Heart Failure

A complex pathophysiologic state described by the inability to generate a cardiac output that is adequate to achieve tissue metabolic requirements. The presence of heart failure has been described as the single most important risk factor for predicting perioperative cardiac morbidity and mortality. Anesthetic management of patients presenting with heart failure can be challenging due to the severely depressed cardiac function and the often debilitated state of the patient.

ANESTHETIC CONSIDERATIONS:

1. Preoperative optimization of heart failure symptoms and medication management.
2. Cautious management of negative inotropic effects of anesthetic medications.
3. Judicious management of preload, afterload, heart rate, rhythm and contractility.
4. Consider end organ dysfunction as a result of long standing heart failure.
5. Consider invasive monitoring: art line, CVP, PA cath, TEE for perioperative optimization of cardiac output.
6. Increased risk of perioperative cardiac morbidity and mortality.

ANESTHETIC GOALS:

1. Preoperative optimization of heart failure symptoms – consider postponing or canceling elective cases if not optimized; consider preoperative investigations if patient unstable or if likely to influence perioperative management according to ACC/AHA 2007 guidelines.
2. Maintain perioperative hemodynamics within 10-20% of pre-induction values.
3. Avoid negative inotropes (propofol, pentothal, high dose volatile agents)
4. Timely recognition of cardiogenic shock and appropriate use of fluids, vasopressors and inotropes to maximize cardiac output.
5. Consider invasive monitoring as required.

HISTORY

- Etiology of cardiac failure if known
  - Ischemic: Detailed history of the status of the coronary arteries including prior bypass grafts or stents
  - Valvular disease, extent of any regurgitation and or stenosis
  - Pulmonary hypertension?
- Symptoms of heart failure and clinical status
  - Clinical signs and symptoms are produced by increased ventricular pressures and subsequent fluid accumulation upstream from the affected ventricle
  - In left-sided heart failure, high LVEDP promotes pulmonary venous congestion
    - Dyspnea and increased WOB on exertion secondary to interstitial pulmonary edema
      - One of the earliest subjective findings of LV failure
      - Quantify by asking the patient how many flights of stairs can be climbed or the distance that can be walked at a normal pace before dyspnea occurs
    - Orthopnea reflects the inability of the failing left ventricle to handle the increased venous return associated with the recumbent position
      - Dry, nonproductive cough that develops when in the supine position and that is relieved by sitting up
    - Paroxysmal nocturnal dyspnea is shortness of breath that awakens a patient from sleep
  - Right-sided heart failure causes systemic venous congestion/hypertension
    - Peripheral edema and congestive hepatomegaly are the most prominent clinical manifestations
    - Anorexia, nausea, or abdominal pain related to increased liver congestion and prerenal azotemia
    - Right-sided heart failure may be caused by pulmonary hypertension or right ventricular myocardial infarction, but the most common cause is left-sided heart failure
  - Fatigue and weakness at rest or with minimal exertion
    - A hallmark of decreased cardiac reserve and low cardiac output
    - During exercise, the failing ventricle is unable to increase its output in order to deliver adequate amounts of oxygen to the muscles
  - Organ system dysfunction related to inadequate CO
    - All patients with significant ventricular dysfunction require a thorough preoperative evaluation of the major organ systems (particularly the renal, hepatic, and central nervous systems) for impairment
  - Decreases in cerebral blood flow may produce confusion, difficulty concentrating, insomnia, anxiety, or memory deficits

New York Heart Association classification:

Class I: Ordinary physical activity does not cause symptoms
Class II: Symptoms occur with ordinary exertion
Class III: Symptoms occur with less than ordinary exertion
Class IV: Symptoms occur at rest

- The severity of the symptoms has an excellent correlation with survival and quality of life.

American College of Cardiology and the American Heart Association Classification

Stage A: Patients at high risk of heart failure but without structural heart disease or symptoms of heart failure
Stage B: Patients with structural heart disease but without symptoms of heart failure
Stage C: Patients with structural heart disease with previous or current symptoms of heart failure
Stage D: Patients with refractory heart failure requiring specialized interventions

- This classification is meant to be complementary to the New York Heart Association classification and to be used in guiding therapy.

