Congestive heart failure: Treat the disease, not the symptom—Return to normalcy

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Pathology is the dilemma that physicians confront daily in patient care. The consequences of any distortion of structure become the symptoms of the disease created by abnormal processes. Treatment is directed toward alleviation of these results that “remodel away” from normal structure and biochemistry. This confrontation over symptoms occurs in the health care community (medicine, surgery, pharmacology, medical devices, and through annual health care costs). Medical students cannot wait to leave their learning curve—established normal principles. To many, these are an astounding roadblock between the basic and clinical areas. The obligatory study of normality is a “brief hurdle” they must pass on the real road to becoming a physician. Fundamental basic sciences are thought to reflect the unimpeachable camp of the “preclinical school.” The motivational objective is to deal with the dilemma of the sick patient.

The purpose of this report is to show that departure from normality and lack of focus on its importance are very expensive and less effective ways to deal with pathologic conditions. The result of this focus on abnormality caused by disease is an explosive development of drugs, operations, machines, and health care personnel to minimize the physical and biochemical derangements of such diseases. A prominent example is *congestive heart failure* (CHF), which affects 4.9 million persons in the United States. The disease constitutes loss of the normal elliptical ventricular architecture, to produce a dilated spherical ventricle with limited contractile and filling capacities. The consequences are fatigue, pulmonary congestion, associated arrhythmias, repeated hospitalizations, and eventually death.

The pathologic derangement is called *remodeling* due to heart diseases in which the blood flow, valve, and muscle itself are damaged. This term *remodel* is used extensively in medical and surgical circles to define the diseased state, but the definition of this word does not appear in many dictionaries. Its antonym is *unhealthy* or *away from normal*. The word *restore* is also absent from the literature. This is defined as “will bring things back to normal.” I use the term *restore* and hope its understanding creates a paradigm shift that may change current treatments of pathologic conditions.

CHF is a change from the normally elliptical left ventricular shape to a spherical dilated muscle, in which the fiber angle for normal contraction is lost and the natural tightness between excitation and resulting contraction becomes abnormal. The collagenase architecture surrounding the muscle, including nerves and vessels, becomes diminished by this distention. Dilatation raises metabolic needs and may cause ischemia because of abnormal flow distribution.¹

The distended, dilated muscle initiates a biochemical reaction in the renin-angiotensin and sympathetic systems. Abnormal levels of angiotensin are present in the renin-angiotensin system to combat distention and produce hypertrophy of working muscle. The sympathetic nervous system is stimulated to increase blood pressure by vasoconstriction to supply organs and enhance heart rate to maximize output. Additionally, aldosterone levels are elevated to cause peripheral constriction to maintain blood pressure and enhance scarring. Reduced urine output results from diminished renal blood flow and sodium retention, and a change in collagen content of the heart produces dilatation by collagenase stimulation through distention.

The *medical* treatments of these consequences are angiotensin-converting en-

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zyme inhibitors (limiting collagen breakdown), β -blockers (decrease sympathetic tone), spironolactone (decreases aldosteronism and fibrosis), and diuretics like furosemide (Lasix) to enhance urinary blood flow and produce decompression of retained fluid. These treatments prolong life expectancy in patients with CHF and help to reduce the classification of CHF from class IV to class II-III. This reflects a moderate enhancement in activities and a rightward shift of the mortality from the 50% 5-year mortality, yet it allows an ongoing downward trend in which, at present, 50% of persons with CHF now live for 8 years. The end result is the same overall mortality, with fewer symptoms and prolonged health care.

Hospitalizations are frequent in patients with class III and class IV CHF, approximately 2 million in 1 year, and approximately 50% of such patients need rehospitalization. The cost today is \$100 billion per year to Medicare and private insurance providers. By 2020 the cost will exceed \$1 trillion per year, as the number of patients older than 65 years will double. This astronomical expense has stimulated the National Institutes of Health, Health Care Financing Administration, pharmacologists, and drug companies to deal with the *symptoms* of CHF. There is *marginal* interest in the disease itself: the role of changing the anatomy of the dilated ventricle.

The *electrical* approach to the sphere includes a wrapping of the diseased muscle, called *cardiomyoplasty*, to prevent dilatation. Electrical defibrillators are implemented to treat arrhythmias caused by this distention. Right ventricular pressure and then pulmonary artery pressure (reflected from right ventricular pressure) are monitored to detect early signs of pulmonary congestion caused by decreased left ventricular emptying. This may limit early rehospitalization before CHF advances to a later stage. Additionally, biventricular electrical pacing restores normal septal contraction as excitation/contraction delay follows distention. These abnormalities occur in both normal muscle (that can recover) and abnormal muscle. The recovery reflex in electromechanical abnormality is corrected by bypassing slower conduction with direct septal, right ventricular, and left ventricular stimulation.

