

ANESTHESIA AND CONGESTIVE HEART
FAILURE: PATHOLOGY, MEDICAL,
AND SURGICAL MANAGEMENT

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Abstract

Congestive heart failure (CHF) is increasingly being recognized as a health problem in the United States. It is estimated that the lifetime risk for CHF is 1 in 5. The clinical anesthesiologist can expect to see several cases involving patients suffering from CHF. Because of the danger associated with surgery in a patient with CHF, a thorough knowledge of the disorder and the potential effects on the delivery of anesthetics must be considered. In addition, knowledge of the disease process and its manifestations is required for smooth guidance of the patient through the perioperative period. The understanding of current pharmacotherapies, surgical procedures and their implications related to interactions with anesthetics are all discussed.

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Congestive Heart Failure

Congestive heart failure (CHF) is a common condition with a poor prognosis. In CHF, the heart is unable to pump blood at a rate commensurate with the requirements of the metabolizing tissues or can do so only from an elevated filling pressure. CHF offers a unique set of challenges for the clinical anesthesiologist.

Epidemiology

Congestive heart failure continues to be a growing problem in the United States and worldwide. It has been determined that the lifetime risk of CHF in the general population to be about 1 in 5¹. This frequency appears to remain constant, even with advancing age. At age 40 years, the lifetime risk for CHF was 21.0% for men and 20.3% for women, and at age 80 years, the lifetime risk was 20.2% for men and 19.3% for women². Heart failure is the only cardiovascular condition that is increasing in incidence, prevalence, and mortality³. The prognosis after development of CHF is grim, with a median survival of 1.7 years in men and 3.2 years in women⁴.

Men have a higher predisposition for CHF than women. Male sex, less education, physical inactivity, cigarette smoking, obesity, diabetes, hypertension, valvular heart disease, and coronary heart disease are all independent risk factors for developing CHF⁵. Of these risk factors, coronary disease appears to be the most influential and contributes the most to the higher frequency of CHF in males. In a recent study, half of the patients who developed CHF had coronary disease⁶.

In addition to gender predisposition, ethnic variation also exists for CHF. National statistics indicate that African Americans are disproportionately affected by CHF. They have increased mortality and hospitalizations resulting from heart failure when compared with other racial/ethnic groups⁷. In African Americans, CHF is characterized by a different natural history, more worrisome prognosis, and potential variances in the response to current medical therapy for heart failure⁸. Compared with whites, blacks suffering from CHF are younger in age,

and have a higher prevalence of hypertension, left ventricular hypertrophy, ejection fraction less than 40%, and readmission rate⁹. It is important to consider these racial differences in the evaluation and management of patients with heart failure.

There are several factors that have been positively linked to CHF. Overt heart disease plus age are the principal determinants of the incidence of CHF. Nearly 90% of patients with CHF have systemic hypertension or coronary heart disease, or both, as the antecedent underlying condition¹⁰. Age also seems to play a significant role in CHF because greater than 80% of patients with chronic heart failure are over the age of 65 years¹¹. Many of these patients go undiagnosed. Current smoking is a powerful independent predictor of morbidity (recurrent heart failure and myocardial infarction) and mortality in CHF patients¹². Smoking cessation appears to have a substantial and early effect (within two years) on decreasing morbidity and mortality in patients.

Genetics

Genetics is beginning to play a much larger role in the detection and treatment of CHF, in particular and heart disease, in general. Several genes with risk for heart disease have been identified, such as the angiotensin converting enzyme (ACE) genotype DD¹³. Elucidation of the human genome and the application of gene mapping techniques to rare monogenic cardiovascular syndromes have provided fundamental insights into the pathogenesis of common cardiovascular diseases. Some of the discoveries have been in areas such as hypertension, hypercholesterolemia, cardiomyopathy with and without conduction system disease, cardiac arrhythmias, and most recently, congenital heart disease¹⁴. Replacement gene therapy as well as use of promoter-specific drugs to act on genetic regulatory elements will encompass the future treatment of cardiovascular disease.

Recently, the role of the genetic background in the onset and development of the disease has been evidenced in heart failure, with and without systolic dysfunction, and in familial and non-familial forms of

this condition. Preliminary studies suggest that the polymorphism of the ACE gene influence the development of left ventricular hypertrophy, a major determinant of heart failure¹⁵. The molecular and cellular changes in hypertrophied hearts that initially mediate enhanced function may contribute to the development of heart failure¹⁶. With prolonged hemodynamic overload, gene expression is altered, leading to re-expression of a pattern of protein synthesis analogous to that seen in fetal cardiac development; other changes are analogous to events that occur during mitosis of normally proliferating cells¹⁷.

Pathophysiology

There are several factors that work concurrently in CHF. Pathologically, the heart muscle exhibits progressive changes in myocyte myofilaments, decreased contractility, myocyte apoptosis and necrosis, abnormal fibrin deposition in the ventricle wall, myocardial hypertrophy, and changes in the ventricular chamber geometry (see Table 1)¹⁸. These changes reduce myocardial function and cardiac output (CO) and lead to increased morbidity and mortality. In addition, neurohumoral and inflammatory processes result in a gradual decline in myocardial function.

