Best Practice & Research Clinical Anaesthesiology 26 (2012) 91–104

Contents lists available at SciVerse ScienceDirect

Best Practice & Research Clinical Anaesthesiology

journal homepage: www.elsevier.com/locate/bean

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Heart failure and mechanical circulatory support

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Keywords:
heart failure
mechanical circulatory support
ventricular assist devices
anaesthesia

Cardiovascular disease (CVD) is defined as one of the following: hypertension, congestive heart failure (HF), stroke, coronary heart disease and congenital heart defects. CVD is the main cause of the disease burden (illness and death) in Europe (23% of all the disease burdens) and the second main cause of the disease burden in those European Union (EU) countries with very low child and adult mortality (17%).[1] Heart disease is a common health problem worldwide. According to the most recent Heart Disease and Stroke Statistics-2011 update,[2] greater than 82 000 000 adults living in the United States of America (USA) have one or more types of CVD. Many resources have been invested in attempting to understand and curtail the progression of congestive HF. This article attempts to address the growing concern over HF by looking at the epidemiology, pathophysiology and available therapies as anaesthesiologists encounter these patients more often nowadays in the operating room and intensive care units. Mechanical circulatory assistance and heart transplantation are two established treatment methods for end-stage HF. In this review, we also address the indications and contraindications for mechanical circulatory assistance, types and spectrum of available ventricular assist devices, efficacy, safety and cost analysis of circulatory support therapy.

Epidemiology of heart failure

Cardiovascular disease (CVD) is the number one cause of death among women and men in Europe. It accounts for nearly half of all deaths in Europe, causing over 4.3 million deaths in Europe every year. In

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doi:10.1016/j.bpa.2012.03.003

the European Union (EU) alone, CVD causes over 2 million deaths every year. CVD is estimated to cost the EU economy more than €192 billion each year.[3] More than 2200 people living in the United States of America (USA) die from CVD each day, which is an average of one death every 39 s[4] The number of medical visits for heart failure (HF) in 2007 exceeded 3.4 million.[5] Of individuals in the USA who suffer from CVD, 5.7 million above the age of 20 years have been diagnosed with diastolic and/or systolic HF. Of those individuals, 3.1 million are male and 2.6 million are female. The incidence for both sexes combined is greater than 600 000 and all mortality is greater than 56 000 people. Furthermore, Rogers et al. reported that HF was mentioned on 277 193 death certificates in 2007 (one in nine deaths).[2] This is similar to the data reported from the National Heart Lung and Blood Institute (NHLBI) and the National Center of Health Statistics (NCHS) from 1995 to 2006, with mortality numbers of 287 000 and 283 000, respectively.

Levy et al., using the data from the Framingham Study, evaluated the incidence of HF and the survival after its onset during four defined time periods.[6] Although the incidence of HF has declined among women, there has been no change among men. They also found that the survival after the onset of HF has improved in both sexes over time. A similar conclusion was reached by Rogers et al.[7] However, both studies report a high death rate of approximately 50% within 5 years of HF diagnosis. Between the years 1970 and 1993, HF, as the contributing factor for death, increased by an average of 10 000 patients per year.

The incidence of HF approaches 10 per 1000 population after 65 years of age and hypertension precedes this syndrome in 75% of cases. As individuals age, there is a greater risk of developing HF symptoms. New HF events have been reported in 15.2/1000 population at age 65 years. In population older than 85 years, HF rates are in excess of 65 per 1000.

After studying a cohort of greater than 6000 patients, Bahrami et al. showed that there is a greater risk of HF among African Americans and Hispanics in USA, generally related to a higher prevalence of diabetes mellitus, hypertension and differences in socioeconomic status. In addition, it was noted that myocardial infarction (MI) was least likely to be the cause of HF in the African American population, while an increase in left ventricular mass had a significant effect on Hispanics and Caucasians.[8] It should be noted that in the Atherosclerosis Risk in Communities (ARIC) study of NHLBI, Loehr et al. described a higher incidence of atherosclerotic risk factors in African Americans as compared with Caucasians.[9]

Pathophysiology of HF

Congestive heart failure (CHF) does not have one cause. There are multiple structural, mechanical and biologic mechanisms that work in tandem to precipitate HF. HF can be designated as systolic or diastolic. The characteristics of the two types of HF are described in Table 1. The current clinical definition of systolic HF taken from a paper published by Davis et al. (2006) is "a clinical syndrome associated with congestive symptoms and/or symptoms of low cardiac output due to impaired ventricular pump function or reduced ejection fraction."[10] Diastolic HF is more controversial in its definition. Satpathy et al. used the following definition: Diastolic HF occurs when signs and symptoms of HF are present but left ventricular systolic function is preserved with an ejection fraction of greater than 45%.[11] Left ventricular systolic dysfunction is the most common cause of HF, occurring in about 60% of patients, the majority of whom have coronary artery disease (CAD), with or without a history of MI.

There are different types of cardiomyopathies that can cause HF. Dilated cardiomyopathy is defined as 'a diminution in the contractile force of the left ventricle (LV) in the absence of pressure overload, volume overload or CAD. The loss of cardiac muscle function results in HF'. Hypertrophic obstructive cardiomyopathy, also referred to as 'idiopathic hypertrophic subaortic stenosis or asymmetric septal hypertrophy' is a disorder in which there is excessive hypertrophy of the interventricular septum. As a result, the septum and the anterior leaflet of the mitral valve can produce LV outflow obstruction, called 'systolic anterior motion of the mitral valve' which can cause a decrease in ejection from the LV, leading to HF. Dilated cardiomyopathies are more common.

HF can occur acutely or chronically. In some cases, an acute exacerbation can be superimposed on chronic HF. The normal heart operates based on the Frank–Starling curve, which was elucidated by Otto Frank and Earnest Henry Starling.[12–15] The Frank–Starling mechanism is 'the ability of the heart to