Surgical treatment follows the same rightward shift of decreasing mortality, yet the downward curve persists. Revascularization alone enhances survival, and long-term mortality relates to preoperative ejection fraction. At 10 years, survival was 20% at an initial ejection fraction of 25% and about 40% at an initial ejection fraction of 35%.² Augmented blood flow relieves angina, but the downward mortality trend persists. A similar prognosis follows valve replacement or repair for valve insufficiency in dilated or ischemic cardiomyopathy. Restoring mitral valve competence by valve replacement (without coronary disease) in patients with an ejection fraction of less than 40% yields a

60% mortality at 10 years³; after valve repair, the 5-year mortality is 50% in patients with ischemic and dilated mitral incompetence.⁴

This symptomatic treatment displaces the mortality curve to the right by altering the downward trend. Patients require frequent visits from nurses to start inotropic drugs to improve cardiac output and prevent rehospitalization. Exercise capacity in patients who have a dilated heart remains limited. Repeated hospitalizations are required to treat recurrent CHF. These limitations account for an astounding health care cost.

Alternate surgical methods to deal with the fatiguing heart include mechanical displacement of the failing ventricle with a left ventricular assist device. These devices involve expensive initial costs, can become infected, and can generate clots because of the absence of an endothelial covering. They reduce the symptoms of CHF but do not function for a prolonged period. They are not available in all centers and require substantial monitoring. The final step, transplantation, is limited by restricted organ availability and subsequent rejection and immunosuppressive treatment to avoid rejection. The treatment for rejection itself includes complications, such as coronary artery disease and tumor formation.^{5,6}

The Diagnostic Dilemma

The approach to the symptoms versus the disease is most evident during characterization of the diagnosis of the pathologic abnormality that reduces left ventricular function and produces CHF. Attention is directed toward how blood is ejected, rather than toward the underlying left ventricular volume and shape responsible for diminished heart function. Everyone accepts that a low ejection fraction is abnormal and recognizes that the heart must deal with this to maintain cardiac output by dilating. Fatigue is recognized as a limitation of decreasing reserve by ventricular dilatation.

Unfortunately, attention is seldom paid to the left ventricular volume itself, the spherical underpinning of CHF. This provides a classic example of this fundamental dilemma, a misunderstanding of the disease despite routine assessment of the left ventricle by ventriculography. This critical information is routine during cardiac catheterization to evaluate for ejection fraction, yet determining left ventricular size has not achieved the critical role it deserves in the CHF entity.

The right anterior ventricular view of left ventricular function is routine or sometimes excluded because of echocardiographic study. Biplane ventriculograms that clearly characterize both ejection fraction and global volume are seldom obtained. Magnetic resonance imaging (MRI) measures left ventricular size and is available but not used in most institutions. The current pattern of left ventricular evaluation persists despite important knowledge of the value of ventricular volume made available since 1987 from

Figure 1. The relative risk for death after myocardial infarction, depicted as the relationship between LVESV and survival. The volume index is approximately 50% of end-systolic volume, or 25 ± 10 mL/m². (From Colucci WS, editor. *Atlas of heart failure: cardiac function and dysfunction*. 2nd ed. Philadelphia: Current Medicine, Inc; 1995, 1999; adapted from White et al.⁷ Reprinted with permission.)

White and associates⁷ (Figure 1). There is a close correlation between increased volume and progressive mortality that is independent of evaluating ejection fraction in many patients (Figure 2).⁷

The value of end-systolic volume indices is unknown to many physicians and surgeons, even though their value has been recognized. Gaudron,⁸ Di Donato,⁹ and their associates reinforced the concept by showing how both changing and altering the left ventricular size affects muscles whose fiber orientation is changed surgically to create a more normal ellipse. This geometric approach excludes muscle affected by myocardial infarction that cannot recover because of akinesia.

The value of left ventricular end-systolic volume index (LVESVI) versus ejection fraction is further characterized by Yamaguchi,¹⁰ Vanovershelde,¹¹ and their colleagues. Unfortunately, these fundamental manuscripts are not in the database of many leaders in the treatment of CHF. Data developed in CHF clinics and worldwide symposiums to deal with the manifold symptoms of CHF persist with this disease. Our patients sustain exhaustive and unfathomed tests that characterize the consequence of low output syndrome from the damaged muscle. The concept of sphericity is recognized but rarely calculated. There is emphasis on the *diastolic* spherical index rather than on systole when ejection fraction occurs. Articles on CHF that do focus on the spherical heart often do not emphasize the essential contraction and *suction* properties of the ellipse.

The National Institutes of Health has exhibited extraordinary interest in CHF. Many of its conceptual leaders focus on the special position of individual knowledge and the "unique responsibility of a certain gene of CHF." A broad extent of genetic factors is stimulated by the "process of CHF," but the capacity to focus on a single factor may

Di Donato M, Dor V. *Centre Cardiothoracique de Monaco 2000*

Figure 2. The relationship between LVESVI and ejection fraction. There is correlation, but great overlap ($r^2 = 0.29$). These values were described by Di Donato and Dor (Centre Cardiothoracique de Monaco, 2000) in patients undergoing restoration.

reflect a real limitation of focal knowledge in understanding the disease.

The first step is to understand the true central theme, the spherical dilated ventricle as the causative factor. As physicians, we must recognize that *remodeling* only enlarges our understanding of this abnormal area. The symptom purveys each patient study of regulated responses and captures our diagnostic and therapeutic interest. The importance of the novel objective to restore the geometry to a more normal ventricular shape should be recognized. Rebuilding fundamental fiber orientation structure will allow more normal muscle function. This is the true end point of our therapeutic approaches. Treat the disease, not the symptom.

