Monitor ECG when available, and compare with baseline or previous tracings.

Evaluation

- Interview and observe for relief of symptoms (weight loss, increased urine output, less extremity edema, easier breathing, improved activity tolerance and self-care ability, slower heart rate).
- Observe serum drug levels for normal or abnormal values, when available.
- Interview regarding compliance with instructions for taking the drug.
- Interview and observe for adverse drug effects, especially cardiac dysrhythmias.

PRINCIPLES OF THERAPY

Goals of Management

The goals for clients with asymptomatic (compensated) HF are to maintain function as nearly normal as possible and to prevent symptomatic (acute, congestive, or decompensated) HF, hospitalizations, and death. When symptoms or decompensation occurs, the goals are to relieve symptoms, restore function, and prevent progressive cardiac deterioration.

Nonpharmacologic Management Measures

1. Prevent or treat conditions that precipitate cardiac decompensation and failure (eg, fluid and sodium retention, factors that impair myocardial contractility or increase cardiac workload).
2. Restrict dietary sodium intake to reduce edema and other symptoms and allow a decrease in diuretic dosage. For most clients, sodium restriction need not be severe. A common order, "no added salt," may be accomplished by avoiding obviously salty foods (eg, ham, potato chips, snack foods) and by not adding salt during cooking or eating. For clients with more severe HF, dietary intake may be more restricted (eg, no more than 2 g daily). A major source of sodium intake is table salt: A level teaspoonful contains 2300 mg of sodium.
3. If hyponatremia (serum sodium <130 mEq/L) develops from sodium restrictions and diuretic therapy, fluids may need to be restricted (eg, 1.5 L/day or less) until the serum sodium level increases. Severe hyponatremia (<125 mEq/L) may lead to dysrhythmias.
4. For clients who are obese, weight loss is desirable to decrease systemic vascular resistance and myocardial oxygen demand.
5. Reduce physical activity in clients with symptomatic HF. This decreases the workload and oxygen consumption of the myocardium. If bed rest is instituted, antithrombotic measures such as compression stockings/devices or heparin therapy should be prescribed to prevent deep vein thrombosis.
6. Administer oxygen, if needed, to relieve dyspnea, improve oxygen delivery, reduce the work of breathing, and decrease constriction of pulmonary blood vessels (which is a compensatory measure in clients with hypoxemia).

Pharmacologic Management

A combination of drugs is the standard of care for both acute and chronic HF. Specific drug components depend on the client's symptoms and hemodynamic status.

1. For **acute HF,** the first drugs of choice may include an IV loop diuretic, a cardiotonic-inotropic agent (eg, digoxin, dobutamine, or milrinone), and vasodilators (eg, nitroglycerin and hydralazine or nitroprusside). This combination reduces preload and afterload and increases myocardial contractility.
2. For **chronic HF,** an ACE inhibitor or ARB and a diuretic are the basic standard of care. Digoxin, a beta-adrenergic blocking agent, and spironolactone may also be added. Although the use of digoxin in clients with normal sinus rhythm has been questioned, studies indicate improved ejection fraction and exercise tolerance in clients who receive digoxin. In addition, in clients stabilized on digoxin, a diuretic, and an ACE inhibitor or ARB, symptoms worsen if digoxin is discontinued.

   Overall, these drugs improve clients' quality of life by decreasing their symptoms and increasing their ability to function in activities of daily living. They also decrease hospitalizations and deaths from HF.
3. **Electrolyte balance** must be monitored and maintained during digoxin therapy, particularly normal serum levels of potassium (3.5 to 5 mEq/L), magnesium (1.5 to 2.5 mg/100 mL), and calcium (8.5 to 10 mg/100 mL). Hypokalemia and hypomagnesemia increase cardiac excitability and ectopic pacemaker activity, leading to dysrhythmias; hypercalcemia enhances the effects of digoxin. These electrolyte abnormalities increase the risk of digoxin toxicity. Hypocalcemia increases excitability of nerve and muscle cell membranes and causes myocardial contraction to be weak (leading to a decrease in digoxin effect).

   In acute HF, there is a high risk of hypokalemia because large doses of potassium-losing diuretics are often given. Serum potassium levels should be monitored regularly and supplemental potassium may be needed. In chronic HF, hypokalemia may be less likely to occur than formerly because lower doses of potassium-losing diuretics are usually being given. In addition, there may be more extensive use of potassium-sparing diuretics (eg, amiloride or triamterene) and spironolactone. Note, however, that hyperkalemia must also be prevented because it is cardiotoxic.