Table 1
Myocardial changes in Hypertrophy/Failure

Structural
Changes in myocyte shape/arrangement
Myofibrillary reduction in myocyte
Interstitial fibrosis
Permanent change in LV shape
Phenotypic Expression
Change in isoforms of troponin ZT, MLC
Decrease β Adrenergic receptors on muscle cell
Functional
Decreased contractile protein response to calcium
Decreased uptake of calcium ions by sarcoplasmic reticulum

Adapted from: Sheppard M, Davies M. Heart Failure. *Practical Cardiovascular Pathology*. New York, NY: Oxford University Press; 1998: 107.

In many pathologic states, the onset of heart failure is preceded by cardiac hypertrophy, the compensatory response of the myocardium to increased mechanical work. The pattern of hypertrophy reflects the nature of the stimulus. Heart failure occurring as a result of pressure-overloaded ventricles (e.g. hypertension or aortic stenosis) develops pressure (also called concentric) hypertrophy of the left ventricle, with an increased wall thickness and a normal to reduced cavity diameter¹⁷. In contrast, heart failure resulting from volume-overloaded ventricles (e.g. mitral or aortic valve regurgitation) develops hypertrophy accompanied by dilation with increased ventricular diameter.

In addition to the physical changes, CHF is characterized by a complex constellation of neurohumoral and inflammatory processes. Catecholamines cause numerous deleterious effects on the myocardium, including direct toxicity to myocytes, induction of myocyte apoptosis, myocardial remodeling, down-regulation of adrenergic receptors, facilitation of arrhythmias, and potentiation of autoimmune effects on heart muscle¹⁹. Arginine vasopressin is known as an antidiuretic hormone and causes both peripheral vasoconstriction and renal fluid retention²⁰. These actions exacerbate hyponatremia and edema in CHF. Atrial and brain natriuretic peptides are also increased in CHF, and may have some protective effect by decreasing preload²¹. Increased serum levels of endotoxin have been found in many individuals with CHF. It is especially common in those with significant peripheral edema, and has been linked to myocyte apoptosis and release of tumor necrosis factor and interleukins²². Tumor necrosis factor is elevated in CHF and contributes to myocardial remodeling, downregulates the synthesis of the vasodilator, nitric oxide, induces myocyte apoptosis, and contributes to weight loss and weakness in individuals with CHF²³. Interleukin-6 in individuals with severe CHF and cardiogenic shock may also contribute to further deleterious immune activation²⁴.

Gross Pathology of CHF

With the onset of CHF, changes occur in the pericardium, cardiac

chambers, valves, epicardial coronary arteries, conduction system, myocardium, and aorta²⁵. The specific changes are dependent upon the nature of the disease. Most heart failure results from progressive deterioration of myocardial contractile function (systolic dysfunction), as often occurs with ischemic injury, pressure or volume overload, or dilated cardiomyopathy; specifically, hypertension and IHD²⁶. At times, however, failure results from an inability of the heart chamber to relax, expand, and fill sufficiently during diastole to accommodate an adequate ventricular blood volume (diastolic dysfunction). This is seen with massive left ventricular hypertrophy, myocardial fibrosis, deposition of amyloid, or constrictive pericarditis²⁷.

Microscopic Pathology of CHF

In severely hypertrophied hearts, structural remodeling occurs continuously and finally leads to heart failure. The remodeling process involves all structural components of the cardiomyocyte and all protein families, consisting of cellular enlargement accompanied by degeneration and the occurrence of fibrosis²⁸. Nuclei are increased in size but the nuclear volume/cell volume ratio is reduced. Transcription and translation are downregulated for contractile and sarcomeric skeleton proteins but both are upregulated for cytoskeletal and membrane-associated proteins²⁹. Chronic degeneration finally leads to cell death by ubiquitin-related autophagy, and acute ischemic cell death (oncosis) is also observed²⁸. Apoptosis seems to be of minor importance.

Clinical Presentation of CHF

Diagnosis of CHF is a relatively straightforward process because of the clinical symptoms that are manifested when the heart fails as a pump (see Table 2). Evaluation of patients with heart failure should include collecting certain historical facts, documenting pertinent physical findings, and obtaining selected laboratory and specialized diagnostic tests³⁰. Information obtained usually allows identification of the disease

causing the cardiac dysfunction and staging the syndrome's severity, thus facilitating prognostication. Most importantly, the information should guide the appropriate therapeutic administration.

