VITEBSK STATE MEDICAL UNIVERSITY

CHAIR OF THE FACULTY THERAPY

**FACULTY THERAPY**

(Methodics work out for foreign students, studying the course of faculty therapy)

Part 1

Vitebsk – 2005

**INFECTIVE ENDOCARDITIS**

Synonyms: Cardiobacterium, native valve endocarditis, prosthetic valve endocarditis, heart infection, bacterial infection, bacteremia

Cardiobacterium hominis is a one of the HACEK (Haemophilus aphrophilus, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, and Kingella kingae) group of fastidious, gram-negative, aerobic bacilli. The HACEK bacteria normally reside in the respiratory tract. They have been associated with local infection in the mouth and, collectively, cause 5-10% of cases of native valve endocarditis in persons who do not abuse intravenous drugs.

**Frequency**

In the US: Endocarditis caused by C hominis accounts for 0.1% of all cases of endocarditis. Of these cases, 75% occur in patients with abnormal valves. The mitral and aortic valves are affected most often.

Mycotic aneurysms are an important cause of morbidity and mortality in endocarditis caused by C hominis. Mycotic aneurysm complicates 2.5-10% of cases of C hominis endocarditis. Embolization occurs during the active stages of endocarditis.

No difference in colonization rates is observed between males and females. C hominis occasionally may be recovered from uterine, cervical, and vaginal cultures in asymptomatic women.

No difference in colonization rates is observed among different age groups.

**Causes**

Bacteremia occurs in the setting of preexisting structural heart disease or a prosthetic heart valve. Many patients have a history of a recent dental procedure or poor dentition.

**Pathophysiology**

C hominis can be isolated from the nose or throat of approximately two thirds of healthy individuals. C hominis is a nonmotile organism that requires 5-10% carbon dioxide for growth. It does not grow on selective media such as MacConkey or eosin methylene blue agar.

In animal studies, C hominis has shown low virulence, with injection of large numbers of organisms failing to produce infection. Nearly all infections reported in humans have been bacteremias or endocarditis.

**Classification**

**Acute bacterial endocarditis (ABE)** is usually caused by S. aureus, group A hemolytic streptococci, pneumococci, or gonococci and by less virulent microorganisms. It can develop on normal valves.

**Subacute bacterial endocarditis (SBE)** is usually caused by streptococcal species (especially viridans streptococci, microaerophilic and anaerobic streptococci, nonenterococcal group D streptococci, and enterococci) and less commonly by Staphylococcus aureus, S. epidermidis, and fastidious Haemophilus sp. SBE often develops on abnormal valves after asymptomatic bacteremias from infected gums or the GU or GI tract.

**Prosthetic valvular endocarditis (PVE)** develops in 2 to 3% of patients within 1 yr after valve replacement and in 0.5%/yr thereafter; it is more common with aortic than mitral valve prostheses and least common with porcine valves (heterografts). Early-onset infections (< 2 mo postsurgery) are caused mainly by antimicrobial-resistant contamination at surgery (eg, S. epidermidis, diphtheroids, coliform bacilli, Candida sp, Aspergillus sp). Late-onset infections are caused mainly by contamination with low-virulence organisms at surgery or by transient asymptomatic bacteremias, most often Streptococcus sp, S. epidermidis, diphtheroids, and the fastidious gram-negative bacilli--Haemophilus sp, Actinobacillus actinomycetemcomitans, and Cardiobacterium hominis. S. epidermidis can be an early or late pathogen.

**Right-sided endocarditis** involving the tricuspid valve and less often the pulmonary valve and artery may result from IV use of illicit drugs or from central vascular lines, which facilitate entry of microorganisms and may damage the endocardium. Organisms may originate from the skin (eg, S. aureus, Candida sp, coliform bacilli).

**CLINICAL**

**History**

The clinical course of endocarditis produced by C hominis tends to be subacute. In a published series, the mean duration of symptoms was 169 days; however, this may reflect the difficulty growing C hominis in older blood culture systems. In this same series, 44% of patients had a history of a dental procedure or oral infection.

**Physical**

Common findings include the following:

Fever (86%)

Splenomegaly (59%)

Peripheral embolic phenomenon (44%)

Petechiae (41%)

Clubbing (19%)

SBE has an insidious onset and may mimic other systemic illnesses with low-grade fever (< 39° C [< 102.2° F]), night sweats, fatigability, malaise, weight loss, and valvular insufficiency. Chills and arthralgias may occur. Emboli may produce stroke, MI, flank pain and hematuria, abdominal pain, or acute arterial insufficiency in an extremity.

Physical examination may be normal or show chronic illness with pallor; fever; a change in a preexisting murmur or a new regurgitant cardiac valvular murmur; tachycardia; petechiae over the upper trunk, conjunctiva, mucous membranes, and distal extremities; painful erythematous subcutaneous nodules about the tips of the digits (Osler nodes); splinter hemorrhages under the nails; or hemorrhagic retinal lesions (particularly Roth's spots--round or oval lesions with small white centers). With prolonged infection, splenomegaly or clubbing of the fingers and toes may also be present.

Hematuria and proteinuria may result from embolic infarction of the kidney or diffuse glomerulonephritis due to immune complex deposition. Manifestations of CNS involvement (in about 35% of patients) may range from transient ischemic attacks and toxic encephalopathy to brain abscess and subarachnoid hemorrhage from rupture of a mycotic aneurysm.

In ABE, symptoms and signs are similar to those of SBE, but the course is more rapid. ABE is marked by the variable presence of high fever, toxic appearance, rapid valvular destruction, valve ring abscesses, septic emboli, an obvious source of infection, and septic shock. Purulent meningitis may occur.

PVE often results in valve ring abscesses; obstructing vegetations; myocardial abscesses; mycotic aneurysms manifested by valve obstruction, dehiscence, and cardiac conduction disturbances; and the usual symptoms of SBE or ABE.

Right-sided endocarditis is characterized by septic phlebitis, fever, pleurisy, hemoptysis, septic pulmonary infarction, and tricuspid regurgitation.

Because symptoms and signs are nonspecific, are highly variable, and may present insidiously, diagnosis requires a high index of suspicion; risk is greatest in patients with a history of cardiac valvular disease, recent invasive medical procedures, or dental work and in drug addicts. Fever and heart murmurs are the most constant finding; although <= 15% of patients may not have fever or a murmur initially, almost all develop both. Patients with bacteremia from organisms known to be frequent causes of infective endocarditis should be examined carefully and repeatedly for new valvular murmurs and signs of embolic phenomena. Any patient with suspected septicemia, especially with fever and a murmur, must have blood drawn for cultures as soon as possible.

**Lab Studies**

Because of continuous bacteremia in intravascular infections, three to five blood cultures (20 to 30 mL each) within 24 h may be needed to isolate the etiologic agent. Identification of the organism and its antimicrobial susceptibility is vital to guide bactericidal treatment. Blood cultures may require 3 to 4 wk incubation for certain organisms; other organisms (ie, Aspergillus sp) may not produce positive cultures, and others require serodiagnosis (Coxiella burnetii, Chlamydia psittaci, Barcella sp, Rochalimaea) or special culture media (Legionella pneumophila).

Other than positive blood cultures, there are no specific laboratory findings. Negative blood cultures may indicate suppression due to prior antimicrobial therapy, infection with organisms that do not grow in routine laboratory culture media, or another diagnosis (eg, noninfective endocarditis, atrial myxoma with embolic phenomena, or a vasculitis).

Older blood culture systems did not support the growth of HACEK bacteria aerobically or anaerobically; blood cultures needed to be held for 2-3 weeks with blind subcultures. Newer blood culture systems support the growth of HACEK bacteria within 5 days. Despite this, some experts continue to recommend holding blood cultures for an extended period (up to a month) when a diagnosis of endocarditis is considered.

In one study, anemia was present in 18 of 23 (78%) patients with C hominis endocarditis

The erythrocyte sedimentation rate is usually elevated and may exceed 100 mm/h.

Urinalysis results may show evidence of glomerulonephritis, including hematuria and proteinuria.

Rheumatoid factor test results may be positive, and this finding is one of the minor criteria for endocarditis.

**Imaging Studies**

Transesophageal echocardiogram (TEE) is considerably more sensitive than transthoracic echocardiography (TTE) for helping detect valvular vegetations. TTE is usually performed first, and, if findings are positive, then TEE is probably unnecessary.

Transthoracic two-dimensional echocardiography detects vegetations in 50% of patients with endocarditis and may eliminate the need for more invasive procedures. Transesophageal echocardiography detects vegetations in > 90% of patients, including those with negative blood cultures, and can detect myocardial abscesses. In established infections, a normocytic-normochromic anemia, elevated ESR, neutrophilia, increased immunoglobulins, circulating immune complexes, and rheumatoid factor are often present.

**Duke criteria for infective endocarditis (IE)**

Major criteria

Blood cultures positive for IE

Typical microorganisms consistent with IE:

Viridans streptococci, Streptococcus bovis, HACEK group, Staphylococcus aureus; or

Community acquired enterococci, in the absence of a primary focus;

or

Microorganisms consistent with IE from persistently positive blood cultures;

or

Single positive blood culture for Coxiella burnetii or phase I IgG antibody titre 1:800

Evidence of endocardial involvement

Echocardiogram positive for IE:

Vegetation

Abscess

New partial dehiscence of prosthetic valve

New valvar regurgitation

Minor criteria

Predisposition, predisposing heart condition, injection drug use

Fever, temperature .38˚C

Vascular phenomena, major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial haemorrhages, Janeway’s lesions

Immunologic phenomena: glomerulonephritis, Osler’s nodes, Roth’s spots, rheumatoid factor

Microbiological evidence: positive blood culture but does not meet a major criterion

**Diagnosis of IE is definite** in the presence of: two major criteria, or one major and three minor criteria, or five minor criteria.

**Diagnosis of IE is possible** in the presence of: one major and one minor criteria, or three minor criteria.

[HACEK, Heamophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella]

**TREATMENT**

Medical Care

Successful treatment requires maintenance of high serum levels of an effective antibiotic and surgical management of mechanical complications and resistant organisms.

**Antibiotic regimens**: Penicillin-susceptible streptococci (penicillin G MIC <= 0.1 µg/mL) include most viridans streptococci, microaerophilic and anaerobic streptococci, and nonenterococcal group D streptococci. Penicillin G 12 to 18 million U/day IV administered continuously or q 4 h and procaine penicillin G 1.2 million U IM q 6 or 12 h for 4 wk have equivalent results. When gentamicin 1 mg/kg IM (up to 80 mg) q 8 h is administered concurrently, treatment should be reduced to 2 wk. Patients allergic to penicillin may be cautiously given ceftriaxone if there is no history of penicillin anaphylaxis or, alternatively, vancomycin. Ceftriaxone 2 g once daily IV for 4 wk through a central line is convenient and effective as outpatient treatment. Oral treatment programs are less reliable and should not be used without close monitoring of serum levels to ensure adequate GI absorption.

Penicillin-resistant streptococci (penicillin G MIC > 0.1 µg/mL) include enterococcal and some other streptococcal strains (including fastidious pyridoxal-requiring viridans streptococci), which are relatively resistant to penicillin G and require a penicillin or vancomycin combined with an aminoglycoside. About 40% of enterococcal strains demonstrate resistance to streptomycin and should be treated with penicillin plus gentamicin. Gentamicin resistance is an increasing therapeutic problem in nosocomial enterococcal endocarditis. Penicillin G 18 to 30 million U/day IV or ampicillin 12 g/day IV administered continuously or q 4 h should be given concurrently with gentamicin 1 mg/kg IV (based on ideal rather than actual body weight in obese persons) q 8 h for 4 to 6 wk. Patients with enterococcal infections lasting > 3 mo with large vegetations or with vegetations on prosthetic valves should be treated for 6 wk. Persons allergic to penicillin may be desensitized or treated with vancomycin 15 mg/kg IV (up to 1 g) q 12 h and gentamicin.

Pneumococcal or group A streptococcal endocarditis should be treated with penicillin G 10 to 20 million U/day IV for 4 wk. S. aureus endocarditis should be treated with penicillin G 15 to 24 million U/day IV if the strain does not produce -lactamase. Ninety-five percent of strains are penicillin-resistant and should be treated with a penicillinase-resistant penicillin (oxacillin or nafcillin) 2 g IV q 4 h for 4 to 6 wk. Staphylococcal strains resistant to the penicillinase-resistant penicillins are also resistant to the cephalosporins, although resistance may be difficult to demonstrate with routine testing. Oxacillin-resistant or nafcillin-resistant staphylococci should be treated with vancomycin 15 mg/kg IV q 12 h. Oxacillin-susceptible or nafcillin-susceptible infections in penicillin-allergic patients may be cautiously treated with cefazolin 2 g IV q 8 h if there is no history of penicillin anaphylaxis, or with vancomycin.

Because S. epidermidis endocarditis occurs most often in patients with prosthetic valves, patients may require antimicrobial drugs and surgery. Penicillin-susceptible or oxacillin-susceptible strains should be treated as outlined above for S. aureus, but for 6 to 8 wk. Oxacillin or nafcillin should be combined with rifampin 300 mg po every 8 h and gentamicin 1 mg/kg IV every 8 h. Oxacillin-resistant strains should be treated with vancomycin 15 mg/kg IV q 12 h plus gentamicin 1 mg/kg IV every 8 h and rifampin 300 mg po q 8 h for 6 to 8 wk.

HACEK microorganisms (Haemophilus parainfluenzae, H. aphrophilus, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae) should be treated with ceftriaxone 2 g/day IV for 4 wk or ampicillin plus gentamicin for 4 wk using the same doses given for enterococcal infections. Coliform bacillary infections often show antimicrobial resistance and should be treated for >= 4 wk with a sensitivity-proven -lactam antimicrobial drug plus an aminoglycoside.

Until recently, the HACEK bacteria were uniformly susceptible to ampicillin. Recently, however, beta-lactamase–producing strains of HACEK have been identified.

Ceftriaxone or cefotaxime should be considered the drug of choice for HACEK endocarditis.

Ampicillin plus an aminoglycoside can be used for susceptible isolates.

In patients unable to take beta-lactams, options include trimethoprim-sulfamethoxazole, fluoroquinolones, or aztreonam. However, little experience has been gained in treating HACEK endocarditis with any of these therapies.

Native valve endocarditis should be treated for 3-4 weeks. Prosthetic valve endocarditis requires 6 weeks of treatment.

**Cardiac valve surgery**: Cardiac valve surgery (debridement and/or replacement of the valve) is frequently required to eradicate infection that is uncontrolled medically, particularly in early-onset PVE. The timing of surgical intervention requires experienced clinical judgment. If heart failure caused by a correctable lesion is worsening (particularly when the organism is S. aureus, a gram-negative bacillus, or a fungus), surgery may be required urgently, but an optimal antibiotic regimen is given for 24 to 72 h before surgery.

Response to treatment: Patients with penicillin-susceptible streptococcal infective endocarditis usually feel better and have a reduction in fever within 3 to 7 days of starting therapy. However, fever may persist for reasons other than continued active infection (eg, drug allergy, phlebitis, infarction from emboli). Staphylococcal infective endocarditis often responds more slowly. Sterile emboli and valve rupture may occur up to 1 yr after successful antimicrobial therapy. Relapse usually occurs within 4 wk; antibiotic retreatment may be effective, but surgery may also be required. Recrudescence of infective endocarditis after 6 wk in patients without prosthetic valves usually is a new infection rather than a relapse.Further Inpatient Care:

Native valve endocarditis should be treated for 3-4 weeks, and prosthetic valve endocarditis requires 6 weeks of treatment.

**Further Outpatient Care**

Patients can be treated in an outpatient setting but should remain on intravenous antimicrobial therapy for the duration of treatment for endocarditis.

Risks of embolic complications may arise during therapy.

Patients should have continuous careful monitoring and prompt access to medical care, including cardiac surgery, in the event of complications.

**Endocarditis Prophylaxis Recommended**

Cardiac conditions.

Prosthetic cardiac valves (including bioprosthetic, homograft, and mechanical).

Previous episode of bacterial endocarditis.

Most congenital cardiac defects (especially cyanotic congenital heart disease, patent ductus arteriosus, ventricular septal defects, and surgically repaired intracardiac defects with residual hemodynamic abnormalities).

Valvular heart disease resulting from rheumatic or other disease (aortic regurgitation and stenosis, mitral regurgitation and stenosis).

Hypertrophic cardiomyopathy.

Mitral valve prolapse with regurgitation.

Dental or surgical procedures.

Dental or surgical procedures that cause gingival or mucosal bleeding, including mechanical dental hygienic procedures.

Tonsillectomy or adenoidectomy.

Surgical procedures involving upper respiratory or gastrointestinal mucosa.

Rigid bronchoscopy.

Sclerotherapy of esophageal varices.

Esophageal dilatation.

