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Mitral Valve Reconstruction in the Patient with Heart Failure

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Abstract. **Secondary MR is a complication of end-stage cardiomyopathy and is associated with a poor prognosis and is due to progressive mitral annular dilation and alteration in LV geometry. A vicious cycle of continuing volume overload, ventricular dilation, progression of annular dilation, increased LV wall tension and worsening MR and CHF occur. The mainstay of medical therapy is diuretics and afterload reduction, and is associated with poor long-term survival in these patients with CHF and MR. However, surgical intervention in the form of undersized, 'overcorrecting' mitral valve repair has shown great promise and is an area of ongoing investigation.**

Key Words. **congestive heart failure, dilated cardiomyopathy, mitral reconstruction**

Introduction

Congestive heart failure (CHF) is one of the leading causes of hospitalization in the United States today and its incidence is increasing. Heart failure will become even more of a medical challenge as average life expectancy continues to rise. Despite improvements with medical management approximately 50% of patients with CHF die within 3 years of presentation. Secondary mitral regurgitation (MR) is a complication of end-stage cardiomyopathy and may affect up to 60% of all heart failure patients as a pre-terminal or terminal event. While heart transplantation is now considered standard treatment for select patients with severe CHF and end-stage heart disease, it is only applicable to a small percentage of patients. Transplantation is limited both by the small number of donor hearts available and its inapplicability in the older patient or those with comorbid medical conditions. In an effort to solve this problem; alternative medical and surgical strategies are evolving.

Anatomy and pathology

In order to address the issue of heart failure and MR, one needs to understand the anatomy of the mitral valve, which is complex. Mitral competence depends on the coordinated function of the components of the mitral apparatus: the leaflets, annulus, papillary muscles, chordae tendinae, and the entire left ventricle (LV).

The mitral valve is the 'inlet' to the LV. The mitral valve consists of two leaflets, the anterior (aortic) and posterior (mural) leaflets. The two leaflets are separated at the annulus by the posteromedial and anterolateral commissures. The anterior leaflet is semicircular and spans the distance between the two commissures. It is attached to the anterolateral wall of the LV in direct continuity with the fibrous skeleton of the heart and with the left and part of the noncoronary aortic valve leaflets. The posterior leaflet is rectangular in shape, and is divided into three portions by clefts in the leaflet.

The mitral annulus represents the junction of the fibrous and muscular tissue that joins the left atrium and ventricle. The average human mitral annular cross-sectional area is 5–11 cm^2. The annulus has two major collagenous structures: the right fibrous trigone (located at the intersection of the membranous septum, mitral and tricuspid valves and aortic root), and the left fibrous trigone (located at the posterior junction of the mitral valve and left coronary leaflet of the aortic valve). During systole the annulus assumes an elliptical shape and is able to contract and decrease in diameter, whereas, in diastole, it assumes a more circular shape. Annular flexibility allows for increased leaflet coaptation during systole and increased annular orifice area during diastole. The anterior aspect of the annulus, which is in continuity with the fibrous skeleton of the heart, has limited flexibility, whereas the posterior aspect of the annulus, which is not attached to any rigid surrounding structures, has more

flexibility. In MR, dilation typically occurs along the more flexible posterior aspect of the annulus.

The anterolateral and posteromedial papillary muscles arise directly from the apical and mid portion of the ventricular wall, and give rise to chordae tendinae that go to both leaflets. The anterolateral papillary muscle receives a dual blood supply from the left anterior descending and from either a diagonal or marginal branch of the circumflex artery. In contrast, the posterolateral papillary muscle has a singular blood supply, either from the right coronary or the circumflex artery, and is therefore more susceptible to ischemia and infarction. The posterior aspect of the LV wall and the papillary muscles together play a very important role in valvular competence and leaflet coaptation. The dynamics of both papillary muscles closely mimic the dynamics of the LV. During LV contraction, the leaflets are pulled downward and together. The LV wall geometry and mechanics play a more significant role in valve competence than the papillary muscles alone. Dilation of the LV may alter the alignment and tension on the papillary muscles and therefore may contribute to valvular incompetence (Fig. 1).

