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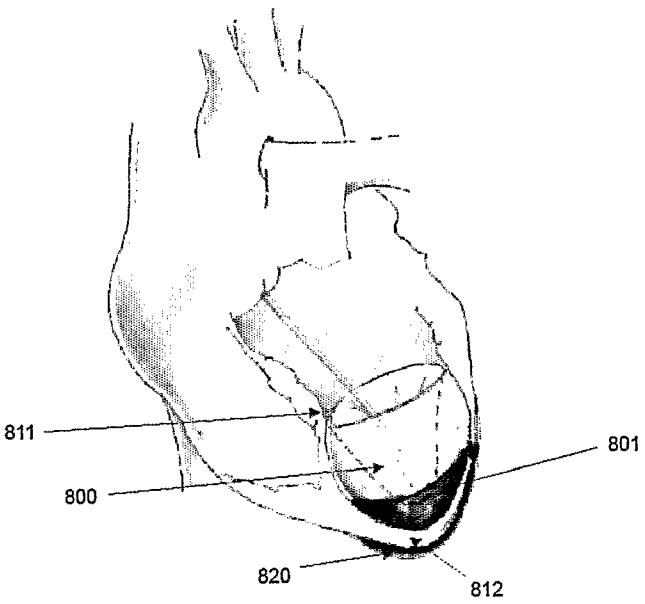

(57) Abstract: Devices and methods are disclosed for repairing a heart chamber, particularly useful for those who have suffered damage from myocardial infarction. A balloon is disclosed aimed at changing the shape of the volume of blood flow through a chamber of the heart from a spherical shape to a natural elliptical shape. This balloon has a magnetic outer wall which communicates with magnets on the outside of the chamber to form supports.

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DEVICES AND METHODS FOR PERCUTANEOUS ENDO-VENTRICULAR RECONSTRUCTION OF THE FAILING HEART

This application claims priority to U.S. Provisional Patent Application Serial No. 60/703,421, filed July 29, 2005, the content of which is hereby incorporated by reference in its entirety into this disclosure.

BACKGROUND OF THE INVENTION

Field of the Invention

The present invention relates generally to heart repair. More particularly, the invention relates to magnetic supports and inflatable implants used in the repair of walls of the heart *in vivo*.

Background of the Invention

About five million people in the United States suffer from congestive heart failure (CHF). This costs around $40 billion each year, which makes up more than 5% of the total U.S. health care dollars. Approximately 70% of the CHF cases suffer from ischemic cardiomyopathy as a complication of myocardial infarction (MI). MI alters cardiac left ventricular regional contraction and thus compromises the cardiac structure/function relationship resulting in loss of the free wall and septal contractility. Modern therapeutic advances in the treatment of acute MI have decreased early mortality. However, survivors go through the process of left ventricular remodeling following in the late development of CHF. Around 20% of the survivors of MI develop symptoms of heart failure within 5 years. In spite of multiple drug therapy, survival with advanced heart failure is approximately only 50% at 1–2 years after diagnosis.

Almost 70% of all infarctions are transmural, while 54% occur in the anterior wall. About 35% to 42% of anterior infarctions produce infarct expansion and wall thinning. The prognosis in patients with expanding infarctions and more severe left ventricular (LV) dysfunction is alarming due to the continuing LV dilatation and the deterioration of the LV function which continues for weeks, months, and even years. Left ventricular remodeling after single or multiple myocardial infarctions (MIs) is a time-dependent process that involves major alterations in cellular and extracellular cardiac structure affecting heart geometry and function.

Remodeled myocardium has been established as dysfunctional, adjacent to an infarct that is normally perfused but hypocontractile; both clinically and experimentally. The postinfarction remodeling process makes this hypocontractility region to extend to normally perfused myocardium, ending in ventricular dilatation and CHF. This is known as border zone myocardium (BZM). Myocardial wall stress is augmented in BZM immediately after MI. This increase is mainly due to reduced endocardial curvature, with a more limited contribution from myocardial wall thinning. In BZM early acute geometric changes produce increased stress and consequently a loss of contractility.

Current data show that expansion, or stretching, of a transmural myocardial infarction initiates a progressive myopathic process in normally perfused myocardium. This phenomenon is initially localized to myocardium immediately adjacent to the infarct. It expands during the remodeling process, however, transforming contiguous, normally perfused, myocardium into hypocontractile, remodeled myocardium. The stretch-induced myopathic

process is the sum of myocyte apoptosis and disruption of the extracellular matrix secondary to activation of matrix metalloproteinases. The stretching of BZ fibers during isovolumic systole may be described as a combination of an intrinsic contractile abnormality of the BZ region and an increase in wall stress.