Transesophageal echocardiography

Gallbladder surgery

Urethral catheterization or urinary tract surgery if infection present

Prostate surgery

I & D of infected tissue

Vaginal hysterectomy

Vaginal delivery in the presence of infection (chorioamnionitis, etc.)

Endocarditis Prophylaxis Not Recommended

Cardiac conditions.

Previous coronary artery bypass surgery.

Mitral valve prolapse without regurgitation. (If MPV is associated with thickening or redundancy of valve leaflets, may have increased risk of endocarditis, especially in men >45 years of age).

Functional or innocuous heart murmurs.

Cardiac pacemakers and implantable defibrillators.

Isolated secundum atrial septal defect.

6 months or more status postsurgical repair of PDA, VSD without residua.

Previous rheumatic heart disease or Kawasaki disease without valve dysfunction.

Dental or surgical procedures.

Dental procedures not likely to cause gingival bleeding such as fillings above the gum line, adjustment of orthodontic appliances.

Injection of intraoral anesthetics.

Shedding of primary teeth.

Tympanostomy tube insertion.

Endotracheal intubation, flexible bronchoscopy with or without biopsy specimens.

Cardiac catheterization.

Endoscopy with or without biopsy.

In absence of infection, urethral catheterization, D&C, uncomplicated vaginal delivery, abortion, sterilization procedures, insertion or removal of an IUD, or laparoscopy.

**Standard Regimens**

Dental, oral, upper respiratory tract. (Total children’s dose should not exceed adult dose).

For adults. Amoxicillin 2 g (children, 50 mg/kg) PO 1 hour before procedure.

In penicillin-allergic patients. Clindamycin 600 mg (children, 20 mg/kg) PO OR Cephalexin or Cefadroxil 2.0 g (children, 50 mg/kg) PO OR Azithromycin or Clarithromycin 500 mg (children, 15 mg/kg) PO 1 hour before procedure

If unable to take oral medications. Ampicillin 2.0 g (children 20 mg/kg) IV or IM 30 minutes before procedure. Alternative: clindamycin 600 mg (children 20 mg/kg) IV 30 minutes before procedure.

In the high-risk, penicillin-allergic patient. Vancomycin 1.0 g IV over 1 hour, starting 1 hour before surgery. A repeat dose is not necessary.

GI or GU procedures. (Total children’s dose should not exceed adult dose).

High risk. Ampicillin 2.0 g IV (children, 50 mg/kg) + Gentamicin 1.5 mg/kg IV (for adults and children, not to exceed 120 mg) 30 minutes before procedure, then amoxicillin 1.0 g (children, 25 mg/kg) PO 6 hours later, or ampicillin 1.0 g (children, 25 mg/kg) IV 6 hours after first dose.

High-risk, penicillin allergic. Vancomycin 1.0 g (children, 20 mg/kg) IV (over 1 hour) starting 1 hour before procedure + Gentamicin 1.5 mg/kg IV (both adults and children, not to exceed 120 mg) 1 hour before. Complete infusion 30 minutes before procedure.

Moderate or low-risk. Amoxicillin 2.0 g (children, 50 mg/kg) PO 1 hour before procedure. Or, Ampicillin 2.0 g (children 50 mg/kg) IM or IV 30 minutes before procedure.

Moderate or Low-risk, penicillin allergic. Vancomycin 1.0 g (children, 20 mg/kg) over 1 hour. Complete infusion 30 minutes before starting procedure.

**MITRAL STENOSIS**

Mitral stenosis (MS) is a narrowing of the outflow path from the left ventricle. Patients with mitral stenosis typically have mitral valve leaflets that are thickened, commissures that are fused, and chordae tendineae that are thickened and shortened.

**Frequency**

In the US: The prevalence of MS has decreased because of the decline in the occurrence of rheumatic fever in the US and developed countries. The mitral valve is the most commonly affected valve in patients with rheumatic heart disease.

Internationally: Progression of MS tends to be more rapid in underdeveloped areas. Occasionally, patients can become symptomatic when younger than 20 years.

Mortality/Morbidity: Without surgical intervention, the progressive nature of the disease results in an 85% mortality rate 20 years after the onset of symptoms.

Sex: Two thirds of all patients with MS are female.

Age: The onset of symptoms usually is between the third and fourth decades of life.

* The most common cause of mitral stenosis is rheumatic fever.

Less common causes include

* congenital mitral stenosis,
* systemic lupus erythematosus (SLE),
* rheumatoid arthritis (RA),
* atrial myxoma,
* bacterial endocarditis.

**Pathophysiology**

Pure MS develops in approximately 40% of all patients with rheumatic heart disease. There is a latency period of 10-20 years, or more, after an episode of rheumatic fever; therefore, the onset of MS symptoms does not occur until then.

The normal area of the mitral valve orifice is 4-6 cm2. When the area of this orifice is reduced to 2 cm2, an increase in left atrial pressure (LAP) is necessary for normal transmitral flow to occur.

Critical MS occurs when the opening is reduced to 1 cm2. At this stage, an LAP of 25 mm Hg is required to maintain a normal cardiac output. This increase in LAP raises pulmonary venous and capillary pressures, resulting in exertional dyspnea.

As the disease progresses, chronic elevation of LAP leads to pulmonary hypertension, tricuspid and pulmonary incompetence, and eventual right heart failure.

Progressive dilation of the left atrium predisposes a patient to 2 further complications.

**One is the development of mural thrombi**. These thrombi embolize in 20% of patients. Patients at high risk for embolization are those older than 35 years, those with atrial fibrillation (Afib) and a low cardiac output, and those having a large left atrial appendage.

The other significant complication is the **development of Afib**, which occurs in up to 40% of patients. Loss of atrial contraction due to the development of Afib decreases cardiac output by 20%. Since cardiac output is related to the heart rate, Afib with a rapid ventricular response decreases diastolic filling time and further compromises cardiac output.

**CLINICAL**

**History**

* History of acute rheumatic fever, although many patients do not recall this
* History of murmur
* Effort-induced dyspnea
* Most common complaint
* Often triggered by exertion, fever, anemia, onset of Afib, or pregnancy
* Orthopnea, which progresses to paroxysmal nocturnal dyspnea
* Effort-induced fatigue
* Hemoptysis, due to the ruptures of thin dilated bronchial veins (late finding)
* Chest pain due to right ventricular ischemia, concomitant coronary atherosclerosis, or a coronary embolism
* Thromboembolism may be the first symptom of MS.
* Palpitations
* Recumbent cough

**Physical**

* The physical examination findings depend on the advancement of the disease and the degree of underlying cardiac decompensation.
* Peripheral and facial cyanosis
* Jugular venous distention
* Respiratory distress, evidence of pulmonary edema (eg, rales)
* Diastolic thrill that is palpable over the apex
* A loud S1 followed by an S2 and the opening snap are best heard at the left sternal border.
* This is followed by a low-pitched, rumbling, diastolic murmur, which is heard best over the apex while the patient is in the left lateral decubitus position.
* Murmur may diminish in intensity as the stenosis increases.
* The duration, but not the intensity, of the diastolic murmur correlates with the severity of the mitral narrowing.
* The holosystolic murmur of mitral regurgitation may accompany the valvular deformity of MS.
* Digital clubbing
* Systemic embolization
* Signs of right heart failure in severe MS include ascites, hepatomegaly, and peripheral edema.
* If pulmonary hypertension is present, there may be a right ventricular lift; an increased pulmonic second sound; and a high-pitched, decrescendo, diastolic murmur of pulmonary insufficiency (ie, Graham Steell murmur).

**Lab Studies**

Complete blood count (CBC) in cases of hemoptysis and to rule out anemia

Blood culture in cases of suspected endocarditis

**Imaging Studies**

**Echocardiography**

It is the most sensitive and specific noninvasive method for diagnosing mitral stenosis.

With two-dimensional echocardiography, the size of the mitral orifice can be measured along with the cardiac chamber sizes.

The addition of color Doppler can evaluate the transvalvular gradient, pulmonary artery pressure, and accompanying mitral regurgitation.

Transesophageal echocardiography (TEE) is useful for detecting vegetations that are smaller than 5 mm or thrombi in the left atrium, which are not seen with transthoracic echocardiography.

**Chest x-ray**

* Signs of pulmonary overload include prominence of pulmonary arteries, enlargement of right ventricle, and evidence of congestive heart failure (eg, interstitial edema with Kerley B lines).
* Left atrial enlargement with straightening of the left heart border
* Double density
* Elevation of the left mainstem bronchus
* Pulmonary venous pattern changes with redistribution of the flow toward the apices
* Prominent pulmonary arteries at the hilum, then they rapidly taper
* Kerley B lines
* Pulmonary edema pattern, which appears late in the disease

**Electrocardiogram**

When the heart is in sinus rhythm an enlarged left atrium is signified by a broad notched P wave, which is most prominent in lead II, with a negative terminal force in V1.

With severe pulmonary hypertension, right axis deviation and right ventricular hypertrophy can be seen.

Atrial fibrilation is a common but nonspecific finding in MS.

**TREATMENT**

Emergency Department Care:

* Upright posture
* Rate control
* Digitalis is of little, if any, benefit to patients who have MS with cardiac sinus rhythm.
* In patients with Afib, digitalis can be effective in slowing the ventricular rate.
* The addition of a beta-blocker to digitalis may be needed to achieve a ventricular rate of 60-70 beats per minute.
* A calcium channel blocker (eg, diltiazem) may be used in patients with a beta-blocker contraindication.
* Diuresis for signs of pulmonary edema
* Anticoagulation
* Anticoagulation is helpful in preventing thrombus formation and embolization in patients with Afib.
* To minimize the risk of a systemic embolization, a period of 3-4 weeks of anticoagulation, when possible, should precede a chemical or electrical cardioversion.

**Further Inpatient Care**

Cardiac catheterization is the ultimate method for detecting the pressure gradient across the mitral valve, pulmonary artery pressure, associated mitral regurgitation, left ventricular function, and coexistent atherosclerosis. It often is performed preoperatively in elderly patients with a history of angina or signs of severe MS, which are demonstrated clinically and by echocardiography.

Balloon valvulotomy results in a decline in LAP; therefore, a prominent and sustained symptomatic improvement occurs. It most commonly is used in young patients without extensive valvular calcification, in pregnant women, and in patients who are unfavorable operative candidates.

Mitral valve replacement is performed if leaflets are immobile or heavily calcified. It also is performed if severe subvalvular scarring is present. Bioprosthetic or artificial mechanical valves can be used as replacements.

**Prevention**

Bacterial endocarditis prophylaxis for dental and invasive procedures must be continued for life.

Appropriate treatment of streptococcal pharyngitis is needed to reduce the occurrence of rheumatic fever.

Prophylaxis against recurrent streptococcal infection, as well as recurrent rheumatic fever, should be given to patients with a history of rheumatic fever.

**Complications**

* Thromboembolism
* Recurrent rheumatic fever
* Bacterial endocarditis
* Pulmonary hypertension
* Pulmonary edema
* Complications of balloon valvulotomy (eg, stroke, cardiac perforation, development of mitral regurgitation)
* Complications of mitral valve replacement (eg, paravalvular leakage, thromboembolia, infective endocarditis, mechanical dysfunction, bleeding due to anticoagulants)

**Prognosis**

The classic history of mitral stenosis includes the following:

Development of the murmur 10 years after the episode of rheumatic fever

Another 10 years until symptoms develop

Another 10 years before the patient develops serious disability

The operative mortality rate is 1-2% for mitral commissurotomy and 2-5% for mitral valve replacement.

**MITRAL REGURGITATION**

Synonyms: MR, mitral incompetence, mitral insufficiency

Mitral regurgitation (MR) is characterized by an abnormal reversal of blood flow from the left ventricle to the left atrium. MR is one of the most common cardiac valvular lesions, but affected persons may remain asymptomatic for many years. The usual etiologies are myxomatous degeneration, ruptured chordae tendineae, collagen-vascular disease, and rheumatic fever. Advances in MR management have resulted in earlier diagnoses, with timely surgical intervention and proper follow up being key to treatment.

**Frequency**

In the US: MR (acute and chronic) affects approximately 5 in 10,000 people. Mitral valve disease is the second most common valvular lesion, preceded only by aortic stenosis. Myxomatous degeneration has replaced rheumatic heart disease as the leading cause of mitral valvular abnormalities. Mitral valve prolapse has been estimated to be present in 4% of the normal population. With the aid of color Doppler echocardiography, mild MR can be detected in as many as 20% of middle-aged and older adults. MR is independently associated with female sex, lower body mass index, advanced age, renal dysfunction, prior myocardial infarction, prior mitral stenosis, and prior mitral valve prolapse. It is not related to dyslipidemia or diabetes.

Internationally: In areas other than the Western world, rheumatic heart disease is the leading cause of MR.

Natural history studies of patients with rheumatic MR have shown 5- and 10-year survival rates of 80% and 60%, respectively. Overall, the operative mortality rate associated with mitral valve replacement ranges from 5-12%. Independent risk factors for surgical intervention are emergent surgery, previous valve surgery, coronary artery disease, and age. The presence of ischemic MR or concomitant coronary artery disease raises the mortality rate to 16%. The operative mortality rate for mitral valve repair is lower than 5%.

**Causes**

**Acute MR**

* Ruptured chordae or papillary muscle due to acute myocardial infarction or trauma
* Perforation of the mitral valve leaflet
* Acute failure of a prosthetic valve

**Chronic MR**

* Mitral valve prolapse
* Rheumatic heart disease
* Coronary artery disease
* Annular calcification
* Connective-tissue disorder
* LV dilatation
* MVP (ie, myxomatous degeneration) accounts for approximately 45% of the cases of mitral regurgitation in the Western world

**Pathophysiology**

The mitral or bicuspid atrioventricular valve is located between the left atrium and the left ventricle of the heart and is a fibrous structure lined by endocardium. The mitral valve is composed of the mitral annulus, the leaflets (a large anterior [aortic] leaflet and a small posterior [mural] leaflet), the chordae tendineae, and the papillary muscles. Abnormalities in any of these structures can cause MR. The leaflets are continuous with each other at their lines of attachment, called commissures, and are tethered to the left ventricle by the chordae tendineae. Chordae tendineae attach to papillary muscles and prevent prolapse of the mitral valve leaflet to prevent reflux of blood into the left atrium.

MR can be caused by

* organic disease (eg, rheumatic fever, ruptured chordae tendineae, leaflet perforation)
* or a functional lesion (ie, a normal valve may regurgitate [leak] because of global annular dilatation, focal myocardial dysfunction, or both).

Congenital MR is rare but is commonly associated with myxomatous mitral valve disease and can be associated with cleft of the mitral valve in persons with Down syndrome.

In acute mitral valve regurgitation, the incompetent mitral valve allows the ventricular ejection fraction to reflux into the left atrium. This volume overload is intensified by the inability of the atrium and ventricle to immediately dilatate, resulting in elevated left atrial and pulmonary venous pressures and acute pulmonary edema. The net reduction in forward stroke volume reduces systemic perfusion, can result in hemodynamic deterioration, and can lead to cardiogenic shock.

In chronic mitral valve regurgitation, the distensibility of the left atrium and ventricle are increased over time. This dilatation of the left atrium decreases left atrial pressures, thus increasing preload. The left ventricle dilatates and, via the process of eccentric hypertrophy, generates a larger stroke volume without a significant rise in wall stress. This results in left ventricular (LV) pressures that remain within the reference range. Because the LV pressure remains in the reference range, LV dilatation can occur without a significant rise in myocardial oxygen demand. The LV dilatation may further prohibit the coaptation of the mitral valve leaflets during systolic ejection, leading to progression of LV dilatation and overload. Thus, patients with compensated MR may remain asymptomatic for years despite the presence of severe volume overload. Ultimately, however, most people with MR decompensate over the long term. Ten years after MR is diagnosed, 90% of patients die or undergo a surgical procedure.

**CLINICAL**

**History**

When associated with coronary artery disease and acute myocardial infarction (typically, inferior myocardial infarction, which may lead to papillary muscle dysfunction), significant acute MR is accompanied by symptoms of impaired LV function, such as dyspnea, fatigue, and orthopnea. In these cases, pulmonary edema is often the initial manifestation because of rapid volume overload on the left atrium and the pulmonary venous system.

Chronic MR often results from a primary defect of the mitral valve apparatus with subsequent progressive enlargement of the left atrium and ventricle. In this state, patients may remain asymptomatic for years. They may have normal exercise tolerance until the gradual impairment of ventricular function causes fatigue because of reduced forward cardiac output. With time, patients may feel chest palpitations if atrial fibrillation develops as a result of chronic atrial dilatation. Patients with LV enlargement and more severe disease eventually progress to symptomatic congestive heart failure with pulmonary congestion and edema. At this stage of LV dilatation, the myocardial dysfunction often becomes irreversible because of the long-standing MR.

**Physical**

* The typical finding associated with MR is **an apical holosystolic murmur,** which radiates **to the left axilla and sternal border**, may be accompanied by a ventricular gallop (signifying LV dysfunction) followed by an early diastolic rumble caused by the large inflow of blood from a dilatated left atrium.