This criterion of unifocalization is known as *knowledge*, and this is defined as "taking apart" or differentiating or analyzing approaches. Of course, the result will be an astounding expertise in one aspect of CHF, but where is the communication between other aspects and this observation? In and of themselves, they will not and cannot deal with any interconnected factors. The synthesis of key points of the aforementioned factors are then "put together" for integration. This solution is called *wisdom*. These concepts are drawn together by a vast degree of underlying knowledge. To do this, we must separate ourselves from the isolated expertise and then use a broad understanding of all aspects of individual factors toward the solid solution that is implemented.

Our role is to control symptoms after normal geometry is restored, not to displace correction of the problem. Conventional approaches deal with the vessels supplying blood to normal muscle, the secondary changes of the ventricle that cause valve insufficiency, but not with the abnormal ventricle directly.

At this time, these architectural changes can occur by surgical correction. In that surgical correction process, we *cannot* superimpose more intraoperative damage during restoration. Unless intraventricular injury from poor protection is avoided, preventable changes will markedly limit the influx of medical/surgical participation in this process and thereby exclude the use of surgical procedures that correct CHF.

The Evolution

The history of our organized escape from understanding normalcy is the 1-year allocation of time for the learning of normalcy (ie, the first year of medical school). This is followed by a 3-year drive to see pathologic conditions, understand them, learn them, and treat them. This information is used for the remainder of medical school training and throughout the physicians' medical lives. The *remodel end point* becomes our *starting point*. Some think it is a failure to return to the point of initial escape—the *normal heart*.

In the heart, for example, we learn about constriction for systole, as described by Harvey, and about dilatation for diastole, also described by Harvey. The concept of twisting and untwisting are unknown to many physicians and surgeons, even though they are precisely how the heart ejects and sucks to fill. These concepts date back to the Greeks, with Erasistratus and then Galen. They were discarded by Harvey, who was simply incorrect. Borelli described the heart as a rope with a vortex or spiral of fibers and clarified that ejection was like twisting a rope to wring out blood. Anatomists like Vesalius, Mall,¹² and others showed that muscle structure for twisting and untwisting occurs. Only with MRI can we now characterize this more precisely. Despite these factors that define normalcy, they are relatively unknown to those who practice medicine, surgery, and treat CHF throughout the health care community.

Instead, the ejection fraction is fundamental, with a near complete absence of either measuring the LVESVI or, more importantly, understanding its meaning when its vital value is given along with the conventional ejection fraction recording. The focus is on a specific number (ie, ejection fraction) rather than on understanding the concept of how the ellipse works to improve function and where fiber angle (not ejection fraction) is critical. This focus defeats our progress. Many experts know calcium, ionic balance, muscle fiber kinetics, electrophysiology, molecular biology, and neuroendocrine responses. Each factor is important, but coalition of these entities is needed.

The Present: A New Focus on the Akinetic Heart

Surgeons and cardiologists read but do not listen to the geometric solutions suggested by Jatene and Dor and Cooley to return ventricular shape toward an elliptical form. Others in the CHF community are unaware of these efforts.

To some, this seems like an “all too simple solution.” Perhaps that is the answer, *for nature is simple*. It is scientists who are complicated, as suggested by Torrent-Guasp (personal communication). We need to learn from understanding normality. The way to understand normality is to remove the current focus *only* on the pathologic remodeled aspect caused by disease.

The anatomic dilemma of CHF is a dilated ventricle. In coronary artery disease, especially with anterior infarction, there is replacement of the anterior wall and septum with either a scar or akinetic muscle with marginal salvage of epicardial regions after reperfusion. There is no aneurysm, but a nonfunctional muscle is left in place. The disease of CHF is related to dilatation of the remaining remote muscle. Its compensatory hypertrophy becomes diminished if coronary artery disease affects vessels supplying functional segments. The later onset of mitral regurgitation is due to increased ventricular distention. A restrictive valve defect develops with or without annular dilatation and compounds the symptoms. The conventional approach to restoring blood supply and mitral valve competence must become amended by rebuilding left ventricular geometry.

The surgical approach in this spherical formation is to exclude the noncontractile area with a patch or direct suture to delete apical and septal loss. The septum always must be excluded if it is diseased. This is a novel surgical approach, especially with nonscarred muscle, as occurs frequently with reperfusion with trabeculated muscle salvage without function. This results in alteration of left ventricular shape to restore an elliptical formation. This resultant geometric change reduces wall stress by limiting radius and increasing thickness. Simultaneously, there is increasing deformation of strain produced by more normal muscle fiber orientation.

An Alternate Objective: The Restored Ventricle

CHF occurs in 4.9 million patients in the United States and 69% have coronary artery disease. Among them, 90% have a myocardial infarction, which results in loss of regional contraction and thereby altered cardiac structure/function relationships. The geometric consequence is loss of the free wall and underlying septum. Left ventricular ejection predominantly is caused by longitudinal shortening relying on obliquely oriented fibers. There is conversion of normal elliptical shape with oblique fiber orientation (where 15% shortening causes 60% ejection fraction) to a spherical chamber where a more transverse fiber orientation reduces contractile forces (ie, 15% fiber shortening causes 30% ejection fraction) in remote viable muscle.¹³ MRI studies show that myocardial stress and strain reduce and decrease contractile shortening in remote myocardium (ie, from 15% to 10%) so that ejection fraction is lowered accordingly.¹⁴ Progressive dilatation of remote muscle further reduces its contractile force and increases CHF and mortality, depending on ultimate left ventricular size.^{7,8,10,15,16}

