A Heart Failure Specialist's Perspective on Cardiac Surgery for Heart Failure

Carl V. Leier, MD

Address
Division of Cardiology, Davis Heart-Lung Research Institute, The Ohio State University, 473 West 12th Avenue, Columbus, OH 43210, USA.
E-mail: leier-1@medctr.osu.edu

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Aside from cardiac transplantation, ventricular assist devices, and the total artificial heart, cardiac surgery now also plays a major role in the overall management of the heart failure patient. For patients with heart failure, cardiac surgery has steadily moved from being a predominant rescue procedure (eg, aneursymectomy, rupture repair, transplantation) to surgical interventions that can prevent or delay the progression of cardiac dysfunction and failure; these operations now include coronary artery bypass surgery, ventricular restoration, and valvular repair/replacement. This article discusses the role and impact of these specific surgical interventions in the setting of ventricular dysfunction and heart failure.

Introduction

Improved techniques in cardiac surgery, complemented by the development of cardiac anesthesiology, have brought surgical intervention into the arena of heart failure management, thus extending our approach to this condition well beyond pharmacotherapy alone.

Surgery must always be considered in conditions in which a surgically remedial lesion(s) has directly resulted in cardiac decompensation. The clinical scenario is frequently, but not exclusively, one of acute or subacute heart failure. The most common entities encountered in developed societies are occlusive coronary artery disease and valvular heart disease.

A much greater challenge resides in determining whether a surgically approachable lesion is contributing significantly to the decompensation and symptoms of the patient with more chronic cardiac dysfunction and failure and whether the surgical correction of that lesion(s) can improve the patient's clinical condition and outcome beyond the risk of the operation and immediate postoperative course. The decision in this setting is usually complex and is influenced by a host of factors (*eg*, severity of the lesion, degree of ventricular dysfunction, comorbidities). Although consultation with the appropriate cardiac surgeon is always an essential component of the decision, the burden of it usually rests on the shoulders of the managing cardiologist.

This article addresses the more common surgically approachable considerations in chronic heart failure, namely coronary bypass surgery, ventricular restoration surgery, and valvular repair or replacement. Restrictions in manuscript length preclude an in-depth dissertation; such is available elsewhere [1••]. Cardiac or cellular transplantation and mechanical intervention (*eg*, ventricular assist devices, total artificial heart) are not discussed because these considerations are less frequently encountered in the day-to-day practice of cardiology and when in question, the patient should simply be referred to a center that regularly evaluates and applies these methodologies.

First Things First: Recognizing the Surgically Remedial Lesion

With the ready availability of several diagnostic tools (*eg*, myocardial imaging, echocardiography), most cardiologists are reasonably equipped and adept at finding and following the common entities of atherosclerotic coronary artery disease and valvular disorders. However, as one of the heart failure cardiologists at a large referral center for heart failure management, transplantation, and ventricular assist devices, I have encountered a number of surgically remedial conditions that frequently escape detection in the setting of chronic heart failure.

Unexplained decompensation in a patient carrying a diagnosis of nonischemic cardiomyopathy (based on prior coronary angiography showing minimal to no coronary artery disease) can be caused by an occlusive coronary lesion(s). Because many of these patients do not have chest pain with their decompensation and because virtually all standard noninvasive myocardial imaging modalities are still quite limited in detecting the new culprit-lesion in an enlarged failing ventricle, repeat coronary angiography must be considered; this is especially appropriate if the previous angiogram was performed 3 or more years before the decompensation. Most of these

patients improve considerably and return to their baseline status after a revascularization procedure. Repeat angiography is also appropriate for patients with known occlusive coronary disease and recent decompensation, but these patients typically undergo repeat angiography by the managing cardiologist.

Silent aortic stenosis can still escape detection, particularly if the patient's standard echocardiogram is technically inadequate. Physical findings (*eg*, markedly diminished or absent carotid pulses), dobutamine echocardiography addressing the aortic valve, and cardiac catheterization can assist in unraveling this diagnosis as a cause of a patient's cardiac failure. This lesion is discussed in more detail later.

Recent-onset, severe tricuspid regurgitation is not an uncommon cause of cardiac decompensation and merits consideration for surgical repair or replacement. This lesion also is discussed later.

