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The Role of Percutaneous Ventricular Restoration Therapy in Heart Failure After Myocardial Infarction

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ABSTRACT

The process of ventricular remodeling has a major role in the pathogenesis of heart failure in patients with myocardial infarction. Angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), antialdosterone diuretics, and beta receptor blockers are drugs that have been widely accepted as anti-remodeling agents. However, sometimes the efficacy of these drugs is inadequate, or their use is constrained by low blood pressure. Percutaneous ventricular restoration therapy is an action that aims to exclude segments of the myocardium that have akinetic or aneurysms by implanting a ventricular partitioning device percutaneously. This technique is expected to prevent the progression of ventricular remodeling through a procedure with a lower risk of intraprocedural mortality than the surgical approach.

1. Introduction

Thanks to advances in invasive therapeutic strategies and current pharmacotherapy, a significant reduction in acute coronary syndrome mortality have been achieved. However, this decrease in mortality was also followed by an increase in the incidence of heart failure in patients who survived the attack of this acute coronary syndrome. It is estimated that 24% of patients with acute myocardial infarction will develop heart failure. In addition, despite recent advances in pharmacological and invasive therapies, a high mortality rate is found in patients with heart failure due to myocardial infarction, which is about 32% at one year and around 50% at five years.[1,2,3]

The process of ventricular remodeling has a major role in the pathogenesis of heart failure in patients with myocardial infarction. Angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), antialdosterone diuretics, and beta receptor blockers are drugs that have been widely accepted as anti-remodeling agents. However, sometimes the efficacy of these drugs is inadequate, or their use is constrained by low blood pressure.[1,4]

Myocardial segment aneurysm is a factor that causes the remodeling process to continue intensively even with combination therapy with anti-remodeling agents. Surgical aneurysmectomy has been the solution to this problem for a long time. This procedure was successful in increasing the survivability ratio of patients with post-infarct heart failure to 60%, but also had a high intra-operative mortality rate (7.7-17.8%).[3] Percutaneous ventricular restoration therapy is an act that aims to exclude myocardial segments that experienced rigid or aneurysm using Implementing ventricular partitioning device percutaneously. This technique is expected to prevent the progression of ventricular remodeling through a procedure with a lower risk of intraprocedural mortality than the surgical approach. [4]

Left ventricular aneurysm after myocardial infarction

The greatest risk factor for the incidence of heart failure with a reduced ejection fraction at this time is a history of myocardial infarction. Regional abnormalities of ventricular wall movement are the basis for the development of heart failure after myocardial infarction. Acutely, this abnormal segmental movement leads to an increase in left ventricular volume and to some extent a decrease in ejection fraction. Chronically, the presence of an akinetic or dyskinetic segment (aneurysm segment) triggers progressive post-infarction remodeling.[1]

The left ventricular aneurysm is one of the complications of myocardial infarction. Clinically, a ventricular aneurysm is defined as a segment of the ventricle that is thinned and protrudes from the cavity during both systole and diastole. On echocardiography, ventricular wall aneurysms are recognized by visualizing segments of the myocardium with thin walls and wide openings, which have dyskinetic movements in the systole phase.[5,6]

The overall incidence of left ventricular aneurysms currently stands at 12%. Multivariate analysis studies show that left ventricular aneurysms are often found in cases, single-vessel disease, total occlusion of the left anterior descending coronary artery (LAD), and patients with anterior myocardial infarction. Furthermore, the area most commonly affected by aneurysms is the anteroapical wall of the left ventricle. Where the incidence of anteroapical wall aneurysms is four times higher than that of inferior or posterior wall aneurysms of the myocardium. Nicolosi had a left ventricular aneurysm and had a poor outcome at 6 months, both in terms of morbidity and mortality. This is because 33% of patients with left ventricular aneurysms will experience progressive remodeling. [4,6,7]

Left ventricular remodeling after myocardial infarction and heart failure

Ventricular remodeling plays an important role in decreasing ventricular systolic function after infarction. This is because progressive remodeling will lead to significant dilatation of the left ventricular cavity. In addition, remodeling is also associated with an increased prevalence of cardiac rupture and arrhythmias. Overall, the process of ventricular remodeling is closely related to poor prognosis after myocardial infarction. [1,8]

