Hypertension: The Silent Killer: Updated JNC-8 Guideline Recommendations

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EDUCATIONAL OBJECTIVES

Upon completion of this activity, pharmacists should be able to:

- Discuss the risk factors for developing hypertension
- Classify a patient's blood pressure according to their systolic and diastolic measurements
- Identify appropriate treatments options for patients
- Recommend alternative therapies for patients who are not successful on their first treatment option

INTRODUCTION

Hypertension (HTN), also known as high blood pressure (BP), affects millions of people. High blood pressure is defined as BP ≥140/90 millimeters of mercury (mmHg). Approximately 77.9 million American adults (1 in 3 people) and approximately 970 million people worldwide have high BP. It is estimated that by 2025, 1.56 billion adults will be living with HTN.[1-7] The overall occurrence is similar between both men and women, but differs with age.[1] For those younger than 45 years old, high blood pressure is more common in men than women. For those 65 years old or older, high blood pressure affects women more than men.[2,3] African Americans (47% in women, 43% in men) develop high blood pressure more often and at an earlier age, followed by Caucasians (31% in women, 33% in men) and Mexican Americans (29% in women, 30% in men). BP values increase with age, and HTN is very common with the elderly. The lifetime risk of developing HTN among those 55 years of age and older who currently have normal BP is 90%.[1,2] HTN costs the nation approximately $47.5 billion each year. This includes the cost of health care services, medications to treat high BP, and missed days of work.[4]

ETIOLOGY

For the majority of patients with high blood pressure, the cause is unknown. This is classified as primary or essential HTN. A small portion of patients have a specific cause of their high blood pressure, which is classified as secondary HTN.[1-3,5]

Over 90% of patients with high blood pressure have primary HTN.[1] Primary HTN cannot be cured, but it can be controlled with appropriate therapy (including lifestyle modifications and medications). Genetic factors may play an important role in the development of primary HTN. This form of high blood pressure tends to develop gradually over many years.[1,2,5]

Less than 10% of patients with high blood pressure have secondary HTN.[1] Secondary HTN is caused by an underlying medical condition or medication (see Table 1). Controlling the underlying medical condition or removing the causative medication(s) will result in a decrease of blood pressure thereby resolving secondary HTN. The most common cause of secondary HTN is associated with kidney impairment such as chronic kidney disease (CKD) or renovascular disease. This form of high blood pressure tends to appear suddenly and often causes higher blood pressure than primary HTN.[1,2,5]

Table 1. Causes of Secondary HTN

Disease States	Drugs and Other Products
• Kidney disease • Adrenal gland tumors • Thyroid disease • Congenital blood vessel disorders • Alcohol abuse or chronic alcohol use • Obstructive sleep apnea	• **Nonsteroidal anti-inflammatory drugs** (NSAIDs) (examples: ibuprofen, naproxen) • **Birth control pills** • **Decongestants** (pseudoephedrine, phenylephrine) • **Cocaine** • **Amphetamines** (eg, amphetamine, methylphenidate, lisdexamfetamine) • **Corticosteroids** (eg, prednisolone, methylprednisolone, dexamethasone, hydrocortisone) • **Food** (foods high in sodium such as canned or processed foods, salad dressings, cheese, chips, sweets) • **Alcohol**

PATHOPHYSIOLOGY

Multiple factors that control blood pressure contribute to developing primary HTN. The two primary factors include problems in either hormonal [natriuretic hormone, renin-angiotensin-aldosterone system (RAAS)] mechanisms or disturbances in electrolytes (sodium, chloride, potassium). Natriuretic hormone causes an increase in sodium concentrations in cells leading to an increase in blood pressure. The RAAS regulates sodium, potassium, and blood volume, which will ultimately regulate blood pressure in the arteries (blood vessels that carry blood away from the heart). Two hormones involved in the RAAS system include angiotensin II and aldosterone. Angiotensin II causes narrowing of the blood vessels, increases release of chemicals that elevate blood pressure, and increases aldosterone production. The constriction of blood vessels increases blood pressure (less space, same amount of blood), which also places pressure on the heart. Aldosterone causes sodium and water to stay

in the blood. As a result, there is a greater volume of blood, which will increase pressure on the heart and elevate blood pressure.[1,5]