Table 2
Syndromes of Cardiac Heart Failure

<i>Syndrome</i>	<i>Clinical and Pathological Features</i>
Acute Heart Failure	Sudden dyspnea and pulmonary edema Left-sided cardiac disease
Circulatory Collapse	Hypotension, oliguria, poor peripheral circulation, cold blue extremities
i. Cardiogenic	i. Acute left ventricular disease, usually infarction
ii. Peripheral	ii. No cardiac muscle disease
→ Blood Loss	
→ Gram-negative septicemia	
Chronic Heart Failure	Dyspnea, poor exercise tolerance Peripheral edema Cardiac disease or chronic lung disease

Adapted from: Sheppard M, Davies M. Atherosclerosis as a Process. *Practical Cardiovascular Pathology*. New York, NY: Oxford University Press; 1998: 106.

There are several symptoms that are found in CHF that are often not unique to CHF, but together should indicate disease presence. The first of the symptoms common to CHF is dyspnea. Dyspnea is both a hallmark of heart failure and other pathologic conditions³⁰. This "shortness of breath" upon exertion is the most common complaint and generally comes early in the development of left-heart failure. The sensation of difficult, labored, and uncomfortable breathing is actually related to pulmonary congestion, which decreases lung compliance and, subsequently, increases work of respiration³¹. It is caused by elevated left atrial, pulmonary venous and pulmonary capillary pressures, and engorgement of the pulmonary capillary-venous bed. This leads to reduced pulmonary compliance and increases the work of breathing, particularly when physical effort amplifies CO₂³². Dyspnea of CHF should be distinguished from other disorders.

Orthopnea should be explored and quantified as well. As a patient with CHF lies supine, intrathoracic blood volume increases and left ventricular filling pressure rises³⁰. This causes the patient to experience labored breathing when lying relatively flat in bed and finds relief by assuming a more upright position. Initially, breathing at night is made easier by elevating the head on two or more pillows. As heart failure progresses, the patient may have to sleep sitting up³³. Similar in general to dyspnea, orthopnea is not specific for heart failure.

Paroxysmal nocturnal dyspnea is a form of orthopnea and may occur with further progression of left ventricular failure³³. In this terrifying symptom, the patient awakens suddenly, feeling suffocated, a few hours after falling asleep. Though simple orthopnea may be relieved by sitting upright at the side of the bed with legs dependent, in the patient with paroxysmal nocturnal dyspnea, coughing and wheezing often persist even in this position³⁴. The sensation may take 30 to 60 minutes to resolve with the patient seated upright³⁰. It should be noted that paroxysmal nocturnal dyspnea, brought on by increasing wheezing and accumulations of secretions when lying flat, can also occur in patients with advanced chronic obstructive pulmonary disease³².

Cough is often associated with these symptoms of breathlessness. This sensation may be interpreted as a “dyspnea equivalent”, is generally non-productive and occurs in settings in which one would expect to see dyspnea, such as with exertion or at night when the patient is recumbent³⁰. It has become more challenging today, however, to ascribe cough to pulmonary congestion in some patients with CHF because of the use of ACE inhibitors (ACEI) (see Table 3)³⁵. These agents can occasionally induce a dry, hacking, forceful cough as a side effect³⁵. This problem is generally more spontaneous than the heart failure cough, which is more common during exertion or at night³². In the large clinical trial, studies of Left Ventricular Dysfunction, cough was seen in 30.2% of patients of placebo-treated patients³⁶. The tussive effect of heart failure is secondary to bronchiolar congestion with excessive mucous production³⁷. Obviously, cough can be caused by primary lung disease as well.

Table 3
Trials of ACE Inhibitors for CHF

Trial	N	Inclusion Criteria	Therapy	Results
CONSENSUS	253	NYHS IV	Enalapril vs Placebo	31% decrease in mortality at 1 year
V-HeFT-II	804	NYHA II-III	Hydralazine/Iso sorbide vs Enalapril	28% decrease in mortality in enalapril vs hydralazine/ isosorbide attributed to a reduction in incidence of sudden death
SOLVED Treatment	2561	NYHA II-III	Enalapril vs placebo	16% decrease in mortality in enalapril group
SOLCVED Prevention	4228		(same as above)	8% mortality decrease in enalapril group (not statistically significant)
SAVE	2231	NYHA I-III	Captopril vs Placebo	20% decrease in mortality in captopril group
AIRE	2006	acute myocardial infarction with or without CHF sympt	Ramipril vs placebo	27% decrease of all-cause mortality in treatment group
ATLAS	3000	Acute myocardial infarction + clinical evidence of CHF NYHA II-IV	High-vs low-dose lisinopril	8% decrease in mortality in high-dose group (not signif)

Adapted from: Van Bakel A, Meuer R. Congestive Heart Failure: Systolic Dysfunction. *Primary Care Management of Heart Disease*. St. Louis, Illinois: Mosby, Inc; 2000: 266.

Angina pectoris is a common complaint of patients suffering from CHF. As congestion develops, ventricular wall stress increases and myocardial oxygen demand rises. Ischemia, therefore, can be exacerbated during heart failure³⁸. Many patients with severe pulmonary hypertension due to LV failure or mitral stenosis complain of typical exertional angina despite having normal coronary anatomy³⁰. The etiology of this pain is not entirely clear, but may relate to right ventricular ischemia secondary to pulmonary hypertension³⁹. These forms of chest pain are usually exertional in nature.