Hockman et al. first used the term remodeling to describe the replacement of necrotic tissue in the infarcted area with healing tissue. Pfeffer et al., in their study of acute post-infarction myocardial remodeling, suggested a post-infarction morphological change in which an increase in the size of the ventricular cavity was found. These morphological changes will lead to ventricular dysfunction. In 2000, the international forum for cardiac remodeling agreed on the definition of remodeling as a change in the molecular, cellular, and interstitial parts of the heart that have clinical manifestations through changes in size, muscle mass, and geometry, and function of the heart, as a result of a response to a condition aggression.[8,9,10]

Post-infarction remodeling is divided into two phases. The initial phase occurs in the first 72 hours post-infarction. The late phase occurs after the first 72 hours post-infarction. In the early phase there is an expansion of the infarct zone, while the later phase is characterized by dilatation and distortion of ventricular morphology, as well as transmural hypertrophy. [11] The initial phase of remodeling is characterized by the expansion of the infarct zone. This process occurs due to the degradation of inter-myocyte collagen. It is

thought that the enzymes responsible for this process are serine proteases and matrix metalloproteinases (MMPs) which are released by neutrophils in the first hours of myocardial injury. This will cause thinning of the walls of the myocardium and dilatation of the ventricles which in turn will cause an increase in systolic and diastolic pressures of the ventricles. [11]

In the advanced phase, there are several neurohormonal changes in the circulation. Increased pressure on the ventricular wall (wall stress) will result in the local release of angiotensin II from the myocardium wall which will then work in a paracrine manner. Decreased cardiac output will also result in activation of the sympathetic nervous system and the Renin-Angiotensin-Aldosterone hormone system. These neurohormonal changes then induce cardiac muscle hypertrophy and serial sarcomere replication. The hypertrophied heart muscle has a morphology that is longer than normal heart muscle. Hypertrophy with such a character is classified as eccentric hypertrophy. This remodeling contributes to changes in ventricular dilatation and morphological distortion that occur over time. [1,11]

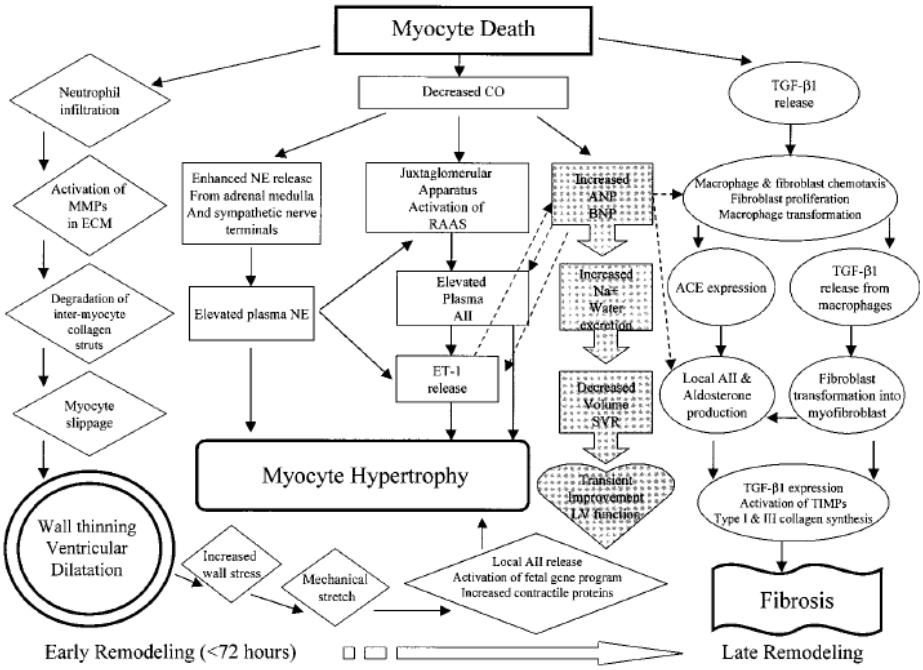

Figure 1. Pathophysiology of ventricular remodeling [11]

Asymmetric ventricular contraction in myocardial infarction also plays a role in the progression of ventricular remodeling. The force generated by the normal myocardium is not matched by the balanced and simultaneous force of the infarcted myocardium. This causes an increase in pressure on the walls of the infarcted ventricle (wall stress). In addition, dyssynchrony contractions of the ventricles also occur, which causes a decrease in stroke volume. [1]