PHYSICAL

- Tachypnea
- Presence of moist rales (may be confined to the lung bases in patients with a mild degree of LV failure, or they may be diffuse in those with acute pulmonary edema)
- Resting tachycardia
- Third heart sound (S3 gallop or ventricular diastolic gallop), produced by blood entering and distending a relatively noncompliant left ventricle
- A narrow pulse pressure with a high diastolic pressure reflects a decreased stroke volume
- Severe heart failure may manifest as systemic hypotension with cool and pale extremities despite peripheral vasoconstriction
- Lip and nail bed cyanosis may be present
- Marked weight loss, also known as cardiac cachexia, is a sign of severe chronic heart failure

- Weight loss is caused by a combination of factors including an increase in the metabolic rate, anorexia and nausea, decreased intestinal absorption of food caused by splanchnic venous congestion, and the presence of high levels of circulating cytokines
- In the presence of right heart or biventricular failure, jugular venous distention may be present or inducible by pressing on the liver (hepatojugular reflux)
  - The liver is typically the first organ to become engorged with blood in the presence of right or biventricular failure
  - The hepatic engorgement may be associated with right upper quadrant pain and tenderness or even jaundice in severe cases
  - Pleural effusions (usually right sided) may be present
  - Bilateral pitting pretibial leg edema is typically present with right ventricular failure and reflects both venous congestion and sodium and water retention

INVESTIGATIONS

LABS:

- BNP
  - Plasma BNP levels below 100 pg/mL indicate that heart failure is unlikely
  - BNP in the range of 100 to 500 pg/mL suggests an intermediate probability for heart failure
  - BNP levels above 500 pg/mL are consistent with the diagnosis of heart failure
  - Plasma levels of BNP may be affected by other factors such as sex, advanced age, renal clearance, obesity, pulmonary embolism, atrial fibrillation, and/or other cardiac tachydysrhythmias
- Lytes, BUN, Cr, AST, ALT, ALP, INR, PTT
  - Decreases in renal blood flow may lead to prerenal azotemia characterized by a disproportionate increase in blood urea nitrogen concentration relative to the serum creatinine concentration
  - When moderate liver congestion is present, liver function tests may be mildly elevated, and when liver engorgement is severe, the prothrombin time may be prolonged. Hyponatremia, hypomagnesemia, and hypokalemia may be present.

IMAGING:

- 12-lead electrocardiogram (ECG) may show evidence of a previous myocardial infarction, LV hypertrophy, conduction abnormalities (left bundle branch block, widened QRS), or various cardiac dysrhythmias, especially atrial fibrillation and ventricular dysrhythmias
- Measuring the LV ejection fraction via echocardiography, radionuclide imaging or ventriculography provides the quantification necessary to document the severity of ventricular systolic dysfunction
- Chest radiography (PA and lateral) may be useful in the evaluation of a heart failure patient by detecting the presence of pulmonary disease, cardiomegaly, pulmonary venous congestion, and interstitial or alveolar pulmonary edema
  - An early radiographic sign of LV failure and associated pulmonary venous hypertension is distention of the pulmonary veins in the upper lobes of the lungs
  - Perivascular edema appears as hilar or perihilar haze- the hilus appears large with ill-defined margins
  - Kerley lines, reflecting edematous interlobular septae in the upper lung fields (Kerley A lines), lower lung fields (Kerley B lines), or basilar regions of the lungs producing a honeycomb pattern (Kerley C lines) may be present
  - Alveolar edema produces homogeneous densities in the lung fields, typically in a butterfly pattern
  - Pleural effusion and pericardial effusion may be observed
- Echocardiography is the most useful test in the diagnosis of heart failure
  - A comprehensive two-dimensional echocardiogram coupled with Doppler flow examination can assess whether any abnormalities of the myocardium, cardiac valves, or pericardium are present
  - Allows assessment of: ejection fraction, LV structure (LA and LV size and wall thickness) and functionality, the presence of other structural abnormalities such as valvular and pericardial disease, the presence of diastolic dysfunction and right ventricular function, and pulmonary artery pressure
  - Assessment of diastolic function provides information about LV filling and left atrial pressure
  - A preoperative echocardiographic evaluation can serve as a baseline for comparison with perioperative echocardiography if a patient's condition deteriorates
- LV ejection fraction can also be measured via radionuclide imaging or ventriculography to document the severity of ventricular systolic dysfunction

