Congestive Heart Failure

:Objectives

1. Define congestive heart failure.
2. Recognise causes of CHF.
3. Describe the pathophysiology of CHF.
4. Identify the clinical manifestations and physical signs of CHF.
5. Recognise the diagnosis of CHF.
6. Identify the management of CHF.
7. Recognise the digoxin toxicity and its treatment.

Definition

Congestive heart failure (CHF) is a clinical syndrome in which the heart is unable to pump enough blood to the body to meet its needs, to dispose of systemic or pulmonary venous return .adequately, or a combination of the two

Causes

The heart failure may arise from diverse causes. Common causes of CHF are volume or pressure overload (or both) caused by

congenital or acquired heart disease and myocardial.1
.diseases

Tachyarrhythmias and heart block can also cause heart.2
failure at any age. By far the most common causes of CHF in
.infancy result from congenital heart defects (CHDs)

Beyond infancy, myocardial dysfunctions of various.3
.etiologies are important causes of CHF

Among the rare causes of CHF are metabolic and endocrine disorders, anemia, pulmonary diseases, collagen vascular diseases, systemic or pulmonary hypertension,
.neuromuscular disorders, and drugs such as anthracyclines

CAUSES OF CONGESTIVE HEART FAILURE RESULTING FROM CONGENITAL HEART DISEASE

Age of Onset Cause

At birth

Hypoplastic left heart syndrome

Severe tricuspid or pulmonary insufficiency

Large systemic arteriovenous fistula

First week

TGA

PDA in small premature infants

HLHS (with more favorable anatomy)

TAPVR, particularly those with pulmonary venous obstruction

Systemic arteriovenous fistula

Critical AS or PS

wk 4–1

COA with associated anomalies

Critical AS

Large left-to-right shunt lesions (VSD, PDA) in premature infants

wk Some left-to-right shunt lesions such as ECD **6–4**

wk–4 mo 6

Large VSD

Large PDA

Others such as anomalous left coronary artery from the PA

Congenital Heart Disease

Volume overload lesions, such as ventricular septal defect (VSD), patent ductus arteriosus (PDA), and endocardial cushion defect (ECD), are the most common causes of CHF in the first 6 months of life.the following also shoud be noted

Children with tetralogy of Fallot (TOF) do not develop CHF unless. 1 they have received a large systemic-to-pulmonary artery shunt .procedure

Atrial septal defect (ASD) rarely causes CHF in the pediatric age. 2 ,group

.although it causes CHF in adulthood

Large left-to-right shunt lesions, such as VSD and PDA, do not cause. 3 CHF before 6 to 8 weeks of age because the pulmonary vascular resistance does not fall low enough to cause a large left-to-right shunt .until this age

Acquired Heart Disease

Acquired heart disease of various causes can lead to CHF. With acquired heart disease, the age at onset of CHF is not as predictable :as with CHD, but the following generalities apply

1. Dilated cardiomyopathy is probably the most common cause of CHF beyond infancy. It may cause CHF at any age during childhood and adolescence. The cause of the majority of dilated cardiomyopathy is idiopathic, but it may be caused by infectious, endocrine, or metabolic disorders; autoimmune diseases; or after antineoplastic treatment (e.g.,anthracycline)
2. Doxorubicin cardiomyopathy may manifest months to years after the completion of chemotherapy for malignancies in children

Cardiomyopathies associated with muscular dystrophy and. 3
.Friedreich's ataxia may cause CHF in older children and adolescents
Myocarditis associated with Kawasaki's disease is seen in children 1. 4
.to 4 years of age
Patients who received surgery for some types of CHDs (surgery for.5
TOF, transposition of the great arteries and other cyanotic defects)
.may remain in or develop CHF after varying period of time
Viral myocarditis tends to be more common in small children older. 6
.than 1year. course with poor prognosis
Acute rheumatic carditis is an occasional cause of CHF that occurs. 7
.primarily in school-age children
Rheumatic valvular heart diseases, usually volume overload lesions. 8
such as mitral regurgitation (MR) or aortic regurgitation (AR), cause
CHF in older children and adults. These diseases are uncommon in
.industrialized counties

Miscellaneous Causes

.Metabolic. 1

.Endocrinopathy such as hyperthyroidism. 2

.Supraventricular tachycardia (SVT) causes CHF in early infancy. 3

Complete heart block associated with structural heart defects. 4

.causes CHF in the newborn period or early infancy

.Severe anemia may be a cause of CHF at any age. 5

.Bronchopulmonary dysplasia seen in premature infants. 6

.Primary carnitine deficiency. 7

Acute systemic hypertension, as seen in acute postinfectious.8

.glomerulonephritis, causes CHF in school-age children

Pathophysiology

Cardiac output is determined by preload, afterload, myocardial contractility, and heart rate. Cardiac output is proportional to filling pressure (preload) and inversely proportional to the resistance against which the heart pumps (afterload).

