INTRODUCTION

The Most frequent cause of heart failure is post infarction ischemic heart disease. Even with early revascularization, there is a subset of patients whom myocardial infarction leaves with ventricular shape and function abnormalities that increase their risk of further ischemic events and of death. The transition from occluded vessel to a scar to dilatation results in a remodeled ventricle. (1)

The first angiographic documentation of ventricular aneurysm in 1951 by Dolly and colleagues prompted surgical interest in treatment of ischemic ventricular dilatation. The first successful surgical resection of a ventricular aneurysm was reported in 1955 by Likoff and Bailey. In 1980, several new reconstructive techniques were developed, the most popular of which was that described by Dor. (2)

The results of surgical ventricular restoration reported by Dor and co-authors showed that the procedure improve left ventricular function, New York heart association functional class, and survival by reducing ventricular volumes and increasing the ejection fraction . These results were observed not only in patients with classic dyskinetic aneurysms, but also in those with dilated ischemic cardiomyopathy and severe left ventricular dysfunction. (3)

According to Dr. Menicanti and Dr. Di Donato they consider the following to be the indications of the procedure: anteroseptal infarction and dilated left ventricle, depressed ejection fraction, left ventricular regional asynergy, symptoms of (angina, heart failure, arrhythmias, or combination of the three), and/or inducible ischemia on provocative tests in asymptomatic patients. (4)

Functional mitral regurge is frequently associated with post ischemic ventricular dilatation, and its presence worsens prognosis. At present it hasn't been established whether, when, and how the

mitral valve should be repaired during surgical ventricular restoration. The presence of moderate to severe mitral regurge and its surgical repair carries a higher operative risk. It is difficult to distinguish whether the higher mortality is due to surgical procedure per sè, because patient with mitral regurge have a worse clinical and hemodynamic condition. (5)

Surgery of left ventricular anterior reconstruction in the setting of ischemic cardiomyopathy has a long history. it has evolved over the years from a linear closure of large aneurysms to present geometric reconstruction by means of endoventricular patch plasty and it is still evolving with more hope for ischemic cardiomyopathy patients.(6)

AIM OF THE WORK

The aim of this study is to describe surgical ventricular restoration as an alternative in treatment of ischemic heart failure. Compare different surgical techniques with more details on the Dor procedure as the most popular technique. And highlight with further data on surgical ventricular restoration with mitral regurge.

MATERIALS AND METHODS

The current paper is intended as practical review of the surgical ventricular restoration procedure as a modality of treatment for ischemic heart failure. We performed a PubMed search for the term "surgical ventricular restoration" in order to identify studies, with focus on those published within the last five years.

PATHOLOGY

Heart failure (HF) remains a major public health problem. Almost 5 million patients in the United States are affected, and 30 to 40% of patients die from HF within 1 year after the diagnosis (7). The incidence and prevalence of heart failure continues to increase due in part to an extended average life expectancy and morbidity and mortality remain high despite improvement in treatment. HF is the leading cause of hospitalization for persons over 65 years of age, and rates of hospital readmission within 6 months range from 25% to 50%, resulting in a large economic burden (8, 9).

Left ventricular remodeling is the process by which mechanical, neurohormonal, and possibly genetic factors, alter ventricular size, shape, and function. Remodeling occurs in several clinical conditions, including myocardial infarction (MI). Myocardial infarcts (MI), particularly large, transmural infarcts, result in a number of structural changes involving both the infarcted and non-

infarcted zones (10). LV remodeling usually begins within the first few hours after a MI and may progress over time. (3)

A model for left ventricular remodeling after infarction. Based on the observations that wall stresses may be abnormal in the infarcted ventricle and the fact that increased diastolic wall stress may initiate a volume- overload type of hypertrophy, a model of left ventricular remodeling after myocardial infarction may be proposed. (11) In this model, the immediate hemodynamic consequences of an acute myocardial infarction on ventricular function include both systolic and diastolic dysfunction. Systolic impairment secondary to loss of contractile function of the infarcted myocardium results in a decreased systolic ejection, increased end-systolic volume, an increase in cardiac size, and a secondary increase in diastolic filling pressure caused by the increase in ventricular volume. Diastolic function is characterized immediately by an increase in diastolic distensibility, (12, 13) which minimizes the rise in filling pressure. However, as necrotic

tissue is replaced by fibrosis, a decrease in distensibility occurs. In addition, there may be upward shift in the diastolic pressure-volume curve secondary to ischemia in the border zone so that filling pressure may tend to be higher for any given volume. Thus diastolic volume increases and diastolic pressure also tends to increase, especially in patients with large infarctions. In the presence of both systolic and diastolic dysfunction, there may be peripheral mechanisms mediated via the sympathetic nervous system and circulating catecholamines to help maintaining a normal arterial blood pressure and cardiac output. These mechanisms may subsequently increase ventricular preload by augmenting venous return and increase ventricular afterload by causing arteriolar vasoconstriction. The resulting increases in ventricular radius and diastolic pressure will, in combination, lead to an increase in end-diastolic wall stress in all parts of the ventricle. (11) Simultaneous with an increase in both end-diastolic wall stress and regional end-systolic wall stress in infarcted

