blood pressure, it is sometimes necessary to use a direct vasodilator. Vasodilators produce relaxation of the vascular smooth muscle, decreasing peripheral resistance and reducing blood pressure. They do not block the reflex tachycardia that occurs when blood pressure drops. Most of the vasodilators are reserved for use in severe hypertension or hypertensive emergencies. They are potent drugs, especially when used in combination with a β-blocker and a thiazide. The vasodilators that might be used to treat severe hypertension include the following:

- **Diazoxide** is used as an intravenous drug in hospitalized patients with severe hypertension. This drug also increases blood glucose levels by blocking insulin release, so it must be used with extreme caution with functional hypoglycaemia.

- **Hydralazine** is available for oral, intravenous and intramuscular use for the treatment of severe hypertension. It is thought to maintain or increase renal blood flow while relaxing smooth muscle.

- **Minoxidil** is an oral agent used only for the treatment of severe and unresponsive hypertension. It is associated with reflex tachycardia and increased renin release leading to volume increase (the oral drug is associated with changes in body hair growth and distribution, which led to a topical preparation for the treatment of baldness).

- **Sodium nitroprusside** is used intravenously for the treatment of hypertensive crisis and to maintain controlled hypotension during surgery; toxic levels cause cyanide toxicity.

## Pharmacokinetics

These drugs are rapidly absorbed and widely distributed. They are metabolized in the liver and primarily excreted in urine. They cross the placenta and enter breast milk. They should not be used during pregnancy unless the benefit to the mother clearly outweighs the potential risk to the foetus. Do not use these drugs during lactation. If they are needed by a nursing mother, then another method of feeding the baby should be selected.

## Contraindications and Cautions

The vasodilators are contraindicated in the presence of known allergy to the drug; with pregnancy and lactation, because of the potential for adverse effects on the foetus or neonate and with any condition that could be exacerbated by a sudden fall in blood pressure such as cerebral insufficiency. Caution should be used in patients with peripheral vascular disease, CAD, CHF or tachycardia, all of which could be exacerbated by the fall in blood pressure.
Adverse Effects

The adverse effects most frequently seen with these drugs are related to the changes in blood pressure. These include dizziness, anxiety and headache; reflex tachycardia, CHF, chest pain, oedema; skin rash and lesions (abnormal hair growth with minoxidil); and GI upset, nausea and vomiting. Cyanide toxicity (dyspnoea, headache, vomiting, dizziness, ataxia and loss of consciousness, imperceptible pulse, absent reflexes, dilated pupils, pink colour, distant heart sounds and shallow breathing) may occur with nitroprusside, which is metabolized to cyanide and which also suppresses iodine uptake and can cause hypothyroidism.

Clinically Important Drug–Drug Interactions

Each of these drugs works differently in the body, so before use, each drug should be checked for potential drug–drug interactions in the latest edition of the BNF.

Sympathetic Adrenergic Agonists

Sympathomimetic drugs bind to sympathetic adrenergic receptors to cause the following effects of a sympathetic stress response: increased blood pressure, increased blood volume

Key Drug Summary: Sodium Nitroprusside

**Indications:** Hypertensive crisis, maintenance of controlled hypotension during anaesthesia, acute or chronic CHF

**Actions:** Acts directly on vascular smooth muscle to cause vasodilation and drop of blood pressure, does not inhibit cardiovascular reflexes and tachycardia, renin release will occur

**Pharmacokinetics:**

<table>
<thead>
<tr>
<th>Route</th>
<th>Onset</th>
<th>Peak</th>
<th>Duration</th>
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<tbody>
<tr>
<td>Intravenous</td>
<td>1–2 min</td>
<td>rapid</td>
<td>1–10 min</td>
</tr>
</tbody>
</table>

$T_{1/2}$: 2 minutes; metabolized in the liver and excretion in urine

**Adverse effects:** Associated with over rapid reduction in blood pressure. Apprehension, headache, retrosternal pressure, palpitations, cyanide toxicity, diaphoresis, nausea, vomiting, abdominal pain and irritation at the injection site

**Abbreviations:**

A = ACE inhibitor

C = calcium-channel blocker

D = thiazide-type diuretic

Black patients are those of African or Caribbean descent, and not mixed-race, Asian or Chinese patients

Figure 42.4 Current guidelines on drug treatment for patients newly diagnosed with hypertension. Reproduced with permission from http://www.nice.org.uk

Reproduced with permission from National Institute for Health and Clinical Excellence
and increased strength of cardiac muscle contraction. These actions increase blood pressure and may restore balance to the cardiovascular system while the underlying cause of the shock (e.g. volume depletion and blood loss) is treated. The sympathomimetic drugs are discussed in Chapter 29 (see Box 42.3).