Preload

According to the Frank-Starling law, as the ventricular end-diastolic volume (or preload) increases, the healthy heart increases cardiac output until a maximum is reached and cardiac output can no .longer be augmented

When the left ventricular (LV) end-diastolic pressure reaches a certain point, however, pulmonary congestion develops with .congestive symptoms (tachypnea and dyspnea)

Afterload

Afterload is the force that resists myofibril shortening during systole, which contributes to total myocardial wall stress (or tension). A decrease in afterload increases cardiac output, and an acute increase in afterload results in decreases in stroke volume and ejection fraction. Indices of afterload include aortic pressure, total systemic vascular resistance, arterial impedance, and myocardial peak wall stress. Afterload reducing increases .cardiac output without increasing oxygen consumption

Wall Stress

According to the law of Laplace, wall tension is the product of .pressure and radium

The Laplace law, although an oversimplification, emphasizes the :following two points

the bigger the LV and the greater the radius, the greater the) 1(wall stress, and (2) at any given radius (LV size), the greater the pressure developed in the LV, the greater the wall stress. Thus, dilated ventricles require more tension in the wall and thus increased oxygen demand to generate the same pressure. A relationship exists between wall stress and preload as well as afterload. Preload can be defined as the wall stress at the end of diastole. The afterload, being the load on the contracting .myocardium, is the wall stress during LV ejection

The increased wall tension in the dilated ventricle leads to ventricular hypertrophy that tends to keep the wall tension low. Well-trained athletes develop cardiac hypertrophy, which helps reduce wall stress, according to Laplace's law. A failing heart will also hypertrophy to ,reduce the increase in wall stress

Compensatory Mechanisms

In the early stages of heart failure, various compensatory mechanisms are evoked to maintain normal metabolic function. Among the compensatory responses are the activation of the sympathetic nervous system and the renin–angiotensin–aldosterone .system

Although these responses are an attempt to preserve cardiovascular homeostasis and thus beneficial initially, chronic stimulation of these systems may be deleterious in the natural history of .myocardial dysfunction

One major compensatory mechanism for increasing cardiac. 1 output is an increase in sympathetic tone secondary to increased adrenal secretion of circulating epinephrine and increased neural .release of norepinephrine

The initial beneficial effects of adrenergic stimulation include increased heart rate and myocardial contractility with a resulting .increase in cardiac output

chronic adrenergic stimulation eventually leads to adverse myocardial effects, including increased afterload, hypermetabolism, .arrhythmogenesis, and direct myocardial toxicity

a. Catecholamines are toxic to cardiac muscle, perhaps by producing .calcium overload or by inhibiting the synthesis of contractile proteins

b. High catecholamine levels decrease the density of β-adrenergic receptors on the surfaces of the myocardial cell,which may be the major cause of the functional loss of the catecholamine-mediated .positive inotropic response

-In clinical settings, the reduction of adrenergic stimulation by the β adrenergic blockers has resulted in clinical improvement in patients with dilated cardiomyopathy, in whom increased levels of .catecholamines have been shown to be present

The reduced blood flow to the kidneys in patients with CHF. 2 causes a marked increase in renin output, and this in turn causes .the formation of angiotensin II

Angiotensin II leads to a further increase in reabsorption of both water and salt from the renal tubules. Angiotensin II may cause atrophic response in vascular smooth muscle (with .vasoconstriction) and myocardial hypertrophy

.Angiotensin II also promotes myocardial fibrosis

Thus, although a hypertrophic response is adaptive by attempting to restore wall stress to normal, angiotensin II plays a maladaptive .role in CHF by initiating fibrosis and altering ventricular compliance

Thus, the reasons for using β-adrenergic blockers and angiotensin converting enzyme (ACE) inhibitors in the treatment of CHF are to block the maladaptive role of adrenergic and .renin–angiotensin–aldosterone systems

Diagnosis

The diagnosis of CHF relies on several sources of clinical findings, including history, physical examination, chest radiographs, and echocardiographic studies. Echocardiographic studies are most helpful

noninvasive study that confirms the diagnosis of heart failure and estimates the severity of heart failure. It may also help identify the .cause of heart failure