Arterial BP is the pressure in the blood vessel, specifically the arterial wall. It is measured in millimeters of mercury (mmHg). The two arterial blood pressure values are systolic blood pressure (SBP) and diastolic blood pressure (DBP). The SBP is the peak (highest) value that is achieved when the heart contracts. DBP is achieved while the heart is at rest (lowest pressure) and the heart chambers are filling with blood.[1,5]

SYMPTOMS

HTN is known as the "silent killer" because it typically has no warning signs or symptoms, and many people do not know they have it.[2] Even when blood pressure levels are dangerously high, most people do not have any signs or symptoms. A small amount of people may experience symptoms such as dull headaches, vomiting, dizzy spells, and more frequent nosebleeds. These symptoms usually do not occur until blood pressure levels have reached a severe or life-threatening stage. The only way to know for certain if a person has HTN is to have a physician or other health care professional measure blood pressure.[2,3]

RISK FACTORS

Various factors increase a person's risk for developing HTN. Risk factors include health conditions, lifestyle, and family history (see Table 2). Some risk factors, such as family history, cannot be controlled. However, there are risk factors such as physical activity and diet that can be controlled to decrease a patient's likelihood of developing HTN.[1-3]

Table 2. Risk Factors for Developing HTN

Risk Factors that can be controlled	Risk Factors that cannot be controlled
• Overweight or obese • Sedentary lifestyle (lack of physical activity) • Tobacco usage • Unhealthy diet (high in sodium) • Excessive alcohol usage • Stress • Sleep apnea • Diabetes	• Age • Race • Family History

CONSEQUENCES OF HTN

The World Health Organization rates HTN as one of the most important causes of premature death worldwide. It is estimated to cause 7.5 million deaths, about 12.8% of the total of all deaths.[7] The excessive high pressure on artery walls caused by HTN can damage blood vessels along with organ function. This increases the risk for developing several dangerous health conditions including heart attack, stroke, chronic heart failure (CHF), and kidney disease.[1-3] Approximately 70% of people who have their first heart attack already have HTN. About 80% of people who have their first stroke have high blood pressure.[2]

High blood pressure causes hardening and thickening of arteries (atherosclerosis), which decreases blood flow and oxygen to the heart. This can also cause chest pain, heart failure, or even a heart attack. Heart failure occurs when the heart cannot pump enough blood and oxygen to meet the body's needs. Heart attacks occur due to the blood supply to the heart being blocked; therefore, the heart does not get the necessary oxygen it needs to survive.[1-3]

High blood pressure can also have damaging effects to the brain, specifically it can cause an aneurysm or stoke. Increased blood pressure may cause blood vessels to weaken and bulge, leading to the formation of an aneurysm. If an aneurysm ruptures, it can have serious life-threatening consequences. Like in the heart, high blood pressure can cause atherosclerosis in blood vessels that supply the brain with blood and oxygen. When this occurs it can cause a stroke. Strokes often led to problems in speech, movement, and other simple activities. Like heart attacks, strokes can be life threatening.[1-3]

CLASSIFICATION

The classification of blood pressure in adults (18 years and older) is based on the average of two or more properly measured blood pressure readings from two or more clinical visits (see Table 3). If the systolic blood pressure and diastolic blood pressure values fall into different categories, the overall classification is determined based on the higher of the two blood pressures. Blood pressure is classified into one of four categories: normal, prehypertension, stage 1 HTN and stage 2 HTN. Prehypertension is not considered a disease, but identifies those who are likely to progress to stage 1 or stage 2 HTN in the future.[1,5]

FIRST-LINE TREATMENT

Treatment for hypertensive patients includes both nonpharmacologic (lifestyle changes) and pharmacologic (medication) therapy to lower blood pressure and prevent cardiovascular (heart) events such as a heart attack. Implementation of lifestyle interventions should be used throughout the management of all patients with high blood pressure.[1,2] According to the updated 2014 Eighth Joint National Committee (JNC-8) guidelines on HTN, evidence from clinical trials indicate that antihypertensive medications (blood pressure medication) should be initiated in patients less than 60 years old if the systolic blood pressure is persistently $\geq$140 mmHg and the diastolic blood pressure is persistently $\geq$90 mmHg despite nonpharmacologic therapy. If a patient is 60 years old and older, antihypertensive therapy should be initiated if the systolic blood pressure is $\geq$150 mmHg and the diastolic blood pressure is $\geq$90 mmHg.[8,9]

