PERIOPERATIVE MANAGEMENT OF HEART FAILURE: THE NEW EPIDEMIC

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Heart failure (HF) is a progressive clinical syndrome involving impaired ventricular function, exercise intolerance, ventricular arrhythmias and reduced life expectancy, which affects 0.4 to 2% of the world’s adult population. Despite great advances in deciphering the pathophysiology of HF, and developing drugs to slow this pathophysiology, morbidity and mortality of HF remain high. Patients with NYHA class IV symptoms have a 1-year mortality of 30-50%, while it is 5% for class I-II. Heart failure can be broadly subdivided into two distinct forms, and distinguishing between the two forms is often difficult. **Diastolic dysfunction** or diastolic heart failure is due to inadequate ventricular relaxation preventing adequate end-diastolic filling (HF with preserved systolic function). This type of heart failure affects the left ventricle (LV).

The second is the more common **systolic dysfunction** or systolic heart failure due to inadequate force generation to eject blood normally. This type of heart failure can affect either ventricle but failure of the left heart is more common.

A four-stage classification of HF is recently recommended by the ACC/AHA to complement the widely-used NYHA classification. The ACC/AHA classification highlights the progressive nature of HF and the importance of neurohormonal antagonism in trying to delay its progression. The NYHA classification, which is based on the severity of symptoms, has a strong correlation with mortality and quality of life.

**Epidemiology.** The incidence and prevalence of HF increase with age and male gender. About 3% of the population segment aged 65-74 have HF, rising to 9% in those over 80. The recent increase in the prevalence of HF to an epidemic scale is attributed to an aging population, and improved survival of patients with heart disease that eventually leads to HF, such as hypertension and coronary artery disease. The population segment aged >65 years is increasing in many nations. Given the high incidence of HF in this age group, these changing demographics indicate the future scale of the problem. In Egypt, the life expectancy at birth has increased from about 50 years in the early 1970s to about 70 years in 2005. The population segment aged 65 years and above is currently about 4% and is expected to increase to 5.4% by 2015.
**Pathophysiology**

HF may be the end result of hypertension, coronary artery disease, valvular heart disease, cardiomyopathy, as well as a variety of other conditions. Other risk factors include male gender, diabetes mellitus and advanced age. A combination of changes in size, shape and function of the LV, commonly termed LV Remodeling (or Remodelling [UK]), is a key event in the development of HF, leading to progressive impairment of function, and ultimately resulting in reduced contractile function and ejection fraction.

In the human heart, aging is associated with a progressive reduction of cardiac myocytes accompanied by a reduction in maximal heart rate and cardiac output.

Interstitial fibrosis results in increased stiffness, and greater contribution of atrial contraction in LV filling. LV pressures are increased because of increased stiffness and delayed relaxation. Reduction in COP (secondary to myocardial injury or aging), results in neurohumoral stimulation mainly of the sympathetic nervous system and the Renin-Angiotensin-Aldosterone system (RAAS), with secondary changes in endothelin receptors, natriuretic peptides and tumor necrosis factor receptors.

After initial improvements, neurohumoral stimulation compounds cardiac injury in HF by stimulating **LV remodeling** and systolic dysfunction. This is related to changes in afterload, preload, stretch, increased wall tension, collagen deposits and direct toxic effects. Myocellular hypertrophy, cell slippage, and sarcomere growth result in chamber enlargement. Increased wall tension and myocardial oxygen consumption, together with reduced subendocardial perfusion, combine to reduce myocyte shortening and with remodeling, the LV dilates and becomes more spherical and less elliptical. The neurohormonal responses to impaired cardiac performance (sympathetic stimulation, salt and water retention, and vasoconstriction) are at first adaptive but later become maladaptive, resulting in pulmonary congestion and excessive afterload. Finally there is a vicious cycle of increased and inefficient cardiac energy expenditure exacerbated neurohumoral stimulation and worsened pump function and tissue perfusion.