Recently it has been emphasized that the way to treat CHF is to restore the shape of the left ventricle (*i.e.*, reestablishing near-normal geometry). Furthermore, left ventricular end systolic volume has been found to be an important prognostic indicator for CHF and death after MI. Specifically, it has been established that the indexed end-systolic volume of the left ventricle (iESV) can be a prognostic factor. IESV of more than 45 mL/m2 often conduces to clinical heart failure, and a resting iESV of more than 60 mL/m2 is related to subsequent cardiac mortality.

As soon as the infarcted scar exceeds 20% of the LV surface area, cardiac function begins to decline whether the scar bulges or not. Dyskinetic and akinetic scar areas present the identical mechanical defect of asynchronous and nonuniform global contraction. This process begins at the border zone where fibrous or necrotic and rigid scar tissue joins more normal muscle resulting in an abnormal wall stress. The change from one shape to another may be the result of progressive ventricular remodeling with the loss of internal bending at the border zone producing severe regional dysfunction.

Cardiac transplantation is the definitive treatment for chronic cardiac failure, including ischemic and nonischemic cardiomyopathy. But it is not widely available due to the shortage of donors and adaptable limitations. Currently left ventricle (LV) volume reduction surgery or LV repair surgery

(LVR) for dilated cardiomyopathy or ischemic cardiomyopathy (ICM) has been considered as a surgical treatment for patients with dilated LV and chronic heart failure. For ICM, improved ejection fraction in the early phase after LVR has been reported. Many studies have been conducted for LV aneurysm repair with good results, particularly for discrete LV aneurysm. Nevertheless, indications, methods of LVR, and long-term results have not been clarified for ICM. In fact, there have been some reports of late redilation of the LV after LVR.

Large dyskinetic or akinetic areas involving the septum are the result of the LAD proximal occlusion compromising the septal branches leading to septal necrosis. The majority of postinfarction left ventricular scar compromises the nonresectable septum; this scar may result in an akinetic region or occasionally in a dyskinetic scar if there is no reperfusion.

Reduction in LV size (ventriculoplasty), by either surgical procedures such as LV aneurysm repair or partial ventriculectomy, has been proposed as a treatment for CHF. Changes in ventricular wall stress are considered to be stimuli for growth and remodeling. Consequently, surgical aneurysm repair is successful when a reduction in wall stress, a subsequent improvement in BZ contractility, and improvement in ventricular function are achieved.

Mathematical modeling predicts that the reduction in end-diastolic, and consequently early systolic, wall stress by reduction of contractile chamber volume produces a decrease of afterload and therefore improved regional systolic performance in the remote normal wall segments. These studies reinforce the clinical experience with surgical LV reconstructive procedures suggesting that ventricular volume reduction with geometrically appropriate 3-

dimensional reconstruction techniques may have an important part in improving outcomes in these difficult patient subsets. Data showed that mortality after aneurysmectomy with linear plasty was a consequence of a considerable reduction and deformation of LV cavity.

Others revealed that some changes of LV shape after the Dor procedure (ventriculoplasty) contribute to increase the LV sphericity, since its longitudinal axis is shortened, with its diameter remaining unchanged. It was observed that severe LV diastolic dysfunction after endoventriculoplasty when the LV length was incorrectly reduced and the patch was too small in high-risk patients.

Electroporation or electropermeabiliztion is an increase in electrical conductivity and permeability of the cell plasma membrane. This is caused by an externally applied electrical field. Electroporation is usually used in molecular biology as a method of introducing substance (*e.g.*, drug, probe, fragment of DNA, etc.) into a cell. The principle capitalizes on the fact that pores form when the voltage across a plasma membrane exceeds its dielectric strength. When the strength and duration of the applied electric field are properly chosen, the pores form and reseal after a short period of time (reversible disintegration of protein). This is made possible because of the weak nature of the phospholipid bilayer's hydrophobic/hydrophilic interactions and its ability to spontaneously reassemble after disturbance. Thus, a quick voltage shock may disrupt areas of the membrane temporarily, allowing polar molecules to pass, but then the membrane may reseal quickly and leave the cell intact. This allows otherwise impenetrable barriers to allow extracellular

compounds to enter into the cell. This process is highly effective for efficient introduction of gene therapy and cell-based therapy.