Increased ventricular wall pressure (wall stress) itself will increase collagen synthesis by cardiac fibroblasts. On the one hand, expansion of the extracellular matrix reduces the pressure on cardiac muscle cells. But on the other hand, it also reduces the contraction function of the ventricles. In addition, the increase in the extracellular matrix also disrupts oxygen diffusion to cardiac muscle cells due to the expansion of the extravascular space. Overall, the extent of myocardial fibrosis was strongly correlated with increased mortality. [1]

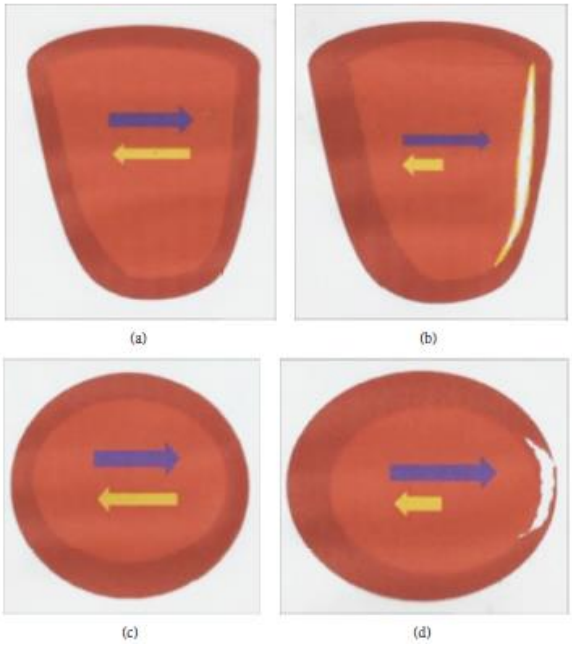

Figure 2: Figures a and c, balanced normal myocardial contractions in each segment. Figures b and d, post-infarction myocardial contraction. The area affected by the infarct stretches because it cannot compensate for the pressure caused by the contraction of the healthy myocardium.

Management of ventricular remodeling after myocardial infarction

As the understanding of the pathophysiology of ventricular remodeling increases, therapeutic modalities to prevent or inhibit this process are increasing. Pharmacological therapies for anti-remodeling that are commonly used today are ACE inhibitors, angiotensin receptor blockers (ARBs), -blockers, and antialdosterone diuretics. While interventional therapy is an option in conditions where there are obstacles in the use of pharmacological agents, or the efficacy of these pharmacological agents has not been able to achieve the expected results. [1,11]

ACE inhibitors and ARBs are pharmacological agents that have the best anti-remodeling efficacy. Interventions in the response of the renin-angiotensin-aldosterone system inhibit continued myocyte hypertrophy and ventricular fibrosis. The combination of one of these agents with an anti aldosterone diuretic has shown better results than the use of one of these agents alone. [1,11]

The combination of an ACEI or an ARB with β – *blocker* has also shown promising results in cases of progressive remodeling despite receiving standard anti remodeling therapy with ACE/ARB. Presumably, β -*blockers* improve autonomic control of the heart by increasing the number and modulating receptors β in cardiac muscle. In addition, β -*blockers* also reduce the incidence of *sudden cardiac death* and arrhythmias. [1,11]

Coronary artery reperfusion, either by thrombolytic, percutaneous coronary intervention, or surgery *by bypass* coronary artery, plays an important role in preventing the remodeling process. This is achieved by reducing the infarct area and restoring the hibernating myocardium. Bautovich and Hackworthy in their study stated that coronary artery reperfusion is the most important factor in changes in ventricular volume within 48 hours to 1 month after infarction. [11,12]

Optimal reperfusion and anti-remodeling medical therapy are the standards for treating ventricular remodeling after myocardial infarction. However, in cases of progressive remodeling, where advanced ventricular dilatation has been found, the efficacy of this modality has not been able to achieve the expected results. In addition, often the use of anti-remodeling drugs with optimal doses is constrained by the patient's low blood pressure. An alternative anti remodeling treatment for this condition that currently exists surgical Anatomical Ventricular Restoration (SAVR). [12,13]