The chordae tendinae are comprised of fibrous connective tissue and attach the leaflets to either the papillary muscles or the LV wall directly. The chordae are divided into three groups. The primary chordae attach directly to the free edge of the leaflet, and ensure that the leaflets coapt without prolapse or flail. The secondary chordae, are more prominent on the anterior leaflet, attach to the leaflet along the line of coaptation, and are important in maintenance of ventricular function. Tertiary chordae are only present on the posterior leaflet, and attach directly to the ventricular wall or to the trabeculae carnae. In addition, there are commissural chordae, which arise directly from either of the papillary muscles and attach to both leaflets.

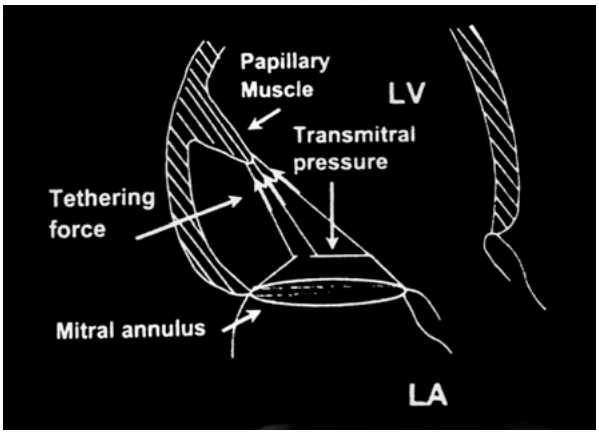

Fig. 1. *Valvular pathology in secondary MR. LV dilation alters the alignment of the papillary muscles and contributes to the development of valve incompetence. In secondary MR the transmitral pressure exceeds the tethering forces of the papillary muscles.*

Maintenance of the chordal, annular, subvalvar continuity and mitral geometric relationships are important in the preservation of overall ventricular function and may be even more important in patients with compromised function. Secondary MR is observed in patients with either idiopathic or ischemic cardiomyopathy and can be caused by many factors. In patients with nonischemic dilated cardiomyopathy, in the absence of intrinsic mitral valve disease, MR is due to a progressive dilation of the annular-ventricular apparatus with altered ventricular geometry, and subsequent loss of leaflet coaptation [1]. In patients with ischemic cardiomyopathy, the mechanisms that contribute to MR are more complex. They may include a combination of both dilation of the annular-ventricular apparatus and LV wall/papillary muscle dysfunction, again with the net result being failure of leaflet coaptation [1]. A large leaflet area is required for coaptation because mitral leaflet area is 2½ times greater than the area of the mitral orifice. As more leaflet tissue is utilized for coverage of the enlarging orifice, a critical reduction in tissue available for coaptation is reached, such that leaflet coaptation becomes ineffective, and a central regurgitant jet of functional or secondary insufficiency develops [1]. Therefore, the most significant determinants of mitral valve coaptation, leaflet orifice area, and MR are the dimensions of the mitral valve annulus. The LV dimension is less important in functional MR, because chordal and papillary muscle length are not significantly altered in people with idiopathic cardiomyopathy with or without MR.

Pathophysiology

MR leads to a cycle of continuing volume overload of the already dilated ventricle, progression of annular dilation, increased LV wall tension, increasing degrees of MR and worsening CHF. Patients with MR refractory to medical therapy have a poor long-term survival. In a study of 28 patients with cardiomyopathy and an ejection fraction of less than 25%, the one-year survival without transplantation was 46% [2].

The pathophysiology of acute MR (from chordal rupture, endocarditis, blunt chest trauma, or myocardial infarction) is different from that of secondary MR. In acute MR, the left atrium is relatively normal with low compliance, and the acute increase in left atrial pressure can lead to

pulmonary edema. This is not the case in secondary MR, where the compensatory changes occur slowly and lead to a gradual increase in left atrial and pulmonary venous compliance, and therefore the signs of pulmonary congestion may not become apparent until much later in the process.