OPTIMIZATION

- Stabilization of ventricular function and treatment for pulmonary congestion are important prior to elective surgery
- Patients with heart failure often require preinduction optimization of intravascular volume status, pharmacologic manipulations of inotropy and afterload, adjustments to pacemaker settings (where present) and on occasion, elective placement of an IABP
- Provide supplemental oxygen and monitor vital signs during the preoperative period
- Pre-operative sedation:
  - The use of sedative and hypnotic agents may be tolerated in patients with mild cardiac failure
  - Beware anxiolysis as sedation may result in hypoventilation, hypoxemia, and respiratory acidosis, which in turn may potentially increase pulmonary vascular resistance which is not tolerated well in heart failure patients

MEDICAL THERAPY:

- The current recommended therapy of chronic heart failure is based on results of large randomized trials and on the American College of Cardiology/American Heart Association and European Society of Cardiology guidelines for the diagnosis and treatment of chronic heart failure
- Treatment options include lifestyle modification, patient and family education, medical therapy, corrective surgery, implantable devices, and cardiac transplantation
- Most medications should be continued throughout the perioperative period especially β-blockers, but the decision as to whether or not to withhold Acei, ARB and diuretics should be made on an individual basis
- **Medical management of systolic heart failure:**
  - Mortality benefit with:
    - ACEi (first-line treatment for HF) or ARB if Acei is not tolerated
    - Aldosterone antagonist: spironolactone in NYHA Class III and IV patients
    - β-blockers
    - Statins
  - Mortality benefit not known but symptomatic control with:
    - Thiazide and loop diuretics
    - Digoxin
    - Vasodilators: hydralazine and nitrates in patients with dilated LV
- **Management of diastolic heart failure:**
  - Prevent development of diastolic heart failure by decreasing risk factors:
    - Treat CAD/HTN/DM
    - Control weight gain

- Allow adequate filling time of left ventricle by decreasing heart rate:
  - BB, CCB, digoxin
- Control volume overload:
  - Diuretics, long-acting nitrates, low-sodium diet
- Restore and maintain sinus rhythm
  - Cardioversion, amiodarone, digoxin
- Decrease ventricular remodeling
  - ACEi, statins
- Control precipitating factors:
  - Aortic valve replacement
  - Coronary revascularization

- **Management of patients who present with acute heart failure for emergency surgery or patients who decompensate intraoperatively:**
  - Characterized by high ventricular filling pressures, low cardiac output, and hyper- or hypotension
  - Interventions to try:
    - Diuretics and vasodilators
      - Loop diuretics can improve symptoms rapidly, but in high doses may have deleterious effects on clinical outcomes
      - Use a combination of a low dose of loop diuretic with an intravenous vasodilator
      - Nitroglycerin and nitroprusside reduce LV filling pressure and systemic vascular resistance and increase stroke volume
    - Inotropic drugs
      - Prinicipally dobutamine or milrinone have been used to treat decompensated HF
      - Side effects of inotropic drugs include tachycardia, increased myocardial energy demand and oxygen consumption, dysrhythmias, worsening of DHF, and down-regulation of β-receptors
    - Mechanical assisted devices (intra-aortic balloon pump, ventricular assist device)
      - If the etiology of acute heart failure is a large myocardial infarction, the insertion of an intra-aortic balloon pump should be considered
      - In severe cardiogenic shock, emergency insertion of LV and/or right ventricular assist devices may be necessary for survival
    - Emergency cardiac surgery
    - Calcium sensitizers
      - Myofilament calcium sensitizers are a new class of positive inotropic drugs that increase contractility without increasing intracellular levels of calcium
      - There is no significant increase in myocardial oxygen consumption or heart rate and no propensity for dysrhythmias
      - The most widely used medication in this class is levosemindan, an inodilator increasing myocardial contractile strength and promoting dilation of systemic, pulmonary, and coronary arteries; It does not worsen diastolic function.
      - Not yet available in Canada
    - Exogenous BNP
      - Nesiritide (Natrecor) is recombinant BNP that binds to both the A- and B-type natriuretic receptors
      - Promotes arterial, venous, and coronary vasodilation, thereby decreasing LVEDP and improving dyspnea
      - Induces diuresis and natriuresis