SUMMARY OF THE INVENTION

The present invention addresses the shortcomings of repair and reconstruction of chambers of the heart by providing an implant to correct the geometry of a damaged chamber. The implant, with a substantially spherical exterior portion and a generally upside-down cone shape carved from the top, is designed to fill in the extra space in a damaged chamber of the heart, lowering the overbearing volume of blood-flow and relieving the tissue walls of unnecessary stress and strain. The implant is designed to fit substantially snug in the proper area of the chamber of the heart so it does not fall out of place or move during normal heart function.

In one particular exemplary embodiment of the present invention, a small, lightweight piece of magnetic film is placed on the outside of the chamber which contains the implant. The implant, having a magnetic outer surface itself, secures itself to the anchor on the outside of the chamber. This connection not only results in no damage to the tissue wall but provides support for it by transferring the stress onto the anchor.

In another particular exemplary embodiment of the present invention, the magnetic connection holding the implant in place is strengthened by a small anti-arrhythmic battery. The current across the tissue wall creates an effect known as electroporation, which simply opens pores in the tissue wall, making it receptive to treatment. Treatment can then be administered to actually repair the tissue wall of the heart chamber.

Furthermore, the present invention can be implanted without resorting to open-heart surgery. The implant can be deployed by maneuvering a slim catheter inside the chamber and then inflating it once inside. The anchor can be installed using a variety of methods, including, but not limited to: (a) percutaneous intravascular transatrial-pericardial sac approach delivery method for the magnetic (*e.g.*, beads, glue, etc.); and (b) thorascopic manipulation tools or video-assisted thorascopic surgery. These methods result in minimal surgical damage and scarring. If the pericardial sac has adhesions between pericardial layers, the anchor may be installed through thoracoscopic manipulation tools or video-assisted thorascopic surgery to deliver the anchorage materials over the parietal pericardium surface.

BRIEF DESCRIPTION OF THE DRAWINGS

FIG. 1A shows mechanical reconstruction of the left ventricle to restore the shape from the dilated (basketball-shape) to a normal elliptical (football-shaped) geometry with the initially collapsed preformed balloon being delivered percutaneously and inflated to the desired shape (left panels) and the magnet from the pericardial space used to anchor the balloon at the level of the apex in accordance with an exemplary embodiment of the present invention.

FIG. 1B shows the external magnet in the pericardial sack at the base of the heart to anchor the balloon in place in accordance with an exemplary embodiment of the present invention.

FIG. 2 shows the deformation of the preformed balloon during the cardiac cycle (end-diastole and suction and end-systole and ejection) in accordance with an exemplary embodiment of the present invention.

FIG. 3A shows a delivery method of the magnetic (*e.g.*, beads, glue, etc.) anchor into the pericardial space.

FIG. 3B shows an interior view of a delivery method of the magnetic anchor in the pericardial space in accordance with an exemplary embodiment of the present invention.

FIG. 4A shows an alternative method for delivery of epicardial magnetic anchor to the pericardium using thorascopic manipulation tools in accordance with an exemplary embodiment of the present invention.

FIGS. 4B-C show an implantation of an epicardial magnetic anchor through video-assisted thorascopic surgery according to another alternate method for delivery in accordance with an exemplary embodiment of the present invention.

FIG. 5 shows deployment of a preformed balloon through the catheter in accordance with an exemplary embodiment of the present invention.

FIG. 6 shows a method of electroporation through a current source to establish an electrical or electromagnetic field whereby transport into the failing myocardium is facilitated resulting in *in vivo* electroporation in accordance with an exemplary embodiment of the present invention.

FIG. 7 shows a biological aspect of an exemplary embodiment of the present invention whereby viral or non-viral transfections and gene delivery can be provided to restore the function of the failing myocardium.

FIG. 8 shows a pre-formed balloon with elliptical left ventricle shape-apical anchorage according to an exemplary embodiment of the present invention.

FIG. 9 shows an anti-arrhythmic drug eluting cover according to an exemplary embodiment of the present invention.

DETAILED DESCRIPTION OF THE INVENTION

Percutaneous endoventricular reconstruction of the remolded left ventricle according to the present invention may be approached by, for example, two combined procedures: (a) Mechanical Reconstruction; and (b) Biologic Reconstruction.