In MR, the regurgitant volume ejected into the left atrium is dependent upon mitral orifice size, ventricular to atrial pressure gradient and heart rate. The regurgitant flow into the left atrium increases left atrial pressure, which leads to atrial enlargement and an increase in compliance, and decreases forward systemic flow. Left atrial pressures rise during systole and decline in diastole. At end diastole, left atrial pressure will remain mildly elevated, representing a flow gradient. In this setting, with only mild elevations in left atrial pressures, increases in pulmonary vascular resistance usually do not occur, and therefore acute pulmonary edema is not frequently seen.

Various interventions can alter the size of the regurgitant orifice area. An increase in preload or afterload, or a decrease in contractility, will result in dilation of the LV and an increase in regurgitant orifice area. In a study of patients with severe CHF, who were managed medically (with diuretics, nitrates and afterload reduction agents), the observed decrease in filling pressure and systemic vascular resistance led to a reduction in the MR associated with their failure. This was attributed to reduction in the regurgitant orifice area related to the decrease in LV volume and annular distension [3]. This complex relationship between mitral annular area and leaflet coaptation may explain why performing a 'valvular' repair, with an undersized annuloplasty ring, can help with a 'muscular' problem. Therefore, in fact, this represents a ventricular solution for a ventricular problem (Fig. 2).

In MR, the impedance to LV emptying (afterload) is reduced and allows the ventricle to adapt to the regurgitant volume by increasing total cardiac output to maintain an adequate forward output. The increases in LV preload, wall tension, diastolic volume, and stroke volume represent ventricular adaptations to severe MR. The increase in preload eventually leads to LV dilation and a change in the shape of the ventricle from an ellipse to a sphere. There is a significant decrease in the efficiency and work expended by the LV to produce flow that ultimately does not contribute to effective forward cardiac output. In these patients, maintenance of forward flow becomes difficult because up to 50% of the stroke volume is ejected into the left atrium before the aortic valve even opens [4]. With elimination of the regurgitant volume, the ventricle no longer has to expend an excessive amount of work on flow that is going in the reverse direction. In cases of severe myocardial dysfunction, the positive effects of the elimination of the regurgitant flow may be even more pronounced. In secondary MR, ventricular mass also increases, and the degree of LV hypertrophy correlates with the amount of chamber dilation. The ratio of LV mass to LV end diastolic volume remains normal. In the setting of decreased afterload, the ejection fraction (a clinical measure of pump performance) may remain in the normal range, even in the presence of significantly impaired intrinsic LV contractility [5]. Many of the commonly used indices of cardiac pump performance are dependent on both preload and afterload, and therefore are not as reliable in the setting of MR. LV end systolic volume is a better parameter as it does reflect changes in systolic ventricular function and is independent of preload and varies directly with afterload [6].

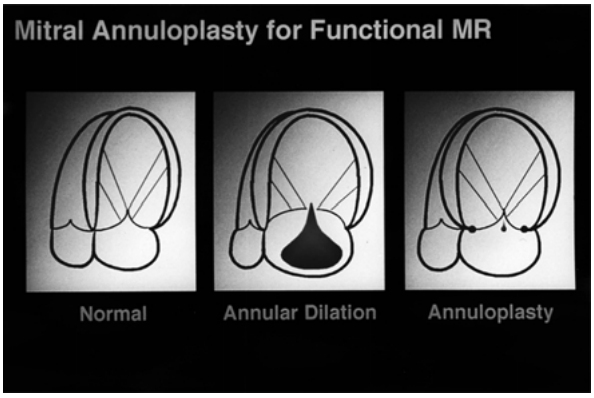

Fig. 2. *Rationale for annuloplasty in secondary MR. As the LV dilates, there is an increase in the regurgitant orifice area and the mitral annulus dilates. An undersized annuloplasty ring facilitates the return of the zone of coaptation to a more normal dimension to correct for the regurgitation.*