**Adverse Effects**

The most common adverse effects associated with this drug are related to the stimulation of \( \alpha \)-receptors and include piloerection, chills and rash; hypertension and bradycardia; dizziness, vision changes, vertigo and headache; and problems with urination.

**Clinically Important Drug-Drug Interactions**

There is a risk of increased effects and toxicity of cardiac glycosides, \( \beta \)-antagonists, \( \alpha \)-adrenergic agents and corticosteroids if they are taken with midodrine. Patients who are receiving any of these combinations should be monitored carefully for the need for a dosage adjustment.

**BOX 42.3** Sympathomimetic Drugs Used to Treat Severe Hypotension and Shock

**Sympathomimetic Drugs (see Chapter 29)**

- dobutamine
- dopamine
- ephedrine
- epinephrine
- isoproterenol
- metaraminol

**BOX 42.4** DRUG THERAPY ACROSS THE LIFESPAN

**Drugs Affecting Blood Pressure**

**CHILDREN**

National standards for determining normal levels of blood pressure in children are quite new. It has been determined that hypertension may start as a childhood disease and more screening studies are being done to establish normal values for each age group.

Children are thought to be more likely to have secondary hypertension caused by renal disease or congenital problems such as coarctation of the aorta.

Treatment of childhood hypertension should be done very cautiously because the long-term effects of the antihypertensive agents are not known. Lifestyle changes should be instituted before drug therapy if at all possible. Weight loss and increased activity may bring an elevated blood pressure back to normal in many children.

If drug therapy is used, a mild diuretic may be tried first, with monitoring of blood glucose and electrolyte levels on a regular basis. \( \beta \)-antagonists have been used with success in some children; adverse effects may limit their usefulness in others. The safety and efficacy of the ACE inhibitors and the ARBs have not been established in children. Calcium channel antagonists have been used to treat hypertension in children and may be a first consideration if drug therapy is needed. Careful follow-up of the growing child is essential to monitor for changes in blood pressure as well as adverse effects.

*When administering any drug to children, always consult the most recent edition of the BNF for Children.*

**ADULTS**

Adults receiving any of these drugs need to be instructed about adverse reactions that should be reported immediately. They need to be reminded of safety precautions that may be needed in hot weather or with conditions that cause fluid depletion (e.g. diarrhoea and vomiting). If they are taking any other drugs, the interacting effects of the various drugs should be evaluated. The importance of other measures to help lower blood pressure – weight loss, smoking cessation and increased activity – should be emphasized.

The safety for the use of these drugs during pregnancy has not been established. ACE inhibitors and ARBs should not be used during pregnancy and women of childbearing age should be advised to use barrier contraceptives to prevent pregnancy while taking these drugs. Calcium channel antagonists and vasodilators should not be used in pregnancy unless the benefit to the mother clearly outweighs the potential risk to the foetus. The drugs do enter breast milk and can have serious adverse effects on the baby. Caution should be used or another method of feeding the baby should be used if one of these drugs is needed during lactation. The main treatment for hypertension during pregnancy is the \( \beta \)-adrenergic blocking agent, labetalol; this drug also has arteriolar vasodilating action, which lowers TPR.

**OLDER ADULTS**

Older adults are frequently prescribed one of these drugs. They are more susceptible to the toxic effects of the drugs and are more likely to have underlying conditions that could interfere with drug metabolism and excretion. Renal or hepatic impairment can lead to accumulation of the drugs in the body. If the patient has renal or hepatic dysfunction, the dosage should be reduced and the patient monitored very closely.

The total drug regimen of the older patient should be co-ordinated, with careful attention to interactions among drugs and alternative therapies.

Older adults need to use special caution in any situation that could lead to a fall in blood pressure such as loss of fluids from diarrhoea or vomiting, lack of fluid intake or excessive heat with decreased sweating that comes with age. Dizziness, falls or syncope can occur if the blood pressure falls too far in these situations. The blood pressure should always be taken immediately before an antihypertensive is administered to an older adult to avoid excessive lowering of blood pressure.

Older patients should be especially cautioned about sustained-release antihypertensives that cannot be cut, crushed or chewed to avoid the potential for excessive dosing if these drugs are inappropriately cut.
Health care providers and patients may want to consult the following Internet sources:

http://cks.library.nhs.uk The National Health Service Clinical Knowledge Summaries provides evidence-based practical information on the common conditions observed in primary care.

http://www.bhf.org.uk Patient information, support groups, diet, exercise and research information on hypertension and other cardiovascular diseases.

http://www.bnf.org.uk The BNF provides UK health care professionals with authoritative and practical information on the selection and clinical use of medicines.

http://www.nhsdirect.nhs.uk The National Health Service Direct service provides patients with information and advice about health, illness and health services.


http://www.medscape.com Specific information on the care of the paediatric hypertensive patient; enter ‘paediatric hypertension’ in the search box.