ANESTHETIC OPTIONS

- All types of general anesthetics have been successfully used in patients with HF, but drug dosages may need to be adjusted
- Regional anesthesia is acceptable for suitable operations in heart failure patients
  - Regional anesthesia is beneficial as the modest decrease in systemic vascular resistance secondary to peripheral SNS blockade may increase cardiac output
  - However, the decreased systemic vascular resistance produced by epidural or spinal anesthesia is not always predictable or easy to control
  - The pros and cons of regional anesthesia must be carefully weighed in heart failure patients

ANESTHETIC SETUP

- Drugs – have inotropes and pressors ready, standard emergency drugs
- Standard CAS monitors plus 5-lead ECG and nerve stimulator
- Consider central venous access for delivery of potent inotropic and vasoactive agents
- Consider pulmonary artery catheterization
  - To follow and optimize trends of CO and other hemodynamic indices, assess the efficacy of pharmacologic interventions to manipulate PVR, measure mixed venous oxygen saturation, and to assess LV filling pressures (a central venous catheter alone can be used to estimate LV filling pressures only if the EF>40%)
  - Intraoperative use of a pulmonary artery catheter may help in evaluation of optimal fluid loading, but in patients with DHF and poor ventricular compliance, accurate assessment of LV end-diastolic volume may be quite difficult
- Consider arterial line monitoring for major operations
- TEE to directly visualize and optimize ventricular filling, monitor ventricular wall motion and valvular function
- Availability of back-up CPB ideal as function of even a severely depressed ventricle can still get worse

MANAGEMENT OF ANESTHESIA

- The hemodynamic consequences of heart failure are decreased cardiac output, increased LVEDP, peripheral vasoconstriction, retention of sodium and water, and decreased oxygen delivery to the tissues with a widened arterial-venous oxygen difference
- Intraoperative management requires a careful titration of anesthetic agents and continuous optimization of hemodynamics

HEMODYNAMIC GOALS:

Contractility

- Optimize and maintain contractility
- Avoid further depression of contractility which will increase myocardial oxygen demand and may cause ischemia

Rate

- Avoid bradycardia with SHF (SV is relatively fixed and any increase in CO is dependent on an increase in hr)
- Avoid tachycardia with DHF (tachycardia can produce a decrease in CO due to inadequate ventricular filling time)

Rhythm

- Maintain NSR (loss of sinus rhythm will cause patient with severely decreased ventricular function to decompensate quickly)
- Ventricular dysrhythmias are common in patients with LV dysfunction

Afterload

- Maintain bp and organ perfusion (avoid hypotension) - use inotropes and vasopressors judiciously at the first sign of refractory hemodynamic instability

- Avoid significant increases in ventricular afterload which will increase myocardial oxygen demand and may cause ischemia

Preload

- Intravascular volume status needs to be carefully considered and continuously optimized for each individual patient (may need PAC or TEE to guide fluid administration)
- Fluid overload during the perioperative period may contribute to the development or worsening of heart failure
- Patients with severe CHF usually present with concurrent diastolic dysfunction, and have pathologic pressure-volume compliance curves and require higher-than-normal filling pressures to obtain normal filling volumes

Other

- Patients with severely decreased ventricular function tend to decompensate quickly from hypercarbia, hypoxemia, and sudden alterations in volume status