This murmur is caused by the rupture of the mitral valve apparatus and, because of the underlying pathology, varies in intensity and radiation over the precordium.

If the MR is caused by LV dilatation and depressed ventricular contractile function, this murmur may be mid, late, or holosystolic and may be accompanied by the aforementioned LV (S3) gallop. In this setting, the murmur is usually grade II/VI or less.

With acute mitral valve regurgitation, a harsh murmur, usually grade III or IV/VI, is heard and is accompanied by a palpable thrill at the apex of the heart.

**Imaging Studies**

**Chest radiograph**

Evidence of LV enlargement due to volume overload may be observed, although pulmonary congestion, represented by increased pulmonary markings, may not be observed until heart failure has developed.

Left atrial enlargement also may be observed as a prominence along the right sternal border.

**Echocardiogram**

With acute mitral valve regurgitation, a ruptured chorda, a flail valve leaflet, or infective endocarditis may be identified as the etiology.

With chronic mitral valve regurgitation, evidence of calcification of the valve leaflets and annulus may be observed. In addition, a depressed ejection fraction with increased end-diastolic and end-systolic dimensions of the ventricle may be observed. These measurements are used as criteria to identify the optimal time for surgical correction, ie, before significant and irreversible myocardial deterioration occurs.

**Electrocardiogram**

Acute MR is often accompanied by acute myocardial infarction, demonstrated by inferior or posterior wall ischemia.

In chronic mitral valve regurgitation, LV dilatation and hypertrophy are observed with increased QRS voltage and ST-T wave changes in the lateral precordial leads.

Left atrial enlargement in chronic mitral valve regurgitation produces a negative P wave in lead V1, but atrial fibrillation may be observed in the late stages.

**Cardiac catheterization**

Left ventriculography confirms mitral valve regurgitation by demonstrating a flow of contrast into the left atrium. LV end-diastolic and end-systolic dimensions can be measured and used to calculate the ejection fraction, LV mass, and regurgitant volume per beat into the left atrium.

Catheterization can also help detect lesions within the aortic valve, coexistent coronary artery disease through selective coronary artery injection, and other cardiac anomalies such as septal defects.

Finally, catheterization may be used to assess global myocardial function along with the pulmonary capillary wedge pressure.

**DIFFERENTIALS**

Calcified aortic stenosis also produces a prominent murmur at the apex and may be confused with mitral valve regurgitation.

Tricuspid regurgitation also causes a holosystolic murmur at the left lower sternal border, but inspiration accentuates the murmur more than in mitral valve regurgitation.

A ventricular septal defect also mimics the harsh holosystolic murmur heard at the lower left sternal border but generally radiates to the right of the sternum compared with the axillary radiation heard with MR.

**TREATMENT**

Medical Care:

**Prehospital care**

Acute mitral valve regurgitation with hemodynamic compromise is usually associated with coronary artery disease and possible myocardial infarction. Close attention to the electrocardiogram tracings and treatment with supplemental oxygen, analgesics for anginal chest pain, and sublingual nitrates for acute myocardial infarction are the components of prehospital care.

If exacerbation of the chronic mitral valve regurgitation with hemodynamic compromise occurs, acute myocardial infarction, although less likely, must be excluded. Treatment involves diuretics for pulmonary congestion and afterload-reducing agents, such as nitrates, to help forward cardiac output.

**Emergency department care**

Any patient with acute or chronic mitral valve regurgitation with hemodynamic compromise should be evaluated for acute myocardial infarction.

Consultations with specialists in cardiology and cardiothoracic surgery should be obtained early during patient stabilization.

Diuretic therapy is continued for individuals with pulmonary congestion, and an echocardiogram must be performed immediately. These patients must be expeditiously transferred to a cardiac critical care unit for central and pulmonary artery pressure monitoring.

Medical therapy

* Afterload-reducing agents, such as nitrates and antihypertensive drugs, are helpful for maintaining the forward-flow state in persons with mitral valve regurgitation.
* If atrial fibrillation is encountered, digitalis therapy is considered.
* Similar to other valvular diseases, prophylactic antibiotics are administered prior to any interventional treatment.

However, the current American Heart Association guidelines for endocarditis prophylaxis in patients with mitral prolapse indicate that patients with no murmur and normal leaflets are at low risk; therefore, antibiotic prophylaxis is not necessary.

* In late-stage mitral valve regurgitation, heart failure develops; diuretics and inotropic agents are administered, and consultation with a specialist in cardiothoracic surgery is arranged.

The use of balloon counterpulsation should be considered as a preoperative measure.

**Surgical Care**

Indications for surgical Intervention

* Acute MR with congestive heart failure or cardiogenic shock
* Acute endocarditis
* Class III/IV symptoms (ie, patient symptomatic while at rest or with minimal activity)
* Class I/II (few or no) symptoms with evidence of deteriorating LV function as evidenced by

1) an ejection fraction less than 0.55 or (55%),

2) fractional shortening less than 30%,

3) either the end-diastolic diameter approaching 75 mm or the end-systolic diameter approaching 50 mm

* Systemic emboli
* End-systolic volume index greater than 60 mL/m2 - Most commonly used parameter

Surgical options

* Mitral valve reconstruction with mitral annuloplasty, quadratic segmental resection, shortening of the elongated chordae, or posterior leaflet resection
* Mitral valve replacement with either a mechanical valve (requiring lifelong anticoagulation) or a bioprosthetic porcine valve

Complications:

* Medical complications –

1. Pulmonary edema,
2. congestive heart failure,
3. thromboembolism resulting from atrial fibrillation
   * Operative risks include
4. bleeding,
5. intraoperative myocardial infarction,
6. stroke.

Prognosis:

Mechanical prosthetic valves have failure-free rates of approximately 98% per year.

The 5-year survival rate is approximately 55-70% for mitral replacement and 75-85% for mitral valve repair.

**AORTIC STENOSIS**

AS, congenital unicuspid or bicuspid valve, rheumatic fever, degenerative calcific changes of the valve.

Aortic stenosis (AS) is the obstruction of blood flow across the aortic valve. AS has several etiologies: congenital unicuspid or bicuspid valve, rheumatic fever, and degenerative calcific changes of the valve.

**Frequency**

This is a relatively common congenital cardiac defect. Incidence is 4 in 1000 live births.

Mortality/Morbidity: Sudden cardiac death occurs in 3-5% of patients with AS. Adults with AS have a 9% mortality rate per year. Once symptoms develop, the incidence of sudden death increases to 15-20%, with average survival duration of less than 5 years. Patients with exertional angina or syncope survive an average of 3 years. After the development of left ventricular failure, life expectancy is slightly greater than 1 year.

Sex: Among children, 75% of cases of AS are in males.

Age: AS usually is not detected until individuals are school aged. AS exists in up to 2% of those who are younger than 70 years. The etiology of AS in those aged 30-70 years can be rheumatic disease or calcification of a congenital bicuspid valve. In those older than 70 years, degenerative calcification is the primary cause of AS. Among people older than 75 years, 3% have critical AS.

**Causes**

The ventricular pressure required to deliver a certain cardiac output at the required perfusion pressure is the pressure gradient across the valve in systole. This pressure gradient defines the degree of aortic valve obstruction.

Newborns with significant AS develop CHF within the first week of life. The left ventricle is often too small to be compatible with life. The newborn heart develops left-to-right shunting through the patent foramen ovale, which leads to worsening CHF.

Congenital AS caused by a congenital unicuspid or bicuspid aortic valve is usually asymptomatic in the otherwise healthy developing child. It often is diagnosed on routine physical examination, although a child may present with angina pectoris with exercise.

As rheumatic fever decreases in frequency, so does rheumatic fever–induced AS. These patients have a fibrous contracture with shortening of the cusps due to recurrent inflammation from rheumatic carditis. Adjacent cusps tend to fuse at the commissures. This causes a form of acquired unicuspid or bicuspid aortic valve. Calcifications may develop, but the primary cause of stenosis is the adhesions that fuse the cusps. In patients older than 70 years, the most common cause of AS is degenerative calcification of the valve. Calcific AS also occurs in older patients with congenital or acquired bicuspid valves. Congenital bicuspid valves cause calcific AS 4 times more frequently than acquired forms do.

**Pathophysiology**

When the aortic valve becomes stenotic, resistance to systolic ejection occurs and a systolic pressure gradient develops between the left ventricle and the aorta. Stenotic aortic valves have a decreased aperture that leads to a progressive increase in left ventricular systolic pressure. This leads to pressure overload in the left ventricle, which, over time, causes an increase in ventricular wall thickness (ie, concentric hypertrophy). At this stage, the chamber is not dilated and ventricular function is preserved, although diastolic compliance may be affected.

Eventually, however, the left ventricle dilates. This, coupled with a decrease in compliance, is associated with an increase in left ventricular end-diastolic pressure, which is increased further by a rise in atrial systolic pressure. A sustained pressure overload eventually leads to myocardial decompensation. The contractility of the myocardium diminishes, which leads to a decrease in cardiac output. The elevated left ventricular end-diastolic pressure causes a corresponding increase in pulmonary capillary arterial pressures and a decrease in ejection fraction and cardiac output. Ultimately, congestive heart failure (CHF) develops.

**CLINICAL**

**History**

AS usually has an asymptomatic latent period of 10-20 years. Symptoms develop gradually. Ultimately, patients experience the classic triad of chest pain, heart failure, and syncope. Typical symptoms include the following:

* Palpitations
* Fatigue (may be an early symptom among children)
* Visual disturbances
* Gradual decrease in physical activity with insidious progression of fatigue and dyspnea on exertion
* Angina pectoris (30-40%)
* Patients may have a higher incidence of nitroglycerin-induced syncope than the general population.
* Always consider AS as a possible etiology for a patient in the ED with particular hemodynamic sensitivity to nitrates.
* Syncope during exertion: Proposed mechanisms include arrhythmias and left ventricular failure with an abrupt decline in cardiac output.
* Symptoms of left ventricular failure (eg, dyspnea on exertion, nocturnal cough, orthopnea, paroxysmal nocturnal dyspnea, hemoptysis) may occur. This is due to an elevation of the pulmonary capillary pressure from left ventricular dilation and reduced compliance.

**Physical examination**

* 1. Palpation reveals a laterally displaced apex reflecting the presence of left ventricular hypertrophy.
  2. A systolic thrill may be palpable at the base of the heart, in the jugular notch, and along the carotid arteries.
  3. Crescendo-decrescendo systolic ejection murmur begins shortly after the first heart sound. The intensity increases toward midsystole, then decreases, and the murmur ends just before the second heart sound. It is generally a rough, low-pitched sound that is loudest at the base of the heart and most commonly is appreciated in the second right intercostal space. An ejection click may be auscultated. This is associated with bicuspid valves.
  4. An audible fourth heart sound indicates the presence of left ventricular hypertrophy in severe AS. Once the left ventricle dilates and fails, a third heart sound may be audible.
  5. Pulsus parvus et tardus: This is an arterial pulse with a delayed and plateaued peak, decreased amplitude, and gradual downslope. A high-pitched, diastolic blowing murmur may be present if the patient has associated aortic regurgitation.

**Imaging Studies**

1. **Chest x-ray**

Chest radiographs may show cardiac enlargement. Minimal enlargement and more subtle signs of concentric hypertrophy without dilatation are present, including mildly enlarged heart size, rounding at the cardiac apex, and slight backward displacement of the heart as seen in lateral view.

In later, more severe stages of AS, roentgenographic signs of left atrial enlargement, pulmonary artery enlargement, right-sided enlargement, and pulmonary congestion are evident.

1. **Echocardiograph**

Two-dimensional transthoracic echocardiography can confirm the clinical diagnosis of AS and provide specific data on left ventricular function. It can show the structure and function of the other valves as well.

The following 3 significant findings can help define the severity of the disease and describe the current hemodynamic significance:

* An echo-dense aortic valve with no cusp motion is indicative of severe AS. This may be unreliable in congenital or rheumatic valvular stenosis.
* A decrease in the maximal aortic cusp separation (<8 mm in the adult) is also indicative of severe AS.
* The presence of otherwise unexplained left ventricular hypertrophy implies significant AS.

Utilizing echo-Doppler techniques, the systolic pressure gradient across the aortic valve can be assessed. Doppler techniques also can help visualize any mitral or aortic regurgitation that might be present.

1. **Electrocardiogram**

Generally, ECG is not a reliable test because of the wide variations seen in AS and other cardiac conditions.

An ECG of a patient with significant AS most likely shows evidence ***of left ventricular hypertrophy*** with or without a strain pattern.

Approximately 25% of patients with significant AS may not show clear ECG evidence of ventricular hypertrophy. This population includes the elderly who have significant myocardial fibrosis and adolescents who may experience ST-segment changes before QRS changes.

Of patients with significant AS, 13% have conduction defects seen on ECG.

These can include

* + first-degree heart block,
  + left bundle-branch block,
  + any other conduction defects.

The presence of left atrial enlargement suggests an associated mitral valve process.

1. **Cardiac catheterization and coronary arteriography**

Perform cardiac catheterization and coronary arteriography on patients who may have surgery, are suspected of having coronary artery disease, or are older than 40 years (even without significant symptoms).

These patients have a 50% incidence of underlying coronary artery disease. This is a significant consideration if the patient may have surgical intervention.

Other considerations in the complete workup of AS include radionuclide studies to evaluate myocardial perfusion at rest and during exertion and exercise studies. Perform these tests cautiously on symptomatic patients. Surgery is recommended routinely in patients with a valve cross-sectional area of 0.8-1.9 cm. However, with improvements in aortic valve replacement, this parameter is becoming more liberal.

**TREATMENT**

* **Emergency Department Care**

Prehospital and ED management is focused on acute exacerbations of the symptoms of AS.

As always, assess and address airway, breathing, and circulation.

A patient presenting with uncontrolled CHF should be treated

* supportively with oxygen,
* cardiac and oximetry monitoring,
* intravenous access,
* loop diuretics,
* nitrates (remembering the potential nitrate sensitivity of patients with AS),
* morphine (as needed and tolerated),
* noninvasive or invasive ventilatory support (as indicated).

Diagnostic studies in the ED should include ECG, chest radiograph, serum electrolytes, cardiac enzymes, CBC, and arterial blood gases (if hypoxemia or a mixed respiratory disease state is suspected). Emergency formal ultrasound may be useful in centers that have this capability.

Vasodilators should be used judiciously in patients with AS, as they may cause a significant drop in blood pressure.

Patients with heart failure due to AS that is resistant to medical management should be considered for emergent surgery.

A patient presenting with angina pectoris requires monitoring and studies as listed above. Measures should be taken to relieve the chest discomfort. This may include administration of nitrates, oxygen, and morphine.

Nitroglycerin-induced syncope occurs more often in patients with AS than in those without AS. This information should be obtained through the history at presentation.

Syncope in the face of AS should be assessed and treated as in any patient presenting with a syncopal episode.

Atrial fibrillation in the setting of AS is considered a medical emergency and should be converted urgently in patients who are hemodynamically unstable. Associated symptoms also should be treated urgently.

**AORTIC REGURGITATION**

Aortic regurgitation is the diastolic flow of blood from the aorta into the left ventricle. Regurgitation is due to incompetence of the aortic valve or any disturbance of the valvular apparatus (eg, leaflets, annulus of the aorta) resulting in diastolic flow of blood into the left ventricular chamber.

**Frequency**

Rheumatic fever and syphilis used to be major causes of aortic regurgitation, but these diseases have diminished in recent years because of the introduction of new antibiotics.

Three fourths of patients with significant aortic regurgitation survive 5 years after diagnosis; half survive for 10 years. Patients with mild-to-moderate regurgitation survive 10 years in 80-95% of the cases.

Average survival after onset of congestive heart failure (CHF) is less than 2 years.

Acute aortic regurgitation is associated with significant morbidity, which can progress from pulmonary edema to refractory heart failure and cardiogenic shock.

Chronic aortic regurgitation often begins in the late 50s and is documented most frequently in patients older than 80 years.

**Causes**

Multiple causes of this valvular abnormality are known, including connective tissue disease and anatomic abnormalities. Acute aortic regurgitation is usually due to aortic dissection, bacterial endocarditis, or trauma, which may be either penetrating or blunt.

* Acute aortic regurgitation

Rheumatic

Infective endocarditis

Ruptured sinus of Valsalva

Trauma, prosthetic valve surgery

Aortic dissection, laceration of the aorta

* Chronic aortic regurgitation

Rheumatic

Infective endocarditis

Syphilis

Aortitis (ie, Takayasu disease)

Marfan syndrome

Bicuspid aortic valve, defect of the interventricular septum or sinus of Valsalva

ing spondylitis

Reiter syndrome

Hypertension

**Pathophysiology**

Incompetent closure of the aortic valve can result from intrinsic disease of the cusp, diseases of the aorta, or trauma. Aortic regurgitation may be a chronic disease process or it may occur acutely, presenting as heart failure. The most common cause of chronic aortic regurgitation used to be rheumatic heart disease, but presently it is most commonly bacterial endocarditis. In developed countries, it is caused by dilatation of the ascending aorta (eg, aortic root disease, aortoannular ectasia).