The principle of surgical ventricular restoration management is the extirpation of the aneurysm or scar segment of the myocardium to reduce the volume and pressure on the ventricular wall. In addition, this action also aims to reconstruct the shape of the ventricles that have tended to be spherical to become elliptical again.[12.14] An early form of SAVR was known as Operation Batista. In this procedure, in addition to removing scar tissue and aneurysms, healthy myocardium is also extirpated after the anterior papillary muscle to the posterior papillary muscle. In the STICH (Study Surgical Treatment for Ischemic Heart Failure), there was no improvement in mortality and rehospitalization for 2 years in the SAVR group.[13,14]

The current SAVR technique is known as the D or technique. In this technique, endoventricular patch saturation is performed on the aneurysm segment through excision of the myocardium. The RESTORE (Reconstructive Endoventricular Surgery Returning Torsion Original Radius Elliptical Shape) study showed improvements in echocardiographic parameters, as well as an improvement in the 5-year survival rate to 68.6%. However, the application of this procedure is limited, because it is major cardiac surgery, requires *a* cardiopulmonary bypass, and a cardiac surgery team that is skilled in this technique.[12,13,14]

Percutaneous ventricular restoration

The basic concept of percutaneous ventricular restoration therapy stems from the idea that, with a percutaneously administered ventricular partitioning device, it is possible to reduce the volume of the left ventricular cavity and restore the geometry of the left ventricular cavity to a more elliptical shape, without having to face the risks of invasive surgery.[4]

Cardiokinetix Corporation, in 2005 began to develop a "" device parachute. The parachute is a transcatheter device that functions to partition the apex aneurysm from the entire left ventricular space. The parachute is currently under advanced research in Europe, the United States and Asia.[14]

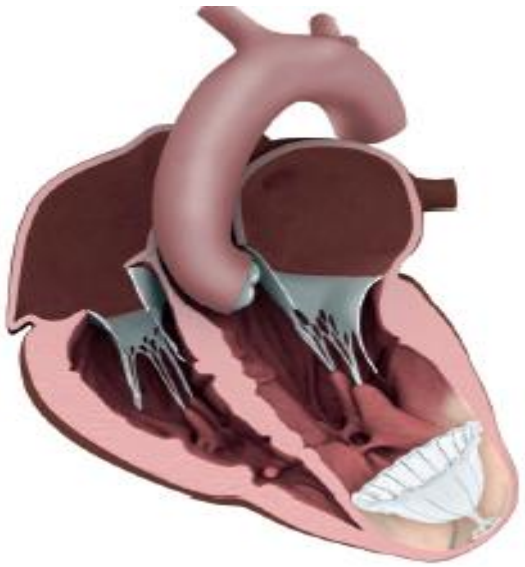

Figure 3: Segment ventricular aneurysms were excluded by parachute device

The main objectives of percutaneous ventricular restoration therapy is a decrease in volume *end-diastolic and* systolic of the ventricles, as well as the pressure drop across the ventricular wall. Some of the effects that are expected with the installation of this device are 1. Substitution of the rough surface of the scar at the apex with a more structure compliant, and improving the ventricular filling process with the "trampoline effect" during diastole. 2. Increase the compliance of the left ventricle, and reduce the end-diastolic pressure of the left ventricular. 3. Changes in the geometry of the left ventricular cavity, thereby reducing the wall stress of the myocardium wall that is not affected by infarction. The expected long-term effect of these various capture points is the reversion of the post-infarction left ventricular remodeling process.[4.14]

Components of parachute equipment

Parachute equipment can simply be grouped into 3 components: 1. Parachute, is a nitinol frame covered by an impermeable membrane of polytetrafluoroethylene (ePTFE). This nitinol frame is shaped like a parachute and has the property of "*self-expanding*". At the top of the parachute are atraumatic polymer feet. 2. Guide Catheter, 3. Delivery system. This Parachute tool itself is available in 8 sizes, namely 65, 65S, 75, 75S, 85, 85S, 95, and 95S.[14]

Figure 4. Delivery system, guide catheter, and parachute[14]

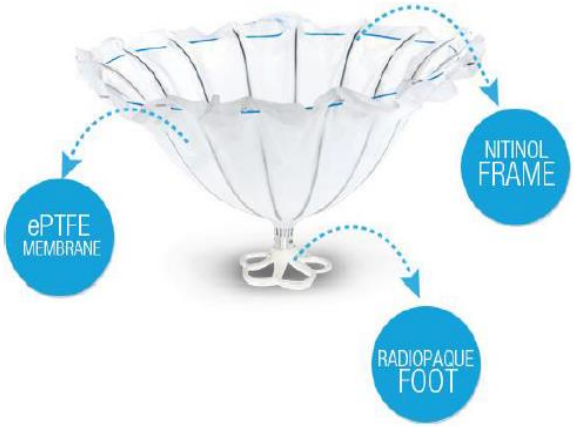

Figure 5. Parachute consisting of a nitinol frame, impermeable membrane, and polymer foot[14]