segments, there is weakening of the normal myocardial structure needed to resist wall stress. As a result, processes of dilatation and thinning progress in the infarct region. At some point, because of continued healing of the infarcted segments with increasing collagenization and the production of a firm scar, the ability of the infarction zone to resist wall stresses is increased and infarct expansion stops. However, one can imagine that before production of a mature scar, if wall stresses are elevated to a sufficient degree and if infarcted segment tensile strength has been sufficiently reduced, myocardial rupture may occur. Finally, in non-infarcted segments, elevation of end-diastolic wall stress may provide the stimulus for volume-overload hypertrophy, (14, 15) in which the combination of fiber elongation and wall thickening result in a return of systolic and diastolic wall stress toward normal. (11)

Hemodynamic consequences of left ventricular remodeling. The hemodynamic changes that occur in the 2 week period after myocardial infarction appear to be in part beneficial, with

improvement in cardiac output despite lower left ventricular filling pressure. This may explain the common clinical observation that occurs early in the course of an infarction, there may be mild clinical congestive heart failure followed by spontaneous improvement. (11) It is notable, however, that these hemodynamic improvements occurred only while chamber volumes increased significantly. Although a portion of the increase in diastolic chamber volume may have been related to the concomitant small decreases in heart rate observed, similar decreases in heart rate occurred both in patients with a greater than 20% increase in end-diastolic volume and in patients with a lesser increase in end-diastolic volume. Thus the degree of volume change was not proportional to fall in heart rate. Although hemodynamic improvement may accompany ventricular remodeling early after infarction, the long-term hemodynamic consequences of remodeling are not known. Data from at least one study have suggested increased morbidity and mortality in post infarction

patients with increased ventricular size.(16) In addition, a recent study has suggested that ventricular dilatation after myocardial infarction may be progressive, continuing for months after the original infarction.(17) Perhaps a more important clinical consequence of infarct remodeling is that it may lead to late decreases in left ventricular performance with depression of both global and regional contractile function. This may be particularly important in the late and often "mysterious" appearance of congestive heart failure seen in patients with infarction, even in the absence of late ischemic events. The etiology of this late ventricular dysfunction could be related to both infarct expansion of infarcted segments and volume-overload hypertrophy of noninfarcted segments. With respect to infarcted segments, the mechanical consequences of infarct expansion place an unusually high burden on the residual functioning myocardium. (18, 19) Moreover, the expanded infarct segment may act as a reservoir and receive blood during systole in competition with aortic outflow, a

condition similar to mitral regurgitation. Perhaps more important than the hemodynamic consequences of infarct expansion are the possible late consequences of volume-overload hypertrophy. In most states of volume-overload hypertrophy (e.g., mitral regurgitation, aortic insufficiency) there is an early phase of adaptive hypertrophy in which contractile function of the hypertrophied myocardium remains normal. However, at some point in the hypertrophy process, there follows a transition when contractile function becomes abnormal.(14, 20) If ventricular remodeling involves volume-overload hypertrophy of noninfarcted segments, it is possible that these segments may show a similar pattern of initial "physiologic" hypertrophy followed by "pathologic" hypertrophy. Since these segments must compensate for the deleterious mechanical consequences of infarct expansion, a loss of function in these segments would be a major factor in the late appearance of clinical congestive heart failure. (11)