Points to Remember

• The cardiovascular system is a closed system that depends on pressure differences to ensure the delivery of blood to the tissues and the return of that blood to the heart.

• Blood pressure is related to heart rate, stroke volume and the TPR against which the heart has to push the blood.

• Peripheral resistance is primarily controlled by constriction or relaxation of the arterioles. Constricted arterioles raise the pressure, whereas dilated arterioles lower the pressure.

• Control of blood pressure involves baroreceptor (pressure receptor) stimulation of the medulla to activate the sympathetic nervous system, which causes vasoconstriction and increased fluid retention when pressure is low in the aorta and carotid arteries and vasodilation and loss of fluid when pressure is too high.

• The kidneys activate the renin–angiotensin system when blood flow to the kidneys is decreased.

• Renin activates conversion of angiotensinogen to angiotensin I in the liver; angiotensin I is converted by ACE to angiotensin II in the lungs; angiotensin II then reacts with specific receptor sites on blood vessels to cause vasoconstriction to raise blood pressure and in the adrenal gland to cause release of aldosterone, which leads to retention of fluid and increased blood volume.

• Hypertension is a sustained state of higher-than-normal blood pressure that can lead to damage to blood vessels, increased risk of atherosclerosis and damage to small vessels in end organs. Hypertension often has no signs or symptoms; therefore, it may be referred to as the silent killer.

• Essential hypertension has no underlying cause and treatment can vary widely from individual to individual. Treatment approaches include lifestyle changes first, followed by careful addition and adjustment of various antihypertensive drugs.

• Drug treatment of hypertension is aimed at altering one or more of the normal reflexes that control blood pressure: diuretics decrease sodium levels and volume; ACE inhibitors prevent the conversion of angiotensin I to angiotensin II; ARBs prevent the body from responding to angiotensin II; calcium channel antagonists interfere with the ability of muscles to contract and lead to vasodilation; and vasodilators directly cause the relaxation of vascular smooth muscle and sympathetic nervous system drugs alter the sympathetic response and lead to vascular dilation and decreased pumping power of the heart.

• Hypotension is a state of lower-than-normal blood pressure that can result in decreased oxygenation of the tissues, cell death, tissue damage and even death. Hypotension is most often treated with sympathomimetic drugs, which stimulate the sympathetic receptor sites to cause vasoconstriction, fluid retention and return of normal pressure.
**CRITICAL THINKING SCENARIO 42-1**

**Initiating Antihypertensive Therapy**

**The Situation**

Clive a 65-year-old man was attending his general practitioner for a routine medical check-up. His examination was normal except for a blood pressure reading of 164/102 mmHg. He was also approximately 9 kg overweight. Urinalysis and blood results were all within normal limits. He was given a 1200-calorie-per-day diet to follow and was encouraged to reduce his salt and alcohol intake, start exercising and stop smoking. He was asked to return in 3 weeks for a follow-up appointment (step 1). Three weeks later, Clive returned with a 3.2 kg weight loss and an average blood pressure reading (of three readings) of 145/92 mm Hg. Discussion was held about starting Clive on a diuretic (step 2) in addition to the lifestyle changes that Clive was undertaking. He was reluctant to take a diuretic and after much discussion, was prescribed a calcium channel antagonist. Clive asked for a couple of more weeks to try to bring his blood pressure down with lifestyle changes before starting the drug.

**Critical Thinking**

- What nursing interventions should be done at this point? Consider the risk factors that Clive has for hypertension and the damage that hypertension can cause.
- What are the chances that Clive can bring his blood pressure within a normal range with lifestyle changes alone?
- What additional teaching points should be covered with Clive before a treatment decision is made?
- What effects could diuretic therapy have on Clive’s day-to-day life?

**Discussion**

Clive was asked to change many things in his life over the last 3 weeks. These changes themselves can be stressful and can increase a person’s blood pressure. Clive’s reluctance to take a diuretic is understandable for a busy man who might not want his day interrupted by many bathroom stops. This may have an impact on Clive’s work and home life. The decision to use a calcium channel antagonist may decrease some of the stress Clive was feeling about the diuretic.

Clive may benefit from trying for a couple more weeks to make lifestyle changes that will help bring his blood pressure into normal range. He will then feel that he has some control and input into the situation and if drug therapy is needed, he may be more willing to comply with the prescribed treatment. The diagnosis of hypertension may be delayed for these 2 weeks while Clive changes his lifestyle. Such a diagnosis should be made only after three consecutive blood pressure readings in the high range are recorded. Clive may be able to have his blood pressure checked at work in a comfortable environment, which will improve the accuracy of the reading.