Secondary MR also impacts on coronary flow characteristics. In a recent study, coronary flow in patients before and after mitral valve reconstruction was assessed. Coronary flow reserve was limited in patients with MR due to an increase in baseline coronary flow, and flow velocity, which was related to LV volume overload, hypertrophy and preload (LV wall stress). The restriction in coronary flow reserve improved following valve reconstruction because of a reduction in the baseline coronary flow and flow velocity once the LV preload, work and mass were reduced [7]. Based on this study, in patients with secondary MR, a restriction in the coronary flow reserve would seem probable and an improvement in flow reserve and velocity would be expected following mitral valve repair. Ultimately mitral valve repair in this setting would lead to improvement in LV geometry.

In the setting of chronic CHF, cardiac reserve is depressed, a number of compensatory mechan-

isms are activated and may account for many symptoms of failure and contribute to its further progression. Some of these mechanisms are responsible for the vasoconstriction seen in heart failure and include stimulation and activation of the neuroendocrine and sympathetic nervous systems. Increases in circulating norepinephrine levels are documented in CHF. Norepinephrine is released and binds to the β-adrenoceptors and results in a positive inotropic response. In CHF, there is an excessive release of norepinephrine from the myocardium, a corresponding increase in plasma levels, and a reduction in its myocardial stores. Following long-term exposure to elevated levels of norepinephrine, the numbers of β-adrenoceptors become downregulated, which results in a reduced positive inotropic effect of β-adrenoceptor agonists [8–10]. Additionally, studies have shown that proinflammatory cytokines (tumor necrosis factor-α, IL-1, IL-2 and IL-6) may be responsible for the myocardial depression in heart failure. TNF-α has been shown to be produced by the heart under stress, has negative inotropic effects, and it may play a role in the development of LV dysfunction, dilated cardiomyopathy, hypotension, and pulmonary edema, all of which can be seen in advanced CHF [11,12].

Clinical presentations

Patients with mild to moderate MR may remain asymptomatic for years as the LV adapts to its increased workload. In contrast, patients with secondary MR usually present with symptoms related to the underlying cardiomyopathy and CHF. Symptoms of decreased cardiac output and pulmonary congestion develop (weakness, fatigue, and dyspnea). These symptoms worsen with the further progression of MR. On physical examination, the cardiac impulse may be hyperdynamic and there is a characteristic blowing apical holosystolic murmur that may radiate to the axilla, back and into the neck.

Diagnostic techniques

Chest X-Ray (CXR)

An enlarged cardiac silhouette is a common radiographic finding in patients with secondary MR and is indicative of LV and left atrial enlargement. Congestive findings in the pulmonary parenchyma are less prominent.

Electrocardiogram (EKG)

Atrial fibrillation is common. Findings consistent with left atrial enlargement and ventricular hypertrophy are typical.

Echocardiogram

Echocardiography provides a non-invasive means of assessment of ventricular function and the severity of MR. Color Doppler analysis yields a semi-quantitative analysis of MR. This assessment of the degree of MR is based on the relative comparisons of size and area of the regurgitant jet to the size and area of the left atrium. The Doppler jet is sensitive to load conditions, driving pressure, jet eccentricity and left atrial size, and therefore can lead to an incorrect estimation of the extent of MR [13]. Proximal flow convergence analysis, which allows for calculation of the regurgitant volume by measuring the flow proximal to the mitral valve orifice, is a useful tool in the assessment of the extent of MR [13]. Transesophageal echocardiography (TEE) is superior to transthoracic echocardiography in that it better