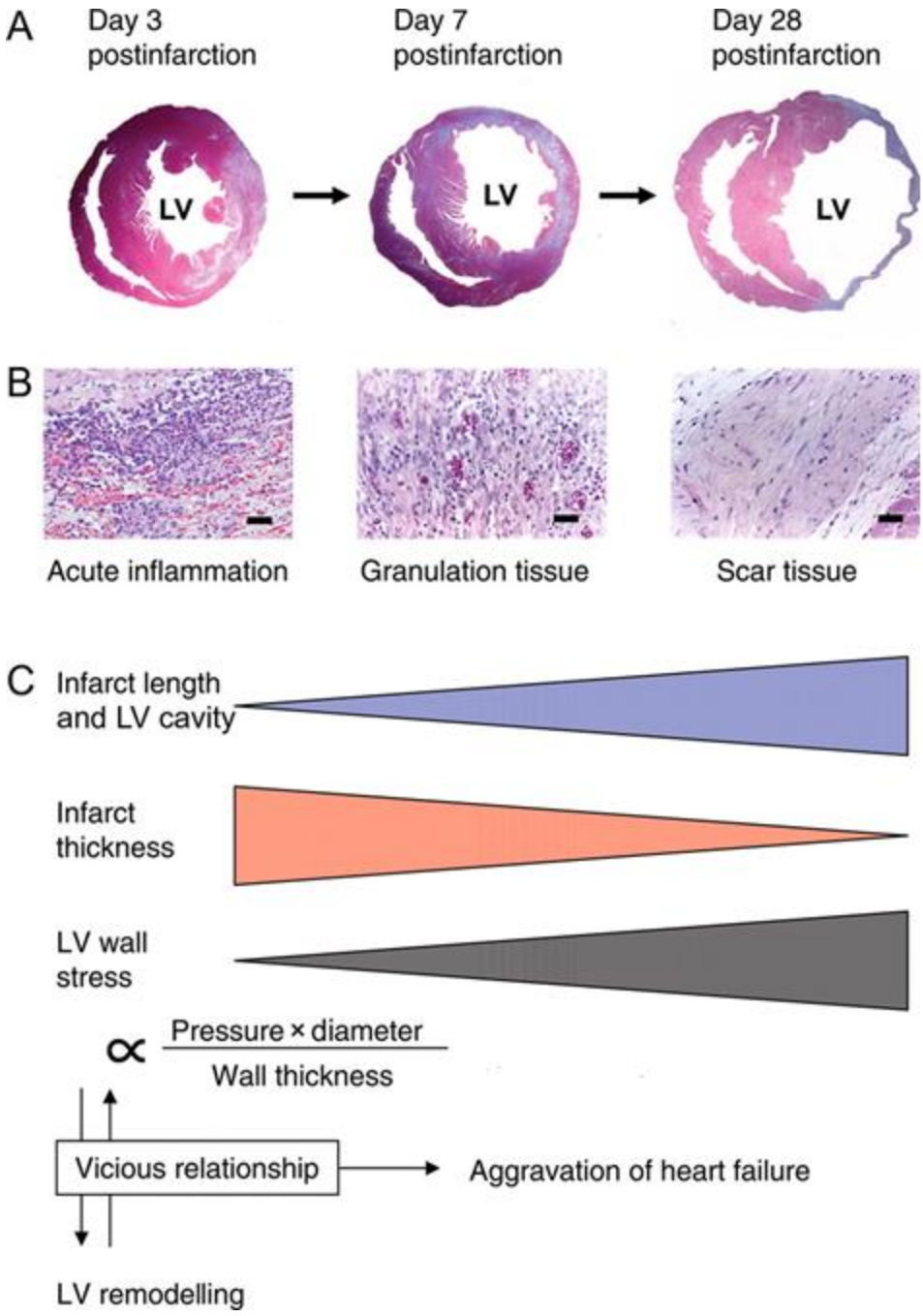

Figure 1: Effect of the postinfarction healing process on cardiac geometry and its relation to wall stress and heart failure. (21)

INDICATION AND EVOLUTION OF THE PROCEDURE

Indication of the procedure: Medical therapy is the mainstay of treatment for congestive heart failure (CHF) of all aetiologies. Myocardial infarction is the leading cause of CHF in Western society. There are, however, many patients for whom medical therapy is not effective and for whom surgical therapy is appropriate. (22) All cardiac surgeons perform surgery for CHF. While many procedures are performed to treat CHF, coronary artery bypass grafting (CABG) and valve replacement/repair are the main procedures performed by most surgeons. Surgeons who specialize in heart failure can also add heart transplantation, mechanical device implantation, and ventricular restorative procedures to their treatment options for the surgical treatment of CHF. Surgical ventricular restoration (SVR) is well within the scope of most cardiac surgeons and can expand the treatment options they offer for patients with post myocardial infarction heart failure. (23)

The goal of SVR is to reverse the adverse anatomic changes that have occurred due to remodelling following myocardial infarction in patients who go on to develop an ischemic cardiomyopathy. Specifically, the operation seeks to restore the left ventricle to a more normal spherical shape and size by reducing the volume in the anterior and septal portions by excluding akinetic and dyskinetic segments. Additionally, complete revascularization and elimination of any mitral regurgitation is performed. Classically, patients are candidates for SVR if they have had an anterior myocardial infarction, have a large area of akinesis or dyskinesia, and have clinical evidence of CHF. Specific characteristics of patients who have successfully undergone SVR are shown in (Table1). Ideal candidates for revascularization have retained function of the basilar and lateral portions of the heart and have good right ventricular function. Relative contraindications include pulmonary hypertension, multiple areas of infarction, loss of

basilar heart function, and unreconstructable coronary artery disease. (24)

Table1. Indications and Contraindications for Surgical Ventricular Restoration (24)
INDICATIONS
• New York Heart Association class III or IV. • Ejection fraction depressed. • Asynergy of >50% of the left ventricle. • End diastolic volume index >150 cc/m2. • End systolic volume index >60 cc/m2. • Candidate for revascularization. • Retained basilar heart function. • Good right ventricular function
CONTRAINDICATIONS
• Multiple areas of infarction. • Loss of basilar myocardial function. • Pulmonary hypertension and right ventricular dysfunction.