In Mechanical Reconstruction, a novel device, a magnetically supported preformed balloon with an elliptically-shaped cavity, is introduced to change the left endoventricular shape, reducing left ventricular volume, to decrease left ventricular wall stress (and strain), to support the infarction Border Zone myocardium **111** and left ventricular septum, and to improve remote myocardial contractility (**Fig. 1**).

The preformed elliptically-shaped balloon **100** which is magnetically supported **101** can transform the left ventricular shape **110** from a spherical into an elliptical shape by filling the preformed balloon with polymers such as: polyurethane, silicon rubber, silastic, PTFE foam, reabsorbable polymers, etc., under TEE (transesophagic echo) or RX control as shown in **Fig. 2**. This approach allows the possibility of increasing or decreasing the volume of the balloon according to the hemodynamic parameters, such as Ejection Fraction, Pressure/Volume Loop, etc., of the patient during the procedure.

This procedure can be delivered with minimal trauma in high risk patients, such as patients with CHF Stage III and IV, chronic renal failure, COPD, elderly patients, etc. The mode of delivery can include: (a) percutaneous through balloon insertion and percutaneous right atrial pericardial sac **313** delivery of magnetic powder, glue, gel or microfilm, magnetic beads or reabsorbable magnetic polymers **330** for balloon magnetic anchorage **301** as shown in **Fig. 3A** and **Fig. 3B** (reference made to co-pending US application, entitled "Percutaneous Intravascular Transatrial-Percardial Sac," Ser. No. 60/817,421, filed June 30, 2006, and incorporated by reference herein in its entirety); or (b) percutaneous balloon insertion and minimal thoracoscopy (5 mm) to deliver to the pericardial sac magnetic powder, glue, gel or microfilm, magnetic beads or reabsorbable magnetic polymers for balloon magnetic anchorage **420** as shown in **Fig. 4A**, **Fig. 4B**, and **Fig. 4C**. Other approaches may also be possible and are within the purview of one having ordinary skill in the art.

The many advantages of this approach include, but are not limited to: (1) systolic and diastolic LV wall support (akinetic or diskinetic walls **112** and septal regions **115** (**Fig. 1A**)); (2) avoidance of open heart surgery and extracorporeal circulation (in high risk patients); (3) combination coronary revascularization by PTCA or mininvasive surgery; (4) support or exclusion of the diskinetic septum scar **115**; (5) delivery of anti-arrhythmic anticoagulant drugs from the external surface of the balloon in contact with the myocardial border zone (**Fig. 2**); and (6) utilization of an electromagnetic field **652** between the subendocardium/external balloon magnetic surface **601** and the epicardial/magnetic surface **620** in order to impose electroporation **652**.

The balloon may be preformed to have different shapes and volumes to accord with the akinetic or diskinetic areas to be filled, tailoring the balloon to the particular needs of a given patient. The balloon is initially totally collapsed to enable percutaneous delivery. Once in place, it can be filled as desired (**Fig. 5**). The balloon has a one-way valve at the tip that ensures containment of fluid after filling. The balloon may be filled with saline or soft gel such as silicone rubber or a polymer containing particles of ferromagnetic material. The balloon materials may be non-thrombogenic (polyurethane, silicon rubber, silastic, PTFE, reabsorbable polymers, etc.) to assist in compatibility with the body. The external balloon surface **520** may be covered or embedded with magnetic compounds (Polyurethane, silicon rubber, silastic, PTFE, reabsorbable polymers, etc., combined with magnetic materials). The right percutaneous ventricular magnetic cover stent patch may be specifically designed for diskinetic septum support.

The balloon should be anchored in the lumen of the heart. One exemplary embodiment is through an epicardium-pericardial sac magnetic support. This can be realized through a magnet powder, gel, glue, gel or microfilm, or magnetic beads to ensure the balloon magnetic anchorage **801** from the pericardial sac. The anchorage of the balloon **801** can be obtained by delivery of magnetic material to the pericardial sac through percutaneous endovascular atrial appendage approach or through minimal thoracoscopy. The anchorage can be realized at the apex as distributed, shown in **Fig. 8**.