The prognosis of CHF after myocardial infarction is related to the volume and shape changes of the left ventricle. Regional infarction of more than 30% of left ventricular circumference causes progressive dilatation of remote viable muscle that tends to convert the normal elliptical left ventricular shape to a sphere.¹⁷ The normal LVESVI of 25 mL/m² increases to exceed 60 mL/m² when more than 40% of muscle becomes nonfunctional. However, an LVESVI of more than 45 mL/m² often progresses to cause clinical CHF.^{7,8,15-18} This onset of clinical symptoms may develop 6 months to 3 years after acute myocardial infarction as the remote muscle dilates.^{8,15} However, the propensity for CHF can be predicted by LVESVI measured 90 minutes after myocardial infarction.¹⁵ Assessment of left ventricular size at 12-month intervals after myocardial infarction is predictive of CHF and mortality.¹⁶ In patients first assessed at a time remote from the acute episode, a resting LVESVI of more than 60 mL/m² reflects a threshold likely to be associated with subsequent cardiac mortality.¹⁵

The surgical approach to scar is established, with 6% of patients who have coronary bypass surgery also undergoing aneurysmectomy. In the past, the conventional surgical decision to address the segment intraoperatively was based on how much scarred noncontracting segment collapsed (ie, dimpled) during left ventricular decompression, signifying that transmural scarred segment. Initial surgical procedures only excluded the bulging segment, as described by Cooley and associates^{18a} in 1959—the linear approach that *did not* exclude the aneurysm. There was negligible approach to the akinetic left ventricle, because exclusion of only anterior wall, without the septum, did not improve CHF symptoms.¹⁹ More recently, the entire thinly scarred region has been excluded, including the free wall and septum, through the work of Jatene²⁰ in 1984, imbricating the scarred region, Dor, Kreitmann, and Jourdan²¹ in 1984 (intraventricular patch), Cooley and associates²² in 1989 (endoaneurysmectomy), and Mickleborough and colleagues²³ in their article in this *Journal*.

Currently, thrombolytic treatment and percutaneous angioplasty for acute myocardial infarction have decreased aneurysm formation so that the scarred left ventricle, especially with dyskinesia, is uncommon. The “open artery” concept produces a wave front of death starting in the endocardium. Recent MRI studies by Bogaert and associates²⁴ confirm the epicardial viability and underlying necrosis of the inner two thirds of the left ventricular wall. A family of damage develops, with 80% to 90% of contractile muscle in the endocardium and midmyocardium lost, covered by a veneer of epicardial salvage to prevent the extensive remodeling with aneurysm formation. However, the incidence of CHF rises progressively, as the akinetic (noncontractile) left ventricle remains and progressive remote dilatation occurs. The infarcted noncontractile section re-

mains akinetic and still involves the free wall and septum, but operatively has a normal surface from epicardial salvage. We must now focus on this akinetic segment.

Surgical Technique

Ventricular reduction surgery is termed *surgical ventricular restoration* and follows the format of established approaches to ventricular scar established by Dor, Jatene, and Cooley and now used in aneurysm surgery. The procedure changes the spherical left ventricular characteristics of CHF to a more normal elliptical shape to lower wall stress and enhance thickening of remote viable muscle, through increasing deformation (ie, torsion), by producing a more helical fiber orientation.

The straightforward surgical approach, before infarct revascularization (ie, open coronary artery), was with the nonfunctional transmural scar that dimpled during left ventricular decompression (venting) to clarify the “free wall” part of injured muscle. The primary approach was toward aneurysms (bulging segment). More recently, these approaches have been applied to thin akinetic (nonparadoxical) scars, as well as conventional aneurysms.²⁰⁻²² There has been restricted approach to the septum. Aspiration defines only transmural scar, but it does not describe the nonfunctional region that causes CHF. Attention to only the thin wall limits septal exclusion, as occurred in only 12% of Mickleborough’s cases,²³ compared with exclusion of 100% in an approach described by Dor²⁵ and Jatene.²⁰ Mickleborough achieved a 6% increase in ejection fraction after restoration of akinetic hearts without septal exclusion versus Dor’s 18% increase in ejection fraction by excluding the septum and 40% reduction in size.²⁵

Because of thrombolysis or percutaneous transluminal coronary angioplasty, there is now salvage of epicardial muscle, preventing dyskinesia (ie, paradox), yet MRI evidence of necrosis of the inner shell of midventricular and endoventricular muscle persists.¹⁴ Akinesia is evident on ventriculogram, echocardiogram, and MRI, yet the epicardial surface, surgically, is normal with occasional trabecular scar. The akinetic wall does not collapse during left ventricular venting, yet the decision for exclusion of the nonfunctional free wall and septum is made *preoperatively*, through nonfunction documented by dynamic assessment (ventriculogram, echocardiogram).

