أ.م.د.أسيل جاسم محمد

Heart failure

Heart failure occurs when the heart cannot deliver adequate cardiac output to meet the metabolic needs of the body.

Cardiac muscle with compromised intrinsic contractility requires a greater degree of dilatation to produce increased stroke volume and does not achieve the same maximal cardiac output as normal myocardium does. The ability of an immature heart to increase cardiac output in response to increased preload is less than that of a mature heart. Premature infants are more compromised by a left-to-right shunt than full-term infants are. The primary determinants of stroke volume are the after load (pressure work), preload (volume work), and contractility (intrinsic myocardial function). Abnormalities in heart rate can also compromise cardiac output and produce both brad arrhythmias and tachyarrhythmia's; the latter shorten the diastolic time interval for ventricular filling. Alterations in the oxygen-carrying capacity of blood (e.g., anemia or hypoxemia) also lead to a decrease in systemic oxygen transport.

There are multiple systemic compensatory mechanisms used by the body to adapt to chronic heart failure. One of the principal mechanisms for increasing cardiac output is an increase in sympathetic tone secondary to increased adrenal secretion of circulating epinephrine and increased neural release of nor epinephrine. The initial beneficial effects of sympathetic stimulation include an increase in heart rate and myocardial contractility, mediated by these hormones' action on cardiac β-adrenergic receptors; both serve to increase cardiac output. Peripheral vasoconstriction can result in decreased renal, hepatic, and gastrointestinal tract function.

CLINICAL MANIFESTATIONS

A critically ill infant or child who has exhausted the compensatory mechanisms to the point that cardiac output is no longer sufficient to meet the basal metabolic needs of the body will be symptomatic at rest. A thorough history is extremely important in making the diagnosis of heart failure and in evaluating the possible causes. Gradually worsening perfusion or increasing cyanosis may not be recognized as an abnormal finding.

Edema may be passed off as normal weight gain, and exercise intolerance as lack of interest in an activity. The history of a young infant should also focus on feeding . An infant with heart failure often takes less volume per feeding, becomes dyspneic while sucking, and may perspire profuselyIn infants, heart failure may be difficult to distinguish from other causes of respiratory distress. Prominent manifestations include tachypnea, feeding difficulties, poor weight gain, excessive perspiration, irritability, weak cry, and noisy, labored respirations with intercostal and subcostal retractions, as well as flaring of the alae nasi. In children, the signs and symptoms of heart failure may be similar to those in adults and include fatigue, effort intolerance, anorexia, dyspnea, and cough.

Many children, however, especially adolescents, may have primarily abdominal symptoms and a surprising lack of respiratory complaints. Attention to the cardiovascular system may come only after an abdominal roentgenogram unexpectedly shows the lower end of an enlarged heart. Orthopnea and basilar rales are variably present; edema is usually discernible in dependent portions of the body, or anasarca may be present. Cardiomegaly is invariably noted. A gallop rhythm is common; when ventricular dilatation is advanced, the holosystolic murmur of mitral or tricuspid valve regurgitation may be heard.

DIAGNOSIS

Roentgenograms of the chest show cardiac enlargement. Pulmonary vascularity is variable and depends on the cause of the heart failure. Fluffy perihilar pulmonary markings suggestive of venous congestion and acute pulmonary edema are seen only with more severe degrees of heart failure.

Echocardiographic techniques are most useful in assessing ventricular function. The most commonly used parameter in children is fractional shortening. Normal fractional shortening is between 28% and 40%. Doppler studies can be used to estimate cardiac output. Magnetic resonance angiography (MRA) can be useful in quantifying left and right ventricular function and mass. .

TREATMENT

The underlying cause of cardiac failure must be removed or alleviated if possible. If the cause is a congenital cardiac anomaly amenable to surgery, medical treatment is indicated to prepare the patient for surgery. With

excellent outcomes of primary surgical repair of congenital heart defects, even in the neonatal period, few children require aggressive heart failure management to "grow big enough for surgery." In contrast, if the cause of heart failure is cardiomyopathy, medical management provides temporary relief from symptoms and may allow the patient to recover if the insult is reversible (myocarditis). If the lesion is not reversible, heart failure management usually allows the child to return to normal activities for some period and delay, sometimes for years, consideration for heart transplantation.

General Measures

Strict bed rest is rarely necessary except in extreme cases, but it is important that the child be allowed to rest during the day as needed and sleep adequately at night. For infants with heart failure, an infant chair may be advisable. After patients begin to respond to treatment, restrictions on activities can often be modified within the context of the specific diagnosis and the patient's ability..

Diet

Infants with heart failure may fail to thrive because of increased metabolic requirements and decreased caloric intake. Increasing daily calories is an important aspect of their management. Increasing the number of calories per ounce of infant formula (or supplementing breast-feeding) may be beneficial.

Severely ill infants may lack sufficient strength for effective sucking because of extreme fatigue, rapid respirations, and generalized weakness. In these circumstances, nasogastric feedings may be helpful , feedings at night, administered by pump, may improve caloric intake while decreasing problems with reflux. Continued malnutrition may be an important factor in the decision to undertake earlier surgical intervention . The use of low sodium formulas in the routine management of infants with heart failure is not recommended because these preparations are often poorly tolerated and may exacerbate diuretic-induced hyponatremia

Digitalis

Digoxin, once the mainstay of heart failure management in both children and adults, is currently used less, as a result of the introduction of newer therapies and the recognition of its potential toxicities. Many cardiologists will use digitalis as an adjunct to ACE inhibitors and diuretics in patients with symptomatic heart failure. The drug crosses the placenta, and therefore a fetus with heart failure (secondary to arrhythmia) can be treated by administering digoxin to the mother. The kidney eliminates digoxin, so dosing must be adjusted according to the patient's renal function.