Diastolic reflux through the aortic valve can lead to left ventricular volume overload. The severity of the aortic regurgitation is dependent on the diastolic valve area, the diastolic pressure gradient between the aorta and left ventricle, and the duration of diastole. An increase in systolic stroke volume and low diastolic aortic pressure produces an increased pulse pressure.

**CLINICAL**

**History:**

* General

The clinical signs of aortic regurgitation are caused by forward and backward flow of blood across the aortic valve, leading to increased stroke volume.

The degree of regurgitation is determined by the degree of valvular incompetence; left ventricular compliance; and end-ventricular, end-diastolic volume.

Acute aortic regurgitation: Symptoms are manifestations of cardiovascular collapse.

* Weakness
* Severe dyspnea
* Hypotension
* Angina
* Chronic aortic regurgitation
* Exertional dyspnea
* Nocturnal dyspnea
* Orthopnea
* Diaphoresis
* Abdominal discomfort
* Uncomfortable awareness of heartbeat
* Palpitations

**Physical**

The hallmark of aortic regurgitation/insufficiency is a high-pitched decrescendo diastolic murmur at the left sternal border after the second heart sound.

* Acute aortic regurgitation

Patients who have CHF or shock associated with severe aortic regurgitation often appear gravely ill.

Tachycardia

Peripheral vasoconstriction

Cyanosis

Pulmonary edema

Arterial pulsus alternans; normal left ventricular impulse

Early diastolic murmur (lower pitched and shorter than in chronic aortic regurgitation) may be present. An Austin-Flint murmur, which is caused by the regurgitant flow causing vibration of the mitral apparatus, is lower pitched and short in duration. The decrescendo diastolic murmur is heard best with the patient leaning forward in full expiration in a quiet room. It is the cardiac murmur most commonly missed.

A murmur at the right sternal border is associated more often with dissection than any other cause of aortic regurgitation.

* Chronic aortic regurgitation

All auscultatory phenomena indicate vasodilatation of peripheral circulation.

Hyperdynamic apical impulse displaced laterally and inferiorly may be associated with an ejection click.

Decrescendo diastolic murmur is heard best while the patient is leaning forward on deep expiration.

Apical middiastolic rumble

Austin-Flint murmur

Pulsus bisferiens; increased pulse pressure; visible, forceful, and bounding peripheral pulses (water hammer)

Corrigan pulse - Quickly collapsing pulses

Musset sign - Bobbing of the head

Quincke sign - Capillary pulsations of the nail bed

Muller sign - Pulsations of the uvula

Hill sign - Systolic pressure in lower extremity greater than systolic pressure in upper extremity by at least 100 mm Hg

Traube sign - Loud systolic sound over femoral arteries

Duroziez sign - Systolic-diastolic murmur produced by compression of femoral artery with a stethoscope

**Lab Studies**

CBC

Prothrombin time (PT)/activated partial thromboplastin time (aPPT)

Type and screen

Electrolytes

Myocardial muscle creatine kinase isoenzyme (CK-MB)

Lactate dehydrogenase panel

Isoenzymes

**Imaging Studies**

1. Chest x-ray

Acute aortic regurgitation

* + Minimal cardiac enlargement
  + Normal aortic root/arch
  + Pulmonary venous pattern increased

Chronic aortic regurgitation

* Marked cardiac enlargement
* Prominent aortic root/arch
* Normal pulmonary venous pattern

2. ECG

* Normal (early in disease)
* Left axis deviation (chronic aortic regurgitation)
* Specific waves
* Prominent Q wave in I, AVF, V3 to V6
* Small R wave in V1
* T wave inverted with ST-segment depression
* P-R prolongation (possible)

3. 2-Dimensional echocardiogram, transesophageal

Acute aortic regurgitation

* Valve anatomy disrupted
* Intimal flap
* Vegetations on valve
* Pericardial effusion

Chronic aortic regurgitation

* Valve anatomy disrupted
* Estimation of degree of regurgitation
* Aortic root size and anatomy
* Left ventricular function

4. Radionuclide techniques

These allow for determination of regurgitant fraction and left ventricular/right ventricular stroke-volume ratio. In the absence of mitral regurgitation and tricuspid regurgitation, a left ventricular/right ventricular stroke-volume ratio of 2.5 or more denotes severe aortic regurgitation.

Demonstration of a fall in ejection fraction with exercise is one of the best indicators for surgery in patients who are asymptomatic.

5. Cardiac catheterization/angiography

Consider for patients with coronary artery disease who are possible candidates for aortic valve replacement, those with a complex lesion associated with a diastolic murmur of unknown cause, and those with left ventricular dysfunction out of proportion to the degree of aortic regurgitation.

Assess the anatomy of the aorta and coronary ostia. Findings are usually normal except for visible reflux of dye from the aortic root into the ventricle.

**TREATMENT**

* Emergency Department Care

General

Provide adequate airway management. Intubate when necessary.

Consider prompt surgical intervention in acute aortic regurgitation.

Acute aortic regurgitation

* Administer a positive inotrope (eg, dopamine, dobutamine) and a vasodilator (eg, nitroprusside). Rarely, administration of cardiac glycosides (eg, digoxin) for rate control may be necessary.
* Avoid beta-blockers in the acute setting.
* Administration of vasodilators may be appropriate to improve systolic function and to decrease afterload.

Chronic aortic regurgitation

* Consider antibiotic prophylaxis for patients with endocarditis when performing procedures likely to result in bacteremia.
* Administration of pressors and/or vasodilators may be appropriate.
* Hemodynamically significant aortic regurgitation may require surgical intervention according to the following criteria:
* End-systolic diameter >55 mm
* End-diastolic radius to myocardial wall thickness ratio >4.0
* Ejection fraction <0.45

Consultations:

Cardiothoracic surgery

**TRICUSPID STENOSIS**

Tricuspid valve dysfunction can result from morphological alterations in the valve or from functional aberrations of the myocardium. Tricuspid stenosis is almost always rheumatic in origin and is generally accompanied by mitral stenosis.

Most stenotic tricuspid valves are associated with clinical evidence of regurgitation that can be documented by performing a physical examination (murmur), echocardiography, or angiography. Stenotic tricuspid valves are always anatomically abnormal, and the cause is limited to a few conditions. With the exceptions of congenital causes or active infective endocarditis, tricuspid stenosis takes years to develop.

**Frequency**

Tricuspid stenosis is rare, occurring in less than 1% of the population. While found in approximately 15% of patients with rheumatic heart disease at autopsy, it is estimated to be clinically significant in only 5% of these patients. The incidence of the congenital form of the disease is less than 1%.

Tricuspid stenosis is found in approximately 3% of the international population. It is more prevalent in areas with a high incidence of rheumatic fever. The congenital form of the disease is rare and true incidence is not available.

The mortality associated with tricuspid stenosis depends on the precipitating cause. The general mortality rate is approximately 5%.

Tricuspid stenosis is observed more commonly in women than in men, similar to mitral stenosis of rheumatic origin.

Tricuspid stenosis can present as a congenital lesion or later in life, precipitated by some other condition. The congenital frequency rate of the lesion is approximately 0.3% of all congenital heart disease cases. The frequency rate in the older population, due to secondary causes, ranges from 0.3-3.2%.

**Causes**

At least 4 conditions can cause obstruction of the native tricuspid valve.

These include

(1) rheumatic heart disease,

(2) congenital abnormalities,

(3) metabolic or enzymatic abnormalities,

(4) active infective endocarditis.

Rheumatic tricuspid stenosis: In this entity, diffuse and fibrous thickening of the leaflets occurs, with fusion of 2 and usually 3 commissures. The chordae tendineae may be thickened and shortened. Calcification of the valve rarely occurs. The leaflet tissue is composed of dense collagen and elastic fibers that produce a major distortion of the normal leaflet layers.

Carcinoid heart disease: Carcinoid valve lesions characteristically manifest as fibrous white plaques located on the valvular and mural endocardium. The valve leaflets are thickened, rigid, and reduced in area. Fibrous tissue proliferation is present on the atrial and ventricular surfaces of the valve structure.

Congenital tricuspid stenosis: These lesions are observed more commonly in infants. They may manifest as incompletely developed leaflets, shortened or malformed chordae, small annuli, abnormal size and number of the papillary muscles, or any combination of these defects.

Infective endocarditis: Large infected vegetations obstructing the orifice of the tricuspid valve may produce stenosis. This condition is relatively uncommon, even in those who abuse intravenous drugs.

Unusual causes: Rare causes of tricuspid stenosis include Fabry disease and giant blood cysts.

Mimickers of tricuspid stenosis: Several conditions may mimic tricuspid stenosis by obstructing flow through the valve, including supravalvular obstruction from congenital diaphragms, intracardiac or extracardiac tumors, thrombosis or emboli, or large endocarditis vegetations. In addition, conditions that impair right-sided filling can reproduce historical and physical findings such as constrictive pericarditis and restrictive cardiomyopathy.

**Pathophysiology**

Tricuspid stenosis results from alterations in the structure of the tricuspid valve that precipitate inadequate excursion of the valve leaflets. The most common etiology is rheumatic fever, and tricuspid valve involvement occurs universally with mitral and aortic valve involvement. In this case, the valve leaflets become thickened and sclerotic as the chordae tendineae become shortened. This restrictive process hampers blood flow into the right ventricle and, subsequently, to the pulmonary vasculature. The obstructed venous return results in hepatic enlargement, decreased pulmonary blood flow, and peripheral edema. Right atrial enlargement is observed as a consequence. Other rare causes include carcinoid syndrome, endocarditis, endomyocardial fibrosis, lupus, and congenital tricuspid atresia.

In the rare instances of congenital tricuspid stenosis, the valve leaflets may manifest various forms of deformity, which can include deformed leaflets, deformed chordae, and displacement of the entire valve apparatus. In the congenital form of the disease, other cardiac anomalies are usually present.

**CLINICAL**

**History**

Fatigue, due to limited cardiac output through the stenosis, may be present. Systemic venous congestion leads to abdominal complaints of discomfort and swelling. Onset is usually gradual, but it can be rapidly increased by the development of atrial fibrillation or flutter. Dyspnea may be present, but it is not severe except with concomitant mitral disease. Patients may complain about prominent pulsations in the neck.

When tricuspid stenosis becomes significant in the presence of mitral stenosis, the decrement of cardiac output to the pulmonary bed may paradoxically diminish dyspnea, hemoptysis, and orthopnea.

Obtain information regarding streptococcal infections, symptoms of the carcinoid syndrome, and possible congenital abnormalities.

**Physical**

With sinus rhythm (more common with tricuspid stenosis than with mitral stenosis), the jugular venous pulse increases and the a wave is prominent (may be confused with arterial pulse). If atrial fibrillation occurs, the a wave is lost. Peripheral edema and ascites are frequent. Without significant mitral pathology, the patient should not be dyspneic and can probably lie flat.

A prominent right atrium may be palpable to the right of the sternum. If not obscured by mitral stenosis sounds, a tricuspid opening snap may be heard. A diastolic murmur along the left sternal border or at the xiphoid will increase with inspiration. Often, tricuspid regurgitation is also present, represented by a systolic murmur in a similar location.

The first heart sound may be split widely. The second heart sound may be single. This single sound is due to the inaudible closure of the pulmonary valve from the decrease in blood flow through the stenotic tricuspid valve.

**Lab Studies**

Complete blood cell count: If the white blood cell count is elevated, infection is present. A disproportionately high hemoglobin (polycythemia) level may be indicative of poor pulmonary blood flow.

Complete chemistry profile: The results of this test may help delineate metabolic abnormalities associated with certain inborn errors of metabolism.

**Imaging Studies**

Chest radiograph: Cardiac size may range from normal to enlarged (ie, cardiomegaly). Right atrial enlargement may be prominent. Findings specific to a particular associated congenital heart disease may also be seen.

Echocardiography: This test has become the procedure of choice for the diagnosis of valvular disorders. The test results help delineate the structure of the tricuspid valve and any other intracardiac pathology that may contribute to the pathophysiology of the process.

Electrocardiogram: Arrhythmias are frequent in this patient population. Because of the enlargement of the right atrium, the presence of atrial flutter and/or fibrillation should not be surprising. In sinus rhythm, right atrial enlargement or abnormality (tall P waves on inferior leads) may be noted.

**Procedures**

Cardiac catheterization: This may be required prior to surgery in older patients to assess for possible coronary artery disease. Right heart catheterization can help determine the gradient across the valve and valve area (ie, severity of stenosis) and can help delineate associated congenital defects (eg, septal defects, shunts, anomalous veins) if present. Assessment of aortic and mitral valves via left heart catheterization is useful in patients with rheumatic disease.

Histologic Findings: Most commonly, stenotic tricuspid valves are secondary to rheumatic fever. These generally demonstrate fibrous tissue proliferation without calcium deposits. The leaflet tissue is composed of dense collagen and elastic fibers, producing major distortions of the normal leaflet layers. Congenitally abnormal valves can show a wide spectrum of incompletely developed leaflets, abnormal chordae tendineae, or dysplastic papillary muscles.

**TREATMENT**

Medical Care: In the treatment of tricuspid stenosis, medical care consists of assessment and treatment of the underlying cause of the valvular pathology.

Treat bacterial endocarditis with the appropriate antibiotics as determined by the sensitivity of the organisms cultured.

Medically address cardiac arrhythmias depending on their characterization.

Decreasing volume overload with diuresis and salt restriction helps decrease symptoms and improve hepatic function.

Surgical Care: Tricuspid stenosis remains a surgical disease and requires either commissurotomy or replacement of the valve if right heart failure or low cardiac output has resulted. Surgery is rarely performed solely on the tricuspid valve. It is usually performed in combination with mitral and/or aortic valve disease repair.

With tricuspid valve replacement, the risk of thrombosis is significant and many surgeons advise warfarin therapy for either mechanical or bioprosthetic valve placement.

Percutaneous balloon valvuloplasty has been used successfully, as long as concomitant regurgitation is not significant.

The therapy chosen depends on the structure of the valve and the degree of deformity encountered.

When possible, excise intracavitary pathology, whether it be tumors or other structural abnormalities.

Redundant portions of the dilated right atrium can be excised during the same procedure for restoring the atrium back to normal size.

Diet:

No specific dietary restrictions are necessary before therapy.

Fluid and sodium restriction is prudent if signs of venous congestion are present.

If a valve replacement is undertaken and the patient must be anticoagulated, dietary instructions must be provided regarding those foods that interfere with anticoagulation and are rich in vitamin K.

Activity:

Activity is usually self-limited by the patient because of easy fatigability secondary to oxygen deprivation.

Once the pathology has been corrected, no activity restrictions are necessary.

Causes: Pure tricuspid regurgitation can be caused by at least 9 conditions.

Rheumatic heart disease

Tricuspid regurgitation secondary to rheumatic involvement is usually associated with mitral valve pathology.

The valve develops diffuse fibrous thickening without commisural fusion, fused chordae, or calcific deposits.

Occasionally, the chordae may be mildly thickened by fibrous tissue. Rheumatic disease is the most common cause of pure tricuspid regurgitation due to deformation of the leaflets.

Endocarditis

This is an important cause of pure tricuspid regurgitation. Precipitating factors that can contribute to infection of the valve include alcoholism, opiate addiction, neoplasms, infected indwelling catheters, extensive burns, and hereditary immune deficiency disease.

The clinical presentation is that of pneumonia from septic emboli rather than CHF. Heart murmurs are frequently absent. Annular abscesses are not uncommon.

Ebstein anomaly

This entity is a congenital malformation of the tricuspid valve characterized by annular insertion of the septal and posterior leaflets displaced apically and atrialization of a portion of the ventricular myocardium.

Prognosis for these patients depends upon the degree of apical displacement of the tricuspid annulus and the severity of the regurgitation.

Prolapse (floppy, redundant)

The incidence of floppy tricuspid valve varies from 0.3-3.2%.

The lesion appears to be associated with prolapse of the mitral valve and does not appear to occur in an isolated fashion.

Histological examination of the floppy tricuspid valve shows alterations on the valve spongiosa.

Carcinoid

Pure tricuspid regurgitation can occur as part of the carcinoid heart syndrome.

Fibrous white plaques form on the ventricular aspect of the tricuspid valve and endocardium, causing the valve to adhere to the RV wall.

Proper systolic coaptation does not occur, resulting in tricuspid regurgitation.

Papillary muscle dysfunction

Papillary muscle dysfunction may result from necrosis (secondary to myocardial infarction), fibrosis, or infiltrative processes.

Although dysfunction secondary to myocardial infarction is less common than the same pathology observed with the mitral valve, the underlying cause must be determined in order to plan treatment.

