SURGERY FOR ADULT CARDIOVASCULAR DISEASE

EDITORIAL: DEFINING THE RELATIONSHIP BETWEEN AKINESIA AND DYSKINESIA AND THE CAUSE OF LEFT VENTRICULAR FAILURE AFTER ANTERIOR INFARCTION AND REVERSAL OF REMODELING TO RESTORATION

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Imprecise definition of a problem may be the largest obstacle to its resolution. Similarly, the solution of a problem is determined by a precise view of its cause. Dor and associates (see page 50) have provided this in their recognition of the cause and consequences of a left ventricular (LV) scar after myocardial infarction and their method of reversing this sequence with the endoventricular circular patch plasty (EVCPP) technique or, more correctly, the Dor procedure.^{1,2}

Dor and his associates have challenged the current understanding of an akinetic LV scar after acute anterior myocardial infarction. This is the most common consequence of occlusion of the left anterior descending (LAD) coronary artery. In doing so, they have simultaneously drawn together the seemingly physiologically and surgically different areas of akinesia versus dyskinesia under a common sequence of regional LV dysfunction (or asynergy) and have shown how exclusion of the nonfunctional segment enhances function. Their technique provides an earlier and clearer understanding of postinfarction aneurysm. Consequently, correction may be attempted earlier, before changes in remote contracting muscle³ limit the benefits of this method of ventricular remodeling to ensure pumping efficiency.

To my surprise, the term “remodeling” does not appear in many editions of *Webster's Dictionary* or in

Roget's Thesaurus. However, the antonym to “model” is “unhealthy” or “imperfect.” In one dictionary, remodel is defined as “to make over, to take away from normal.” The term “restore” is defined everywhere as “to bring back to health, revive, reestablish perfection, or to give back something taken away.” Thus restoration reverses remodeling, and that is a surgical objective of the Dor procedure.

The traditional concept of the results of LAD occlusion, with or without revascularization (medical or surgical), is the production of a scar that does not contract (i.e., akinesia) or that paradoxes during systole (i.e., dyskinesia). This is defined by ventriculography as nonparadoxing (i.e., akinetic) or bulging or paradoxing (i.e., dyskinetic) segments without quantification. The ejection fraction (EF) is evaluated separately and sometimes divided into the global EF and the contractile EF (i.e., the nonischemic area observed to shorten during systole).⁴ This definition is furthered intraoperatively, as infarcted muscle has the appearance of a scar (i.e., transmural collagen tissue that collapses on LV venting) or damaged muscle that is paler than normally perfused muscle and contains clear linear patches of yellow scarred segments. We have previously decided not to treat the akinetic segment and to reconstruct the dyskinetic region by the linear method only if significant paradox is evident on the ventriculogram.^{5,6}

The decision to address this segment of the anterior wall is usually based on how much the noncontractile scarred segment collapses during LV venting. Small segments with akinetic muscle are frequently left alone surgically, because multiple reports suggest improvement in the symptoms of LV failure, and reduction in cardiac diameter on chest x-ray films occurs only when large dyskinetic regions are removed by the linear approach.^{5,6} This “standard” method (1) excises the region of the collapsed

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anterior LV scar and approximates the "border" to the more rigid anterior septal and lateral wall borders and (2) excludes direct approaches to the septum.

Since 1984, Dor has used a technique (1) to exclude the septum in ventricular remodeling, (2) to narrow the aneurysm neck, and (3) to crystallize our concept of scar by using the area of asynergy (i.e., number of centerline segments that are > 2 standard deviations [SD] from normal).^{4,7} Consequently, the akinetic and dyskinetic regions are similar (i.e., both > 2 SD for normal), have a grade of zero, and differ only in bulge or noncontraction that is recorded as A% or percent muscle greater than 2 SD from normal.⁷

The centerline ventriculographic definition makes akinesia and dyskinesia the same consequence of LV scar and leaves the appearance of bulge or akinesia as only a radiologic or surgical definition. More important, this changes completely the conceptual understanding of akinesia versus dyskinesia and defines aneurysm as a noncontractile segment. The value of this more global definition is that the operation must exclude the noncontractile septum. The EVCPP repair, or Dor procedure, does precisely this.

This more physiologic concept exceeds the limitations of current ventriculographic and surgical descriptions, which do not quantify the muscle involved. The centerline method describes motion of the septum and all LV segments. Dor's description does not include positron emission tomography or transesophageal echocardiography with dobutamine stimulation data as predictors of wall improvement by revascularization. These tests reportedly demonstrate that contraction increases marginally (1 SD) in akinetic segments. Consequently, only severely hypokinetic or possibly moderately hypokinetic muscle develops after revascularization.