Additional examples include anomalous coronary anatomy (origin of the left coronary artery from the pulmonary artery) and large arteriovenous malformations, but most of these conditions are relatively uncommon in occurrence.

Occlusive Coronary Disease

For patients with occlusive coronary artery disease of a single vessel other than the left main coronary artery, percutaneous coronary intervention (PCI) with stent (drug-eluting) placement is currently the preferred option irrespective of accompanying ventricular function. This approach is especially apropos in patients at high surgical risk (*eg*, prior open heart surgery, age older than 70 years, left ventricular ejection fraction [LVEF] less than 35%, recent myocardial infarction, and requirement for intra-aortic balloon-counterpulsation support) [2]. The recurrence rate of angina pectoris is generally higher in PCI-treated patients compared with those undergoing coronary artery bypass-graft surgery (CABG); however, the data comparing these two options were obtained before the routine use of drug-eluting stents.

For patients with angina pectoris and left main or multivessel occlusive coronary disease, two major randomized trials have shown that CABG, compared with medical therapy, improves survival (out to 10 years) and reduces the recurrence rate of angina and the need for another revascularization procedure [3,4]. Despite a higher operative risk, these trials uncovered the greatest survival benefit with CABG in patients with left ventricular (LV) dysfunction (ejection fraction [EF] 35% to 50%). One can argue that medical therapy was not optimal during the time period of these trials (1970–1990), but neither was CABG, anesthesiology, and postoperative care. Similar trials have not been performed in patients with LV dysfunction without angina, and little data are available regarding the effects of CABG versus medical therapy on LV remodeling, volumes, and EF, which are major determinants of long-term outcomes in patients with LV dysfunction and heart failure.

With the rapidly evolving advances in CABG and PCI, it is difficult to definitively declare the role of each in treating multivessel coronary disease in patients with heart failure. Many of the trials were performed before the use of stents (*ie*, angioplasty only), much less drug-eluting stents, and most of the prospective randomized trials required angina for inclusion and excluded patients with a substantial reduction in EF [5,6•]. Outside of left main coronary disease, trials have yet to demonstrate any major differences in cardiovascular event rates, including survival (out to 2 years) between the CABG and PCI interventions in patients with heart failure and mild to moderate reduction of LVEF (35% to 50%) [2,5,6•,7,8]. Patients at high risk for CABG (elderly, recent myocardial infarction, LVEF < 30%) may be better served with PCI.

Few data are available in patients with LVEF less than 30%, and no data are from prospective randomized trials. A major limb of the STICH (Surgical Treatment for Ischemic Heart Failure) trial is addressing medical therapy alone versus CABG plus medical therapy in patients with operable occlusive coronary artery disease and LVEF less than 35%.

In a small retrospective study of 117 patients with multivessel disease and LVEF less than 30%, Toda *et al*. [9] found CABG reduced cardiac events out to 3 years and increased LVEF compared with PCI; however, the treatment groups were neither randomized nor well-matched. But when risk-adjusted, the patients who were aged younger than 65 years (*ie*, less surgical risk) with high-grade proximal lesions of the left anterior descending artery seemed to fare better clinically with CABG.

For all patients with multivessel coronary disease and heart failure, the clinical response to any revascularization procedure will likely be related to the amount of reversible ischemic myocardium and the extent of revascularization achieved to relieve the ischemic burden [10]. This is particularly important for the patients with the lowest LVEF, which places them at highest risk for PCI or CABG. Myocardial viability studies and assessment of cardiac performance pre- and postprocedure become important aspects of any investigation of interventions for occlusive coronary disease in heart failure. Such evaluations are part of the protocol of the STICH trial.