All hypertensive patients should be counseled on the appropriate lifestyle modifications needed to help lower blood pressure. Evidence has shown that societies in which the average sodium intake is high (more than 2.3 grams per day) have a greater amount of patients diagnosed with HTN. High amounts

Table 3. Classification of Blood Pressure in Adults (age ≥18 years)			
Classification	**Systolic Blood Pressure (mmHg)**		**Diastolic Blood Pressure (mmHg)**
Normal	<120	AND	<80
Prehypertension	120-139	OR	80-89
Stage I HTN	140-159	OR	90-99
Stage 2 HTN	≥160	OR	≥100

of sodium intake lead to increased volume in the bloodstream. This places increased pressure on the heart to pump blood throughout the body. As a result, blood pressure can become elevated.[1,2,9] The American Heart Association (AHA) recommends limiting sodium intake to less than 1500 mg per day (1.5 grams). Since most dietary salt is found in packaged and processed foods, limiting their intake and finding healthier alternatives is beneficial for blood pressure reduction. Restrictive diets, like the Dietary Approaches to Stop Hypertension (DASH) diet, have been found to help lower blood pressure. The DASH diet emphasizes a food plan high in fruits, vegetables, whole grains, poultry, and fish while limiting sweets, sugar-sweetened beverages, and red meat. Furthermore, the DASH diet recommends that men restrict alcohol intake to two or fewer drinks a day and women to one or less. This recommendation is based on evidence indicating that patients who excessively drink alcohol have had a higher incidence of high blood pressure compared to those that drink alcohol in moderation. In addition to dietary modifications, exercise is recommended.[10] Both aerobic exercise and resistance training have been shown to lower blood pressure and improve overall cardiovascular health. Examples of aerobic exercise include walking, jogging, swimming, and biking. The AHA recommends an average of 40 minutes of moderate to vigorous intensity aerobic exercise three to four times a week to help lower blood pressure.[11]

If nonpharmacologic treatment is ineffective in managing high blood pressure, pharmacological therapy is initiated. Initial pharmacological therapy for HTN includes thiazide diuretics, long-acting calcium channel blockers (CCB), angiotensin-converting enzyme (ACE) inhibitors, and angiotensin II receptor blockers (ARBs). Blood pressure goals for HTN are specific for a patient's age and comorbid diseases (see Table 4). It is important to note that these goals are updated from those previously recommended in past guidelines (Seventh Joint National Committee Guidelines).[1,2,8,9]

Past HTN guidelines (JNC-7 guidelines) recommended five medication classes for HTN treatment in the general population with thiazide-type diuretics being first line therapy. The five medication classes recommended for HTN were thiazide-type diuretics, calcium channel blockers, angiotensin-covering enzyme inhibitors, angiotensin receptor blockers, and beta-blockers. However, the updated JNC-8 guidelines do not include beta-blockers as initial treatment and treatment is addressed separately based on ethnicity.[1,2,8]

Table 4. Blood Pressure Goals	
Population	**Blood Pressure Goal (Systolic/Diastolic)**
< 60 years old	<140/90 mmHg
> 60 years old	<150/90 mmHg
Chronic Kidney Disease (CKD)	<140/90 mmHg
Diabetes	<140/90 mmHg

The JNC-8 guidelines recommend that the general nonblack population's (including those with diabetes) initial pharmacologic therapy should include a thiazide-type diuretic, calcium channel blocker, angiotensin-converting enzyme inhibitor, or angiotensin receptor blocker. In contrast, the general black population's (including those with diabetes) initial therapy should include a thiazide-type diuretic or calcium channel blocker. The black population's recommendations are different form the nonblack population based on evidence that black patients have a smaller reduction in blood pressure when given ACEI or ARB medication therapy.[8]

The objectives of initiating drug therapy are to reach and maintain the goal blood pressure. If a patient's goal blood pressure is not reached after a month of therapy, the initial drug's dose can be increased or a second drug can be added from one of the classes recommended. Combination therapy (with two different classes of medications) can be used as initial therapy if the SBP ≥160 mmHg and/or the DBP is >100 mmHg or the SBP is >20 mmHg above goal and/or the DBP is >10 mmHg above goal. If two medications are not sufficient to meet the blood pressure goal, a third medication can be added. Alternative agents can be utilized for HTN if the blood pressure goal is not achieved with first-line agents (thiazides, CCB, ACEI, ARBs).[8]