The left ventricle can be opened with conventional cardioplegic approaches (used for coronary bypass grafting and mitral valve repair when needed) to define an endocardial scar. Absence of visible scar is not problematic, because the aortic clamp is removed to allow the heart to beat so that digital palpation (not vision) allows decisions about separation between noncontracting free wall and septum and furthermore documents viable contracting remote muscle (ie, inferior or lateral wall and septum) that was made dysfunctional by myocardial stretching. This digital (not

Figure 3. Relationship between preoperative LVESVI (mL/m²) and survival to 10 years. Note increased long-term survival if LVESVI is more than 120 mL/m². These patients underwent surgical ventricular restoration at Centre Cardiothoracique de Monaco by Dor.

visual) separation allows a circumferential Fontan stitch to be placed to reduce cavity size and create a “surgical neck” for exclusion of noncontracting free wall and septal non-functioning regions. The ellipse is restored by inserting an oval patch (Dacron, pericardium, septal flap), or by using interrupted sutures to deal with the nonscarred, nonfunctional, more muscular suture bed.

The technique of placing sutures into muscle rather than scar may include interrupted sutures with pledgets for hemostatic security. This mechanical technique is firmly established surgically, as the method mirrors, precisely, what is used for valve replacement. Here, the oval orifice within the ventricle, without moving parts, becomes substituted for the valve orifice when a prosthesis is placed in patients with valve disease. The surgical oval site and suture techniques are commonplace. There is no risk for air embolism in the beating open heart, as this technique has been used for many years with antiarrhythmic approaches during intraventricular surgery.

The improved myocardial protection afforded during restoration by either conventional cardioplegia or the beating state becomes coupled with rebuilding a more normal elliptical geometry to augment ejection fraction intraoperatively and postoperatively. Consequently, mechanical support is infrequently needed in these high-risk patients with severe left ventricular dysfunction.

Another factor, which is important in evaluating the role of surgical ventricular restoration, is that medical therapy for treating CHF has advanced significantly. Randomized, placebo-controlled blinded studies have shown that β -blockers, when added to digoxin, diuretics, and angiotensin-converting enzyme inhibitors, decreased mortality by at least 30%, with annual mortality rates for patients with class II and III CHF on β -blockers less than 15%. In addition, there is similarly impressive reduction in frequency of hospitalizations for cardiovascular diseases and decreased pred-

gression for CHF. Furthermore, trials of coronary bypass grafting with left ventricular failure in which ejection fraction is less than 35% show low operative mortality (~3%) and 65% to 75% 5-year survival.^{2,26} Despite these encouraging survival results, approximately 50% of patients have CHF at the 5-year follow-up interval,²⁶ and 10-year mortality is 60% to 80% if ejection fraction is either less than 35% or less than 25%.²

In 1998, an international cooperative effort of surgeons and cardiologists to investigate the surgical vascular restoration (surgical ventricular restoration) procedure was organized to include leading centers in the United States, Europe, Asia, and South America. Nine centers have collected and shared information on 439 consecutive patients undergoing the surgical ventricular restoration procedure.²⁷ The team is called RESTORE (rebuilding normal elliptical architecture). The mean ($\pm$ SD) age of these patients was 63 ± 10 years; baseline ejection fraction was $28\% \pm 10\%$ with a median LVESVI of 110 mL/m^2 . Akinesia was present in 67% and dyskinesia in 33%. One hundred one of the patients (23%) also had mitral valve repair and 96% had bypass grafting. Only 7% required intra-aortic balloon pumping. Operative mortality was 5.7%, estimated 1.5-year mortality was 10%, and rehospitalization for CHF was 7%. Postoperative ejection fraction was $39\% \pm 12.6\%$ (an increase of 10.5 ± 0.5 percentage points) with a median LVESVI of 68 mL/m^2 (a 34.5-mL/m^2 decrease).

The left ventricular shape, independent of ejection fraction, defines prognosis after surgical treatment to restore a smaller size and increase elliptical shape during rest and exercise. The operative mortality was only 5.7% in 439 patients in a recent series, yet the large left ventricular shape, even after successful surgical treatment of the three components in CHF (coronary bypass grafting, mitral valve repair, and left ventricular shape) determined long-term prognosis after follow-up over 10 years in all survivors (Figure 3).

Studies of left ventricular shape in the RESTORE patients, which supplement the extensive experience of Vincent Dor and Marisa Di Donato, show surgical restoration of size will reduce LVESVI from 110 to 68 mL/m^2 . Our collaborative surgical/medical team has taken the steps suggested in my editorial in 1998,²⁸ in which restoration becomes a component of surgical treatment of the failing akinetic heart. The extent of postoperative reduction is related to initial LVESVI. Clearly, the extent of irreversible changes from chronic dilatation leading to remote muscle scar will become greater as the volume of viable muscle becomes larger. Conversely, the incidence of CHF and mortality without restoration exceeds 30% in 1 year if LVESVI is more than 60 mL/m^2 immediately after myocardial infarction.¹⁵ These LVESVI signposts immediately after infarction can be useful to *both treat* CHF and *predict*

CHF in left ventricles with systolic dysfunction and increased volume. A change in geometry may occur in postinfarction patients undergoing revascularization with a large ventricle *before* CHF symptoms develop.