When patients with CHF lie supine, a redistribution of blood flow occurs and renal perfusion increases⁴⁰. Furthermore, with diminished global oxygen demands, peripheral vasoconstriction becomes less intense and renal perfusion transiently improves³⁰. This can lead to nocturnal diuresis because of the fluid overload. Likewise, urine output decreases when upright because of increased systemic vascular resistance³².

Fatigue and weakness are cardinal complaints in patients with CHF due to low blood perfusion. Those complaints are nonspecific, but in the setting of breathlessness with ventricular dysfunction, they become a critical component of a patient's history⁴¹. Exercise capacity is reduced by the limited ability of the failing heart to increase its output and deliver oxygen to the exercising muscle³⁴.

Some patients with CHF may manifest emotional disorders stemming from their perception of their illness and the limited activity it imposes. Any diminution of cerebral vascular blood flow can cause alteration in mental acuity. Patients, or their close companions, may note confusion, disorientation, or memory impairment³².

Medical Management

The treatment of CHF begins with correction of reversible causes such as systemic hypertension, valvular heart disease, anemia, or beta blockade. When symptoms persist despite correction of reversible causes, the three cornerstones of pharmacological treatment of CHF are digitalis, diuretics (see Table 4), and vasodilators⁴². The present trend in treatment of CHF is to optimize cardiac function by manipulating the peripheral circulation with a vasodilator⁴³. ACE inhibitors are becoming the drug of choice for chronic CHF. Nevertheless, digitalis and diuretics remain the initial therapy for most patients with left ventricular failure, and vasodilators serve as adjunctive drugs⁴⁴.

Table 4
Pharmacological Characteristic of Oral Loop Diuretics

Diuretic	Dosage	Comments
Furosemide	40-400 mg/d	Higher doses required with progressive renal insufficiency
Bumetanide	1-5 mg/d	Risk of ototoxicity relate to peak plasma levels Risk is low with even high doses of these two medications due to prolonged absorption time and lower peak plasma
Torsemide	Half the furosemide dose	May be useful when resistance develops to other drugs
Ethacrynic acid	50-100 mg/d	High ototoxicity rush but can use in sulfa-allergic patients
Metolazone	2.5-20 mg/d	Promotes significant kaliuresis Most often used in combo with loop diuretic
<i>Thiazide diuretics</i>		
Hydrochlorothiazide	25-100 mg/d	Not as potent as loop diuretics
Chlorthalidone	25-200 mg/d	
Chlorothiazide	500-1000 mg/d	
Spirolactone	50-400 mg/d	Efficacy depends on level of aldosterone secreted

Adapted from: Van Bakel A, Meyer R. Congestive Heart Failure: Systolic Dysfunction: Fathman, Elizabeth, eds. *Primary care Management of Heart Disease*: St Louis, Missouri. Mosby, Inc; 2000: 268.

Since patients with CHF almost always have signs and symptoms of pulmonary and/or systemic congestion, diuretic agents play an integral role in the therapeutic approach to this syndrome. Diuretics are usually the first agents used in the treatment of CHF⁴⁵. The goal of diuretic treatment is to promote sodium and water excretion by the kidney. Although there is limited evidence from clinical trials to document their efficacy in patients with advanced CHF and volume overload, their obvious impact as demonstrated by clinical experience has made them a standard mode of treatment⁴⁶. In some instances, diuretic therapy also may increase CO⁴⁷. Cardiac output can be increased with diuretic therapy when a reduction in ventricular volume helps to reduce LV afterload and when relief of pulmonary congestion leads to a reduction to neurohormonal stimuli, which cause peripheral vasoconstriction⁴⁸.

One question that has been raised is whether or not diuretic agents are

also needed in patients who are already receiving an ACEI. Although the data are not extensive, one trial demonstrated that in patients with heart failure and evidence of fluid retention, ACEI therapy alone did not produce adequate diuresis⁴⁹. In this study, the addition of a diuretic agent was required to achieve this. Thus, the effects of diuretics and the ACEIs in patients with CHF and evidence of volume overload appear to be additive.

The diuretics used most often in CHF, for both their potency and low side-effects, are the loop diuretics such as furosemide or bumetanide⁵⁰. Diuretics that act on other portions of the nephron may be used in association with one of the loop diuretics to accomplish specific purposes. For instance, a diuretic like metolazone, which acts on the distal collecting tubule, can be used with a loop diuretic to help promote diuresis in otherwise resistant patients⁵⁰.