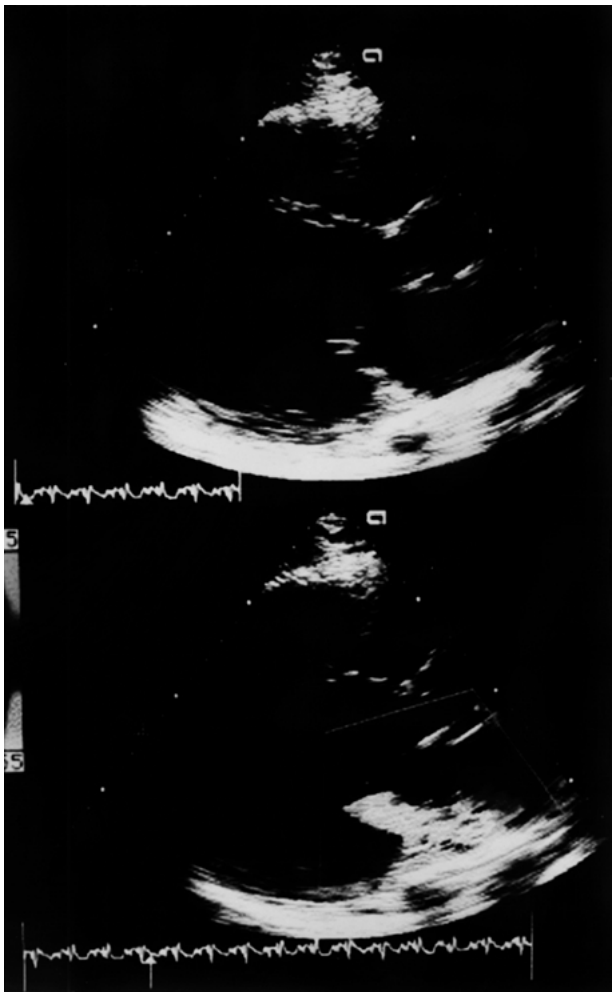

Fig. 3. *Transesophageal Echocardiogram. TEE and color Doppler analysis to demonstrate annular dilation and the presence of secondary MR.*

defines the details of mitral valve pathology and anatomy and the severity of the regurgitation (Fig. 3).

Cardiac catheterization

Cardiac catheterization is not necessary to establish the diagnosis of secondary MR, however, it provides data regarding secondary processes and associated cardiac pathology. Left ventriculography, while it does not permit a truly accurate assessment of the mitral valve or subvalvar apparatus, does allow a calculation of ejection fraction.

Management

The mainstay of medical management of patients with cardiomyopathy and secondary MR is the treatment of the underlying CHF, with the use of diuretics and afterload reduction agents. By reducing the aortic ejection impedance, the regurgitant volume into the left atrium is reduced and pulmonary congestion is relieved. This strategy reduces LV volume and increases forward stroke volume, which results in a smaller regurgitant orifice area. An area of current investigation for newer modalities of medical treatment includes the use of recombinant agents directed at TNF-α and the specific TNF-α receptors.

Historically, the surgical approach to patients with MR was mitral valve replacement and little was understood of the adverse consequences that interruption of the annulus-papillary muscle continuity had on LV systolic function. This procedure was associated with high mortality rates. It is in this population of patients that the concept of the 'pop off' effect of MR originated, that is reversal of blood flow was somehow beneficial to the patient in failure. It has been demonstrated in a number of studies that preservation of the annulus-papillary muscle continuity is of paramount importance to preservation of LV function [14,15]. It was the excision of and disruption of the subvalvar apparatus that accounted for the significant loss of systolic function due to the destruction of the LV that led to the poor outcome in the earlier patients who underwent valve replacement [16]. Preservation of the mitral apparatus and LV in mitral valve repair has been demonstrated to enhance and maintain LV function and geometry with an associated decrease in wall stress [17]. This procedure has been shown to be safe with good long-term outcomes [18]. In fact, it has been shown that there is no 'pop-off' effect, but the mortality ascribed to these patients from mitral replacement was due to disruption of the subvalvar apparatus and loss of LV function.