Restoration: Hibernation and Mitral Valve

The surgical rebuilding of left ventricular volume excludes the infarct region that can be dyskinetic, akinetic, or significantly hypokinetic from epicardial viability from recent revascularization.¹⁴ The restoration value of exclusion becomes even more apparent if LVESVI is greater than 75 mL/m² and positron emission tomography or dopamine viability exists in a nonfunctioning or severely hypokinetic scar that is conventionally thought to be hibernating. Revascularization alone does not change left ventricular volume¹¹ in large ventricles (>75 mL/m²) so that the potential area of regional hibernation does not recover. In small hearts (<75 mL/m²) the delay of increased contraction of the infarcted region is 1 to 6 months. Only then increased regional shortening of remote muscle follows to improve ejection fraction. In contrast, exclusion of the nonfunctional area restores immediate contractility (independent of initial volume)²⁵ because the left ventricular size is reduced surgically, the remote muscle develops a reduced radius and becomes thickened, and ejection fraction rises immediately after the restoration procedure.

Currently, there is a “biphasic approach” toward either the mitral valve or the ventricle. Mickleborough and associates²³ deal with the left ventricle and do not change the mitral valve. In contrast, Bolling and associates⁴ changed the mitral valve and did not change the ventricle. They reported reduced sphericity, increased New York Heart Association function, and low operative mortality, but the 5-year results showed approximately 50% mortality. Left ventricular volumes were not reported, and I presume that changes in cardiac size accounted for the late mortality. These results paralleled late valve replacement (aortic and mitral) results, with approximately 60% mortality if ejection fraction was less than 40%, even though the surgical procedures provided a perfectly competent valve.³

It seems clear that the large ventricle determines ultimate prognosis. Mitral insufficiency is a dynamic process that worsens with exercise, as the left ventricle dilates and changes tethering of the chords and papillary muscle to decrease coaptation. Certainly, left ventricular restoration improves mitral valve function (81%), yet some insufficiency persists.²³ Consequently, the residual insufficiency can produce a *secondary* increase in cavity size that can potentially offset the value of restoration unless *both mitral and ventricular* aspects are corrected. The surgical approach to CHF is therefore a *trilogy* to deal with the vessels, valve, and ventricle.

Evaluation Changes

Classic symptoms of CHF (fatigue or shortness of breath) can be characterized most clearly through physiologic measurements of peak oxygen uptake—how cardiac output increases to deliver flow to meet metabolic needs with exercise. This physiologic measurement of peak oxygen consumption correlates with progression of, at rest, left ventricular size/time in patients with CHF (independent of resting ejection fraction or volume) and progressive mortality.²⁹ After conventional medical treatment (angiotensin-converting enzyme inhibitors, β -blockers) mortality is high at 18% if oxygen consumption increases, and doubles to approximately 40% if oxygen consumption falls during sequential yearly measurements.^{16,29} Of equal importance to resting shape, the dynamic shape with exertion determines exercise capacity.³⁰ Oxygen consumption rises if the left ventricle becomes more elliptical (reduced LVESVI) and falls if the left ventricle becomes more spherical (increased LVESVI). The measurement provides a physiologic marker of ventricular mechanics for CHF progression. A more elliptical shape during systole improves fiber orientation to augment ejection (ie, MRI twisting).³⁰ Similarly, early diastolic filling needed to augment venous return for cardiac output is enhanced after increased ellipse during exercise; demonstrating untwisting on MRI will improve diastolic suction.³¹

Approximately 30% of patients with an ejection fraction of less than 35% in a recent series had moderate (2+) mitral regurgitation with increased spherical chamber formation.²⁷ Exercise capacity (ie, peak oxygen consumption) is reduced if there is only mild (1+) mitral regurgitation under resting conditions.³² Mild mitral regurgitation at rest restricts oxygen consumption during exercise and thereby accounts for the increased CHF symptoms during exertion. This physiologic measurement may be useful in preoperative prediction of need for mitral valve repair in patients undergoing coronary bypass alone or in combination with a restoration procedure.

Arrhythmic Approaches

The predominant cause of late death in CHF is either progressive hemodynamic deterioration or sudden death. Distention is a key factor to late arrhythmia development. In the past, correction of electrophysiologic defects responsible for sudden death included cryotherapy to the unexcised muscle and endocardectomy. This method was used by Mickleborough²³ and Dor,²⁵ resulting in few patients dying of late ventricular tachycardia. The role of decompression alone in reducing late arrhythmias is not yet known. Many surgeons test patients postoperatively by programmed ventricular stimulation and insert an automatic implantable cardiac defibrillator or use antiarrhythmic drugs. Subsequent comparisons will define which approach is more appropriate.

TABLE 1. Potential CHF changes in diagnosis, treatment, and management

	Conventional	Alternative
Diagnosis	EF	ESVI, percent asynergy
Medical treatment	CABG + MVR	Conventional + surgical ventricular restoration
Evaluation of treatment	Symptoms	VO ₂ , PAP

The "alternative" pathway means LVESVI and asynergy (noncontractile area) are measured in addition to ejection fraction (EF). Surgical treatment will amend coronary bypass grafting ± mitral valve repair (CABG ± MVR) with restoration. A more precise prognosis will supplant New York Heart Association class with measurements of oxygen consumption (VO₂) during exercise and changes of pulmonary artery pressure (PAP) by echocardiogram.