Clive must receive regular follow-up and frequent blood pressure checks; it may be a good idea to allow him to take some control and continue lifestyle changes. If at the end of the 2 weeks no further progress has been made or Clive’s blood pressure has risen, drug therapy should be considered. Teaching should be aimed at helping Clive to incorporate the drug effects into his lifestyle, to improve his compliance and tolerance of the therapy.
Answers to the questions in this chapter may be found in the Answer Key in the back of the book.

Multiple Choice
Select the most appropriate response to the following:

1. The baroreceptors are the most important factor in minute-to-minute control of blood pressure. The baroreceptors
   a. are evenly distributed throughout the body to maintain pressure in the system.
   b. sense pressure and immediately send that information to the medulla in the brain.
   c. are directly connected to the sympathetic nervous system.
   d. are as sensitive to oxygen levels as to pressure changes.

2. Essential hypertension is the most commonly diagnosed form of high blood pressure. It is usually
   a. caused by a tumour in the adrenal gland.
   b. associated with no known cause.
   c. related to renal disease.
   d. caused by liver dysfunction.

3. Hypertension is associated with
   a. loss of vision.
   b. strokes.
   c. atherosclerosis.
   d. all of the above.

4. The stepped-care approach to the treatment of hypertension would include
   a. lifestyle modification, including exercise, diet and decreased smoking and alcohol intake.
   b. use of a diuretic, beta-blocker, or ACE inhibitor to supplement lifestyle changes.
   c. a combination of antihypertensive drug classes to achieve desired control.
   d. all of the above.

5. ACE inhibitors work on the renin–angiotensin system to prevent the conversion of angiotensin I to angiotensin II. Because this blocking occurs in the cells in the lung, which is usually the site of this conversion, use of ACE inhibitors often results in
   a. spontaneous pneumothorax.
   b. pneumonia.
   c. unrelenting cough.
   d. respiratory depression.

6. A client taking an ACE inhibitor is scheduled for surgery. The nurse should
   a. stop the drug.
   b. alert the surgeon and mark the client’s chart prominently, because the blockage of compensatory angiotensin II could result in hypotension after surgery that would need to be reversed with volume expansion.
   c. cancel the surgery and consult with the prescriber.
   d. monitor fluid levels and make sure the fluids are restricted before surgery.

7. A patient who is hypertensive becomes pregnant. The drug of choice for this patient would be
   a. an angiotensin II receptor blocker.
   b. an ACE inhibitor.
   c. a diuretic.
   d. a calcium channel blocker.

Extending Matching Questions
Select all that apply.

1. Pressure within the vascular system is determined by which of the following?
   a. Peripheral resistance
   b. Stroke volume
   c. Sodium load
   d. Heart rate
   e. Total intravascular volume
   f. Rate of erythropoietin release

2. The renin–angiotensin system is associated with which of the following?
   a. Intense vasoconstriction and blood pressure elevation
   b. Blood flow through the kidneys
   c. Production of surfactant in the lungs
   d. Release of aldosterone from the adrenal cortex
   e. Retention of sodium and water in the kidneys
   f. Liver production of fibrinogen

Matching
Match the following drugs with their appropriate class of antihypertensive agents. (Some classes may be used more than once.)

1. __________________ candesartan
2. __________________ quinapril
3. __________________ losartan
4. __________________ nitroprusside
5. __________________ lisinopril
6. __________________ valsartan
7. _______________ nicardipine
8. _______________ minoxidil
9. _______________ amlodipine

A. ACE inhibitor
B. Angiotensin II receptor antagonist
C. Calcium channel antagonist
D. Vasodilator
E. Ganglionic antagonist

True or False

Indicate whether the following statements are true (T) or false (F).

_____ 1. The cardiovascular system is an open system that depends on pressure differences to ensure the delivery of blood.

_____ 2. Blood pressure is related to heart rate, stroke volume and the TPR.

_____ 3. Constricted arterioles lower pressure; dilated arterioles raise pressure.

_____ 4. Control of blood pressure involves baroreceptor (pressure receptor) stimulation of the medulla to activate the parasympathetic nervous system.

_____ 5. The kidneys activate the renin–angiotensin system when blood flow to the kidneys is decreased.

_____ 6. Renin activates angiotensinogen to angiotensin I in the lung using ACE.

7. Hypertension is a sustained state of higher-than-normal blood pressure.

8. Essential hypertension has no underlying cause and treatment can vary widely.

9. Angiotensin II receptor antagonists prevent the body from responding to angiotensin II and blocking calcium channels.

10. Hypotension can result in decreased oxygenation of the tissues, cell death, tissue damage and even death.

Bibliography and References

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