Trauma

Occasionally, trauma to the right ventricle may damage the structures of the tricuspid valve, resulting in insufficiency of the structure.

More commonly associated with stab wounds, projectile destruction of the valve can also occur.

Connective-tissue diseases

Patients with Marfan syndrome or other connective-tissue diseases (eg, osteogenesis imperfecta, Ehlers-Danlos syndrome) may have pure tricuspid regurgitation.

Other valvular dysfunction is also observed in the same patient.

The etiology of the regurgitation can be attributed to a floppy tricuspid valve and a mildly dilated tricuspid valve annulus.

Anatomically normal tricuspid valves

A common etiology of tricuspid regurgitation is dilatation of the RV cavity.

The valve structures are normal; however, because of enlargement of the cavity and dilatation of the annulus, proper coaptation of the leaflets is not possible.

**TRICUSPID REGURGITATION**

The causes of pure tricuspid regurgitation are multiple, and this lesion is the fifth most frequently excised native cardiac valve in patients older than 15 years. Tricuspid regurgitation may result from structural alterations of any one or all of the components of the tricuspid valve apparatus. Components include the leaflets, chordae tendinea, annulus, and papillary muscles or adjacent right ventricular (RV) muscle. The lesion may be classified as primary when it is caused by an intrinsic abnormality of the valve apparatus or as secondary when it is caused by RV pressure or volume overload.

**Frequency**

In the US: Incidence appears to be 0.9%.

Internationally: Incidence appears to be less than 1%.

The morbidity and mortality of the disease process are secondary to the underlying cause. In rheumatic disease, mortality rates with treatment are less than 3%. In Ebstein anomaly, mortality depends upon the severity of the deformity and the feasibility of correction. Mortality rates with correction are approximately 10%. Tricuspid regurgitation resulting from myocardial dysfunction or dilatation has a mortality rate of up to 50% at 5 years.

Ebstein anomaly can be detected at birth and during early childhood. In patients older than 15 years, the most common form of tricuspid regurgitation is rheumatic valvular disease. In the adult population, other predisposing factors, including carcinoid, bacterial endocarditis, and CHF, take precedence.

**Causes**

Pure tricuspid regurgitation can be caused by at least 9 conditions.

* Rheumatic heart disease
* Tricuspid regurgitation secondary to rheumatic involvement is usually associated with mitral valve pathology.
* The valve develops diffuse fibrous thickening without commisural fusion, fused chordae, or calcific deposits.
* Occasionally, the chordae may be mildly thickened by fibrous tissue. Rheumatic disease is the most common cause of pure tricuspid regurgitation due to deformation of the leaflets.
* Endocarditis
* Ebstein anomaly
* Prolapse (floppy, redundant)
* Carcinoid
* Trauma
* Connective-tissue diseases

**Pathophysiology**

The pathophysiology of tricuspid regurgitation focuses on the structural incompetence of the valve. The incompetent nature of the valve can result from primary structural abnormalities of the leaflets and chordae or from secondary myocardial dysfunction and dilatation.

Tricuspid valve insufficiency is generally found in combination with tricuspid stenosis. The most common cause of this problem appears to be rheumatic in origin. Ebstein anomaly accounts for the most common congenital form of this abnormality.

In tricuspid regurgitation, chronic right atrial overload results in right-sided congestive heart failure (CHF) manifested by hepatic congestion, peripheral edema, and ascites. Because of the impedence of flow to the pulmonary vasculature, hypoxemia, cyanosis, and polycythemia may result.

**CLINICAL**

**History**

The patient with tricuspid regurgitation presents with the signs and symptoms of right-sided heart failure. The spectrum of presenting symptoms is dependent upon whether the condition is secondary to left ventricular (LV) dysfunction. If it is, dyspnea on exertion and paroxysmal nocturnal dyspnea accompany ascites and peripheral edema as common presenting complaints. Exercise intolerance and hypoxemia may also be observed. The patient rarely reports angina, which may be present in the absence of coronary artery disease secondary to RV overload and strain.

These patients must be questioned regarding drug use and history of rheumatic fever and febrile episodes because bacterial endocarditis is a common cause of tricuspid valvular disease.

**Physical**

* S3 gallop is present, and the following physical findings may be found:
* Jugular venous distention with a prominent V wave: When present, a pansystolic murmur is heard along the lower left sternal border with inspiratory accentuation.
* Diminished peripheral pulse volume secondary to impaired forward blood flow: Patients with this sign may have relative hypotension secondary to therapeutic interventions used to decrease volume overload.
* Pulmonary rales associated with LV dysfunction or mitral stenosis
* RV heave and gallop that increases with inspiration
* Ascites
* Peripheral edema
* Cachexia, cyanosis, and jaundice
* Atrial fibrillation
* A high-pitched pansystolic murmur (loudest in the fourth intercostal space in the parasternal region): The murmur is usually augmented during inspiration and is reduced in intensity and duration in the standing position and during a Valsalva maneuver. A short, early diastolic flow rumble may be present.

**Lab Studies**

Chemistry findings may show abnormal liver function and hyperbilirubinemia secondary to liver congestion.

**Imaging Studies**

* Chest radiography

Marked cardiomegaly is evident.

Evidence of elevated right atrial pressure may include distention of the azygous vein and pleural effusions.

Ascites with diaphragmatic elevation may be present.

Pulmonary arterial and venous hypertension is common.

Echocardiography

The right ventricle is dilated.

Paradoxical motion of the ventricular septum is observed and is similar to that found in an atrial septal defect.

Delayed closure of the tricuspid valve is observed.

Prolapse of the tricuspid valve may be evident, as well as vegetations if endocarditis is present.

* Electrocardiography

Findings are usually nonspecific.

Incomplete right bundle branch block, Q waves in lead V1, and atrial fibrillation are found.

**Procedures**

Cardiac catheterization

Right atrial pressure and RV end-diastolic pressure are elevated. A rise or no change in right atrial pressure on deep inspiration is characteristic of tricuspid regurgitation.

The use of angiography in this setting is controversial.

**TREATMENT**

Medical Care

For patients in whom tricuspid regurgitation is secondary to left-sided heart failure, treatment centers on adequate control of fluid overload and failure symptoms. Diuretic therapy with interventions to address the primary pathology is of paramount importance.

Surgical Care

Surgical intervention is indicated when structural deformity of the valve (eg, Ebstein anomaly) exists, when the valve is destroyed by bacterial endocarditis, or when ventricular dilatation is severe and uncontrolled with medical therapy.

Tricuspid regurgitation associated with mitral valve disease and pulmonary hypertension

Assess the severity of the regurgitation by palpation of the valve at the time of mitral valve intervention. Patients with mild tricuspid regurgitation do not require intervention.

As pulmonary vascular pressures fall with successful mitral valve therapy, the tricuspid regurgitation tends to disappear.

Severe regurgitation has been successfully treated with tricuspid annuloplasty.

Organic disease of the tricuspid valve

Corrective measures for organic disease of the tricuspid valve usually involve valve replacement. Because of the increased incidence of prosthetic valve thrombosis in this low-flow position, a porcine heterograft is the valve of choice.

Tricuspid valve replacement has been used in carcinoid heart disease and cardiogenic shock with RV infarction, and after cardiac transplantation.

Tricuspid valve endocarditis

Total excision of the tricuspid valve without immediate replacement is recommended and is well tolerated.

Diseased valvular tissue is excised to eradicate the endocarditis, and antibiotic treatment is continued. Most patients tolerate loss of the tricuspid valve well.

If medical management does not control the tricuspid regurgitation well and the infections have been controlled, an artificial valve can be inserted.

Ebstein anomaly: If this anomaly produces uncontrollable tricuspid regurgitation, then tricuspid valve replacement is necessary.

**Further Inpatient Care**

Inpatient care requires control of any heart failure and treatment of any infectious process that may have affected the valve. Postoperative care encompasses these principles. If the valve has been replaced, the control of arrhythmias is paramount because patients succumb to this problem even if heart failure has been adequately controlled.

Anticoagulation is generally in order if valve replacement has been undertaken because of the low flow state of the right side of the heart. Maintenance of the INR between 2.5-3 should prove sufficient to prevent thrombosis and embolization.

**Further Outpatient Care**

Patients should be carefully monitored for control of any heart failure. Repeat echocardiography is indicated at 6-month intervals for patients in whom the valve has been removed. Annual echocardiography should be considered in patients whose valve has been replaced.

**In/Out Patient Meds**

Digitalis, diuretics (including potassium-sparing agents), ACE inhibitors, and anticoagulants are all indicated in the care of these patients. Antiarrhythmics are added as needed to control atrial fibrillation.

**Complications**

Complications of tricuspid regurgitation include cardiac cirrhosis, ascites, thrombus formation, and embolization. Complications of operative intervention can include heart block, arrhythmias, thrombosis of the prosthetic valve, and infection.

**Prognosis**

Prognosis in these patients is fair. If the cause of the regurgitation is infection, removal of the valve generally cures the problem, provided that the inciting cause is removed (eg, poor dentition, illicit drug use). For patients with accompanying pulmonary hypertension or cardiac dilatation, the prognosis is directly associated with the prognosis for these problems.

**HEART FAILURE**

Synonyms: congestive heart failure, CHF, myocardial failure, circulatory failure

Heart failure is the pathophysiologic state in which the heart, via an abnormality of cardiac function (detectable or not), fails to pump blood at a rate commensurate with the requirements of the metabolizing tissues and/or pumps only from an abnormally elevated diastolic filling pressure.

Heart failure may be caused by myocardial failure but may also occur in the presence of near-normal cardiac function under conditions of high demand. Heart failure always causes circulatory failure, but the converse is not necessarily the case because various noncardiac conditions (eg, hypovolemic shock, septic shock) can produce circulatory failure in the presence of normal, modestly impaired, or even supranormal cardiac function.

**Frequency**

CHF is a worldwide problem, but few accurate financial data are available. CHF is the fastest-growing clinical cardiac disease entity in the United States, affecting 2% of the population. As discussed elsewhere, the most common cause of CHF in industrialized countries is ischemic cardiomyopathy. Other causes, including Chagas disease, assume a more important role in underdeveloped countries than in the United States.

Despite recent advances in the management of patients with heart failure, morbidity and mortality rates remain high, with an estimated 5-year mortality rate of 50%.

Assigning figures for inpatient mortality rates is difficult because the causes and the severity of heart failure vary considerably. The most recent estimates of inpatient mortality rates indicate that death occurs in up to 5-20% of patients.

Hypoxemia that occurs in decompensated CHF, which may be severe, may result in myocardial ischemia or infarction.

*Race*: The incidence and prevalence of CHF are higher in African Americans, Hispanic persons, Native Americans, and recent immigrants from nonindustrialized nations, Russia, and the former Soviet republics.

The higher prevalence of CHF in African Americans, Hispanic persons, and Native Americans is directly related to the higher incidence and prevalence of hypertension and diabetes. This problem is particularly exacerbated by a lack of access to health care and to substandard preventive health care of the most indigent of these and other groups; many persons within these groups are without adequate health insurance coverage.

The higher incidence and prevalence of CHF among recent immigrants from nonindustrialized nations is largely due to a lack of prior preventive health care and to a lack of treatment or to substandard treatment for common conditions such as hypertension, diabetes, rheumatic fever, and ischemic heart disease.

*Sex*: Men and women have equivalent incidence and prevalence of CHF. CHF in women tends to occur later in life compared to men.

*Age*: The prevalence of CHF increases with age, being most common in individuals older than 65 years. Nonetheless, CHF can occur at any age, depending on the cause.

**Causes**

From a clinical standpoint, it is useful to classify the causes of heart failure into 3 broad categories:

1) underlying causes, comprising structural abnormalities (congenital or acquired) that affect the peripheral and coronary arterial circulation, pericardium, myocardium, or cardiac valves, thus leading to the increased hemodynamic burden or myocardial or coronary insufficiency responsible for heart failure;

2) fundamental causes, comprising the biochemical and physiological mechanisms, through which either an increased hemodynamic burden or a reduction in oxygen delivery to the myocardium results in impairment of myocardial contraction;

3) precipitating causes, including the specific causes or incidents that precipitate heart failure in most episodes of heart failure.

Note that most patients who present with significant heart failure do so because of an inability to provide adequate cardiac output in that setting. This is often a combination of the causes listed above in the setting of an abnormal myocardium. The list of causes responsible for presentation of a patient with a CHF exacerbation is very long, and it is important to search for the proximate cause in order to optimize therapeutic interventions.

Overt heart failure may be precipitated by progression of the underlying heart disease. A previously stable compensated patient may develop heart failure that is clinically apparent for the first time when the intrinsic process has advanced to a critical point, such as with further narrowing of a stenotic aortic valve or mitral valve. Alternatively, decompensation may occur as a result of failure or exhaustion of the compensatory mechanisms but without any change in the load on the heart in patients with persistent severe pressure or volume overload.

Precipitating causes of heart failure

Inappropriate reduction of therapy: The most common cause of decompensation in a previously compensated patient with heart failure is inappropriate reduction in the intensity of treatment, whether dietary sodium restriction, physical activity reduction, drug regimen reduction, or, most commonly, a combination of these measures.

* Arrhythmias
* Tachyarrhythmias, most commonly atrial fibrillation
* Marked bradycardia
* Atrioventricular dissociation
* Abnormal intraventricular conduction
* Systemic infection or development of unrelated illness
* Pulmonary embolism
* Cardiac infection and inflammation
* Myocarditis or infective endocarditis may directly impair myocardial function and exacerbate existing heart disease. The anemia, fever, and tachycardia that frequently accompany these processes are also deleterious.
* In the case of infective endocarditis, the additional valvular damage that ensues may precipitate cardiac decompensation.
* Excessive intake of water and/or sodium
* Administration of cardiac depressants or drugs that cause salt retention
* Development of a second form of heart disease

Patients with one form of underlying heart disease that may be well compensated can develop heart failure when a second form of heart disease ensues.

For example, a patient with chronic hypertension and asymptomatic LV hypertrophy may be asymptomatic until a myocardial infarction develops and precipitates heart failure.

**Pathophysiology**

Inadequate adaptation of the cardiac myocytes to increased wall stress in order to maintain adequate cardiac output following myocardial injury (whether of acute onset or over several months to years, whether a primary disturbance in myocardial contractility or an excessive hemodynamic burden placed on the ventricle, or both), is the inciting event in CHF.

Most important among these adaptations are the

1) Frank-Starling mechanism, in which an increased preload helps to sustain cardiac performance;

2) myocardial hypertrophy with or without cardiac chamber dilatation, in which the mass of contractile tissue is augmented;

3) activation of neurohumoral systems, especially the release of norepinephrine (NE) by adrenergic cardiac nerves, which augments myocardial contractility and the activation of the renin-angiotensin-aldosterone system (RAAS) and other neurohumoral adjustments that act to maintain arterial pressure and perfusion of vital organs.

In acute heart failure, the finite adaptive mechanisms that may be adequate to maintain the overall contractile performance of the heart at relatively normal levels become maladaptive when trying to sustain adequate cardiac performance.

* The primary myocardial response to chronic increased wall stress includes myocyte hypertrophy and remodeling, usually of the eccentric type.
* The reduction of cardiac output following myocardial injury sets into motion a cascade of hemodynamic and neurohormonal derangements that provoke activation of neuroendocrine systems, most notably the above-mentioned adrenergic systems and RAAS. The release of epinephrine (E) and nor epinephrine (NE), along with the vasoactive substances endothelin-1 (ET-1) and vasopressin (V), causes vasoconstriction, which increases afterload, and, via an increase in cyclic adenosine monophosphate (cAMP), causes an increase in cytosolic calcium entry. The increased calcium entry into the myocytes augments myocardial contractility and impairs myocardial relaxation (lusitropy).
* The calcium overload may also induce arrhythmias and lead to sudden death. The increase in afterload and myocardial contractility (known as inotropy) and the impairment in myocardial lusitropy lead to an increase in myocardial energy expenditure and a further decrease in cardiac output. The increase in myocardial energy expenditure leads to myocardial cell death, resulting in heart failure and further reduction in cardiac output, thus starting an accelerating cycle of further increased neurohumoral stimulation and further adverse hemodynamic and myocardial responses as described above.
* In addition, the activation of the RAAS leads to salt and water retention, resulting in increased preload and further increases in myocardial energy expenditure. Increases in renin, mediated by decreased stretch of the glomerular afferent arteriole, reduced delivery of chloride to the macula densa, and increased beta1-adrenergic activity as a response to decreased cardiac output, results in an increase in angiotensin II (Ang II) levels and, in turn, aldosterone levels. This results in stimulation of release of aldosterone. Ang II, along with ET-1, is crucial in maintaining effective intravascular homeostasis mediated by vasoconstriction and aldosterone-induced salt and water retention.