General conclusions and recommendations

With the profound paucity of powered data, definitive statements and firm conclusions become tempered into recommendations and suggestions. Nevertheless, one can start by stating that successful reversal of substantial myocardial ischemia with PCI or CABG in patients with occlusive coronary disease and resultant heart failure will generally enhance event-free survival. CABG is indicated in patients whose occlusive coronary disease involves the left main or left main equivalent (left anterior descending

plus left circumflex artery) arteries. Although PCI is often reflexly selected for single-vessel coronary disease, CABG using the left internal mammary artery merits consideration in patients aged less than 65 years with high-grade proximal disease of the left anterior descending artery. For the patient with an LVEF less than 30% and proximal occlusive coronary disease involving vessels other than left main or left main equivalent arteries, demonstration of a substantial amount of threatened myocardium is important, along with other factors (*eg*, experience and skill of the surgeon or PCI operator, feasibility of adequate revascularization, the need for another surgical procedure [*eg*, mitral repair], patient's preference), in the decision whether to send the patient into any high-risk coronary procedure. These general recommendations are in agreement with published guidelines of the American College of Cardiology/American Heart Association [11••].

Ventricular Surgery

The natural course of a transmural myocardial infarction is local akinesis or dyskinesis (± aneurysm) and eventual remodeling of the ventricle into an enlarged chamber with global systolic dysfunction. Surgery in this area has previously been directed at aneurysmectomy in patients whose heart failure became refractory to optimal medical management. The role of ventricular surgery in heart failure is now moving from rescue from heart failure to the prevention of such. The development of LV enlargement and dysfunction is unequivocally linked to poor outcome (symptoms and mortality) [12,13]. Clinical data are now available to support surgical correction of regions of dyskinesis, and perhaps akinesis, as a means of blunting the LV remodeling response to infarction, and thus, to avert or at least delay the evolution of ventricular enlargement, global systolic dysfunction, symptomatic heart failure, and death [14–18,19•,20•,21,22]. As a result, the movement afoot is to bring ventricular surgery, now referred to as surgical ventricular restoration (SVR), into a much earlier phase of the heart failure course [17,19•,20•, 22].

Surgical ventricular restoration is most often directed at excluding anterior-apical infarction-dyskinesis (which frequently extends into the distal ventricular septum and inferoapical segment), representing the distribution region of the left anterior descending coronary artery. SVR is almost invariably performed in conjunction with CABG and occasionally with mitral valve surgery. The thrust to bring SVR into heart failure management and even prevention is related to several factors and developments [16–18,19•,20•,21,22]. First, with more surgical experience and advances, the operative mortality rates at experienced centers (< 7%) are becoming acceptable for a major elective operation and should decrease further because the operation is being applied to a less ill population. A major impetus is the quest to surgically restructure the left ventricle back into a more natural, efficient, ellipsoid chamber with an apex. Consistently demonstrable SVR augmentation of LV performance and the general impression of improved overall clinical outcomes with SVR are also major factors fostering SVR as a consideration in select heart failure patients.

The specific techniques applied in SVR vary between institutions and surgeons. The standard aneurysmectomy has largely been replaced by methods to exclude most of the infarct-dyskinetic zone (including septal), reducing LV volume into a reasonable range and restructuring the chamber into an ellipsoid shape with an apex [16–18,19•,20•,21,22]. Use of a ventricular sizing device and placement of a patch (Dor procedure) within the LV chamber facilitate the exclusion of most of the infarcted-fibrotic-dyskinetic myocardium and the attainment of a more ideal chamber size and configuration [16,19•,20•,21,22]. A more detailed description of the SVR procedure itself is available elsewhere [22].

Another stratum of the ongoing STICH trial is addressing the outcome of SVR plus CABG versus CABG alone [19•]. This trial should provide laboratory and clinical outcome data from its prospective, randomized controlled design to correct the dearth of such.

The caveats of SVR include operative and in-hospital mortality rates that are far from benign and the risk of leaving behind the disturbed hemodynamics and heart failure of diastolic dysfunction. Both threats are being reduced as surgical experience and advances evolve over time.

Other surgical methods directed at averting or reversing the enlargement and sphericity of the failing left ventricle are being studied; noteworthy examples include placement of epicardial mesh support and transventricular tension wires [20•].

Mitral Valve Surgery

Innumerable articles have been written addressing the indications and timing of mitral valve surgery for mitral regurgitation. These issues are even more complex and unresolved in the setting of chronic cardiac failure.