This geometric change in mechanical muscle formation markedly diminishes the requirements for *electrical* treatment of heart failure. Wrapping the muscle, cardiomyoplasty, is not needed because volume is diminished surgically. Automatic implantable cardiac defibrillators are uncommon, due to diminished dilatation and resultant ectopy. Increased pulmonary artery pressure becomes limited, because function of the left and right sides of the heart is improved by restoring an elliptical shape. Consequently, electrical monitoring of pulmonary artery pressure to avoid rehospitalization becomes uncommon. The septum of the restored heart beats spontaneously immediately after the operation if myocardial protection is adequate.³³ Consequently, the need for biventricular pacing is limited or avoided.

The Future

We must more properly measure how the left ventricle functions to eject and fill. This amends standard clinical signs of dyspnea, fatigue, and edema. Sufficient twisting for ejection must provide enough cardiac output to meet metabolic needs during exercise, while appropriate untwisting for suction of venous return avoids raised filling pressure to cause lung congestion. These consequences of ejection and filling are not currently measured but are achievable through evaluating peak oxygen uptake during exercise and noninvasive pulmonary artery pressure by echocardiography. Organized use of these available evaluation methods can enlarge precision in the extent of CHF and its alteration by treatment.

I have summarized 3 events (Tables 1 and 2) that reflect a fundamental alteration in how the ventricle is viewed (ie, change from only fiber shortening to include evaluation of size and shape), how dysfunction is treated (ie, evaluating how geometric changes of size complement conventional medical and surgical treatment), and how treatment is evaluated by supplementing symptomatic assessment by measuring peak oxygen uptake during exercise.

TABLE 2. If restoration is useful, this challenge may alter the way outcome is evaluated, treated, and measured in patients with CHF

Introduce to:	
Cardiologists	Importance of LV size
Surgeons	When to alter LV geometry
CHF clinicians	Efficacy of exercise VO ₂ measurements

CHF, Congestive heart failure; LV, left ventricular; VO₂, oxygen consumption.

These geometric observations on the mechanical adjustment may reduce the current *symptomatic* treatment of the consequent disease of a spherical heart. The surgical restoration of the left ventricular ellipse may reduce the extraordinary expansion of drugs, surgery, and electrical devices toward morbidity and mortality related to focus *only* on remodeling away from normal. The mechanical solution of that course needs further testing. All must recognize the normal cardiac helical structure, and our interest is to strive to restore the geometric configuration responsible for heart function. This may augment reserve and may avoid medical, surgical, electrical, and health care costs currently related to only reducing morbidity. The hope is to separate this treatment from the present mortality and morbidity end point, where the disease is left intact, but only modified. Our central focus may change, but this can occur only when we begin to understand, and then restore, the normal structure with its attendant functional counterpart. Normality, rather than less disease, should prevail to achieve the solution to CHF.

References

1. Helmer GA, McKirnan MD, Shabetai R, Boss GRRJ Jr, Hammond HK. Regional deficits of myocardial blood flow and function in left ventricular pacing-induced heart failure. *Circulation*. 1996;94:2260-7.
2. Trachiotis GD, Weintraub WS, Johnston TS, Jones EL, Guyton RA, Craver JM. Coronary artery bypass grafting in patients with advanced left ventricular dysfunction. *Ann Thorac Surg*. 1998;66:1632-9.
3. Durate IG, Murphy CO, Kosinski AS, Jones EL, Craver JM, Gott JP, et al. Late survival after valve operation in patients with left ventricular dysfunction. *Ann Thorac Surg*. 1997;64:1089-95.
4. Bolling SF, Pagani FD, Deeb GM, Bach DS. Intermediate-term outcome of mitral reconstruction in cardiomyopathy. *J Thorac Cardiovasc Surg*. 1998;115:381-6.
5. Thompson JG. Production of severe atheroma in a transplanted human heart. *Lancet*. 1969;2:1088.
6. Bieber CP, Reitz BA, Jamieson SW, Oyer PE, Stinson EB. Malignant lymphoma in cyclosporin A treated allograft recipients [letter]. *Lancet*. 1980;1:43.
7. White HD, Norris RM, Brown MA, Brandt PW, Whitlock RM, Wild CJ. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. *Circulation*. 1987;76:44-51.
8. Gaudron P, Eilles C, Kugler I, Ertl G. Progressive left ventricular dysfunction and remodeling after myocardial infarction: potential mechanisms and early predictors. *Circulation*. 1993;87:755-63.
9. Di Donato M, Sabatier M, Toso A, Barletta G, Baroni M, Dor V, et al. Regional myocardial performance of non-ischaemic zones remote