As the availability of transplantation for end-stage cardiomyopathy is limited, there has been recent interest in the altered geometry of the LV in patients with severe dysfunction. This innovative work was initially described by Batista, who states that all mammalian hearts share the same ratio of mass to diameter, regardless of the size of the heart ($M = 4 \times R^3$). Batista proposed that for those hearts that do not comply with this relationship, an operative procedure should be performed to restore the ratio back to normal [19]. Surgeons have attempted to renormalize this relationship by LV myoreduction surgery (the Batista procedure). Batista initially reported an operative mortality of 5%, a 30-day mortality of 22%, and a two-year survival of 55%. Unfortunately, complete and long-term follow-up was not available [20]. This procedure has met with varying degrees of success in the United States and worldwide. The Cleveland Clinic series of 62 patients reported a 3.5% in-hospital mortality, with 7 late deaths, and with a one-year actuarial survival of 82% [21]. Of significance, in all of these cases, a mitral valve repair or replacement occurred routinely as part of the myoreduction procedure. It is therefore difficult to discern what the exact role of correction of MR plays in the overall success of the procedure.

At the University of Michigan (1993–1999), 92 patients with end-stage cardiomyopathy and refractory MR underwent mitral valve repair with an undersized annuloplasty ring. The overall operative mortality was 5% and one- and two-year actuarial survivals were 80% and 70%, respectively. In patients undergoing mitral valve reconstruction for myopathy there is re-establishment of a more normal LV mass to volume ratio without the loss of myocardial mass [22,23]. The average LV volume at 24-month follow-up in this study was over 200 ml, still quite large, while with the LV myoreduction procedure Batista has demonstrated acute reduction of volumes to 90–100 ml at the time of operation [24]. In a recent study that assessed acute cardiovascular changes that occur with the Batista procedure, significant elevations in LV end-diastolic pressure and end-diastolic elastance were noted, indicative of persistent post-operative depressed diastolic function [25]. There is no loss of ventricular mass with mitral valve reconstruction alone, however, an appropriate mass/volume ratio is restored. In addition, a decrease in sphericity index and LV volume measurements were demonstrated post-operatively, and it is in these patients that the negative cycle of CHF is interrupted and the surgical unloading of the LV is achieved. These patients may be undergoing a slow self-remodeling from the alteration of the angulation of the base of the

heart, stabilization of the mitral annulus or LV unloading, all contributing to a more favorable ventricular geometry. Importantly, there is an acceptable surgical mortality, both at 30 days and 1 year, for mitral valve reconstruction, which is equivalent to or lower than what has been reported for LV myoreduction procedures [20,21,24].

There are numerous factors that explain why this cohort of CHF patients with MR, previously thought to be 'inoperable' have done well.

Reduction in regurgitant orifice

In a recent study of patients with severe heart failure, who were managed with pharmacologic agents (diuretics, nitrates and afterload reduction agents), the observed decrease in filling pressure and systemic vascular resistance led to a reduction in the dynamic mitral regurgitation associated with their heart failure. This was attributed to a reduction in the mitral regurgitant orifice area, related to a decrease in left ventricular volume and annular distension [26]. This complex relationship between mitral annular area and leaflet coaptation may explain why, paradoxically, an undersized 'valvular' repair can help a 'muscular' problem. Although significant undersizing of the mitral annulus was employed in our study to over correct for the zone of coaptation, no mitral stenosis was induced nor was any SAM noted. Systolic Anterior motion (SAM) was avoided due to widening of the aorto-mitral angle and increased left ventricular size seen in myopathic patients. In addition, acute remodeling and re-angulation of the base of the heart from the undersizing of the mitral annular ring may also contribute to the improvement seen in these myopathic hearts. This may reestablish the ellipsoid shape and somewhat normal geometry to the base of the left ventricle.

Ventricular adaptations

Increases in left ventricular preload, wall tension, diastolic volume, and stroke volume are all documented ventricular adaptations to severe mitral regurgitation. There is a significant decrease in the efficiency of the left ventricular contraction and the work expended by the left ventricle to produce flow that ultimately does not contribute to effective forward cardiac output. In these patients, maintenance of forward flow becomes more difficult because up to 50% of the stroke volume is ejected into the left atrium before the aortic valve even opens [27]. With elimination of the regurgitant volume, the ventricle no longer has to expend an excessive amount of work on flow that is going in the reverse direction. All blood flow will be in the forward direction, and will contribute to forward flow and effective cardiac output. In cases of severe myocardial dysfunction, the positive effects of the elimination of the regurgitant flow may be even more pronounced.