Much of the quandary regarding mitral surgery in chronic heart failure resides in the fact that mitral regurgitation can cause and be caused by cardiac failure. Significant mitral regurgitation results in progressive ventricular remodeling, enlargement, and global systolic dysfunction. The ventricular enlargement results in further annular dilatation and reorientation of the papillary-chordal-valvular structures to exacerbate the degree of regurgitation. The hemodynamics of mitral regurgitation in heart failure contribute to the progressive loss of LV systolic function, LV failure, increase of LV diastolic, pulmonary capillary, and pulmonary artery pressures, right heart failure, further impairment of overall cardiac performance, and accentuation of the heart failure syndrome.

Table 1. Clinical and laboratory manifestations of problematic tricuspid regurgitation in chronic heart failure

Otherwise unexplained decompensation of clinically stable chronic heart failure
Subacute development or exacerbation of fatigue (+ dyspnea), malaise, and weakness
Increased somnolence
Evolving cachexia
Prominent V waves on jugular examination
Pulsatile liver ± ascites
Increasing peripheral edema or ascites with increased diuretic requirement and refractoriness, and frequently, striking elevation of serum blood urea nitrogen and creatinine levels as diuretic dosing is advanced
Reduction in serum albumen, increase in prothrombin time and hepatic enzymes
Echocardiographic findings of severe tricuspid regurgitation

In patients whose mitral regurgitation has resulted in or is clearly exacerbating heart failure, mitral surgery becomes an important intervention to interrupt or at least blunt the downhill course of this lesion. The same consideration is appropriate in patients whose LV enlargement and systolic dysfunction is complicated by severe mitral regurgitation; typically, the mitral regurgitation and resultant increased pulmonary artery and right pressures cannot be adequately treated medically (*ie*, pharmacologically) in most of these patients. The intent of surgical intervention is to avert or delay the progression to an end-stage dysfunctional heart and advanced heart failure. Using less than ideal baseline data or historical controls, mitral valve repair appears to improve clinical outcomes in these patient subgroups [23–31]. Surgical in-hospital mortality ranges from 1% to 15%, largely influenced by cardiac and noncardiac comorbidities and the extent of the overall operation performed. Most studies report a mortality less than 7%.

In the setting of LV enlargement, reduced EF, and clinical heart failure, some have advocated mitral surgery for mild to moderate regurgitation, particularly if optimal heart failure therapy has not reduced the degree of regurgitation to a modest level with concomitant reduction in pulmonary artery and right heart pressures [23,24,26]. Mitral surgery certainly merits consideration for moderate or more regurgitation in heart failure patients undergoing CABG with or without SVR [19•,20•,26,28,31]. In my experience, it is not wise to assume that the degree of regurgitation will decrease substantially with CABG (which theoretically should reduce papillary ischemia and dysfunction) and/or SVR (consequent improvement of ventricular-papillary anatomy).

Better outcomes have generally been achieved with mitral repair (annuloplasty ± restructuring of valve), compared with mitral valve replacement. However, the results of the latter approach are improving with retention and proper positioning of the chordae tendinae and native valve tissue [27–29]. The specific surgical approaches and techniques vary among surgeons, and clinical science is still evolving in this area. At this point, I obtain consultation from surgeons experienced with mitral annuloplasty or repair for moderate to severe mitral regurgitation in the heart failure patient.

There are several caveats to mitral surgery in chronic heart failure, in addition to still noteworthy operative and in-hospital mortality rates. A few patients simply do not improve clinically. In my experience, these patients tend to be elderly with a history of systemic hypertension and "preserved systolic function." It is likely that after mitral surgery these patients are still left with diastolic-dysfunction heart failure. On rare occasion, the valve is inadvertently converted from regurgitation to stenosis.