**CLINICAL**

1. History

Breathlessness, a cardinal sign of LV failure, may manifest with progressively increasing severity as

1) exertional dyspnea,

2) orthopnea,

3) paroxysmal nocturnal dyspnea,

4) dyspnea at rest,

5) acute pulmonary edema.

The New York Heart Association (NYHA) Classification of Heart Failure, which varies slightly from the above categorization of CHF symptoms, is widely used in practice and in clinical studies to quantify clinical assessment of CHF.

***1. Exertional dyspnea***

The principal difference between exertional dyspnea in subjects who are healthy and exertional dyspnea in patients with heart failure is the degree of activity necessary to induce the symptom. As heart failure first develops, exertional dyspnea may simply appear to be an aggravation of the breathlessness that occurs in healthy persons during activity.

As LV failure advances, the intensity of exercise resulting in breathlessness progressively declines; however, subjective exercise capacity and objective measures of LV performance at rest in patients with heart failure are not closely correlated. Exertional dyspnea, in fact, may be absent in sedentary patients.

***2. Orthopnea***

This early symptom of CHF may be defined as dyspnea that develops in the recumbent position and is relieved with elevation of the head with pillows. As in the case of exertional dyspnea, the change in the number of pillows required is important.

In the recumbent position, decreased pooling of blood in the lower extremities and abdomen occurs. Blood is displaced from the extrathoracic to the thoracic compartment. The failing LV, operating on the flat portion of the Starling curve, cannot accept and pump out the extra volume of blood delivered to it without dilating. As a result, pulmonary venous and capillary pressures rise further, causing interstitial pulmonary edema, reduced pulmonary compliance, increased airway resistance, and dyspnea.

***3. Paroxysmal nocturnal dyspnea***

Attacks of paroxysmal nocturnal dyspnea usually occur at night. This symptom of CHF is defined by a sudden awakening of the patient, after a couple hours of sleep, with a feeling of severe anxiety, breathlessness, and suffocation. The patient may bolt upright in bed and gasp for breath. Bronchospasm increases ventilatory difficulty and the work of breathing and is a common complicating factor of paroxysmal nocturnal dyspnea. On chest auscultation, the bronchospasm associated with a CHF exacerbation can be difficult to distinguish from an acute asthma exacerbation, although other clues from the cardiovascular examination should lead the examiner to the correct diagnosis. Both types of bronchospasm can be present in the same individual.

In contrast to orthopnea, which may be relieved by immediately sitting up in bed, attacks of paroxysmal nocturnal dyspnea may require 30 minutes or longer in this position for relief. Episodes of this may be so frightening that the patient may be afraid to resume sleeping, even after the symptoms have abated.

***4. Dyspnea at rest - Mechanisms of dyspnea in heart failure***

Decreased pulmonary function

Decreased compliance

Increased airway resistance

Increased ventilatory drive

Hypoxemia due to increased pulmonary capillary wedge pressure (PCWP)

Ventilation/perfusion (V/Q) mismatching due to increased PCWP and cardiac output

Increased carbon dioxide production

Respiratory muscle dysfunction

Decreased respiratory muscle strength

Decreased endurance

Ischemia

Fatigue and weakness

These symptoms are often accompanied by a feeling of heaviness in the limbs.

Fatigue and weakness are generally related to poor perfusion of the skeletal muscles in patients with a lowered cardiac output. Although generally a constant feature of advanced CHF, episodic fatigue and weakness are common in earlier stages.

* ***Nocturia***

Nocturia may occur relatively early in the course of heart failure. Recumbency reduces the deficit in cardiac output in relation to oxygen demand; renal vasoconstriction diminishes and urine formation increases. This may be troublesome for the patient with heart failure because it may prevent the patient from obtaining much-needed rest.

Oliguria is a late finding in CHF and is found in patients with markedly reduced cardiac output from severely reduced LV function.

* **Cerebral symptoms**

Cerebral symptoms: Confusion, memory impairment, anxiety, headaches, insomnia, bad dreams or nightmares, and rarely, psychosis with disorientation, delirium, or hallucinations may occur in elderly patients with advanced heart failure, particularly in those with cerebrovascular atherosclerosis.

* **Predominant right-sided heart failure**

Ascites,

congestive hepatomegaly,

anasarca

due to elevated right-sided heart pressures transmitted backward into the portal vein circulation may result in increased abdominal girth and epigastric and right upper quadrant (RUQ) abdominal pain. Other gastrointestinal symptoms, owing to congestion of the hepatic and gastrointestinal venous circulation, include anorexia, bloating, nausea, and constipation. In preterminal heart failure, inadequate bowel perfusion can cause abdominal pain, distention, and bloody stools. Distinguishing right-sided CHF from hepatic failure is often clinically difficult.

Dyspnea, prominent in LV failure, becomes less prominent in isolated right-sided heart failure because of the absence of pulmonary congestion. On the other hand, when cardiac output becomes markedly reduced in patients with terminal right-sided heart failure (as may occur in isolated RV infarction and in the late stages of primary pulmonary hypertension and pulmonary thromboembolic disease), severe dyspnea may occur as a consequence of the reduced cardiac output, poor perfusion of respiratory muscles, hypoxemia, and metabolic acidosis.

**Physical**

General appearance

* Patients with mild heart failure appear to be in no distress after a few minutes of rest, but they may be obviously dyspneic during and immediately after moderate activity. Patients with LV failure may be dyspneic when lying flat without elevation of the head for more than a few minutes. Those with severe heart failure appear anxious and may exhibit signs of air hunger in this position.
* Patients with recent onset of heart failure are generally well nourished, but those with chronic severe heart failure are often malnourished and sometimes even cachectic.
* Chronic marked elevation of systemic venous pressure may produce severe tricuspid regurgitation and may lead to visible pulsation of the eyes and of the neck veins.
* Central cyanosis, icterus, and malar flush may be evident in patients with severe heart failure.
* In mild or moderate heart failure, stroke volume is normal at rest; in severe heart failure, it is reduced, as reflected by a diminished pulse pressure and a dusky discoloration of the skin.

With very severe heart failure, particularly if cardiac output has declined acutely, systolic arterial pressure may be reduced. The pulse may be weak, rapid, and thready; the proportional pulse pressure (pulse pressure/systolic pressure) may be markedly reduced. The proportional pulse pressure correlates reasonably well with cardiac output. In one study, when pulse pressure was less than 25%, it usually reflected a cardiac index of less than 2.2 L/min/m2.

Evidence of increased adrenergic activity

Increased adrenergic activity is manifested by tachycardia, diaphoresis, pallor, peripheral cyanosis with pallor and coldness of the extremities, and obvious distention of the peripheral veins secondary to venoconstriction.

Diastolic arterial pressure may be slightly elevated.

Pulmonary rales

Rales heard over the lung bases are characteristic of CHF of at least moderate severity. With acute pulmonary edema, rales are frequently accompanied by wheezing and expectoration of frothy, blood-tinged sputum.

* Edema

Although a cardinal manifestation of CHF, edema does not correlate well with the level of systemic venous pressure. In patients with chronic LV failure and low cardiac output, extracellular fluid volume may be sufficiently expanded to cause edema in the presence of only slight elevations in systemic venous pressure.

Edema, in the absence of dyspnea or other signs of LV or RV failure, is not solely indicative of heart failure and can be observed in many other conditions, including chronic venous insufficiency, nephrotic syndrome, or other syndromes of hypoproteinemia or osmotic imbalance.

* Hepatomegaly

Hepatomegaly is prominent in patients with chronic right-sided heart failure, but it may occur rapidly in acute heart failure.

* Hydrothorax (pleural effusion)

Hydrothorax is most commonly observed in patients with hypertension involving both systemic and pulmonary systems. Hydrothorax is usually bilateral, although when unilateral, it is usually confined to the right side of the chest.

When hydrothorax develops, dyspnea usually intensifies because of further reductions in vital capacity.

* Ascites

This finding occurs in patients with increased pressure in the hepatic veins and in the veins draining into the peritoneum.

Ascites usually reflects long-standing systemic venous hypertension.

Protodiastolic (S3) gallop: This is the earliest cardiac physical finding in decompensated heart failure in the absence of severe mitral or tricuspid regurgitation or left-to-right shunts.

* Cardiomegaly

A nonspecific finding, cardiomegaly nonetheless occurs in most patients with chronic heart failure.

Notable exceptions include heart failure from acute myocardial infarction, constrictive pericarditis, restrictive cardiomyopathy, valve or chordae tendineae rupture, or heart failure due to tachyarrhythmias or bradyarrhythmias.

* Cardiac cachexia

Cardiac cachexia is found in long-standing heart failure, particularly of the RV, because of anorexia from hepatic and intestinal congestion and sometimes because of digitalis toxicity. Occasionally, impaired intestinal absorption of fat and (rarely) protein-losing enteropathy occur.

Patients with heart failure may also exhibit increased total metabolism secondary to augmentation of myocardial oxygen consumption, excessive work of breathing, low-grade fever, and elevated levels of circulating TNF.

**Lab Studies**

CBC count: This study aids in the assessment of severe anemia, which may cause or aggravate heart failure. Leukocytosis may signal underlying infection. Otherwise, CBC counts are usually of little diagnostic help.

Electrolytes

* Serum electrolyte values are generally within reference ranges in patients with mild-to-moderate heart failure before treatment. However, in severe heart failure, prolonged, rigid sodium restriction, coupled with intensive diuretic therapy and the inability to excrete water, may lead to dilutional hyponatremia, which occurs because of a substantial expansion of extracellular fluid volume and a normal or increased level of total body sodium.
* Potassium levels are usually within reference ranges, although the prolonged administration of diuretics may result in hypokalemia. Hyperkalemia may occur in patients with severe heart failure who show marked reductions in GFR and inadequate delivery of sodium to the distal tubular sodium-potassium exchange sites of the kidney, particularly if they are receiving potassium-sparing diuretics and/or ACE inhibitors.

Renal function tests

* BUN and creatinine levels can be within reference ranges in patients with mild-to-moderate heart failure and normal renal function, although elevated BUN and BUN/creatinine ratios may also be present.
* Patients with severe heart failure, particularly those on large doses of diuretics for long periods, may have elevated BUN and creatinine levels indicative of renal insufficiency because of chronic reductions of renal blood flow from reduced cardiac output. Diuretics may aggravate renal insufficiency when these patients are overmedicated with diuretics and become volume depleted.

Liver function tests

* Congestive hepatomegaly and cardiac cirrhosis are often associated with impaired hepatic function, which is characterized by abnormal values of aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactic dehydrogenase (LDH), and other liver enzymes.
* Hyperbilirubinemia, secondary to an increase in both the directly and indirectly reacting bilirubin, is common. In severe cases of acute RV or LV failure, frank jaundice may occur.
* Acute hepatic venous congestion can result in severe jaundice, with a bilirubin level as high as 15-20 mg/dL, elevation of AST to more than 10 times the upper reference range limit, elevation of the serum alkaline phosphatase level, and prolongation of the prothrombin time. Both the clinical and the laboratory pictures may resemble viral hepatitis, but the impairment of hepatic function is rapidly resolved by successful treatment of heart failure. In patients with long-standing heart failure, albumin synthesis may be impaired, leading to hypoalbuminemia and intensifying the accumulation of fluid.
* Fulminant hepatic failure is an uncommon, late, and sometimes terminal complication of cardiac cirrhosis.

B-type natriuretic peptide

* BNP is a 32-amino acid polypeptide containing a 17-amino acid ring structure common to all natriuretic peptides. Unlike ANP, whose major storage sites are in both the atria and ventricles, the major source of plasma BNP is the cardiac ventricles, suggesting that BNP may be a more sensitive and specific indicator of ventricular disorders than other natriuretic peptides. The release of BNP appears to be in direct proportion to ventricular volume expansion and pressure overload. BNP is an independent predictor of high LV end-diastolic pressure and is more useful than ANP or NE levels for assessing mortality risk in patients with CHF.
* BNP levels correlate closely with the NYHA Classification of Heart Failure as well as the Goldman Activity Classification of Heart Failure.

**Imaging Studies**

* Chest radiography

Chest radiographs are very helpful in distinguishing cardiogenic pulmonary edema (CPE) from other pulmonary causes of severe dyspnea.

Classic radiographic findings demonstrate cardiomegaly (in patients with underlying CHF) and alveolar edema with pleural effusions and bilateral infiltrates in a butterfly pattern. The other signs are loss of sharp definition of pulmonary vasculature, haziness of hilar shadows, and thickening of interlobular septa (Kerley B lines).

Chest radiographs in patients with abrupt onset are usually helpful but can be limited because a delay of as long as 12 hours is possible from the onset of dyspnea due to acute heart failure to the development of classic abnormal findings on x-ray films.

* Echocardiography

This is the easiest and least-expensive method of determining LV function, both systolic and diastolic. Echocardiography is also the easiest and least-expensive method of determining the presence of valvular heart disease, LV wall thickness, chamber sizes, presence of pericardial disease, and regional wall motion abnormalities that may suggest ischemic coronary artery disease as the cause. Echocardiography is very reliable in diagnosing the cause or causes of heart failure.

Transesophageal echocardiography is particularly useful in patients who are on mechanical ventilation or morbidly obese and in patients whose transthoracic echocardiogram was suboptimal in its imaging. It is an easy and safe alternative to conventional transthoracic echocardiography and provides superior imaging quality compared to conventional transthoracic echocardiography.

* Radionuclide multiple gated acquisition scan

Radionuclide multiple gated acquisition (MUGA) scan is a very reliable imaging technique for determining global heart function. LV ejection fraction, as determined by MUGA scanning, is often used for serial assessment of LV function because of its reliability.

However, this study is limited in its assessment of valvular heart disease and pericardial disease.

**Other Tests**

* Arterial blood gases

ABGs usually reveal mild hypoxemia in patients who have mild-to-moderate heart failure. ABGs are more accurate than pulse oximetry for measuring oxygen saturation. Patients with severe heart failure may have signs and symptoms ranging from severe hypoxemia, or even hypoxia, along with hypercapnia, to decreased vital capacity and poor ventilation.

* Pulse oximetry

Pulse oximetry is highly accurate at assessing the presence of hypoxemia and, therefore, the severity of heart failure.

Patients with mild-to-moderate heart failure show modest reductions in oxygen saturation, whereas patients with severe heart failure may have severe oxygen desaturation, even at rest.

Pulse oximetry is useful for monitoring the patient's response to supplemental oxygen and other therapies.

* **Electrocardiography**

The presence of left atrial enlargement and LV hypertrophy is sensitive (although nonspecific) for chronic LV dysfunction.

ECG may suggest an acute tachyarrhythmia or bradyarrhythmia as the cause of heart failure.

ECG may aid in the diagnosis of acute myocardial ischemia or infarction as the cause of heart failure or may suggest the likelihood of prior myocardial infarction or presence of coronary artery disease as the cause of heart failure.

ECG is of limited help when an acute valvular abnormality or LV systolic dysfunction is considered to be the cause of heart failure; however, the presence of left bundle branch block (LBBB) on an ECG is a strong marker for diminished LV systolic function.

**Procedures**

* Right-sided heart catheterization

PCWP can be measured by using a pulmonary arterial catheter (Swan-Ganz catheter), and this helps differentiate cardiogenic causes of decompensated heart failure from noncardiogenic causes such as ARDS, which occurs secondary to injury to the alveolar-capillary membrane rather than to alteration in Starling forces. A PCWP exceeding 18 mm Hg in a patient not known to have chronically elevated left atrial pressure is indicative of cardiogenic decompensated heart failure. In patients with chronic pulmonary capillary hypertension, capillary wedge pressures exceeding 30 mm Hg are generally required to overcome the pumping capacity of the lymphatics and produce pulmonary edema.

* Left-sided heart catheterization and coronary angiography

Left-sided heart catheterization and coronary angiography should be undertaken when the etiology of heart failure cannot be determined by clinical or noninvasive imaging methods or when the etiology is likely to be due to acute myocardial ischemia or myocardial infarction. Coronary angiography is particularly helpful in patients with LV systolic dysfunction and known or suspected coronary artery disease in whom myocardial ischemia is thought to play a dominant role in the reduction of LV systolic function and the worsening of heart failure. As a general rule, most patients with clinically significant CHF should undergo cardiac catheterization to exclude the reversible causes listed above.

Specific rationales for right- and left-sided heart catheterization include the need to determine the etiologic significance and severity of mitral and/or aortic valvular disease in patients with heart failure in whom the cause-effect relationship of valvular heart disease with regard to heart failure is unclear. Furthermore, right- and left-sided heart catheterization should be performed in patients in whom constrictive pericarditis is considered a likely cause of heart failure.

**Staging**

A classification of patients with heart disease based on the relation between symptoms and the amount of effort required to provoke them has been developed by the NYHA.

Class I: No limitations. Ordinary physical activity does not cause undue fatigue, dyspnea, or palpitations.

Class II: Slight limitation of physical activity. Such patients are comfortable at rest. Ordinary physical activity results in fatigue, palpitations, dyspnea, or angina.