**Management**

The drug therapy of **chronic HF** has evolved dramatically during the last 20 years, where digoxin has moved to the end of the list, Angiotensin Converting Enzyme (ACE) inhibitors are first line drugs, and β-adrenergic blockers that were classically contraindicated, are now known to reduce mortality and symptomatology. The rationale is towards antagonism of the neurohumoral changes that initiate, perpetuate and aggravate ventricular dysfunction.

On the other hand, the treatment of **acute, decompensated HF** did not change much in the last 20 years, probably due to the failure of the current therapies in protecting the
myocardium from further damage. This may lead to a shift in therapeutic paradigms of acute HF to include cardioprotection as a therapeutic target and not only focus on hemodynamics.

**Disease modifying therapy of chronic HF** includes treatment with ACE inhibitors, ß-adrenergic blockers (carvedilol, bisoprolol, metoprolol), Aldosterone antagonists, Angiotensin II receptor blockers (where ACE inhibitors are contraindicated), Diuretics (symptomatic relief), and Digoxin (mainly for patients with atrial fibrillation). Additional pharmacological therapies, of which some have debatable roles include aspirin, warfarin, statins, erythropoietin and iron (anemia). Lifestyle modification (smoking, alcohol), aerobic exercise, fluid and salt restriction are also of benefit.

**Non-transplant surgical and device options** include revascularization of hibernating myocardium, Ventricular Restoration Surgery (Left Ventricular Reshaping) or the Dor Procedure (LV becomes more elliptical), correction of mitral regurge, cardiac support devices that are intended to arrest progressive LV dilatation by external constraint (Acorn CorCap, and Heart Net), Cardiac Resynchronization Therapy, AICD, and Ventricular Assist Devices.

**Anesthetic Considerations**

A comprehensive preoperative evaluation of coronary, valvular and myocardial pathology and the presence of pulmonary hypertension, is needed for ensuring hemodynamic stability during induction and maintenance of anesthesia. The anesthetic plan should take into account other extracardiac comorbidities (hepatic, renal, cerebral).

**Preoperative optimization** (fine tuning) is usually achieved by afterload reduction, and ß-adrenergic blockers, and diuretics. Therapeutic choices should be individualized based on data from clinical trials. ACE inhibitors inhibitors deserve special consideration owing to reports of refractory hypotension during anesthesia, associated with their use.

Withdrawal of ß-adrenergic blockers from patients chronically treated with them may be dangerous. ß-adrenergic blockers have definite benefits in certain high risk populations to protect from and treat hypertension, ischemia and arrhythmia in the perioperative period. However, they may have deleterious effects in other patient populations.

Diuretics should be titrated to “normalize” intravascular volume and relieve pulmonary congestion and peripheral edema, while trying to avoid hypovolemia and hypokalemia especially in elderly patients. Perioperative discontinuation of digoxin could be justified because of the long half life and narrow therapeutic margin. Preoperative anxiolytic/sedative medication should be chosen cautiously to avoid hypoxemia, respiratory acidosis, increased pulmonary vascular resistance, and reduction of sympathetic tone.
Currently there are no data to support the choice of specific anesthetic agents or techniques. The choice of anesthetic drugs seems to be less important than management of fluids, medical therapy, transfusion choices, pacemaker and aortic balloon adjustments, to achieve sound hemodynamic goals. Nevertheless, large doses of myocardial depressant and vasodilator anesthetics should be avoided. High concentrations of inhalational agents are poorly tolerated. Sevoflurane, and Isoflurane could be used to supplement opioids.

Invasive hemodynamic monitoring and transesophageal echocardiography (TEE) should be used. TEE is especially superior in monitoring volume status, compared to intracardiac pressure measurement in this patient population with abnormal and changing ventricular pressure/volume relationship (compliance).

Cardiac surgical procedures intended to improve cardiac function usually involve cardiopulmonary bypass. During the prebypass period, optimization of preload, afterload, inotropic state and cardiac rhythm is essential to avoid deterioration of function, increase oxygen demand and myocardial ischemia.

Adequate postoperative pain management is needed to avoid tachycardia, stress and increased oxygen consumption, which may not be tolerated by patients with markedly impaired LV function. Systemic opioids, epidural techniques, or intrathecal opioids are valid options for this patient population.

Further reading: