The second is pharmacologic therapy.

Lifestyle Change

Last year, the AHA published an update to its dietary guidelines for preventing and treating hypertension. These guidelines form the nucleus of lifestyle change to lower blood pressure and other risk factors for heart disease.

Lifestyle modification can have a significant impact on blood pressure reduction, whether practiced alone or in combination with medications. The goal of weight loss is achievement of a normal body mass index (<25 kg/m²). For every 10 kg in weight loss, systolic blood pressure can fall 5 mm Hg to 20 mm Hg.

The AHA guidelines recommend stringent sodium restriction: a limit of 1.5 g/day of sodium (65 mmol). The previous recommendation from the JNC was 2.4 g/day of sodium (100 mmol). Limiting dietary sodium to 100 mmol/day can reduce systolic blood pressure by 2 mm Hg to 8 mm Hg. The 65 mmol/day limit would further reduce blood pressure.

A primary tool for sodium reduction is the DASH diet. The acronym stands for Dietary Approaches to Stop Hypertension, and the eating plan is available at www.nhlbi.nih.gov/health/public/heart/hbp/dash/new_dash.pdf. The DASH diet focuses on fruits, vegetables, low-fat dairy products and reduced saturated fat and cholesterol. Following the DASH diet can reduce systolic blood pressure by 8 mm Hg to 14 mm Hg.

Moderation of alcohol consumption is also recommended as an important strategy for blood pressure reduction. Men should limit alcohol consumption to two drinks or less per day (1 ounce of alcohol, 24 ounces beer, 10 ounces wine or 3 ounces 80-proof whiskey). Women should limit alcohol ingestion to one drink or less daily (0.5 ounces alcohol, 12 ounces beer, 5 ounces wine or 1.5 ounces 80-proof whiskey). These alcohol recommendations are the same as those included in JNC guidelines. Limiting alcohol consumption can reduce systolic blood pressure by 2 mm Hg to 4 mm Hg.

The AHA guidelines — along with numerous other scientific sources — recommend physical activity of at least 30 minutes daily on most days of the week. Such regular exercise may reduce blood pressure by 4 mm Hg to 9 mm Hg.

Pharmacologic Therapy

To treat hypertension in patients with CKD, the NKF recommends the initiation of preferred drugs. These include loop or thiazide diuretics, angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs). Calcium channel blockers and beta blockers may be prescribed as second-line therapy, and the NKF states that alpha blockers may be added as necessary until blood pressure control is achieved.

Thiazide Diuretics

Thiazide diuretics inhibit sodium and chloride reabsorption in the distal convoluted tubule. This results in a decrease in extracellular fluid volume and a decrease in cardiac output, which results in a decrease in peripheral vascular resistance and a lowering of blood pressure.

Thiazides are appropriate for patients with mild to moderate renal insufficiency when GFR is 30 mL/min/1.73 m² or greater. Commonly prescribed thiazides include hydrochlorothiazide (Hydrodiuril), chlorothiazide (Diuril), metolazone (Zaroxolyn), indapamide (Lozol).

Studies show that a reflexive increase in plasma renin may occur when thiazide diuretics are given alone or in combination with a dihydropyridine calcium channel blocker. The compensatory rise in angiotensin II causes an increase in blood pressure and can be reversed with the addition of an ACE inhibitor.

Thiazides may potentiate other antihypertensive drugs and are often used in combination with a loop diuretic to enhance diuresis. They are less effective in the presence of excessive salt intake or nonsteroidal anti-inflammatory drugs (NSAIDs).

As discussed, the implementation of a reduced-sodium diet is important to achieve maximal benefit from diuretics. Patients should be monitored for signs of hypokalemia and worsening kidney function.

Some studies suggest that the thiazide diuretics contribute to impaired glucose tolerance, impaired fasting glucose or diabetes mellitus. The JNC's seventh report, as well as the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) and a meta-analysis of 42 clinical trials of antihypertensives, each concluded that at low doses (12.5 mg to 25 mg), thiazide diuretics are safe and have little or no effect on metabolic status.

Loop Diuretics

Loop diuretics block chloride reabsorption in the thick segment of the ascending loop of Henle and result in loss of sodium, chlorides and potassium in the urine. As GFR falls, thiazides become ineffective. When this occurs, loop diuretics are indicated because they increase renal blood flow. Higher doses may be required to achieve diuresis in the presence of significant peripheral edema or heart failure. These doses may be given once or twice daily and in combination with a thiazide diuretic to enhance diuresis.

Electrolytes and kidney function should be monitored closely in patients who take diuretics. As blood pressure is lowered in patients with renal insufficiency, a temporary decrease in renal function may occur. Patients should therefore be careful about using NSAIDs, which decrease the effectiveness of a diuretic by blocking prostaglandins, which results in vasoconstriction.

Potassium-Sparing Diuretics

Potassium-sparing diuretics and aldosterone antagonists interfere with the ability of aldosterone to promote sodium reabsorption and potassium secretion. As a result, sodium is excreted along with water, and potassium is retained.

Commonly prescribed potassium-sparing diuretics and aldosterone antagonists are spironolactone (Aldactone), eplerenone (Inspra), amiloride (Midamor), triamterene (Dyrenium).

Potassium-sparing diuretics and aldosterone antagonists should be used with caution in patients with CKD. They should also be used carefully in patients with a GFR less than 30 mL/min/1.73 m² and in patients who take an ACE inhibitor or an ARB or have a history of hyperkalemia. Both drug classes may produce hyperkalemia in patients with GFR less than 30 mL/min and are not recommended in patients with CKD.

ACE Inhibitors and ARBs

ACE inhibitors reduce blood pressure by blocking the conversion of angiotensin I to angiotensin II. Angiotensin II is a potent vasoconstrictor that also stimulates the release of aldosterone. Renin levels are