Plasma levels of natriuretic peptides, atrial natriuretic peptide (ANP) and B-type natriuretic peptide (BNP), are increased in most adult patients with heart failure. They are important makers of heart failure and may help distinguish dyspnea caused by heart failure and pulmonary disease in adult patients. ANP is stored mainly in the RA and is released when the atrial distending pressure increases. BNP is stored in ventricular myocardium and appears to be released when the ventricular filling pressure increases. Both peptides exhibit vasodilating effects and natriuretic effects on the kidneys and counteract the water-retaining effects of the

The plasma levels of these peptides are elevated in newborns and in the first weeks of life but decrease to the levels observed in normal adults. Increased levels of BNP and the *N*-terminal segment of its prohormone (NT-ProBNT) have been reported in most children with either pressure or volume overload cardiac lesions compared with the levels .seen in normal children

History

Poor feeding of recent onset, tachypnea that worsens. 1
during feeding, poor weight gain, and cold sweat on the
.forehead suggest CHF in infants

Older children may complain of shortness of breath,.2
especially with activities, easily fatigability, puffy eyelids,
.or swollen feet

.**Physical Examination**

The following are found as compensatory responses to impaired. 1 :cardiac function

a.*Tachycardia*, *gallop rhythm*, and weak and thready pulses are .common

b.*Cardiomegaly* is almost always present. Chest radiographs are more .reliable than physical examination in demonstrating cardiomegaly

c. There are signs of increased sympathetic discharges (e.g., *growth .failure*; *perspiration*, cold and wet skin)

Pulmonary venous congestion (from left-sided failure). 2

a. *Tachypnea* is common and is an early manifestation of CHF in .infants

b. Dyspnea on exertion (equivalent to poor feeding in small infants) is .common in children

.c. Orthopnea may be seen in older children

.d. Wheezing and pulmonary crackles are occasionally audible

Systemic venous congestion (caused by right-sided failure). 3
:results in
a. Hepatomegaly is common, but it is not always indicative of CHF. A large liver may be palpable in conditions that cause hyperinflated lungs (asthma, bronchiolitis, during hypoxic spells) and in infiltrative liver disease. Conversely, the absence of hepatomegaly does not rule out CHF; hepatomegaly may be .absent in (early) left-sided failure
.b. Puffy eyelids are common in infants
c. Distended neck veins and ankle edema, which are common in .adults
d. Splenomegaly is not indicative of CHF; it usually indicates .infection

Radiography

The presence of cardiomegaly should be demonstrated by chest .radiographs

The absence of cardiomegaly almost rules out the diagnosis of CHF. The only exception to this rule is when the pulmonary venous return is obstructed; in such cases, the lung parenchyma will show pulmonary edema or venous congestion

Electrocardiography

Electrocardiography helps determine the type of heart defect causing heart failure but is not helpful in determining whether CHF .is present

Echocardiography

Echocardiographic studies may confirm enlargement of ventricular chambers and impaired LV systolic function (decreased fractional shortening or ejection fraction) as well as impaired diastolic function .by the use of Doppler techniques

A more important role of echocardiography may be its ability to determine the cause of CHF. Echocardiography is also helpful in serial .evaluation of the efficacy of therapy

Management

The treatment of CHF consists of

,elimination of the underlying causes) 1 (

treatment of the precipitating or contributing causes (e.g.,)2(

infection, anemia,arrhythmias, fever), and

control of heart failure state. Eliminating the underlying) 3(

.causes is the most desirable approach whenever possible

Treatment of Underlying Causes or Contributing Factors

When surgically feasible, surgical correction of underlying CHDs. 1
.and valvular heart disease is the best approach for complete cure
If hypertension is the underlying cause of CHF, antihypertensive. 2
.treatment should be given
If arrhythmias or advanced heart block is the cause of or. 3
contributing factor to heart failure, antiarrhythmic agents or
.cardiac pacemaker therapy is indicated
If hyperthyroidism is the cause of heart failure, this condition. 4
.should be treated
Fever should be controlled with antipyretics. 5
When there is a concomitant infection, it should be treated with. 6
.appropriate antibiotics
For anemia, a packed cell transfusion is given to raise the. 7
.hematocrit to 35% or higher

.**General Measures**

.A “cardiac chair” keep infants in a semiupright position. 1

Oxygen (40%–50%) with humidity is administered to infants with. 2
.respiratory distress

Adequate calories and fluid should be provided to permit. 3 appropriate weight gain. Infants in CHF need significantly higher caloric intakes than recommended for average children. The required calorie intake may be as high as 150 to 160 kcal/kg/day for infants in .CHF

a. Increasing caloric density of feeding may be required and it may be .accomplished with fortification of feeding

b. Frequent small feedings are better tolerated than large feeding in .infants

c. If oral feedings are not well tolerated, intermittent or continuous .nasogastric (NG) feeding is indicated

d. Salt restriction in the form of a low-salt formula and severe fluid .restriction are not indicated in infants