INDUCTION

- The failing heart is chronically compensated by a heightened adrenergic state, and removal of that sympathetic tone may lead to rapid decompensation with cardiovascular collapse during anesthetic induction
- High-dose opioid with a NMB agent has been used traditionally for patients with severely depressed cardiac function
    - Opioids seem to have a particularly beneficial effect in heart failure patients because of their effect on the δ-receptor, which inhibits adrenergic activation
    - May provide hemodynamic stability but may not provide adequate amnesia and may get significant bradycardia and initial chest wall rigidity
- Etomidate (0.2-0.3 mg/kg iv) is usually the induction agent of choice in these patients because it causes neither a significant reduction in SVR nor a significant decrease in myocardial contractility
- The decreases in vascular tone and myocardial contractility with propofol induction make this drug unsuitable for those with severely depressed cardiac function
- Thiopental also causes myocardial depression and venodilation with consequent decreases in CO, thus should not be used for these patients
- Ketamine induction is useful in patients with severely decreased ventricular function(1-2.5 mg/kg iv or 2.5-5 mg/kg im) followed by a maintenance infusion (50-100 mcg/kg/min) will usually provide excellent hyemodynamic stability while assuring adequate analgesia and amnesia
- Midazolam is generally provided prior to giving ketamine in an attempt to lessen the potential postemergence psychiatric side effects that may occur in some patients

MAINTENANCE

- Generally, high doses of inhalational agents are poorly tolerated (all are myocardial depressants)
    - Avoid enflurane and halothane (particularly potent myocardial depressants)
    - Isoflurane, sevoflurane, and desflurane are more likely to be compatible with hemodynamic stability in the well-optimized patient, although isoflurane and desflurane must be used cautiously due to their particular tendency to decrease SVR
    - Sevoflurane appears to cause less myocardial depression and decrease in SVR compared to the others
- Inhaled agents may also affect myocardial automaticity, impulse conduction, and refractoriness potentially resulting in reentry phenomena and arrhythmias
- **Effects of mechanical ventilation in heart failure:**
    - Improves PaO_2
    - Influences DO_2 through its effects on CO
    - Suppression of spontaneous respirtory efforts may substantially decrease the work of breathing and improve the oxygen supply-demand relationship
    - In isolated LV failure, improvements in cardiac performance may be achieved by the use of PPV with PEEP
        - In particular, patients with elevated LV filling pressures, mitral regurgitation, and reversible ischemic dysfunction may improve from afterload reduction related to increased airway and intrathoracic pressures
        - Rise in intrathoracic pressure caused by PPV or PEEP is associated with a decrease in CO; however in the presence of CHF, raised intrathoracic pressure has the potential to favourably affect the detrminants of global cardiac performance
        - Raised intrathoracic pressure may significantly improve LV performance as a result of the reduced transmural pressure required to generate an adequate systemic bp (afterload reduction)
        - Positive pressure ventilation and positive end-expiratory pressure may be beneficial in decreasing pulmonary congestion and improving arterial oxygenation
    - In right and biventricular heart failure, the increase in the airway pressure caused by ventilatory support should be kept at a minimum compatible with adequate gas exchange
        - Avoid high levels of PEEP
            - Large tidal volumes and high levels of PEEP increase PVR which can decrease CO and cause further RV dilation in RV failure
            - Decreased RV preload secondary to PPV or PEEP can be overcome by fluid administration, leg elevation, or even vasopressors to raise the systemic venous pressure
            - Preload augmentation must be done cautiously if there is RV failure
        - Trials of decreased inspiratory times
        - Decreased flow rates
        - Decreased tidal volumes
        - Consider breathing modes that emphasize spontaneous efforts (SIMV, pressure support, CPAP)

EMERGENCE

- Continue to optimize hemodynamic goals and volume status
- In patients with CHF, on weaning ventilatory support, changes of the loading conditions of the heart brought about by the resumption of spontaneous ventilation can induce a vicious cycle resulting in hypoxemia and pulmonary edema
- In the patient with severe ventricular dysfunction who may have received considerable fluid therapy to augment preload and improve cardiac performance, may require diuretic therapy to reduce this hypervolemia and vasodilator therapy to reduce ventricular wall stress, before these patients are exposed to the afterload stress of ventilatory weaning