Digitalis is the only orally effective positive inotropic drug in common use, having a long history of efficacy (more than 200 years) in the treatment of CHF⁴⁴. Digitalis improves contractility, shifting the ventricular function curve so that at any given level of ventricular filling pressure, more stroke work is generated⁵¹. Digoxin, the most widely used of the digitalis preparations, has well documented inotropic effects and it can block impulse transmission across the AV node⁵². There are more potent inotropic agents, but their use is avoided because they may actually have a deleterious effect on CHF patients⁵³. By blocking the impulse transmission across the AV node, it can provide additional benefits by slowing the ventricular response rate in patients with atrial fibrillation and atrial flutter. The patients who seem to benefit most from digoxin have more chronic and severe heart failure, dilated left ventricle, reduced ejection fraction, and S₃-gallop⁵¹.

The activation of the rennin-angiotensin system plays an important role in the pathogenesis of CHF. In general, elevations in plasma-renin activity correlate with the severity of the condition⁵⁴. A reduction in CO and renal perfusion appears to be a major factor in the process⁵⁵. Patients with asymptomatic LV dysfunction, however, may have increased levels of plasma-renin activity, particularly if they are being treated with diuretic agents⁵⁵. The adverse consequences of activation of the rennin-

angiotensin system provide a rationale for the use of ACEIs in the treatment of CHF.

As a result of their ability to dilate peripheral vessels and to reduce the afterload of the heart, the ACEIs have been shown to improve cardiac function⁵⁶. Initially, there is little change in exercise capacity despite the increase in CO and reduction in ventricular filling pressure. However, over a period of weeks to months, exercise duration has been shown to improve in patients with mild to moderate CHF⁵⁷. Long-term therapy has also been shown to improve CHF symptoms. ACEIs have been able to enhance endothelial derived relaxing factor (EDRF) activity in an experimental model of CHF, and this factor could help account for the improved peripheral vasodilation and clinical benefits seen during long-term therapy⁵⁸.

Anesthetic Considerations and Operative Management

Patients with CHF do not make good candidates for elective surgery. The presence of CHF has been described as the single most important factor for predicting postoperative cardiac morbidity⁵⁹. Evidence of left-sided heart failure is associated with a poor prognosis. Patients with ejection fractions of less than 40 percent, determined by radioisotope imaging, have a 1-year cumulative mortality of 30 percent⁶⁰. If surgery is necessary, the drugs and techniques chosen to provide anesthesia are selected with the goal of optimizing CO.

Clinical anesthesiologists are commonly involved in the management of patients treated with ACEIs. The role of ACEIs and their possible interaction with anesthetic agents must be an integral part of clinical decision-making during anesthesia. Hemodynamic variation during anesthesia is mainly related to specific effects of anesthetic agents on the sympathetic nervous system⁶⁰. Those patients with volume depletion and extended sympathetic blockade can have reduced vascular capacitance resulting in decreased venous return, reduced CO and severe arterial hypotension⁶⁰. Normally, Angiotensin II (AII), a potent vasoconstrictor, balances this effect. However, during ACE inhibition,

AI cannot counterbalance this effect. Practitioners should be vigilant and readily have vasopressors, necessary fluids, and other resuscitative measures for treatment of unexpected hemodynamic instability during anesthesia and surgery.

The use of diuretics, especially spironolactone, can also raise certain concerns for the clinical anesthesiologist. Spironolactone, being used as a potassium-sparing diuretic, should be carefully monitored before surgery. Serum potassium concentration should be measured immediately before operation to detect hyperkalemia in heart failure patients treated with spironolactone. renal insufficiency, advanced age, potassium supplementation, decompensated CHF, and a spironolactone dose larger than 25 mg/d increase the risk of hyperkalemia as a consequence of spironolactone therapy⁶¹.

The debate whether regional anesthesia is preferable to general anesthesia for patients with congestive heart failure undergoing non-cardiac surgery remains ongoing. Several studies have been conducted comparing the outcomes of general versus regional anesthesia⁶². The results show no statistically significant difference between groups (general versus regional) in incidence of combined cardiac events, death, myocardial infarction, unstable angina, or CHF⁶².

Administration of a volatile anesthetic must be done cautiously, in view of the dose-dependent cardiac depressant effects produced by these drugs⁶². The cardiac depression produced by the combined effects of a volatile anesthetic and CHF is greater than that effect in the absence of CHF⁶³. Opioids, benzodiazepines, and etomidate are acceptable considerations, since they produce little or no direct myocardial depression⁶². It must be remembered, however, that the addition of nitrous oxide to opioids or the combination of benzodiazepines and opioids is associated with significant depression of CO and blood pressure⁶⁴. Conversely, nitrous oxide added to diazepam does not seem to produce cardiac depression⁶⁵. In the presence of severe CHF, the use of opioids as the only drug for maintenance of anesthesia may be justified⁶². Monitoring is adjusted to the complexity of the operation.

A study was performed to test the cardiovascular effects of volatile anesthesia in patients with CHF⁶⁶. The effects of isoflurane and halothane

anesthesia on CHF were studied. In controls, each anesthetic agent produced dose-related decreases in mean arterial pressure and increases in heart rate, but not significant changes in CO⁶⁶. As CHF developed, there was an attenuation of the heart rate response to anesthesia⁶⁶. Halothane, but not isoflurane, significantly reduced CO in more advanced stages of CHF. Administration of the ACEI, enalaprilat, reversed the CO effects of halothane when the combination of halothane and enalaprilat resulted in severe circulatory depression⁶⁶.