Thiazide and thiazide-like diuretics have been the mainstay of HTN management for a longer period than any other antihypertensive agent. Their continued use is based on consistent evidence in their ability to reduce the risk of heart disease, stroke, heart attack, and death.[1,8,9,12] Thiazide-type diuretics used for HTN include metolazone, chlorthalidone, hydrochlorothiazide, and indapamide. Out of these four, the two most commonly used thiazides for HTN are hydrochlorothiazide and chlorthalidone.

However, metolazone may be effective in patients with poor renal (kidney) function when other thiazide medications are not effective. Thiazide diuretics inhibit sodium and chloride absorption in the kidney. As a result of water and electrolyte (sodium and chloride) loss, there is less volume in the blood as well as lowered pressure on the heart. Overtime, the diuretics cause blood vessels to dilate (widen blood vessels) which contributes to long-term blood pressure reduction. Chlorthalidone is more potent (~ 2 times stronger acting) and longer acting (24-72 hours verses 6-12 hours) compared to hydrochlorothiazide.[1,2,8,9,12] Controversy has developed on whether chlorthalidone is superior to hydrochlorothiazide. While there are no studies that compare patients taking either chlorthalidone or hydrochlorothiazide, indirect comparisons have shown that chlorthalidone is superior in lowering blood pressure and reducing cardiovascular events.[8,14] Currently, the JNC-8 guidelines do not distinguish whether one is preferred over the other. Guidelines recommend hydrochlorothiazide to be initiated at 12.5 to 25 mg per day with a target dose of 25-50 mg. Due to the shorter duration of action, a higher dose of hydrochlorothiazide can be separated into two doses per day. Chlorthalidone should be initiated at 12.5 mg with a target dose between 12.5-25 mg.[8] Common side effects include increased thirst, increased urination, dizziness, and low blood pressure. The increased urination is common during initiation of therapy, but decreases overtime. Patients should be counseled to take the medication in the morning to prevent nighttime urination. Serious side effects of thiazide therapy include electrolyte imbalance (low potassium, increased uric acid, low magnesium, increased glucose). The risk of having the electrolyte imbalance is not significant with the lower doses used to treatment HTN (12.5-25 mg per day).[1,2,8,9,12-15]

Table 5. Thiaazide Diuretics

Generic (Brand) Name	Dose (mg/day)
Chlorthalidone (Thalitone®)	12.5-25 mg
Hydrochlorothiazide (Microzide®)	12.5-50 mg
Indapamide	1.25-2.5 mg
Metolazone (Zaroxolyn®)	2.5-5 mg

Calcium channel blockers (CCBs) used for the treatment of HTN include amlodipine, felodipine, isradipine, nicardipine sustained-release, nifedipine long-acting, nisoldipine.[1,2,8,12,13,15] Normally, calcium enters the muscle cells in the blood vessels. Calcium channel blockers bind to calcium channels found in the blood vessels. As a result of the calcium channel blockade, CCBs causes vasodilation (widening) of the blood vessels. This places less pressure on the heart and lowers blood pressure. Dosing for HTN is listed below (see Table 6). Common side effects include headache, dizziness, flushing, and swelling in the legs and arms. Serious side effects include chest pain that has occurred when CCBs are initiated.[1,2,12,13]

Table 6. Calcium Channel Blocker Dosing

Generic (Brand) Name	Dose (mg/day)
Amlodipine (Norvasc®)	2.5-10 mg
Felodipine (Plendil®)	2.5-10 mg
Isradipine sustained-release (DynaCirc SR®)	5-10 mg
Nicardipine sustained-release (Cardene SR®)	60-120 mg
Nifedipine long-acting (Adalact CC®, Procardia XL®)	30-90 mg
Nisoldipine (Sular®)	17-34 mg