DISPOSITION & MONITORING

- Patients who have evidence of acute heart failure during surgery should be transferred to an intensive care unit where invasive monitoring can be continued postoperatively
- Patients should have their usual medications restarted as soon as possible.
- May be necessary to optimize intravascular volume status and employ pharmacologic manipulations of afterload and contractility

ANALGESIA:

- Pain should be aggressively treated since its presence and hemodynamic consequences may worsen heart failure

- Patients with severely decreased ventricular function will not tolerate the stress resonse and tachycardia that accompany postoperative pain due to the increased myocardial oxygen demand (potentially leading to ischemia) and decreased diastolic filling time (potentially leading to decreased stroke volume); this combination is especially deleterious in patients with poor ventricular function and will exacerbate hemodynamic instability
- Most often post-op pain management in this population is with intermittent boluses of opioids delibered by a nurse or via PCA
- Regional techniques (eg. continuous epidural infusions and single-shot intrathecal opioids) are becoming popular
    - High doses of epimorph (7.5-10 mcg/kg in subarachnoid space or 75-100 mcg/kg in the epidural space) will generally be well tolerated and can provide adequate analgesia for approximately 16-24 hours following cardiac surgical procedures (watch for potential late respiratory depression)

COMPLICATIONS

- According to the ACC/AHA guidelines, congestive heart failure is one of six independent predictors of major cardiac complications described in the Lee Revised Cardiac Risk Index in stable patients undergoing elective major noncardiac surgery
    - The other predictors include high risk surgery, ischemic heart disease, cerebrovascular disease, preoperative insulin-dependent diabetes mellitus, and preoperative serum creatinine greater than 2.0 mg/dL
    - The presence of several risk factors increases the incidence of postoperative cardiac complications such as cardiac death, cardiac arrest/ventricular fibrillation, complete heart block, acute MI, and pulmonary edema
- CHF at the time of surgery has also been identified as a significant predictor of postoperative respiratory complications
    - May require prolonged ventilatory support

PATHOPHYSIOLOGY

- The initiating mechanisms of heart failure are:
    - Pressure overload (aortic stenosis, essential hypertension)
    - Volume overload (mitral or aortic regurgitation)
    - Myocardial ischemia/infarction
    - Myocardial inflammatory disease
    - Restricted diastolic filling (constrictive pericarditis, restrictive myocarditis)
- In the failing ventricle, various adaptive mechanisms are initiated to maintain a normal cardiac output:
    - Frank-Starling relationship
    - Activation of the SNS- arteriolar and venous constriction increase systemic afterload and venous return
    - Activation of the renin-angiotensin-aldosterone system to increase stroke volume
    - Alterations in the inotropic state, heart rate, and afterload
    - Humoral-mediated responses- ANP and BNP production promotes diuresis, natriuresis, vasodilation, antihypertrophy, anti-inflammation, and inhibits the RAAS and SNS
- In more advanced stages of heart failure, these mechanisms become maladaptive and ultimately lead to myocardial remodeling (myocardial hypertrophy, myocardial dilation and wall thinning, increased interstitial collagen deposition, myocardial fibrosis, and scar formation due to myocyte death)
- In acute heart failure, LVEDP is elevated which reduces coronary blood flow because of decreased coronary perfusion pressure

Systolic and Diastolic Heart Failure

- Decreased ventricular systolic wall motion reflects systolic dysfunction, whereas diastolic dysfunction is characterized by abnormal ventricular relaxation and reduced compliance
- Clinical signs and symptoms do not reliably differentiate systolic dysfunction from diastolic dysfunction

Systolic Heart Failure

- Causes of SHF:
    - CAD
    - Dilated cardiomyopathy
    - Chronic pressure overload (aortic stenosis and chronic hypertension)
    - Chronic volume overload (regurgitant valvular lesions and high output cardiac failure)
- A decreased ejection fraction is closely related to the increase in the diastolic volume of the left ventricle