Drug interactions in patients treated with digitalis should be anticipated. Any drug that can abruptly increase parasympathetic nervous system activity, could theoretically have additive effects with digitalis⁶². Nevertheless, clinical experience does not support the occurrence of an increased incidence of cardiac dysrhythmias in patients treated with digitalis and receiving succinylcholine⁶⁵. Sympathomimetics with beta agonist effects, as well as pancuronium, may increase the likelihood of cardiac dysrhythmias in patients treated with digitalis⁶².

Although propofol is commonly used to induce and maintain anesthesia and sedation for surgery, systemic hypotension and reduced CO can occur in patients with or without intrinsic cardiac disease⁶⁷. This is a significant consideration in the patient with CHF. Propofol reduces total arterial resistance and increases total arterial compliance derived from aortic impedance in CHF patients⁶⁸. The myocytes in the CHF patient are more sensitive to the negative effects of propofol on velocity of shortening. In general, propofol has a direct and negative effect on myocyte contractile processes in the setting of CHF. This is more pronounced than that on healthy myocytes at reduced propofol concentrations⁶⁷. Etomidate is the drug of choice for induction. Ketamine may also be used for the induction of anesthesia in patients with CHF. However, as reported previously, the clinical anesthesiologist must remember that volatile anesthetics should be administered cautiously because of the dose-dependent cardiac depression associated with these drugs⁶⁹.

Regarding operative management of the patient with CHF, the medical community has witnessed an increase in available surgical procedures. Despite the advances in inotropic medications, the surgical management of heart failure is the fastest growing aspect of

cardiovascular surgery⁷⁰. Thus, when medical therapy is not successful in improving the functional class in heart failure patients, surgical alternatives must be examined⁷¹.

There is a wide array of surgical options available for the treatment of CHF. Such procedures range from traditional coronary artery bypass grafting to a total artificial heart transplant (see Table 5). The operation chosen depends on the severity of the disease and the preference of the patient. In addition, some surgeries are indicated for patients with moderate disease and prevent progressive heart failure, while other procedures are performed on patients who will only survive with high-risk surgery⁷².

Table 5
Patient Selection for Cardiac Transplantation

<p>End-Stage Congestive Heart Failure:</p> <ul style="list-style-type: none"> - Cardiogenic shock or low output state requiring mechanical assistance - Refractory heart failure or low output state requiring continuous inotropic support - NYHA (New York Heart Association) (Class III or IV heart failure in ambulatory patients of marked functional limitation and poor 12-month survival) <p>Refractory angina pectoris is not responsive to maximal medical therapy and not suitable for revascularization:</p> <ul style="list-style-type: none"> - Severe ischemic symptoms limiting daily activities - Objective evidence of ischemia within the first two stages of a Bruce exercise - Recurrent hospitalizations for unstable angina - Life-threatening coronary anatomy <p>Severe hypertrophic or restrictive cardiomyopathy with NYHA Class IV symptoms</p> <p>Recurrent symptomatic life-threatening ventricular arrhythmias uncontrolled by all appropriate medical and surgical procedures</p> <p>Cardiac tumors with low likelihood of metastases</p>

Adapted from: Van Bakel A, Meyer R. Congestive Heart Failure: Fathman, Elizabeth, eds. *Primary Care Management of Heart Disease*: St Louis, Missouri. Mosby, Inc; 2000: 273.

The clinical anesthesiologist must be able to identify which class, based on The New York Heart Association (NYHA) classification, the patient with CHF is in. This system is the most commonly used to identify

staging of heart failure. It is based completely on the patient's subjective reporting of activity (see Table 6)⁷³. The patient population currently eligible for surgical management consists of those patients with end stage heart disease (NYHA-functional class IV), a predicted life expectancy of less than six to twelve months and an ejection fraction less than 20% on maximal medical therapy⁷³.

Table 6
The New York Heart Association classification

<p>Class I - No limitation and ordinary physical activity does not cause symptoms.</p> <p>Class II - Slight limitation of physical activity and ordinary activity will result in symptoms.</p> <p>Class III - Marked limitation of physical activity and less than ordinary activity will lead to symptoms.</p> <p>Class IV - Inability to carry on any activity without symptoms and symptoms are present at rest.</p>

Adapted from: Delphin ES, Koch T. Anesthesia for the Surgical Management of Congestive Heart Disease: Thys DM, Hillel Z, Schwartz AJ, eds. *Textbook of Cardiothoracic Anesthesiology*. New York, NY: The McGraw-Hill Companies, Inc; 2001: 743.