Coronary flow characteristics

Further evidence of underlying mechanisms explaining why mitral valve repair for patients with cardiomyopathy may be successful is taken from a study of the coronary flow characteristics in patients with mitral regurgitation, in the absence of coronary artery disease. This study assessed coronary flow characteristics in patients before and after mitral valve reconstruction. Coronary flow reserve was limited in patients with mitral regurgitation due to an increase in baseline coronary flow and flow velocity which was related to left ventricular volume overload, hypertrophy and preload (left ventricular wall stress). The restriction in coronary flow reserve improved following mitral valve reconstruction because of a reduction in the baseline coronary flow and flow velocity once the left ventricular preload, work and mass were reduced [28]. Based on this study, in patients with mitral regurgitation and cardiomyopathy, a restriction in the coronary flow reserve would seem probable and an improvement in flow reserve and velocity would be expected following mitral valve repair. Ultimately the mitral valve repair in this setting would lead to an improvement in the left ventricular geometry.

Neurohumoral alterations

In the setting of chronic congestive heart failure, when cardiac reserve is depressed, various mechanisms compensate for the reduction in cardiac performance to maintain cardiovascular homeostasis. Some of the well-recognized compensatory mechanisms include an increase in sympathetic nervous system activity with an increase in release of several neurohumoral factors. In response to the increased sympathetic activity, norepinephrine is released from the myocyte into the synaptic cleft and it binds to the β-adrenoceptors. These receptors then couple with adenyl cyclase, which results in an increase in cyclic AMP (cAMP) production. This leads to the stimulation of protein kinase A and ultimately results in a positive inotropic response [29]. In heart failure there is an excessive release of norepinephrine from the myocardium, a corresponding increase in plasma norepinephrine levels and a reduction in myocardial stores of norepinephrine [30]. Following long-term exposure to elevated levels of norepinephrine, the numbers of β-adrenoceptors become downregu-

lated [30,31], the receptors become desensitized, and the post-receptor signal transducing pathway becomes altered [32]. This results in a decrease in cAMP production and a reduced positive inotropic effect of β-adrenoceptor agonists [32,33]. In addition, while in the setting of heart failure, the positive inotropic effects of phosphodiesterase inhibitors, when used as a single agent, are reduced in comparison to that of the nonfailing heart [29,34] due to diminished basal cAMP production in the failing heart [35]. It would therefore seem that the administration of β-adrenoceptor agonists or cAMP phosphodiesterase inhibitors would not be the best treatment in patients with heart failure. Additionally, the use of β-adrenoceptor agonists in the acute setting is associated with a high incidence of arrhythmias. In our study population we advocate the simultaneous use of norepinephrine and milrinone. The exogenous norepinephrine administration acts to replete the diminished myocardial stores of norepinephrine and it stimulates cAMP production which has been shown to restore the positive inotropic effects of phosphodiesterase inhibitors [36,37].