Tricuspid Valve Surgery

Little has been studied or written about this intervention in general, and especially in the setting of chronic cardiac failure. There are no prospective, randomized, controlled trials to guide us. In my experience, tricuspid regurgitation can be a very threatening clinical condition in heart failure; however, it is rarely recognized as such. The development of tricuspid regurgitation is one of the causes of clinical decompensation in patients with previously stable chronic heart failure. Table 1 presents the major clinical and laboratory clues for problematic tricuspid regurgitation. In general, the finding of prominent V waves on jugular examination, a pulsatile liver, early ascites and increasing peripheral edema in a patient with subacute exacerbation of fatigue, weakness, and malaise, increased somnolence, and evolving cachexia indicate that tricuspid regurgitation has become a major threat to the patient's clinical course. Increasing diuretic therapy frequently results in problematic elevation in serum blood urea nitrogen and creatinine without resolution of the peripheral edema and ascites. Echocardiography is helpful in confirming the presence and severity of the lesion, assessing right ventricular volume and function, and excluding remedial left heart lesions.

In my experience, the short-term prognosis without surgical intervention is poor at best; there is no long-term

survival. These patients become refractory to diuretic and standard heart failure therapy with a miserable course of worsening edema, ascites, and cachexia. Because of the dismal outcome of these patients without surgery, they merit careful diagnostic and hemodynamic evaluation. A complete Doppler echocardiogram and a comprehensive cardiac catheterization are recommended to exclude a previously undetected left heart lesion or condition (*eg*, moderate to severe mitral regurgitation, diastolic dysfunction, occlusive coronary artery disease), to assess the degree of pulmonary hypertension, and to evaluate the status of the right ventricle. The development of problematic tricuspid regurgitation is not uncommon in patients with a history of mitral surgery for mitral regurgitation or rheumatic mitral disease. Careful assessment of the integrity of the prior mitral valve repair or replacement is essential in this scenario because the patient may require valve replacement in the mitral position in addition to the tricuspid surgery.

In the absence of a nonreparable left-sided condition and moderate to severe pulmonary hypertension (pulmonic systolic pressure > 50 mm Hg), I obtain consultation from a cardiac surgeon experienced with tricuspid valve surgery. I have been impressed with the short- and long-term (> 2 years) clinical results in a limited number of patients (n = 4). Two additional patients who had advanced to cardiac cirrhosis and severe refractory ascites before tricuspid surgery fared much worse; both died within 6 months of cardiac surgery. For patients with moderate to severe pulmonary hypertension and nonreparable left heart conditions, attempts should be made to pharmacologically reduce the level of pulmonary artery pressure by whatever means possible (*eg*, diuresis, endothelin blockade, sildenafil, dihydropyridine, nitrate) to not only allow reconsideration for tricuspid surgery, but also to decrease the severity of regurgitation and improve the clinical status of this unfortunate patient subgroup.

Because tricuspid valve surgery may later restrict placement of right ventricular leads for pacemaker or defibrillator intervention, consideration should be given to epicardial lead placement at the time of tricuspid surgery.

Aortic Valve Surgery

Aortic stenosis

Aortic valvular stenosis takes a ventricle from pressure-overload concentric LV hypertrophy to diastolic dysfunction heart failure to eventual remodeled enlargement with added global systolic dysfunction. Aortic valve replacement is indicated along this entire course, with expected improvement in virtually all clinical (symptoms, survival) and laboratory (cardiac structure and performance) parameters [32–39]. Operative mortality is directly related to the degree of ventricular systolic dysfunction, heart failure functional class, and cardiac (*eg*, coronary artery disease, another valvular lesion) and noncardiac (*eg*, age, renal dysfunction) comorbidities. The operative mortality is 7% to 21% for symptomatic patients, even the most compromised [33–39]. Clinical outcomes after aortic valve replacement exceed those of medical management alone, although no prospective, randomized, controlled trial has ever been (or ever will be) performed to definitively declare such.

For the patient with a depressed LV function (LVEF < 30%), demonstration of contractile reserve (augmentation of systolic function) with dobutamine portends a lower operative mortality rate and a more favorable short- and long-term clinical response, compared with the patient whose aortic valvular disease has resulted in irreversible ventricular damage and dysfunction and no contractile reserve [38,40,41]. The outlook for the latter patient is quite dismal irrespective of whether medical or surgical options are selected, but valve replacement might still be considered, particularly if the patient is otherwise not a candidate for cardiac transplantation. A long recovery period (months) after surgery is typical for those who survive the operation and the immediate postoperative period.