In older children, salt restriction (<0.5 g/day) and avoidance of. 4 salty snacks and table salt are recommended. Bed rest remains an important component of management. The availability of a television screen and computer games for entertainment ensures .bed rest in older children

If respiratory failure accompanies cardiac failure, intubation and. 5 positive pressure ventilation are occasionally required. Respiratory failure usually signifies that surgical intervention will be needed for .CHDs when the patient is stabilized

.Daily weight measurement is essential in hospitalized patients. 6

Diuretics

Diuretics remain the principal therapeutic agent to control pulmonary and systemic venous congestion. Diuretics only reduce preload and improves congestive symptoms, but do not improve cardiac output or myocardial contractility. There are three main classes of diuretics that .are commercially available

1. Thiazide diuretics (e.g., chlorothiazide, hydrochlorothiazide), which .act at the proximal and distal tubules
2. Rapid-acting diuretics, such as furosemide and ethacrynic acid, are the drugs of choice. They act primarily at the loop of Henle (“loop .diuretics”)
3. Aldosterone antagonists (e.g.spironolactone) act on the distal tubule to inhibit sodium-potassium exchange. Aldosterone antagonists have value in preventing hypokalemia produced by other diuretics and .thus are used in conjunction with a loop diuretic

Rapidly Acting Inotropic Agents

In critically ill infants with CHF, in those with renal dysfunction (e.g., infants with coarctation of the aorta), or in postoperative cardiac patients with heart failure, rapidly acting catecholamines with a short duration of action are preferable to digoxin. This class of agents includes dopamine, dobutamine, isoproterenol, and epinephrine. These agents possess inotropic and vasodilator actions and thus are .useful in acute situations

Dobutamine has fewer chronotropic effects than dopamine. Dopamine in high doses causes α- receptor stimulation with .vasoconstriction and reduction of renal blood flow

Drug Dosage and Route Side Effects

Epinephrine (Adrenalin) 0.1–1 µg/kg/min IV
Hypertension, arrhythmias
Isoproterenol (Isuprel) 0.1–0.5 µg/kg/min IV
Peripheral and pulmonary vasodilatation
Dobutamine (Dobutrex) 2–8 µg/kg/min IV Little
tachycardia and vasodilatation, arrhythmias
Dopamine (Intropin) 5–10 µg/kg/min IV Tachycardia,
arrhythmias, hypertension or hypotension
:Dose-related cardiovascular effects (µg/kg/min)
Renal vasodilatation: 2–5
Inotropic: 5–8
Tachycardia: >8
Mild vasoconstriction: >10
Vasoconstriction: 15–20

Digoxin

The main mechanism of action of digoxin is to inhibit the sodium-potassium ATPase pump in the myocardium. This promotes sodium-calcium exchange and increases intracellular calcium and thus contractility. Digoxin also has beneficial modulating effects in the neurohormonal system: improvement in baroreceptor function, increased vagal tone, sympathoinhibitory effects, decreased circulating norepinephrine levels, and possibly aldosterone antagonistic effects

ORAL DIGOXIN DOSAGE FOR CONGESTIVE HEART FAILURE

Age	**Total Digitalizing µg/kg/day**	**Maintenance Dose (µg/kg)**
Premature infants	20	5
Newborn infants	30	8
2< yr	40–50	
2> yr	30–40	8–10

The maintenance dose is 25% of the total digitalizing dose in two divided doses. The intravenous dose is 75% of the oral dose. In addition to inotropic action, digoxin also has parasympathomimetic action with slowing of heart rate, reducing sinoatrial firing, and .slowing the atrioventricular (AV) conduction

Several earlier studies have shown that digoxin reduces circulating .norepinephrine, renin, and aldosterone levels

Digoxin is a diuretic agent as well. Thus, digoxin can,increase inotropy .without increasing myocardial oxygen consumption

How to digitalize

Loading doses of the total digitalizing doses are given over 12 hours followed by maintenance doses. This results in a pharmacokinetic steady state in 3 to 5 days. The intravenous route is preferred over the oral route, particularly when dealing with infants in severe heart failure

When an infant is in mild heart failure, the maintenance dose may be administered orally without loading doses; this results in .a steady state in 5 to 8 days

The following is a suggested step-by-step method of digitalization:

1. Obtain a baseline ECG (rhythm and PR interval). Hypokalemia and hypercalcemia predispose to digitalis toxicity.
2. Calculate the total digitalizing dose.
3. Give half the total digitalizing dose immediately followed by one fourth and then the final fourth of the total digitalizing dose at 6- to 8-hour intervals.
4. Start the maintenance dose 12 hours after the final total digitalizing dose.