Class III: Marked limitation of physical activity. Although patients are comfortable at rest, less-than-ordinary activity leads to fatigue, dyspnea, palpitations, or angina.

Class IV: Symptomatic at rest. Symptoms of CHF are present at rest; discomfort increases with any physical activity.

The Goldman Activity Classification of Heart Failure is based on estimated metabolic cost of various activities, and classes correlate to NYHA classes.

Class I: Patients can perform to completion any activity up to 7 metabolic equivalents (METS).

Class II: Patients can perform to completion any activity up to 5 METS of activity but cannot perform to completion any activities equal to or more than 7 METS.

Class III: Patients can perform to completion any activity up to 2 METS of activity but cannot perform to completion any activities equal to or more than 5 METS.

Class IV: Patients cannot perform to completion activities equal to or more than 2 METS.

**TREATMENT**

Medical Care: Medical therapy of heart failure focuses on 3 main goals:

(1) preload reduction,

(2) reduction of systemic vascular resistance (afterload reduction),

(3) inhibition of both the RAAS systems and vasoconstrictor neurohumoral factors produced by the sympathetic nervous system in patients with heart failure.

The first 2 goals provide symptomatic relief. While reducing symptoms, inhibition of the RAAS and neurohumoral factors also results in significant reductions in morbidity and mortality rates.

Preload reduction results in decreased pulmonary capillary hydrostatic pressure and reduction of fluid transudation into the pulmonary interstitium and alveoli. Afterload reduction results in increased cardiac output and improved renal perfusion, which allows for diuresis in the patient with fluid overload. Inhibition of the RAAS and sympathetic nervous system results in favored vasodilation and reduction of neurohumoral vasoconstrictors, thereby increasing cardiac output and reducing blood volume and myocardial oxygen demand.

Patients with severe LV dysfunction or acute valvular disorders may present with hypotension. These patients may not tolerate medications to reduce their preload and afterload and may require inotropic support to maintain adequate blood pressure.

Patients who remain hypoxic despite supplemental oxygen or who demonstrate severe respiratory distress require mechanical ventilation, in addition to maximal medical therapy.

Preload reduction

* Nitroglycerin

Nitroglycerine (NTG) is the most effective, predictable, and rapid-acting medication available for preload reduction.

Multiple studies comparing NTG to furosemide or morphine sulfate have demonstrated greater efficacy and safety and a faster onset of action for NTG.

Use of sublingual NTG is associated with preload reduction within 5 minutes and some afterload reduction.

Topical NTG may be as effective as sublingual NTG in most patients with heart failure, but it should be avoided in patients with severe LV failure because of poor skin perfusion (manifesting as skin pallor or mottling) and resultant poor absorption.

Intravenous NTG at higher dosages provides rapid and titratable preload and afterload reduction and has been demonstrated to be an excellent single-agent therapy for patients with severe decompensated CHF.

* Loop diuretics

Loop diuretics are the cornerstone of heart failure treatment and have been considered as such for many decades. Furosemide is most commonly used. Bumetanide has a higher bioavailability and may be more effective in patients with severe CHF.

Loop diuretics are presumed to decrease preload through 2 mechanisms: diuresis and direct pulmonary artery vasodilation and venodilation.

In most patients, diuresis does not occur for at least 20-90 minutes; thus, the effect is delayed.

In some patients with heart failure, particularly those with diastolic heart failure who are minimally fluid overloaded, continued diuretic use after resolution of acute symptoms may be associated with adverse outcomes, including electrolyte derangements and hypotension.

Use of medications that decrease preload (eg, NTG) and afterload (eg, ACE inhibitors), either concomitantly or before the administration of loop diuretics, can prevent potential adverse hemodynamic changes.

* Potassium-sparing diuretics

Numerous studies have shown spironolactone to be as beneficial in the management of CHF as loop diuretics.

Some of the beneficial effects of spironolactone may be due to its neurohormonal actions.

* Morphine sulfate

Morphine sulfate use in acute CHF for preload reduction has been commonplace for many years.

Use should be weighed against potential adverse effects (eg, nausea/vomiting, local or systemic allergic reactions, respiratory depression) that may outweigh any potential benefit, especially given the availability of much more effective medications for preload reduction (eg, NTG).

Any beneficial hemodynamic effect probably is due to anxiolysis, with a resulting decrease in catecholamine production and systemic vascular resistance.

Vasodilators (combined afterload and preload reducers)

* ACE inhibitors

Although initial studies focused on the efficacy of ACE inhibitors in the treatment of chronic CHF, recent studies have demonstrated excellent results for treatment of acute decompensated CHF.

Studies demonstrate that the use of ACE inhibitors in acute heart failure is associated with reduced admission rates to ICUs and decreased endotracheal intubation rates.

Hemodynamic effects of ACE inhibitors include reduced afterload, improved stroke volume and cardiac output, and reduced preload.

ACE inhibitors must be initiated with extreme care in individuals presenting with borderline hemodynamic parameters.

When administered by intravenous (enalapril 1.25 mg) or sublingual routes, hemodynamic and subjective improvements are noted within 10 minutes; improvements occur more slowly with the oral route.

ACE inhibitors prolong survival in heart failure. Furthermore, compared to the combination of hydralazine and long-acting nitrates, ACE inhibitors showed a trend to a greater prolongation of survival, had improved hemodynamics, and were better tolerated.

* Ang II receptor inhibitors

Ang receptor inhibitors, such as losartan and candesartan, are highly recommended alternatives to ACE inhibitors in patients who cannot tolerate ACE inhibitors because of adverse effects, most notably, coughing.

Furthermore, these agents have gained wider use based on their low adverse effect profile and early study findings, which indicated that combined ACE inhibition and Ang II receptor inhibition is beneficial.

* Hydralazine

Hydralazine was the first oral balanced (afterload and preload reduction) vasodilator and was popular before the availability of ACE inhibitors. It is a direct vasodilator, unlike ACE inhibitors or Ang receptor inhibitors, which are vasodilators through inhibition of the RAAS system.

When combined with long-acting nitrates, hydralazine was shown, in the Veterans Administration Heart Failure Trial (VHEFT) studies, to prolong survival in patients with CHF.

Hydralazine has one main advantage over ACE inhibitors in that it is safe in pregnancy. It also is not known to worsen renal function in patients with heart failure who have reduced renal function and is not associated with the risk of hyperkalemia. Additionally, hydralazine use is recommended in patients who cannot tolerate ACE inhibitors.

Hydralazine, as a single agent, has less reduction in myocardial oxygen demand than ACE inhibitors because of a slight increase in heart rate that usually results from its use.

* Nitroprusside

Nitroprusside results in simultaneous preload and afterload reduction through direct smooth muscle relaxation, although it has a greater effect on afterload.

Afterload reduction is associated with increased cardiac output.

Potency and rapidity of onset and offset of effect make this an ideal medication for patients who are critically ill.

It may induce precipitous falls in blood pressure; intraarterial blood pressure monitoring often is recommended.

Use nitroprusside cautiously in the setting of acute myocardial infarction because of its potential to induce hypotension.

If nitroprusside is used, convert patients to oral or alternative intravenous vasodilator therapy as soon as possible because prolonged use is associated with thiocyanate toxicity.

Use in pregnancy is associated with fetal thiocyanate toxicity.

* Inotropic support

Digoxin (cardiac glycoside)

Digoxin has been a cornerstone for the treatment of heart failure for decades and is the only oral inotropic support agent currently used in clinical practice.

Digoxin acts by inhibiting the Na+/K+–ATPase transport pump and inhibits sodium and potassium transport across cell membranes. This increases the velocity and shortening of cardiac muscle, resulting in a shift upward and to the left of the ventricular function (Frank-Starling) curve relating stroke volume to filling volume or pressure. This occurs in healthy as well as failing myocardium and in atrial as well as ventricular muscle. The positive inotropic effect is due to an increase in the availability of cytosolic calcium during systole, thus increasing the velocity and extent of myocardial sarcomere shortening.

No evidence indicates that digoxin affects peripheral vascular resistance or systemic blood pressure.

All evidence suggests that digoxin provides, even in the short term, a moderate and metabolically efficient positive inotropic effect, an important consideration in ischemic cardiomyopathies.

Although the incidence and severity of digitalis intoxication is decreasing, vigilance for this important complication of therapy is essential. Drugs that interact with digoxin are numerous and include amiodarone, propafenone, quinidine, verapamil, nifedipine, diltiazem, levothyroxine, cyclosporine, flecainide, disopyramide, omeprazole, tetracycline, and erythromycin. These agents affect clearance or absorption of digoxin, thus necessitating dose alteration of digoxin in patients taking these medications. Furthermore, patients with renal insufficiency may need to have their digoxin dose adjusted downward to avoid digitalis intoxication.

Numerous studies confirm that digoxin does not prolong survival in patients with systolic heart failure, but it is associated with reduced hospital admissions, improved functional class, reduced symptoms of heart failure, and improved quality of life.

Digoxin is also an effective agent against atrial tachyarrhythmias at rest in patients with LV dysfunction, but it has limited efficacy in controlling the ventricular rate of atrial arrhythmias during exertion.

Dobutamine (sympathomimetic agent)

Dobutamine mainly serves as a beta1-receptor agonist, although it has some beta2-receptor and minimal alpha-receptor activity.

Intravenous dobutamine induces significant positive inotropic effects with mild chronotropic effects. It also induces mild peripheral vasodilation (decrease in afterload).

The combination effect of increased inotropy with decreased afterload results in a significant increase in cardiac output.

Combination use with intravenous NTG may be ideal for patients with myocardial infarction and decompensated heart failure and mild hypotension in order to provide simultaneous preload reduction with increased cardiac output. In the setting of acute myocardial infarction, dobutamine use could increase infarct size because of the increase in myocardial oxygen consumption that may ensue.

In general, avoid dobutamine in patients with moderate or severe hypotension (eg, systolic blood pressure <80 mm Hg) because of the peripheral vasodilation.

Dopamine (sympathomimetic agent)

Vascular and myocardial receptor effects are dose dependent.

Low dosages (0.5-3 mcg/kg/min) cause stimulation of dopaminergic receptors within the renal and splanchnic vascular beds, causing vasodilation and increased diuresis.

Moderate dosages (3-10 mcg/kg/min) cause stimulation of beta-receptors in the myocardium, resulting in increased cardiac contractility and heart rate.

High dosages (10-20 mcg/kg/min) cause stimulation of alpha-receptors, resulting in peripheral vasoconstriction (increased afterload), increased blood pressure, and no further improvement in cardiac output.

As with other inotropic agents, moderate and high dosages are arrhythmogenic and also result in increased myocardial oxygen demand (potential for myocardial ischemia); therefore, use dopamine only in patients with heart failure who cannot tolerate the use of dobutamine because of severe hypotension (eg, systolic blood pressure <60-80 mm Hg).

* Phosphodiesterase inhibitors (milrinone, amrinone)

Phosphodiesterase inhibitors (PDIs) increase intracellular cAMP, which results in a positive inotropic effect on the myocardium and peripheral vasodilation (decreased afterload) and a reduction in pulmonary vascular resistance (decreased preload).

PDIs, unlike catecholamine inotropes, are not dependent on adrenoreceptor activity; therefore, patients are less likely to develop tolerance to these medications. Tolerance to catecholamine inotropes can develop rapidly through down-regulation of the adrenoreceptors.

PDIs are less likely than catecholamine inotropes to cause adverse effects that are typically associated with adrenoreceptor activity (eg, increased myocardial oxygen demand, myocardial ischemia).

Several studies directly comparing the use of PDIs (milrinone, amrinone) to dobutamine in patients with heart failure have demonstrated that milrinone produced equal or greater improvements in stroke volume, cardiac output, PCWPs (preload), and systemic vascular resistance (afterload). They are also associated with less tachycardia and myocardial oxygen consumption. However, PDIs have been associated with a significantly greater incidence of adverse events (eg, tachyarrhythmias) than has dobutamine.

At present, oral PDIs have no role. Their use was associated with a 53% increase in mortality rates in patients with NYHA Class IV heart failure in the Prospective Randomized Milrinone Survival Evaluation (PROMISE) trial, prompting an early termination of that study.

Unfavorable results were also evident in a smaller trial that compared oral milrinone to digoxin or placebo. Furthermore, sustained hemodynamic improvement with oral milrinone was lacking, and the incidence of adverse events, particularly cardiac arrhythmias, was greater.

* Beta-adrenergic blocking agents (metoprolol, carvedilol)

A large and increasing body of evidence indicates that these agents improve symptoms, exercise tolerance, cardiac hemodynamics, and LV ejection fraction and that they decrease mortality rates in patients with heart failure, particularly those with both ischemic and idiopathic cardiomyopathy.

A growing body of evidence suggests that long-term beta-adrenergic antagonist administration improves cardiac function, reduces myocardial ischemia, improves ventricular-arterial coupling, and decreases myocardial oxygen consumption. These agents may also reduce the incidence of sudden death due to primary ventricular arrhythmias in patients with heart failure, although this latter benefit has yet to be definitively proven.

Detectable improvements in ventricular function are usually not apparent for a minimum of 1-3 months, and longer-term structural changes, such as a decline in ventricular volume or mass, may take 12-18 months.

Beta-adrenergic antagonists with vasodilator activity, such as carvedilol and labetalol, have the added benefit of further afterload reduction because of arterial vasodilation from alpha1-receptor blockade.

Treatment of heart failure with predominant diastolic dysfunction: The therapeutic approach to diastolic dysfunction has 2 major components. The first involves attempts to reverse the abnormal cardiac diastolic properties. The second is directed toward reducing LV filling pressure and thereby venous congestion.

Surgical Care

Kantrowitz initially described intraaortic balloon pumping (IABP) in 1953, but the procedure was first used clinically in 1969 in a patient with cardiogenic shock. Since the 1980s, IABP has been increasingly used in various clinical situations as a lifesaving intervention to obtain hemodynamic stabilization prior to definite therapy.

The intraaortic balloon pump is inserted percutaneously via the femoral artery using a modified Seldinger technique. The distal end of the pump is placed just distal to the aortic knob and the origin of left subclavian artery.

Fluoroscopy may be used for correct positioning of the balloon, and a subsequent chest radiograph should be obtained to document satisfactory balloon placement.

Proper timing of IABP for optimal hemodynamic support

Proper timing of counterpulsation is necessary for maximum hemodynamic support. The timings of balloon inflation and deflation are best evaluated and adjusted at a pump frequency of 1:2.

* Cardiac transplantation

Diet

Patients admitted with heart failure or pulmonary edema should maintain a low-salt diet in order to minimize fluid overload. Monitor fluid balance closely.

Activity

Patients with decompensated heart failure should be placed on complete bed rest until their decompensation is resolved. This is necessary to maximally reduce myocardial oxygen demand and to avoid exacerbation of the abnormal hemodynamics and symptoms of heart failure.

Once the patient with heart failure has been stabilized, activity should be gradually and progressively increased. Emphasize the importance of cardiac rehabilitation to all patients with heart failure who require improved cardiac fitness. Encourage patients to exercise daily for at least 20-30 minutes in a low-intensity, endurance-enhancing activity such as walking, biking, or swimming. Regular exercise improves the quality of life for these patients and improves efficiency of oxygen utilization at the tissue level, thus reducing the workload of the heart in the role of oxygen delivery to end organs and muscles.

**Further Inpatient Care**

After the patient has been initially stabilized and the decompensation of heart failure has been resolved, further inpatient care depends on the underlying cause of CHF.

Place patients with heart failure in a monitored bed to watch for acute dysrhythmias. Pay strict attention to the patient's fluid balance by closely monitoring fluid input and output. Maintain patients who are fluid-overloaded in negative fluid balance through the use of diuretics, or, if necessary in patients with renal failure, hemodialysis with ultrafiltration.

Check cardiac enzymes to evaluate for myocardial infarction. Slight elevations in cardiac enzymes can occur with decompensated heart failure in the absence of myocardial infarction because of coronary thrombosis.

Perform coronary angiography on patients whose decompensated heart failure resulted from an acute coronary syndrome, either unstable angina or myocardial infarction. Stress testing can also be performed later during hospitalization to evaluate for reversible ischemia in patients without acute coronary syndromes but who have prior symptoms of angina or who have a high likelihood of coronary artery disease as the cause of LV dysfunction.

Order echocardiography at the earliest possible moment to evaluate for evidence of acute valvular dysfunction and wall motion abnormalities and to assess the patient's systolic and diastolic function. Since the long-term therapy of patients with heart failure differs significantly between those with predominantly systolic dysfunction and those with predominantly diastolic dysfunction, it is absolutely essential that all patients with heart failure have echocardiographic evaluation of cardiac function, chamber size, and valve function.