The patient with severe systolic dysfunction (LVEF < 30%) and "silent" or low-gradient aortic stenosis deserves special attention. Any clinical or laboratory evidence of aortic valve disease should lead to a carefully performed dobutamine echocardiogram (and occasionally, dobutamine catheterization) to try to evoke a significant transvalvular gradient, to document a reduced aortic valve area if present, and to assess LV contractile reserve [38–41]. This provocation usually distinguishes patients whose low valvular pressure gradients are principally secondary to the low flow and stroke volume of marked LV systolic dysfunction alone from those whose low gradients are secondary to reduced LV function evoked by long-standing severe aortic stenosis. The latter patients generally still benefit from aortic valve replacement.

The clinical and laboratory clues for significant aortic stenosis in patients with the silent or low-gradient presentation (and thus, who merit dobutamine echocardiography) are presented in Table 2. These include historical, physical, and laboratory findings that raise suspicion for this underdiagnosed lesion.

For patients with functional class III to IV heart failure from aortic stenosis, preoperative care should include measures to improve the marked congestion/volume overload, low cardiac output, and systemic hypoperfusion of these patients. Careful administration of nitroprusside, occasionally supplemented by dobutamine, can greatly assist in enhancing the patient's clinical and hemodynamic status before the major operative procedure [42].

Aortic regurgitation

Although aortic valve replacement for the heart failure of acute aortic regurgitation can be quite dramatic, the operation for moderate to severe chronic heart failure caused by long-standing aortic regurgitation is often not very

Table 2. Clinical and laboratory findings that serve as clues for significant aortic stenosis (and the need for further evaluation) in patients with "silent" or "low-gradient" aortic stenosis and severe LV dysfunction and failure

A history of an aortic outflow murmur
Any aortic outflow murmur in a patient with markedly depressed LV systolic function
Loss of the aortic component of the second heart sound
Greatly diminished or absent carotid pulses in the absence of occlusive carotid disease
Any defect of the aortic valve, any aortic valvular pressure gradient, or unexplained, inappropriately increased LV mass on echocardiography
Otherwise unexplained LV hypertrophy, typically with a "strain" pattern, on electrocardiography

LV—left ventricular.

impressive, but neither is medical management [43–49]. For this reason, aortic valve replacement should never be delayed to the point of moderate to severe LV systolic dysfunction or any symptoms beyond mild. Although there are no prospective randomized trials addressing the timing of valve surgery to avert a compromised long-term clinical outcome, several studies examining short- and long-term surgical results indicate that any sign of deteriorating LV function should move the patient to valve replacement [43,46–49]. In patients with minimal to no symptoms, such evidence might include an LVEF less than 45%, LV end-diastolic short axis diameter 75 mm or more, or systolic diameter 55 mm or more. Because of the poor clinical course of heart failure caused by chronic aortic regurgitation, I occasionally lean toward valve replacement before any of these criteria are strictly met.

For the patient whose LV function and clinical status have deteriorated into refractory heart failure (with or without valve replacement), few options remain. These include cardiac transplantation, and for those who are not eligible for such, ventricular assist device (with surgical closure of the aortic valve).

Conclusions

All patients who present with cardiac failure must be evaluated for surgically correctable, remedial lesions and conditions irrespective of the severity of LV dysfunction. Surgical advances, expanded surgical experience, and improvement in anesthesiology and perioperative management have brought several procedures into consideration in the overall approach and care of the patient with chronic heart failure. Symptomatic heart failure and an LVEF less than 30% are no longer viewed as contraindications to CABG, SVR, and/or valvular surgery. When properly applied, these procedures can retard the progression of cardiac dysfunction and heart failure, and some may also render a survival benefit. It is important to consider these options long before the patient is forced to queue up in the lines for cardiac transplantation, mechanical support (ventricular assist device or total artificial heart), continuous infusions of cardiovascular-active medications, or heart failure death.

Over three decades of clinical work and investigation in human heart failure, witnessing the unrelenting clinical deterioration over time of all heart failure patients regardless of pharmacotherapeutic achievements, has made me more aggressive (not less, as expected from a "medical person") with respect to considering surgical intervention that can favorably impact the clinical course of this patient population. This view was fueled by the advances in surgical methods and experience over the years.

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