As alluded to previously, the goal of the clinical anesthesiologist in preoperative care is to maintain the balance of pharmacologic and medical therapy in an attempt to support the patient's circulatory status until surgery⁷³. In addition, it is important to remember metabolic acidosis, hyperkalemia and hypocalcemia are the most common acid-base and electrolyte disorders seen in low-output states⁷⁴. The medical practitioner, in general, and the clinical anesthesiologist, in particular, should also understand the role of nutritional supplementation. It is known that CHF depletes the myocardium of carnitine, coenzyme Q10 (CoQ10) and taurine and that these substances influence mitochondrial function and cell calcium⁷⁵. Recent literature has indicated that nutritional supplementation containing carnitine, CoQ10 and taurine results in higher levels of these in the myocardium. Further, a reduction in left ventricular end-diastolic volume in patients with left ventricular dysfunction before revascularization was discovered. Thus, because the risk of death for surgical revascularization is related to preoperative left ventricular end-

diastolic volume, supplementation may improve outcomes⁷⁵.

Once the type of surgical procedure has been chosen, the clinical anesthesiologist can prepare the treatment regimen accordingly. For example, heart transplantation is still the most effective therapy for end-stage heart disease. The ten-year survival rate after transplantation approaches 50%⁷⁶. Recent advances, specifically in immunosuppressive therapy, have significantly improved the outcome of cardiac allotransplantation⁷³.

Anesthetic management of the cardiac donor is critical. If care is not optimal, the potential for post-transplantation cardiac dysfunction and failure can increase. As a result of hemodynamic lability following brain death, the clinical anesthesiologist must invasively monitor the donor's arterial pressure and central venous or pulmonary arterial pressure⁷³. Mean arterial pressure must be maintained between 80 to 90 mmHg with the use of vasodilators, vasopressors or inotropes. The urine output should be kept between 100 to 200 mL/h, therefore, volume resuscitation and diuresis may be indicated. Hypothermia and resultant ventricular arrhythmias may develop and warm intravenous fluids can be used to maintain core temperature. Finally, management of ventilation must occur and positive end-expiratory pressure might be required to provide appropriate oxygenation⁷³.

Regarding the anesthetic management of the recipient, the current laboratory data must be obtained and analyzed. Pulmonary artery catheters that allow continuous mixed venous saturation and/or intermittent or continuous cardiac output monitoring can assist in assessing hemodynamic function⁷³. High-dose narcotics or benzodiazepines titrated with cardiostable muscle relaxants (vecuronium or cisatracurium) and 100% oxygen are commonly used during induction. During or following induction, the clinical anesthesiologist should be prepared to use inotropic and vasoactive drugs if hemodynamic depression occurs⁷³.

Implantation of the cardiac allograft is most often performed orthotopically, meaning most, if not all, of the endogenous heart is replaced by the donor graft⁷³. The "new" denervated heart, lacking

autonomic input, still responds to circulating catecholamines. However, without autonomic stimuli, the heart cannot respond to hypotension by increasing heart rate. It must rely on isoproterenol for the chronotropic stimulation. Isoproterenol is also a potent inotrope and dilator of the pulmonary vasculature⁷³.

Postoperatively, the clinical anesthesiologist must remember that rejection is still a significant cause of morbidity and mortality following transplantation. Long-term complications in transplant patients are a direct result of immunosuppressive therapy⁷³. Therefore, since infection remains a constant threat, perioperative and postoperative anti-bacterial prophylaxis are administered. Additionally, the clinical anesthesiologist must recognize that approximately two-thirds of cardiac transplant patients have hypertension as a result of corticosteroid and cyclosporine use. To date, no therapy has been proven effective in the treatment of this form of hypertension⁷³.

Despite the success in treating CHF with heart transplantation, the critical shortage of donor organs is still the limiting factor in heart transplantation⁷³. Since the efforts to increase the supply of donor organs have failed to improve the shortage, alternatives to cardiac allotransplantation have developed. An example of another option is the placement of left ventricular assist devices⁷⁶. These mechanical devices have been used as a bridge to heart transplantation for patients who may otherwise die while waiting for a new heart. Interest has also focused on using these devices to assist patients in a full recovery or for destination therapy, thereby circumventing the need for a heart transplant⁷⁶.

The key objectives in using ventricular assist devices (VAD) are to support and maintain perfusion, support myocardial recovery by decreasing myocardial work and/or optimizing the patient's condition until heart replacement⁷³. The principal indications for the use of mechanical assistance are cardiogenic shock after cardiac surgery or myocardial infarction with cardiac failure secondary to other causes. Two examples of such causes are myocarditis or a rejection crisis in a transplanted heart⁷³.

In general, the types of assist devices can be categorized as providing short-term support or long-term support⁷³. Examples of short-

term support devices are the intraaortic balloon pump (IABP), extracorporeal continuous flow pumps (roller and centrifugal), The Hemopump (Medtronic, Minneapolis, MN) and The Abiomed BVS 5000 (Abiomed Cardiovascular, Inc, Danvers, MA). Long-term support devices include total artificial hearts, The Thoratec ventricular assist device (Thoratec Corporation, Pleasanton, CA), The HeartMate Implantable Pneumatic (IP) and Vented Electric (VE) (Thermo Cardiosystems, Inc, Woburn, MA) systems and the Novacor N 100 (Baxter Healthcare Corporation, Oakland, CA)⁷³.