In most patients with decompensated heart failure, oral vasodilator therapy, most commonly ACE inhibitors, can be used as first-line therapy to reverse the cardiac decompensation and to restore optimal cardiac function. The clinician must be extremely cautious with vasodilator therapy only in patients with severe aortic or mitral stenosis or in those with obstructive cardiomyopathy. Patients who required intravenous inotropic support should be weaned off as quickly as possible and should have their vasodilator therapy maximized quickly in order to avoid the risk of adverse cardiac events from increased myocardial oxygen consumption leading to ischemia.

Patients in whom pulmonary edema was caused by dietary factors or medication noncompliance need strict counseling and education to help prevent recurrence.

**Further Outpatient Care**

Focus further outpatient care of patients with heart failure on maximizing some or all of the medical modalities used in their treatment. Undertake further assessment of the clinical and hemodynamic effects of that therapy fairly soon after discharge and at regular intervals.

Precise definition and aggressive treatment of all reversible causes for heart failure is absolutely essential. For instance, patients with myocardial ischemia (particularly those with reduced systolic function) should be promptly evaluated with noninvasive and/or invasive evaluations of coronary perfusion, and they should be promptly referred for revascularization if they are suitable candidates for such revascularization. Similarly, patients with severe valvular disease, assessed clinically and echocardiographically, should be promptly referred for cardiac catheterization. If a patient is a suitable candidate for valve replacement or repair, he or she should undergo prompt surgical therapy.

Patients with nonreversible NYHA class IV heart failure who are younger than 65 years and facing the likely prospect of death within the next 6-24 months, despite maximal medical therapy, and who are not candidates for beneficial surgical therapy, should be promptly referred to a cardiac transplant center for consideration of cardiac transplantation.

Screen patients with cardiomyopathy and heart failure for candidacy for cardioverter/defibrillator implantation because the risk of sudden death in these patients is considerable.

**Complications**

The major complications associated with heart failure are sudden cardiac death from ventricular tachyarrhythmias or bradyarrhythmias and pump failure with cardiovascular collapse. Approximately half of patients with heart failure eventually die from fatal ventricular arrhythmias. Prompt diagnosis and treatment usually prevent this complication in the acute setting. Prompt diagnosis of CHF and prompt treatment to reduce pulmonary venous congestion, reduce afterload, and improve cardiac output is essential in preventing cardiovascular and respiratory failure.

**Prognosis**

In general, the inpatient mortality rate for patients with heart failure is 5-20%.

Heart failure associated with acute myocardial infarction is associated with an inpatient mortality rate of 20-40%; mortality approaches 80% in patients who are also hypotensive (eg, cardiogenic shock).

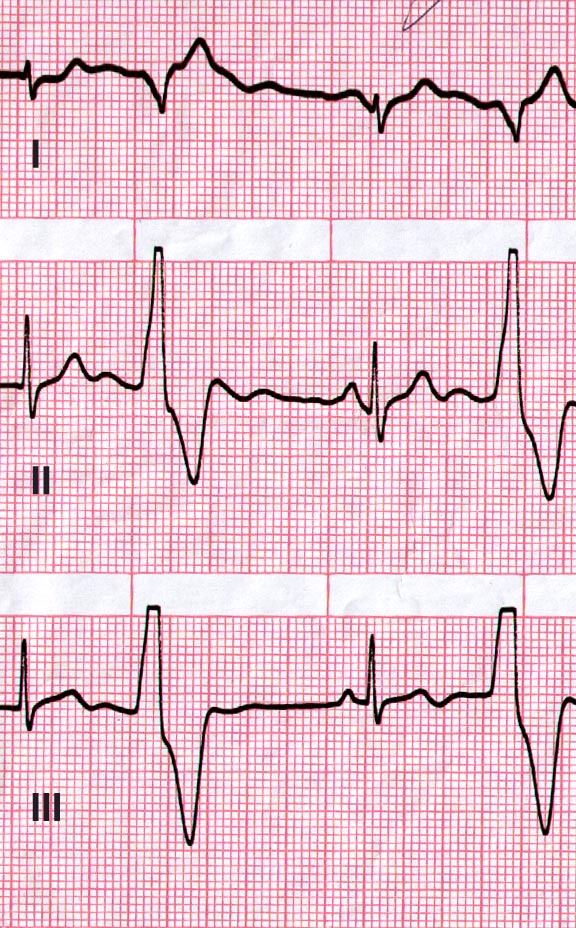
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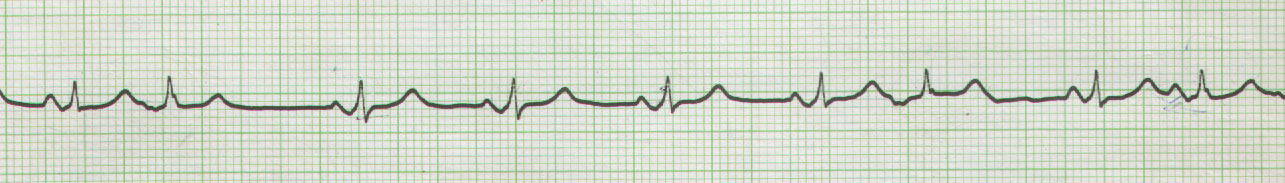
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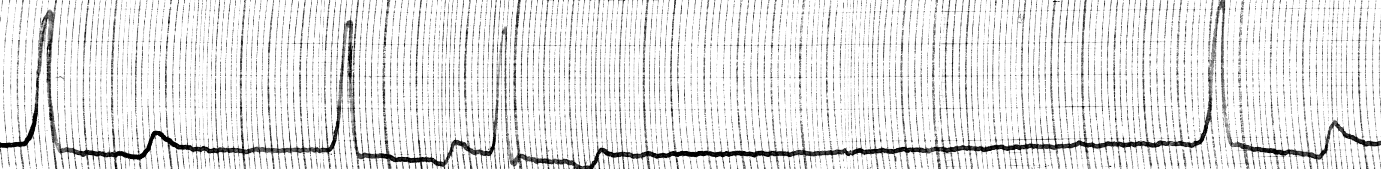
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**App. 1.**



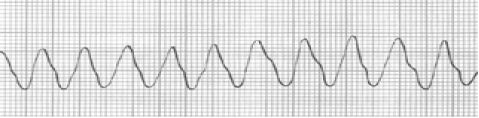


Pic. 2. Ectopic supraventricular arrhythmias.

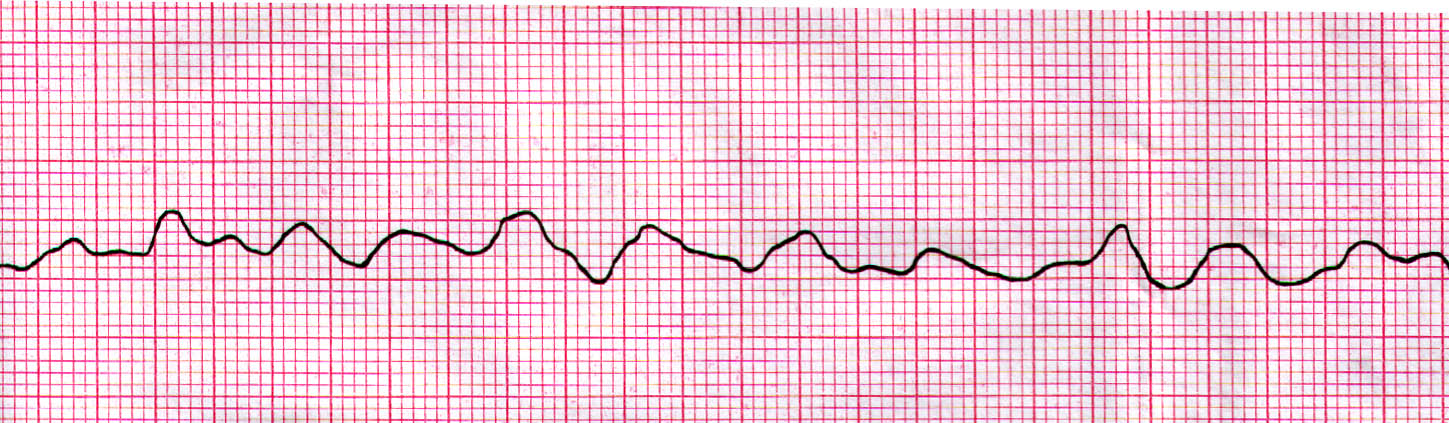


Pic. 3. Atrial fibrillation**.**

Pic. 1. Ectopic ventrical arrhythmias.



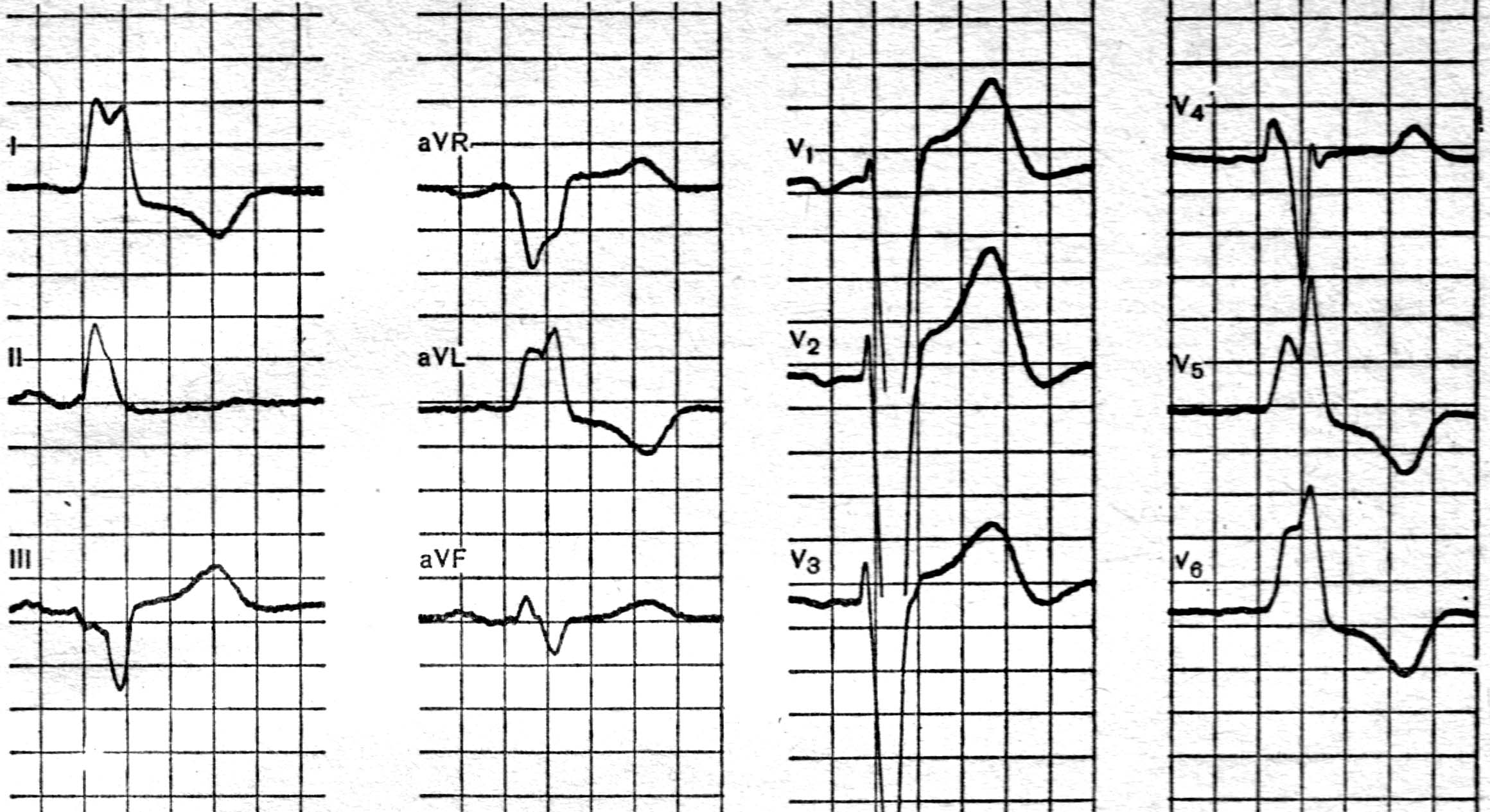
Pic. 4. Ventricular tachycardia.

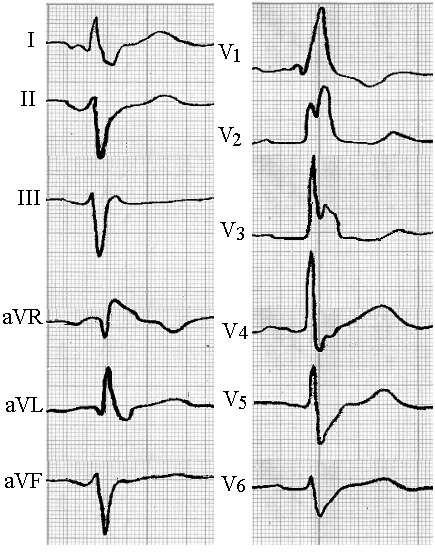


Pic. 5.Ventricular fibrillation.

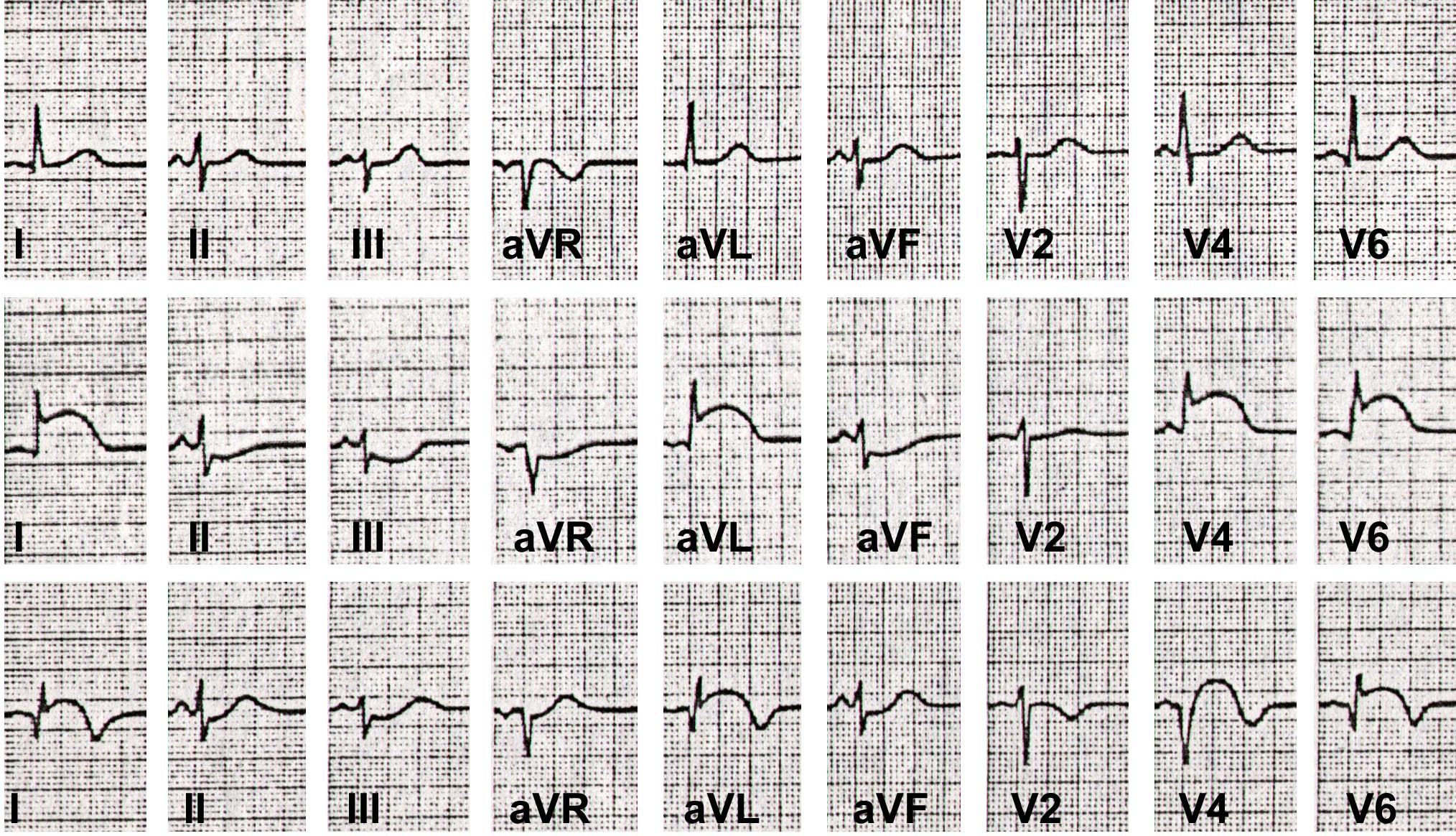


Pic. 6. Atrioventricular block (third degree).





Pic. 7. Left bundle branch block Right



Pic. 8. Miocardial infartion (inferiol wall)

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