Indications for action

The use of this device is still in an advanced research phase. The PARACHUTE I, II, and III studies used inclusion criteria; post-infarct heart failure patients, with anthropical segment kinetics or dyskinesia, have an ejection fraction of 15-40% despite receiving optimal medical therapy for at least 3 months.[2,14,15]

Contraindications to action

From the studies that have taken place, several things that cause failure or complications in this procedure include:[2,14,15] severe heart valve disease, apex aneurysm filled with thrombus, calcified apical aneurysm weight, and dilated cardiomyopathy.

Procedure action

Parachute implantation at the apex of the left ventricle was performed percutaneously through the femoral artery access. Anesthesia is performed locally, in addition to light sedation. The entire parachute device is conveyed to the left ventricle via the femoral artery by sheath 14/16 F. After entering the left ventricle, the parachute is continuously pushed until the polymer foot touches the ventricular apex. This procedure was performed under transthoracic echocardiography and left ventriculography. Once the polymer foot touches the apex, the balloon on the delivery system is inflated to aid in the expansion of the parachute. After the parachute is successfully implanted, the balloon is deflated and withdrawn with the delivery catheter.[14.15]

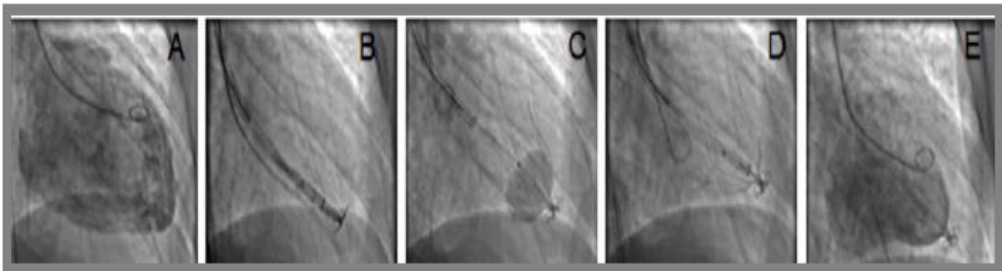

Figure 6. The stages of the procedure. a. Left ventriculography before implantation. b. The parachute is pushed to the apex until the polymer foot touches the apex wall. c. The parachute is inflated with the help of a balloon. d. Guide catheter was pulled out. e. Post-implantation ventriculography[4]

Combination antiplatelet aggregation therapy and anticoagulation should be given after the procedure. Costa and colleagues, in the PARACHUTE I study, used a combination of aspirin for 1 year, clopidogrel for 6 months, and warfarin for 3 months. Thomas and colleagues, in the PARACHUTE III study, used a combination of low-dose aspirin and warfarin for 1 year.[2.15]

From the studies that have been conducted, several complications can arise either Intra procedurally or post-procedurally. Intra-procedural complications include; aortic valve injury, hemorrhage, mitral valve injury and embolization. While the observed complications that may arise post-processing include; device detachment, device infection, ventricular tachycardia, and thromboembolic stroke.[14]

The role of imaging modalities in successful Parachute implantation

Imaging modality support is crucial in the selection of candidates for Parachute implantation procedures. Besides being useful in determining which patients are indicated for this procedure or not, imaging modalities also play an important role in estimating the success rate of the procedure, and in selecting the size of the device.[16]

Transthoracic echocardiography is a commonly used imaging modality before this procedure. There are several findings in imaging studies that indicate a good percentage of the success of the procedure. These findings include:[16] (1) The left ventricular end-diastolic chamber diameter is 55-70 mm. The smaller diameter increases the chances of the parachute device not fully inflating. While the larger diameter increases the likelihood of this device detaching from its fixation. (2) Ventricular wall thickness >3.5 mm. Parachute implantation in patients with a ventricular wall < 3.5 mm carries a high risk of perforation. (3) Left ventricular apex end-diastolic diameter 4 cm from apex apex > 5cm. This area is where the parachute frame is attached. The size of the diameter in this area which is < 5cm will inhibit the development of the parachute. (4) Normal ventricular wall trabeculation. Hypertrabeculation will inhibit the attachment of the parachute frame to the ventricular wall.