Following placement of the device, the clinical anesthesiologist must remember that infection remains a significant risk in these patients. Therefore, antimicrobial, antifungal and antibiotic treatment may be indicated. Malignant ventricular arrhythmias also occur at high incidence in patients with VAD support⁷³. The return to a regular rhythm improves hemodynamics and atrial activity decreases the potential for thromboembolic complications. Despite these risks, the use of assist devices have proven to be effective and reliable in the treatment of patients with end-stage CHF⁷³.

The partial left ventriculectomy (PLV) is another surgical procedure available for patients with CHF. PLV has been advocated as a surgical alternative for certain patients with dilated cardiomyopathy (see Table 7)⁷⁷. A study performed in 2001 revealed that among other noteworthy improvements, left ventricular ejection fraction increased after this type of surgery from 14.1% +/- 4.7% to 24.1% +/- 3.1% ($p < 0.05$, t-test). This result persisted up to three years following the operation. Thus, results of PLV compare favorably to reports of similar groups of patients treated with medical therapy alone⁷⁷.

Table 7
Different Cardiomyopathies

Specific	Primary Myocardial
Ischemic	Dilated
Valvar	Hypertrophic
Hypertensive	Restrictive
Inflammatory (myocarditis)	Obliterative
Systemic disorders	ARV cardiomyopathy

Adapted from: Sheppard M, Davies M. The cardiomyopathies. *Practical Cardiovascular Pathology*. New York, NY: Oxford University Press; 1998: 109.

The PLV was recently introduced by Batista and coworkers⁷³. The surgical procedure attempts to decrease left ventricular dimensions and consequently volume. In addition to left ventricular volume reduction, the elimination of mitral regurgitation (see Table 8) with repair or replacement of the mitral apparatus may occur. The standard technique now is to place the heart in cardioplegic arrest for ventricular resection⁷³.

Table 8
Mechanisms of Mitral Regurgitation

Cusps
Perforated
Bacterial endocarditis
Retracted
Chronic rheumatic disease, SLE
Expanded
Floppy valve
Chordae
Short
Chronic rheumatic disease
Long/broken
Floppy Valve
Papillary Muscle
Rupture
IHD
Fibrosed
IHD, Cardiomyopathy
Ring Dilated
Marfan's Disease
Ring Rigid
Mitral ring calcification

Ventricle Dilated
Functional mitral regurgitation

Adapted from: Sheppard M, Davies M. Atherosclerosis as a Process. *Practical Cardiovascular Pathology*. New York, NY: Oxford University Press; 1998: 19-20.

The anesthetic management of the patient undergoing a PLV is very similar to that of the patient receiving a cardiac transplant or left ventricular assist device insertion. Peripheral and pulmonary catheters should be placed and routine monitoring performed. Vasopressors and inotropic support may also be required. To prevent excessive stress on the ventriculectomy suture line, mean arterial pressure should be maintained between 60 to 70 mmHg and volume overload avoided in the ventricle⁷³.

Recent literature has indicated that although the Batista operation is mainly performed for non-ischemic dilated cardiomyopathy, the procedure was very effective when applied to ischemic dilated cardiomyopathy⁷⁸. Another study found that early survival in patients receiving a PLV for dilated cardiomyopathy is significantly affected by the amount of myocardial inflammation. Patients with more severe or continuous inflammation may have poor clinical outcomes. Thus, chronic myocarditis could play a vital role in the etiology and pathophysiology of idiopathic dilated cardiomyopathy⁷⁹. In addition, recent data have concluded that cardiomyocyte apoptosis and proliferation occur in a large number of patients with idiopathic dilated cardiomyopathy. Increased numbers of apoptotic cardiomyocytes and apoptotic interstitial cells are significantly related to a poor late outcome following PLV⁸⁰.

In closing, the PLV could become a preferred strategy implemented as a bridge to mechanical assist device implantation, transplantation, or possibly an alternative to transplantation⁷³. However, regardless of the surgical procedure chosen to treat the CHF patient, one thing seems probable. The future role of surgical treatment in CHF will be strongly affected by the advent of gene therapy, cell therapy and engineered

artificial myocardial tissue⁸¹. It is paramount that the clinical anesthesiologist remains knowledgeable concerning the surgical treatments available to the patient with CHF.

Conclusion

A clinical anesthesiologist will encounter patients with CHF. Systemic effects and current therapy create unique problems in properly anesthetizing these patients. Careful preoperative assessment and appropriate monitoring are necessary to avoid potential complications. Thorough history taking, emphasis on physical examination and medication profile are all essential in planning, maintaining and providing the highest level of safety, care and comfort for this patient population.

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