Diastolic Heart Failure

- Causes of DHF:
    - Abnormalities of myocardial relaxation
        - Ischemia
        - Hypertrophy
        - Long-standing essential hypertension
        - Valvular heart disease eg. progressive aortic stenosis
    - Abnormalities of myocardial compliance
        - Aging
        - Fibrosis
        - Hypertrophy
        - Diabetes mellitus
        - Metabolic syndrome
        - Infiltrative disorders – amyloidosis
        - Cardiomyopathies
        - Constrictive pericarditis
        - Pressure overload
- Symptomatic heart failure in patients with normal or near-normal LV systolic function is most likely due to diastolic dysfunction
- DHF may co-exist in patients with SHF
- DHF can be classified into four stages:
    - Class I DHF is characterized by an abnormal LV relaxation pattern with normal left atrial pressure
    - Classes II, III, and IV are characterized by abnormal relaxation as well as reduced LV compliance resulting in an increase in LV end-diastolic pressure (LVEDP)
    - As a compensatory mechanism, the pressure in the left atrium increases so that LV filling can occur despite the increase in LVEDP

(TABLE 6–1 from Coexisting) **-- Characteristics of Patients with Diastolic Heart Failure and Patients with Systolic Heart Failure**

Characteristic	Diastolic Heart Failure	Systolic Heart Failure
Age	Frequently elderly	Typically 50–70 years old
Sex	Frequently female	More often male

Left ventricular ejection fraction	Preserved, ≥40%	Depressed, ≤40%
Left ventricular cavity size	Usually normal, often with concentric left ventricular hypertrophy	Usually dilated
Chest radiography	Congestion ? cardiomegaly	Congestion and cardiomegaly
Gallop rhythm present	Fourth heart sound	Third heart sound
Hypertension	+++	++
Diabetes mellitus	+++	++
Previous myocardial infarction	+	+++
Obesity	+++	+
Chronic lung disease	++	0
Sleep apnea	++	++
Dialysis	++	0

Acute and Chronic Heart Failure

Acute heart failure

- Defined as a change in the signs and symptoms of heart failure requiring emergency therapy
  - Due to a sudden decrease in cardiac output, systemic hypotension is typically present without signs of peripheral edema
  - Acute heart failure encompasses three clinical entities: (1) worsening chronic heart failure; (2) new-onset heart failure such as with cardiac valve rupture, large myocardial infarction, or severe hypertensive crisis; and (3) terminal heart failure, which is refractory to therapy

Chronic heart failure

  - Present in patients with long-standing cardiac disease
  - Typically accompanied by venous congestion, but blood pressure is maintained

Low-Output and High-Output Heart Failure

Low-output failure

- May be difficult to diagnose low-output heart failure because a patient may have a cardiac index that is nearly normal in the resting state but does not respond adequately to stress or exercise
- The most common causes of low-output heart failure are CAD, cardiomyopathy, hypertension, valvular disease, and pericardial disease

High-output failure

- Causes of high cardiac output include anemia, pregnancy, arteriovenous fistulas, severe hyperthyroidism, beriberi, and Paget's disease
- The ventricle fails not only because of the increased hemodynamic burden placed on it but also because of direct myocardial toxicity as caused by thyrotoxicosis and beriberi and myocardial anoxia caused by severe and prolonged anemia