The problem with using echocardiography as the sole imaging modality to support this procedure is that it is operator-dependent. CT-scan technology and Magnetic Resonance Imaging (MRI) provide a more objective imaging alternative. However, the use of MRI is often constrained due to the large percentage of candidates who have undergone pacemaker installation. Dynamic cardiac multidetector computed tomography (MDCT) is the imaging modality of choice to support the success of this procedure.[16]

The pilot study conducted by Sievert and Sagic, which used only preprocedural transthoracic echocardiography imaging, showed implantation success rates of 83% and 91%, respectively. Costa in the PARACHUTE I study, in which the initial 15 cases used only preprocedural echocardiographic imaging, reported an explantation rate of 3 out of 39 subjects who underwent this procedure. PARACHUTE III, which uses multi-slice CT in addition to echocardiography, has a 97% success rate for the procedure. Ince used a *dynamic cardiac* multidetector CT scan accompanied by more stringent inclusion criteria, getting an implantation success rate of 100%.[2,15,16]

Clinical studies of percutaneous ventricular restoration therapy

Parachute

Parachute is the first clinical study of percutaneous ventricular restoration in humans. This study was designed to assess the safety and feasibility of a percutaneous ventricular restoration procedure with a parachute device in patients with heart failure due to myocardial infarction.[15]

The primary endpoint in this study was to assess the safety of this procedure. The presence or absence of major adverse cardiac events (MACE) within 6 months is the starting point for assessment. The incident was classified as MACE, among others:[15] of sudden cardiac arrest, emergency heart surgery, erosion devices, cardiac tamponade, peripheral embolism, new heart failure or worsening heart failure, endocarditis and migration of the device.

Efficacy was the secondary endpoint in this study. Efficacy assessment was carried out by transthoracic echocardiography examination and the calculation of the Minnesota Living with Heart Failure (MLWHF) score. Efficacy assessment was carried out at 6, 12, 24 and 36 months.[15]

Table 1. Minnesota living with heart failure[15]

In the past month, have your heart problems prevented you from living the life you wanted? Why?		
No	Symptoms	Score (0-5)
1	Causes swelling of the legs and feet	
2	Causes having to sit or lie down for long periods	
3	Makes walking or climbing stairs difficult	
4	Makes doing daily household chores difficult	
5	Makes going outside difficult	
6	Makes it difficult to sleep well at night	
7	Causes disruption of activities with family and friends	
8	Causes difficulty finding a livelihood	
9	Causes difficulty in recreation	
10	Causes difficulty with sexual activity	
11	Causes decreased appetite	
12	Causes feelings of tiredness, fatigue, and loss of energy	
13	Causes hospitalization	
14	Causes large expenditures for health care	
15	Causes shortness of breath	
16	Causes many symptoms side effects of medication	
17	Causes feelings of being a burden to family and friends	
18	Causes feelings of loss of control over life	
19	Causes feelings of worry	
20	Causes difficulty concentrating and craving at	
21	Causes feelings of depression	

The study was conducted from October 2005 to June 2009 in the United States and Europe. 39 NYHA II-IV heart failure patients with an akinetic or dyskinetic antero-apical segment of myocardium agreed to participate in this study. 31 subjects completed the study (observation time for 3 years). [15] Results from the primary endpoint study showed that this procedure had a good safety rating, with a MACE-free rate of 85.3% for 6 months. Where the incident occurred was urgent cardiac surgery for evacuation of the consequent device; detachment of the parachute's polymer foot from the cardiac apex, failure to inflate the nitinol frame, and the presence of a lymphatic abscess increase the risk of endocarditis. [15] Results from the secondary endpoint showed symptomatic improvement in 52% of the subjects. Transthoracic echocardiography also revealed an improvement in left ventricular volume that persisted at 3 years of follow-up.[15]

Parachute III

Parachute III is an observational study to assess the long-term safety and efficacy of percutaneous ventricular restoration procedures with parachute devices. This study was conducted in Europe with a study population of patients with heart failure after anterior myocardial infarction. [2] As with the first PARACHUTE study, the safety of the procedure was the primary endpoint of this study. However, the PARACHUTE III study uses Major Adverse Cardiac Cerebral Events (MACCE) as a safety measure. The secondary endpoint of this study was efficacy. The assessment was carried out with a 6-minute walk test procedure and evaluation of echocardiographic parameters.[2]