Obtaining an ECG strip before starting the maintenance dose is advised.

Electrocardiographic Changes Associated with Digitalis Effects

Shortening of QTc, the earliest sign of digitalis effect

Sagging ST segment and diminished amplitude of T wave (the)T vector does not change

Slowing of heart rate

Toxicity

Prolongation of PR interval: sometimes a prolonged PR interval is seen in children without digitalis, making a baseline ECG mandatory; the prolongation may progress to second-degree AV block

Profound sinus bradycardia or sinoatrial block

Supraventricular arrhythmias, such as atrial or nodal ectopic beats and tachycardias

Serum digoxin levels

Therapeutic ranges of serum digoxin levels for treating CHF are 0.8 to 2 ng/mL. Blood for serum digoxin levels should be drawn at least 6 hours after the last dose or just before a scheduled dose; samples obtained earlier than 6 hours after the last dose will give a falsely elevated level .Serum digoxin levels may be elevated when administered concomitantly with other drugs such as quinidine, verapamil, amiodarone, beta-blockers,tetracycline, and erythromycin. Lower serum levels have been noted with rifampin, ...kaolin-pectin, neomycin, and cholestyramine

Factors That May Predispose to Digitalis Toxicity

High Serum Digoxin Level

High-dose requirement, as in treatment of certain arrhythmias

Decreased renal excretion

Premature infants

Renal disease

Hypothyroidism

Drug interaction (e.g., quinidine, verapamil, amiodarone)

Increased Sensitivity of Myocardium (without High)Serum Digoxin Level

Status of myocardium

Myocardial ischemia

Myocarditis (rheumatic, viral)

Systemic changes

Electrolyte imbalance (hypokalemia, hypercalcemia)

Hypoxia

Alkalosis

Adrenergic stimuli or catecholamines

The diagnosis of digitalis toxicity is a clinical decision and
:usually is based on the following clinical and laboratory findings
.The patient has a history of accidental ingestion. 1
Noncardiac symptoms appear in digitalized children; these. 2
symptoms include anorexia, nausea, vomiting, diarrhea,
restlessness, drowsiness, fatigue, and visual disturbances in older
.children
.Heart failure worsens. 3
ECG signs of toxicity probably are more reliable and appear early. 4
5. An elevated serum level of digoxin (>2 mg/mL) is likely to be
associated with toxicity in a child if the clinical findings suggest
.digitalis toxicity

Afterload-Reducing Agents

Vasoconstriction that occurs as a compensatory response to reduced cardiac output seen in CHF may be deleterious to the failing .ventricle

Vasoconstriction is produced by a rise in sympathetic tone and circulating catecholamines and an increase in the activity of the renin–angiotensin system. Reducing afterload tends to augment the stroke volume without a great change in the contractile state of the heart and therefore without (increasing myocardial oxygen (consumption

Afterload-reducing agents may be divided into three groups based on the site of action: arteriolar vasodilators, .venodilators, and mixed vasodilators

Arteriolar vasodilators (hydralazine) augment cardiac output by. 1 acting primarily on the arteriolar bed, with resulting reduction of the .afterload

Venodilators (nitroglycerin, isosorbide dinitrate) act primarily by. 2 dilating systemic veins and redistributing blood from the pulmonary to .the systemic circuit

,Mixed vasodilators include ACE inhibitors (captopril, enalapril). 3 nitroprusside, and prazosin. These agents act on both arteriolar and .venous beds

β-Adrenergic blockers

Beneficial effects of β-adrenergic blockers were reported in adult .patients with dilated cardiomyopathy

Metoprolol was added to standard anticongestive medicines in patients with chronic CHF from dilated cardiomyopathy. Metoprolol increased LV

fractional shortening and ejection fraction and improved symptoms.

Carvedilol, when added to standard medical therapy for CHF, has been shown to be beneficial in children with dilated cardiomyopathy .

Carvedilol and metoprolol have been studied most often. There is a theoretical advantage of carvedilol over metoprolol. Carvedilol is a nonselective beta-blocker that inhibits β1-, β2-, and α1-adrenoceptors with additional vasodilatory and antioxidant properties.

Contraindications to the use of β-adrenergic blockers include symptomatic bradycardia or heart block, significant hypotension, .active asthma, and severe bronchial disease