Right Heart Failure

- Certain aspects of the physiology of the RV make it different from the LV
  - Normally the RV free wall receives its blood flow during systole and diastole (LV only during diastole)
  - Systemic hypotension or increased RV systolic and diastolic pressures may cause supply-dependent depression of contractility when myocardial oxygen consumption is increased while coronary perfusion pressure is decreased
  - The normal thin-walled RV is at least twice as sensitive to increases in afterload as is the LV
  - Relatively modest increases in outflow impedence from multiple causes can exhaust preload reserve, causing a decrease in RVEF with ventricular dilation
  - RV pressure overload may be complicated by volume overload caused by functional tricuspid regurgitation
- RV failure can cause LV failure
  - Decreases in RV stroke volume will diminish LV filling
  - Dilation of the RV can cause a leftward shift of the interventricular septum, interfering with diastolic filling of the LV (ventricular interaction)
  - A distended RV limited by the pericardial cavity further decreases LV filling
  - Also, RV failure has the potential to affect LV performance by decreasing pulmonary venous blood flow, decreasing diastolic distending pressure, and decreasing LV diastolic compliance
  - The resulting decrease in LV output will further impair RV pump function
  - Once established, RV failure is self-propagating and aggressive treatment interventions may be needed to interrupt the vicious cycle
- Diagnosis
  - Low CI with increased RA pressure disproportionately compared with changes in left-sided filling pressures
    - The PAOP may also increase because of ventricular interaction but the relationship of RA pressure to PAOP stays close to or higher than 1.0
    - The absence of a step-up in pressure in going from the RA to the pulmonary artery (mean), provided PVR is low, suggests that RV failure is severe and the Rt. side of the heart is acting only as a conduit
    - Venous waveforms are accentuated with a prominent Y-descent similar to constrictive pericarditis, suggesting reduced RV compliance
      - Large V-waves may also be discernible and may relate to tricuspid regurgitation
  - PA catheter: increased end-diastolic volume in association with a decreased RVEF indicates decompensation (but not accurate when TR is present)
  - TEE allows a qualitative interpretation of RV size, contractility, and configuration of the interventricular septum, and can provide a definitive diagnosis of RV dysfunction or failure
    - TEE is also useful to determine if the increased RA pressures open a PFO, producing a R to L shunt
    - In this case, don't use PEEP or larger tidal volumes to treat hypoxemia, as these maneuvers will only inrease the afterload of the RV and potentially increase the shunt and hypoxemia
- Treatment
  - Preload should be increased to the upper range of normal
    - However, the Frank-Starling relationship is flat in RV failure, and to avoid ventricular dilation the CO response to an increasing CVP should be determined
    - Volume loading should be stopped when the CVP exceeds 10 mmHg and CO does not increase despite increases in this pressure
    - If a PA cath is in use, an increase in the end-diastolic volume with unchanged or declining RVEF suggests there will be no advantage to further volume loading
    - The CVP should not be permitted to exceed the PAOP, because if these pressures equalize, any increase obtained in pulmonary blood flow will be offset by decreased diastolic filling in the LV by means of ventricular interdependence
  - Maintain NSR
    - The atrial contribution to RV filling is important when the ventricle is dilated and noncompliant
    - Use atrial pacing if required
  - Attempt to decrease RV outflow impedence

        - Vasodilators may lead to cardiovascular collapse in RV infarction as a result of decreases in RV filling and coronary perfusion
        - However, if PVR is increased and pulmonary HTN exists, pulmonary vasodilation may be beneficial
        - IV vasodilators reduce systemic bp, mandating simultaneous administration of a vasoconstrictor
            - One way to reduce the pulmonary effects of the vasoconstrictor is to administer it through a LA catheter
            - PDE inhibitors are commonly used for their effect on the pulmonary vasculature and RV function, but this also usually requires systemic norepinephrine
            - Aerosolized pulmonary vasodilators (nitric oxide, protaglandin I_2 – epoprostenol or prostacyclin, milrinone) may reduce the undesirable systemic vasodilation
    - Avoid hypercarbia or increased $PaCO_2$
    - IABP may be of substantial benefit by increasing coronary perfusion, even in patients in whom the RV is mainly responsible for circulatory decompensation
    - Right heart assist devices have a place as a temporizing measure in severe intractable failure

PHARMACOKINETICS

- Decreased hepatic and renal clearance secondary to decreased cardiac output and organ perfusion
- Decreased drug distribution secondary to increased sympathetic activity
- Decreased intramuscular absorption secondary to increased plasma norepinephrine
- Decreased GI absorption due to altered regional perfusion
- Volume of distribution changes with changes in extracellular fluid volume and protein binding
- Decreased drug metabolism with visceral congestion

CONSIDERATIONS IN THE PREGNANT PATIENT

- It has been suggested that acute MI complicated by refractory CHF should prompt an early cesarean delivery
    - In two case reports, symptoms of CHF improved promptly after cesarean delivery with eventual resolution of LV dysfunction, suggesting that early C-section may help protect stunned but viable myocardium and thereby prevent further damage

REFERENCES

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- Chestnut Pg. 888