In this study, 100 subjects were found to be willing to participate. Implantation was successful in 97 subjects. 2 subjects experienced complications of intraprocedural left ventricular perforation, 1 subject did not achieve a good implantation position.[2] In the follow-up 1-year, 93 subjects had no MACCE incidence. However, 11 subjects had bleeding complications, of which 4 of them required transfusion of more than 4 units of blood. The MACCE incidents that occurred included 3 aortic valve injuries requiring surgical repair, and 1 case of unstable bradycardia. All MACCE incidents occurred no later than 10 days post-processing. [2] There is an increase in the score of the 6-minute walking test, from an average of 372 meters to 397 meters within 1 year. An improvement in echocardiographic parameters was also found, namely the end-diastolic volume index from 117.3 $\pm$ 26 to 99.1 $\pm$27.3 with $p > 0.0001$.[2] This PARACHUTE III study is still ongoing for 36 months of observation.[2]

Acute Effects of Parachute Implantation on Hemodynamics

Tobias Schmidt et al. investigated the effect of acute parachute implantation on patient hemodynamics. From July 2012 to November 2013 16 subjects participated. Invasive hemodynamic measurements were performed with a Swans Ganz catheter for the right heart and a pigtail catheter for the left heart. Measurements were taken before and after the implantation procedure.[3] These measurements revealed a significant increase in stroke volume (25.4% / 12.3 $\pm$ 2.7 ml) post-implantation. There was also an increase in the stroke volume index (26.5% / 6.9 $\pm$ 1.5 ml/m^2/beat) and cardiac output (25.8% / 0.8$\pm$0.1 l/min). In addition, there was also a slight decrease in systemic vascular resistance (11.2%). There were no significant changes in other invasive hemodynamic parameters.[3]

Comparison of the results of studies of surgical ventricular restoration therapy and percutaneous ventricular restoration therapy

The studies on ventricular restoration therapy that have been carried out to date have demonstrated the safety of the procedure of percutaneous ventricular restoration therapy over surgical ventricular restoration therapy (SAVR). There was no intra-procedural or 30-day post-procedural death on percutaneous ventricular restoration therapy. Meanwhile, Castelvecchio in the RESTORE study stated that the 30-day post-SAVR mortality rate was 5.3%. Tadashi et al. reported an intraprocedural SAVR mortality rate of 4.3% in elective surgery and 18% in emergency surgery.[2,15,17,18]

From the available studies, it appears that both surgical and percutaneous ventricular restoration therapy give a positive outcome. Although the STICH study failed to demonstrate the efficacy of adding the SAVR procedure compared to CABG alone, the RESTORE study on SAVR showed increased 5-year survivability of up to 68.6% in patients undergoing this SAVR procedure. The RESTORE study also showed improvements in post-procedure echocardiographic parameters, namely an increase in ejection fraction and a decrease in end-systolic volume. However, no change in end-diastolic volume was reported in this study.[17,19]

Studies show survivability PARACHUTE first 3 years of percutaneous ventricular restoration procedure by 86%. Meanwhile, the PARACHUTE III study showed 1-year post-procedure survivability of 90%. Although these two studies did not show a significant increase in ejection fraction post parachute implantation. A significant decrease in end-diastolic volume was found at the end of the second report of this study.[2,15]

2. Conclusion

Surgical ventricular restoration therapy (SAVR) is a procedure developed to inhibit the progressive remodeling of post-infarction ventricular aneurysms. The RESTORE study using the latest SAVR technique showed improvement in 5-year mortality, but still with significant 30-day mortality. Percutaneous ventricular restoration therapy aims to reduce left ventricular volume and exclude aneurysmal or akinetic segments at the apex, similar to SAVR but with a less invasive approach. This procedure is performed with a parachute device consisting of the parachute itself, a *guide catheter*, and a delivery system. Parachute implantation is performed percutaneously via the femoral artery access. Existing studies of this procedure demonstrate greater intraprocedural safety than SAVR. There was also a significant improvement in clinical and echocardiographic parameters. The decrease in left ventricular volume persisted for 1 year and 3 years, indicating the efficacy of this procedure in inhibiting the remodeling process.

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