Progress in Cardiovascular Diseases 54 (2011) 115–131
www.onlinepcd.com

Surgical Treatment of Advanced Heart Failure: Alternatives to Heart Transplantation and Mechanical Circulatory Assist Devices

Timothy J. George, George J. Arnaoutakis, Ashish S. Shah*

Division of Cardiac Surgery, The Johns Hopkins Medical Institutions, Baltimore, MD 21287

Abstract Although orthotopic heart transplantation is the gold standard for definitive surgical treatment of end-stage heart failure, other operative therapies exist for dealing with severe systolic left ventricular dysfunction. The choice of surgical intervention depends on the etiology and functional characteristics of the patient's ventricular dysfunction. In patients with ischemic cardiomyopathy, surgical revascularization improves survival. Patients with mitral regurgitation experience significant functional improvement from mitral valve repair and replacement. In patients with aortic valve dysfunction, aortic valve replacement results in improved survival and functional status. Although surgical ventricular reconstruction is controversial, significant data exist suggesting that it is an effective therapy in a subset of patients with left ventricular dysfunction. Finally, passive restraint devices are effective at halting further ventricular dilation. Although cardiac surgery in patients with severe ventricular dysfunction can be complicated by significant morbidity and mortality, experienced centers have demonstrated acceptable outcomes in carefully selected patients. (Prog Cardiovasc Dis 2011;54:115-131)

Keywords: Heart failure; Ventricular dysfunction; Surgery

Although much progress has been made in the medical treatment of heart failure (HF), optimal medical therapy remains associated with significant morbidity and mortality. The ultimate treatment of end-stage HF is orthotopic heart transplantation. Unfortunately, organ shortages and strict exclusion criteria severely limit the use of this lifesaving intervention. Moreover, the immunosuppression required to maintain allograft function places patients at risk of infection, sepsis, and death. Mechanical circulatory support systems, particularly ventricular assist devices (VADs), offer another surgical solution. Despite improvements in this technology, VAD therapy remains an expensive solution with its own significant morbidity and mortality, as well as a potential need for lifelong anticoagulation.

Nevertheless, other surgical options are available for patients with HF. Although patients who suffer from the clinical syndrome of HF may have different types and degrees of cardiac dysfunction, it is patients with severe left ventricular systolic dysfunction who form the basis of this review. Depending on the etiology of their ventricular dysfunction, patients can benefit from coronary artery bypass grafting (CABG), mitral and aortic valve (AV) interventions, surgical ventricular reconstruction, and passive restraint devices.

Coronary artery bypass grafting

Coronary artery disease (CAD) is the most common cause of HF.[1] Even with optimal medical therapy, patients with ischemic cardiomyopathy (ICM) can have a 1-year survival as low as 54%.[2,3] Physicians have long

Statement of Conflict of Interest: see page 127.

* Address reprint requests to Ashish S. Shah, MD, Associate Professor of Surgery, Division of Cardiac Surgery, The Johns Hopkins Hospital, 600 N. Wolfe St., Blalock 618, Baltimore, MD 21287.
E-mail address: ashah29@jhmi.edu (A.S. Shah).

0033-0620/$ – see front matter © 2011 Elsevier Inc. All rights reserved.
doi:10.1016/j.pcad.2011.04.001

Abbreviations and Acronyms

AR = aortic regurgitation

AS = aortic stenosis

AV = aortic valve

AVA = aortic valve area

AVR = aortic valve replacement

CABG = coronary artery bypass grafting

CAD = coronary artery disease

CMRI = cardiac magnetic resonance imaging

DCM = dilated cardiomyopathy

DSE = dobutamine stress echography

EF = ejection fraction

FDG-PET = 18-fluorodeoxyglucose positron emission tomography

HF = heart failure

IABP = intraaortic balloon pump

ICM = ischemic cardiomyopathy

LCOS = low cardiac output syndrome

LGAS = low-gradient aortic stenosis

LV = left ventricle

LVEDV = left ventricular end-diastolic volume

LVEF = left ventricular ejection fraction

LVESVI = left ventricular end-systolic volume index

MI = myocardial infarction

MR = mitral regurgitation

MV = mitral valve

MVR = mitral valve repair

NYHA = New York Heart Association

PLV = partial left ventriculectomy

PSAS = pseudosevere aortic stenosis

SPECT = single photon emission computed tomography

STICH = Surgical Treatment of Ischemic Heart Failure

SV = stroke volume

SVR = surgical ventricular reconstruction

TAVI = transcatheter aortic valve implantation

TSAS = true severe aortic stenosis

VAD = ventricular assist device

recognized the theoretical benefit of revascularization; however, early experiences with CABG in patients with poor ejection fraction (EF) were associated with poor outcomes.[4] Impaired left ventricular (LV) function is an independent predictor of mortality after cardiac surgery[5] and has been shown to adversely affect both short- and long-term survival after CABG.[6] In 1984, the Veterans Affairs Cooperative Study Group published the first large randomized clinical trial demonstrating the superiority of CABG over medical therapy in the treatment of CAD.[7] Since that time, an increasing understanding of the pathophysiology underlying ICM and multiple trials of CABG in patients with poor EF has demonstrated significant improvements in the morbidity and mortality of patients with LV dysfunction.

Pathophysiology and hibernating myocardium

Although myocardial damage caused by CAD was historically considered irreversible, more recent observations demonstrate that this is not always the case. Myocardial dysfunction noted preoperatively has been observed to improve after revascularization.[8] Moreover, inotropic stimulation can evoke functional movement from dysfunctional myocardium.[8] The observation that some dysfunctional myocardial tissue is actually viable led to the theories of myocardial stunning and hibernation. *Myocardial stunning* is a transient, hypocontractile state after temporary ischemic injury in which viable myocytes regain contractile function spontaneously.[9] *Myocardial hibernation* refers to viable myocytes that are hypocontractile secondary to ongoing ischemia; these myocytes remain poorly functional until the oxygen supply and demand imbalance can be rectified, usually by revascularization.[8,9] After revascularization, stunned myocardium demonstrates early recovery of function without further improvement.[10] In contrast, hibernating myocardium not only shows some immediate recovery but also demonstrates continued improvement over time.[8,10] Therefore, if surgeons can revascularize nonfunctional but viable tissue, hibernating myocardium can regain contractility, thereby restoring ventricular function and ameliorating the sequelae of HF.

Hibernating or viable myocardium differs from nonviable tissue in terms of its preserved perfusion, ongoing glucose metabolism, intact mitochondria, and cell membrane integrity.[11] These differences can be detected by a number of noninvasive tests. Viability can be ascertained by evoking a response from dysfunctional tissue with inotropic stimulation, catecholamine infusion, or afterload reduction with nitrates while simultaneously scanning with any of the following modalities: thallium Tl 201, technetium Tc 99m sestamibi, single photon emission computed tomography (SPECT), 18-fluorodeoxyglucose positron emission tomography (FDG-PET), or dobutamine stress echography (DSE).[8] More recently, cardiac magnetic resonance imaging (CMRI) has also been used to document viability.[12] No single technique has been definitively shown to be superior (Table 1). Moreover, each technique has advantages and disadvantages, revealing slightly different information about the tissue. Thallium Tl 201 scans have been shown to predict 3-year mortality.[13] Technetium Tc 99m sestamibi scans can predict the degree of improvement in left ventricular ejection fraction (LVEF).[14] Single photon emission computed tomographic scans help identify patients who will have improved LVEF and HF symptoms.[15] Finally, in post–myocardial infarction (MI) patients, DSE has been shown to generate wall motion scores predictive of LVEF improvement.[16] Overall, studies suggest that 50% of