Simultaneously, by inclusion of the endoventricular surgical suture suggested by Fontan,⁸ the aneurysmal neck is systematically narrowed to help restore the more normal circular architecture. The patch, with either a Dacron or mobilized scar of the endocardial aneurysm, provides the only nonfunctional component. Before the endoventricular monofilamentous suture technique was developed by Fontan,⁸ this aneurysmal neck was reduced by Dor by limiting the size of the patch relative to the visible opening of the internal border of the junction between the scar and normal muscle.

Dor's more organized reduction of LV volume and increasing EF from 24% to 42% occurred

similarly with the EVCPP approach in both akinetic and dyskinetic asynergic aneurysms. Dor reports a time delay between infarct and ventricular remodeling of between 37 (dyskinetic) and 48 (akinetic) months, so that progressive changes may occur in remote muscle. This added time delay may account for the number of patients with EFs less than 30% in this article.

Review of the report by Gaudron and associates³ shows that the trend toward increased LV end-diastolic pressure and volume occurs within 6 months after infarction. This observation suggests that a more precise analysis of patients having an acute myocardial infarction with LAD walls that are akinetic (i.e., the majority of patients with or without angioplasty or thrombolysis) should serve as a warning to allow us to intervene earlier. Furthermore, we must recognize that current reperfusion methods with normal blood accomplish the two goals of reducing arrhythmias and improved remodeling; they do not avoid scar.

The provision of new nourishment immediately after acute infarction reduces mortality, because opening the infarct vessel converts a potential transmural scar to one that is limited by salvage of an intact subepicardial border zone. This viable region can (1) cause angina even though it does not contract and (2) prevent dyskinesia. The development of a paradoxical scar is also unlikely without revascularization of the LAD territory, because aneurysmectomy is now uncommon.

The common consequence of akinetic regions leaves very few patients with the type of muscle surgeons would currently consider for restoration. This is due to our heretofore imprecise definition of the pathophysiology of LV scar; that is, we have yet to appreciate that akinesia and dyskinesia are similar and that they differ only by the systolic "bulge." We do, however, recognize that the paradoxical segment may steal stroke volume during systole and further reduce the ability of remote muscle to compensate hemodynamically.

The classic definition of LV failure is that in the absence of mitral insufficiency there is insufficient viable muscle to generate a satisfactory cardiac output. Our role surgically is to limit this inability by restoring the LV architecture as close to normal as possible while preserving the geometry of cardiac curvature. This occurs with the EVCPP. We must question (1) why the ventricle was allowed to become so large before intervention and (2) how to define more precisely the LV aneurysm neck so that

sufficient remote volume is retained to further improve EF. This may be possible only with the earlier restoration approaches that are recommended.

Dor has pursued these studies for 13 years in more than 700 patients, and he has shown that akinesia and dyskinesia are part of the same process; they do not affect the type of restoration used. Clearly, an anterior aneurysm is a noncontractile or asynergic muscle that includes the septum as well as the anterior wall and apex. We must therefore reclassify our concept of aneurysm to indicate scar or noncontractile segment. This asystolic region makes the remaining ventricle enlarge and compensate for the inability of the scarred segment to help generate cardiac output. When this occurs, we will then recognize that akinesia versus dyskinesia is a verbal and not an operative description; both lead to remodeling and subsequent cardiac failure.

Dor has made this point to me repeatedly since our first meeting, but it took me a while to fully appreciate the role of this noncontractile ventricular region. Fontan reiterated this concept in extension of new knowledge in his honored guest's address before The American Association for Thoracic Surgery in 1990. Fontan addressed primarily aneurysms, and he contrasted Dor's work with that of Cooley⁹ and Jatene.¹⁰ Each of them also addressed bulging aneurysms and did not deal with the akinetic segment. Fontan's desire was for cardiac surgeons to build on the current database with new knowledge provided by Dor. The test of time supports the EVCPP approach, by showing improved EF in failing hearts.

Dor reported a 12% mortality in those patients with EFs less than 30%. This mortality may be reduced with different techniques of myocardial protection and also may limit the need for intraaortic balloon pumps, which are currently needed in 19% of patients with EFs less than 20%.^{4,7} Reduced intraoperative damage from cardioprotective methods may increase the use of the Dor procedure and provide greater confidence of avoiding damage during the learning process in patients with poorly contracting ventricles.

We must listen to lessons learned from more than 700 cases in 13 years and begin to change. We must (1) redefine our concept of aneurysm, (2) eradicate the concept that the septum should be excluded,

because indeed the septum must be included, (3) recognize that heart failure is treated best by excluding the scarred segment responsible for its presence (i.e., nonresectable septum), (4) understand that ventricular restoration reverses the adverse effect of remodeling after postinfarction scar, and (5) recognize that restoration and revascularization are part of the same surgical process and cannot be separated. Dor has provided us with these views, and in doing so he has made a remarkable and fundamental contribution to our knowledge, which paves the way for surgical restoration of the remodeled LV.

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