Cytokine response

Heart failure is a clinical syndrome derived from chronic work overload of the myocardium, ischemia, inflammation and additional etiologies such as cardiomyopathy. Studies have shown that the proinflammatory cytokines tumor necrosis factor-α (TNF-α), IL-1, IL-2 and IL-6 may be responsible for the myocardial depression seen in the complex syndrome of heart failure [38–41]. TNF-α has been shown to be produced by the heart under stress and has negative inotropic effects. Studies have demonstrated that this proinflammatory cytokine may play a role in the development of left ventricular dysfunction, dilated cardiomyopathy, hypotension, and pulmonary edema, all of which can be manifestations of advanced heart failure [42–45]. There are two forms of TNF-α specific receptors: TNF-R1 and TNF-R2. Both receptors are found in equal proportions in the normal myocardium and TNF-α binds with equal affinity to both receptors. The negative inotropic effects of TNF-α are mediated primarily by its interaction with TNF-R1. The expression of the myocardial TNF-Rs is downregulated in the presence of heart failure, similar to that seen with the β-adrenergic receptors [42–46]. The circulating, or soluble, forms of the TNF-Rs are elevated in patients with heart failure suggesting that these receptors may be 'shed' from the myocardial cells [47]. The circulating TNF-Rs can neutralize the biological effects of circulating TNF-α [48]. Based on these observations, it has been postulated the cardiac tissue response to increased TNF-α levels is to increase the level of soluble TNF receptors in order to decrease the amount of bioactive TNF-α that can potentially stimulate cardiac cells [42]. At the University of Michigan we have measured levels of TNF-α, soluble TNF-R1 and TNF-R2, IL-6 and IL-6 receptors in the pre- and post-operative period in patients undergoing mitral valve repair in the presence of heart failure, these data are recorded in Table 2. Six months following mitral reconstruction, the levels of these cytokines and their respective soluble receptor levels were decreased. While this data supports the role of TNF-α in the development of heart failure, further studies are being conducted in which the soluble recombinant form of TNF-R is being administered to heart failure patients in the hope of decreasing some of the manifestations of failure [42].

Prognosis

For patients with dilated cardiomyopathy, mortality is directly related to severity of ventricular systolic dysfunction [20]. In addition, increased chamber sphericity, the presence of MR [24], and an increase in LV end-diastolic volume are markers of a poor prognosis. In review of these types of patients, 1-year mortality has been reported between 54% and 70% [25]. Mitral valve surgery for secondary MR relieves symptoms, increases long-term survival, helps prevent further progression of LV dysfunction and improves overall ventricular function.

Table 1. *Preoperative and postoperative echocardiographic data*

ECHO parameter	Pre-operative	Post-operative (24 months)	*P* Value
End diastolic volume (ml)	281 ± 86	206 ± 88	< 0.001
Ejection fraction (%)	16 ± 5	26 ± 8	0.008
Regurgitant fraction (%)	70 ± 12	13 ± 10	< 0.001
Cardiac output (l/min)	3.1 ± 1.0	5.2 ± 0.8	0.001
Sphericity index (D/L)	0.82 ± 0.10	0.74 ± 0.07	0.005

Table 2. *Pre-operative and post-operative cytokine levels*

	TNF-α (pg/ml)	TNF-R1 (pg/ml)	TNF-R2 (pg/ml)	IL-6 (pg/ml)	lL6-R (ng/ml)
Pre-op	3.5 ± 1.3	931 ± 187	1989 ± 381	61 ± 6.6	36.0 ± 5.2
Post-op (6 mo.)	2.8 ± 1.1	774 ± 177	1450 ± 254	2.9 ± 1.8	31.2 ± 8.1
P value	0.02	< 0.01	< 0.01	0.13	0.15

Summary

Secondary MR is a significant complication of end-stage cardiomyopathy. The MR is thought to occur due to progressive dilation of the annular-ventricular apparatus, altered ventricular geometry, loss of leaflet coaptation and LV wall/papillary muscle dysfunction. In secondary MR, there is an increase in preload and a decrease in afterload, which eventually leads to LV dilation and remodeling. Medical management consists primarily of diuretics and afterload reduction, which is met with poor long-term survival. Mitral reconstruction via an annuloplasty ring effectively corrects MR in cardiomyopathy patients, is a safe procedure in a high-risk population, and has an acceptable operative mortality rate. Both survival and functional status have improved for these patients. The effects of this procedure with severe myocardial dysfunction may be attributed to a decrease in the regurgitant orifice area, better effective forward flow, and an increase in coronary flow reserve. These changes all contribute to restoration of the normal LV geometric relationship. While longer-term follow-up is necessary, mitral reconstruction offers a new strategy for end stage cardiomyopathy.

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