Newer agents utilized for HTN treatment include angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs). ACE inhibitors used for HTN include benazepril, captopril, enalapril, fosinopril, perindopril, lisinopril, moexipril, quinapril, ramipril, and trandolapril.[1,2,8,9,15] ACE inhibitors prevent the formation of angiotensin II by blocking the enzyme that converts angiotensin I into angiotensin II. Angiotensin II is a hormone in the body that causes constriction (narrowing) of the blood vessels. Furthermore, angiotensin II stimulates the release of another hormone called aldosterone, which holds both sodium and water in the body. Both narrowing of the blood vessels and increased volume (due to sodium and water) elevates blood pressure. By inhibiting the formation of angiotensin II, blood pressure is lowered. ACE inhibitors have been shown to prevent death in patients with heart failure after a heart attack and in all patients at high risk for heart complications. They have also been shown to reduce proteinuria (excess protein in urine) in diabetic patients. Individual dosing for HTN is listed below (see Table 7).[1,8,9,12,15] Patient's prescribed ACE inhibitors can develop a cough, which usually begins within the first two weeks of therapy. If this occurs, therapy should be discontinued. After the agent is stopped, the cough resolves within a week. Common side effects include low blood pressure, headache, and reduction in glomerular filtration rate (test used to check how kidneys are working). Serious side effects of ACE inhibitors include a risk of angioedema (swelling that occurs under the skin that is similar to hives) and high potassium levels. If a patient has a prior history of angioedema with one ACEI, ACE inhibitor therapy with the same ACE inhibitor or different ACE inhibitor should not be used for HTN. ACE inhibitor therapy should not be used in pregnant women since this agent has an increased risk of fetal complication.[12,13]

Angiotensin II receptor blockers (ARBs) are medications that act similarly to ACE inhibitors. ARBs used for HTN include azilsartan, candesartan, eprosartan, irbesartan, losartan, olmesartan, telmisartan, and valsartan.[1,8] Like ACE inhibitors, these agents prevent angiotensin's action on blood pressure. However, instead of preventing angiotensin II's formation, it blocks angiotensin II's binding to the angiotensin II receptor. For angiotensin to work, it

Table 7. ACE Inhibitor Dosing	
Generic (Brand) Name	**Dose (mg/day)**
Benazepril (Lotensin®)	20-80 mg
Captopril (Capoten®)	25-50 mg
Enalapril (Vasotec®)	2.5-40 mg
Fosinopril (Monopril®)	10-80 mg
Lisinopril (Prinivil®, Zestril®)	10-40 mg
Moexipril (Univasc®)	7.5-30 mg
Perindopril (Aceon®)	4-16 mg
Quinapril (Accupril®)	10-80 mg
Ramipril (Altace®)	2.5-20 mg
Trandolapril (Mavik®)	1-8 mg

Table 8. ARBs Dosing	
Generic (Brand) Name	**Dose (mg/day)**
Azilsartan (Edarbi®)	40-80 mg
Candesartan (Atacand®)	8-32 mg
Eprosartan (Teveten®)	400-800 mg
Irbesartan (Avapro®)	150-300 mg
Losartan (Cozaar®)	25-100 mg
Olmesartan (Benicar®)	20-40mg
Telmisartan (Micardis®)	20-80mg
Valsartan (Diovan®)	80-320 mg

must bind to the receptor. Since ARBs prevent angiotensin II from binding to the receptor, angiotensin II cannot exude its blood pressure increasing effects. Because ARBs and ACE inhibitors have similar mechanisms, these two medications should not be used together for HTN treatment. Individual dosing for ARBs are listed in below table (see Table 8).[1,8,9,12,15] In comparison to ACE inhibitors, ARBs have been shown to be equally effective as ACE inhibitors, but with less side effects. Common side effects include cough, low blood pressure, headache, and reduction in glomerular filtration rate. Serious side effects include the risk of angioedema and high potassium levels. The risk of both cough and angioedema is significantly lower with ARB therapy as compared to ACE inhibitors. Like ACE inhibitors, these agents should not be used during pregnancy because of the fetal risks.[12,13,15]