Rapid digitalization of infants and children in heart failure may be carried out intravenously. The electrocardiogram must be closely monitored and rhythm strips obtained before each of the three digitalizing doses. Digoxin should be discontinued if a new rhythm disturbance is noted. Serum digoxin determination is helpful when digitalis toxicity is suspected, although it may be less reliable in infants. Baseline serum electrolyte levels should be measured before and after digitalization. Hypokalemia and hypercalcemia exacerbate digitalis toxicity. Because hypokalemia is relatively common in patients receiving diuretics, potassium levels should be monitored closely in those receiving a potassium-wasting diuretic (e.g.furosemide) in combination with digitalis. In patients with active myocarditis. Patients who are not critically ill may be given digitalis initially by the oral route, and in most instances digitalization is completed within 24 hr.

Hypokalemia, hypomagnesemia, hypercalcemia, cardiac inflammation secondary to myocarditis, and prematurity may all potentiate digitalis toxicity.

Diuretics

These agents interfere with reabsorption of water and sodium by the kidneys, which results in a reduction in circulating blood volume and thereby reduces pulmonary fluid overload and ventricular filling pressure. Diuretics are most often used in conjunction with digitalis therapy in patients with severe congestive heart failure. Careful monitoring of electrolytes is necessary with long-term furosemide therapy because of the potential for significant loss of potassium. Potassium chloride supplementation is usually required unless the potassium-sparing diuretic spironolactone is given concomitantly.

Afterload-Reducing Agents and ACE Inhibitors

This group of drugs reduces ventricular after load by decreasing peripheral vascular resistance and thereby improving myocardial performance. Some of these agents also decrease systemic venous tone, which significantly reduces preload. Blood pressure must be continuously monitored because sudden hypotension can occur. ACE inhibitors have additional beneficial effects on cardiac structure and function that may be independent of their effect on afterload. Adverse reactions to captopril include hypotension and its sequelae (syncope, weakness, dizziness, hyperkalemia). A maculopapular pruritic rash is encountered in 5–8% of patients, but the drug may be continued because the rash often disappears spontaneously with time. Neutropenia, renal toxicity, and chronic cough also occur.

α- and β-Adrenergic Agonists(Dopamine,Dobutamine and epineprines).

These drugs are usually administered in an intensive care setting, where the dose can be carefully titrated to hemodynamic response.

Phosphodiesterase Inhibitors

Milrinone is useful in treating patients with low cardiac output who are refractory to standard therapy and has been shown to be highly effective in managing low-output state in children after open heart surgery.

Chronic Treatment with β-Blockers

Studies in adults with dilated cardiomyopathy show that β-adrenergic blocking agents, introduced gradually as part of a comprehensive heart failure treatment program, improve exercise tolerance, decrease hospitalizations, and reduce overall mortality. The agents are metoprolol, a β1-adrenergic receptor selective antagonist, and carvedilol, an agent with both α- and β-adrenergic receptor blocking.

Cardiogenic Shock

Cardiogenic shock may occur as a complication of

(1) severe cardiac dysfunction before or after cardiac surgery. (2) septicemia.(3) severe burns.(4) anaphylaxis.(5) cardiomyopathy, (6) myocarditis, (7) myocardial infarction or stunning, and (8) acute central nervous system disorders. It is characterized by low cardiac output and hypotension and therefore results in inadequate tissue perfusion.

Heart failure in infants and children results in some degree of Hepatomegly and occasionally Splenomegaly.

The heart should be examined in a systematic manner starting with inspection and palpation. A precordial bulge to the left of the sternum with increased precordial activity suggests cardiac enlargement; such bulges can often best be appreciated by having the child lay supine with the examiner looking up from the child's feet. A hyper dynamic precordium suggests a volume load such as that found with a large left-to-right shunt.

Often, the 2nd heart sound seems to be single during expiration. The presence of a normally split 2nd sound is strong evidence against the diagnosis of atrial septal defect. A 3rd heart sound is best heard with the bell at the apex in mid-diastole. A 4th sound occurring in conjunction with atrial contraction may be heard just before the 1st heart sound in late diastole.

Auscultation for murmurs should be carried out across the upper precordium, down the left or right sternal border, and out to the apex and left axilla. Auscultation should also always be performed in the right axilla and over the back. Systolic murmurs are classified as ejection, pansystolic, or late systolic according to the timing of the murmur in relation to the 1st and 2nd heart sounds. The intensity of systolic murmurs is graded from I to VI: I, barely audible; II, medium intensity; III, loud but no thrill; IV, loud with a thrill; V, very loud but still requiring positioning of the stethoscope at least partly on the chest; and VI, so loud that the murmur can be heard with the stethoscope off the chest. Pansystolic or holosystolic murmurs begin almost simultaneously with the 1st heart sound and continue throughout systole, on occasion becoming gradually decrescendo.