- from anterior wall left ventricular aneurysm. *Eur Heart J*. 1995;16:1285-2.
10. Yamaguchi A, Ino T, Adachi H, Murata S, Kamio H, Okada M, et al. Left ventricular volume predicts postoperative course in patients with ischemic cardiomyopathy. *Ann Thorac Surg*. 1998;65:434-8.
 11. Vanoverschelde JL, Depre C, Gerber BL, Borgers M, Wijns W, Robert A, et al. Time course of functional recovery after coronary artery bypass graft surgery in patients with chronic left ventricular ischemic dysfunction. *Am J Cardiol*. 2000;85:1432-9.
 12. Mall FP. On the muscular architecture of the ventricles of the human heart. *Am J Anat*. 1911;11:211.
 13. Ingels NB Jr. Myocardial fiber architecture and left ventricular function. *Technol Health Care*. 1997;5:45-52.
 14. Bogaert J, Bosmans H, Maes A, Suetens P, Marchal G, Rademakers FE. Remote myocardial dysfunction after acute anterior myocardial infarction: impact of left ventricular shape on regional function—a magnetic resonance myocardial tagging study. *J Am Coll Cardiol*. 2000;35:1525-34.
 15. Migrino RQ, Young JB, Ellis SG, White HD, Lundergan CF, Miller DP, et al. End-systolic volume index at 90 to 180 minutes into reperfusion therapy for acute myocardial infarction is a strong predictor of early and late mortality. The Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries (GUSTO)-I Angiographic Investigators. *Circulation*. 1997;96:116-21.
 16. Florea VG, Henein MY, Anker SD, Francis DP, Chambers JS, Ponikowski P, et al. Prognostic value of changes over time in exercise capacity and echocardiographic measurements in patients with chronic heart failure. *Eur Heart J*. 2000;21:146-53.
 17. Pfeffer MA, Lamas GA, Vaughan DE, Parisi AF, Braunwald E. Effect of captopril on progressive ventricular dilatation after anterior myocardial. *N Engl J Med*. 1988;319:80-6.
 18. Hammermeister KE, DeRouen TA, Dodge HT. Variables predictive of survival in patients with coronary disease: selection by univariate and multivariate analyses from the clinical, electrocardiographic, exercise, arteriographic, and quantitative angiographic evaluations. *Circulation*. 1979;59:421-30.
 - 18a. Cooley DA, Henly WS, Ahmad KH, Chapman DW. Ventricular aneurysm following myocardial infarction: results of surgical treatment. *Ann Surg*. 1959;150:595-612.
 19. Mangschav A. Akinetic versus dyskinetic left ventricular aneurysms diagnosed by gated scintigraphy: difference in surgical outcome. *Ann Thorac Surg*. 1989;47:746-51.
 20. Jatene AD. Left ventricular aneurysmectomy: Resection or reconstruction? *J Thorac Cardiovasc Surg*. 1985;89:321-31.
 21. Dor V, Kreitmann P, Jourdan J. Interest of “physiological” closure (circumferential plasty on contractive areas) of left ventricle after resection and endocardectomy for aneurysm or akinetic zone comparison with classical technique about a series of 209 left ventricular resections [abstract]. *J Cardiovasc Surg*. 1985;26:73.
 22. Cooley DA, Frazier OH, Duncan JM, Reul GJ, Krajcer Z. Intracavitary repair of ventricular aneurysm and regional dyskinesia. *Ann Surg*. 1992;215:417-23.
 23. Mickleborough LL, Carson S, Ivanov J. Repair of dyskinetic or akinetic left ventricular aneurysm: results obtained with a modified linear closure. *J Thorac Cardiovasc Surg*. 2000;121:675-82.
 24. Bogaert J, Maes A, Van de Werf F, Bosmans H, Herregods MC, Nuyts J, et al. Functional recovery of subepicardial myocardial tissue in transmural myocardial infarction after successful reperfusion: an important contribution to the improvement of regional and global left ventricular function. *Circulation*. 1999;99:36-43.
 25. Dor V, Sabatier M, Di Donato M, Montiglio F, Toso A, Maioli M. Efficacy of endoventricular patch plasty repair in large postinfarction akinetic scar and severe left ventricular dysfunction: comparison with a series of large dyskinetic scar. *J Thorac Cardiovasc Surg*. 1998;116:50-9.
 26. Luciani GB, Montalbano G, Casali G, Mazzucco A. Predicting long-term functional results after myocardial revascularization in ischemic cardiomyopathy. *J Thorac Cardiovasc Surg*. 2000;120:478-89.
 27. Athanasuleas CL, Stanley AWH Jr, Buckberg GD, Dor V, Di Donato M, Blackstone EH, and the RESTORE Group. Surgical anterior ventricular endocardial restoration (SAVER) in the dilated remodeled ventricle following anterior myocardial infarction. *J Am Coll Cardiol*. In press.
 28. Buckberg GD. Surgery for adult cardiovascular disease. *J Thorac Cardiovasc Surg*. 1998;116:47-9.
 29. Florea VG, Henein MY, Anker SD, Francis DP, Gibson DG, Coats AJ. Relation of changes over time in ventricular size and function to those in exercise capacity in patients with chronic heart failure. *Am Heart J*. 2000;139:913-7.
 30. Tischler MD, Niggel J, Borowski DT, LeWinter MM. Relation between left ventricular shape and exercise capacity in patients with left ventricular dysfunction. *J Am Coll Cardiol*. 1993;22:751-7.
 31. Tischler MD, Ashikaga T, LeWinter MM. Relation between left ventricular shape and Doppler filling parameters in patients with left ventricular dysfunction secondary to coronary artery disease. *Am J Cardiol*. 1995;76:553-6.
 32. Tada H, Tamai J, Takaki H, Ohnishi E, Okano Y, Yoshioka T. Mild mitral regurgitation reduces exercise capacity in patients with idiopathic dilated cardiomyopathy. *Int J Cardiol*. 1997;58:41-5.
 33. Athanasuleas CL, Stanley AWH Jr, Buckberg GD. Restoration of contractile function in the enlarged left ventricle by exclusion of remodeled akinetic anterior segment: surgical strategy, myocardial protection, and angiographic results. *J Card Surg*. 1998;13:418-28.