SECOND-LINE TREATMENT

Other medications utilized for the treatment of HTN include beta-blockers, aldosterone antagonists, alpha-blockers, and direct renin inhibitors. Beta-blockers used for HTN treatment include atenolol, bisoprolol, metoprolol tartrate, metoprolol succinate extended release, carvedilol, labetalol (see Table 9). Beta-blockers stop the beta-receptors on the heart from being activated. Normally, stimulation of these receptors will cause the heart rate to increase and put pressure on the heart. By blocking these receptors, there is less stress on the heart and blood pressure is reduced.[1,9,12,13,15] Beta-blockers (BB) were not indicated for initial treatment of HTN. The reason beta-blockers are second-line therapy is based on studies showing that beta-blockers had a higher incidence of heart attack or stroke when used for HTN in patients without a specific indication for use (example: recent heart attack or stroke). According to the JNC-8 guidelines, beta-blockers should be initiated if first-line therapy is not effective in lowering blood pressure. However, beta-blockers should be used as primary therapy if a patient has a compelling indication (recent stroke or heart attack).[8,9,15,16]

Aldosterone antagonists are another second-line treatment for HTN. Aldosterone antagonists including spironolactone and eplerenone block the actions of aldosterone. Normally, aldosterone increases the absorption of salt and water in the kidney, which increases volume in the blood stream and elevates blood pressure. By blocking aldosterone, blood pressure is reduced due to lowered pressure.[2,13]

Alternative agents not mentioned in the guidelines include alpha-1 blockers, central alpha-2 blockers, and direct renin inhibitors. The first direct renin inhibitor aliskerin became available in 2007. This medication works similarly to both ARBs and ACE inhibitors. Aliskerin inhibits renin, an enzyme that converts a precursor of angiotensin (angiotensinogen) into angiotensin I (which will turn into angiotensin II in the body). As a result, it blocks the formation of angiotensin II. Like ACE inhibitors and ARBs, this agent should not be used in pregnancy.[1,2,13] Alpha-2 agonists including clonidine, guanfacine, and methyldopa work centrally in the brain to block neurotransmitters (chemicals that communicate to the body) from increasing the heart rate and blood pressure. However, the agent's side effects (dizziness, drowsiness, fatigue, headache) limit their use.[1,12,13] Alpha-1 antagonists (doxazosin, prazosin, terazosin) cause small blood vessels to remain open, which lowers blood pressure. A trial comparing doxazosin to other antihypertensive medications found that doxazosin had a higher incidence of heart failure and cardiovascular events. As a result, alpha-1 blockers should not be used as first-line therapy.[1,9,15,17] Lastly, vasodilators including minoxidil and hydralazine work by widening the blood vessels to reduce blood pressure. These agents should be used as a last-line option to treat HTN.[2,13]

Table 9. Beta-Blockers	
Generic Name	**Brand Name**
Atenolol	Tenormin®
Bisoprolol	Zebeta®
Betaxolol	Kerolone®
Metoprolol tartrate	Lopressor®
Metoprolol succinate extended release	Toprol XL®
Nadolol	Corgard®
Propranolol	Inderal LA®, Inderal XL®
Timolol	Blocadren®
Acebutolol	Sectral®
Penbutolol	Auxilium®
Pindolol	
Carvedilol	Coreg®
Labetalol	Normodyne®
Nebivolol	Bystolic®

Table 10. Other Secondary HTN Treatment	
Generic Name	**Brand Name**
Spironolactone	Aldactone®
Eplerenone	Inspra®
Aliskerin	Tekturna®
Clonidine	Catapres®
Guanfacine	Tenex®
Methyldopa	Aldomet®
Doxazosin	Cardura®
Prazosin	Minipress®
Terazosin	Hytrin®
Minoxidil	Loniten®
Hydralazine	Apresoline®

CONCLUSION

HTN is one of the most prevalent disease states that occur in approximately one in three adults. HTN is caused by malfunctions in both hormonal regulation such as angiotensin and aldosterone as well as disturbances in electrolytes such as sodium and water. Ways to prevent the development of high blood pressure include eating a well-balanced diet, exercising, and maintaining a healthy weight. If a patient is diagnosed with HTN, both pharmacologic and nonpharmacologic therapies should be utilized for treatment.[1,2,8,12] First-line pharmacologic treatment for HTN includes thiazide diuretics, calcium channel blockers, angiotensin-converting enzyme, and angiotensin II receptor blockers.[8,9,12] It is important to control blood pressure to prevent complications such as heart attack, stroke, impaired kidney function, and even death.[1,2]

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