As shown in **Fig. 1**, the combination of a metallic material **101** along with a corresponding magnet **120** may be used to correct the structure of a deformed or injured heart. As used in this example, heart failure patients

have misshaped hearts that may be in the form of a spheroid or other unnatural or harmful shape. To correct such a harmful shape, a peripheral balloon may be introduced into the heart cavity through a percutaneous catheter. This specialized metallic balloon **100** may have some metallic material or coating or other imbedded particles **101** that allow the balloon to interact with a magnetic sheet **120** that may be positioned outside of the cavity of the heart, for example, on the pericardial segment exterior **111** of the left ventricle **110**. The size, shape and geometry of the intraventricular metallic pouch **100** may be predetermined and made to the specific dimensions of a particular patient's heart after examination of MRI (or other common tool) images of the heart. The pouch is then deflated to fit within the lumen of a percutaneous catheter **560**. After the catheter **560** is led into the proper position within the ventricular cavity **110** using conventional techniques known to one of ordinary skill in the art, the preformed (polyurethane or silastic, etc.) balloon catheter **100** with external metallic portion metallic pouch **101** is released and positioned within the cavity. A magnetic sheath **120** is then used to properly position the pouch within the cavity **110**. A saline or other suitable solution is then injected within the pouch to give the pouch a proper form in order to reduce the spherical shape of the ventrical cavity **110** making it more elliptical. The catheter **560** is then removed. To assist the pouch in grabbing onto and anchoring its position within the intraventricular cavity **110**, barbs or other securing means may be used to gently grip the interior cavity tissue **111**.

The volume of filling of the pouch **500** within the ventricle is determined by the intraventricular volume for the patient and how much this volume is

above the intended volume for that patient. The pouch may be filled to decrease the available blood volume within the ventricle and allow more normal blood pumping function. Such a volume for the pouch may be subsequently increased or decreased in time as the patient's ventricular volume is assessed by a health care worker. Although the present example was made using the left ventricle as an example, the present technique may be applied in a similar fashion to any of the chambers of the heart, taking into account that such a pouch and its shape should not interfere with any of the heart valves or their ability to function.

Alternatively, the balloon/pouch may be a biocompatible polymer having no metallic substance or material imbedded therein. Instead, the solution that is used to fill up the pouch contains metallic particles such that the magnet is drawn to the solution within the pouch instead of the pouch itself per se. A combination of metallic pouch and metal-containing solution is also possible.

Using the present technique, the whole procedure of introducing an organ restructuring system may be made without harmful invasive technique of opening up the chest to operate on the beating heart. With practice, the procedure may be performed in outpatient services, allowing the patient rapid recovery and gradual and safe reshaping of the heart to its natural shape from its deformed spherical shape.

In another aspect of the present invention, biological reconstruction may also be performed. A number of biological therapeutics can be added at the interface of the preformed balloon **100** and the magnetic anchor **120** such as: Virus transfection Genes (Retrovirus and adenovirus, Gene transfer

VEGF, FGF, etc.); Non Virus transfection Genes (calcium phosphate, liposome, particle bombardment, electroporation, magnetic nanotubes or any combination of these methods; hybrid procedures/magnetic nanoparticles cells BMC, etc.); Cardiac Progenitor cells, etc. For the biological reconstruction aspect of the present invention, there are at least three different ways of delivery: (1) Coronary artery delivery **770**; (2) Coronary sinus venous delivery **771**; (3) Pericardial sac delivery (**Fig. 7**).

An electromagnetic field **652** may be imposed through a current source **650** between external balloon surface/endocardium **601** and epicardial/pericardial sac **620** (creating a sandwich effect). This can initiate electroporation **652** to enhance uptake of drugs and therapeutics (see **Fig. 6**). This initiates an important advance in biotechnology and medicine for the controlled introduction of macromolecules, such as gene constructs and drugs, seeding of myoblasts or myocytes into various cells of the remnant myocardial tissue isles on the akinetic or diskinetic areas **111**, **112**.

A number of advantages of this biological approach include, but are not limited to: (1) the versatility of electroporation **652** as an effective method of uptake with nearly all cell and species types; (2) high efficiency where a large majority of cells take in the target DNA or molecule; (3) smaller amount of DNA than is required for other methods; and (4) the procedure may be performed *in vivo* on intact tissue.

Anti-arrhythmic agents can be used as a coating over the balloon surface for slow release of drugs (**Fig. 9**). This includes at least one polymer and one therapeutic agent and is used as a drug-eluting device for the myocardium similar to those used in vessel stents. The anti-arrhythmic

coating may be total cover **905** or bands **906**, and allow slow release to the myocardium to diminish or abolish ventricular arrhythmias usually initiated in ischemic areas. The anti-arrhythmic drugs may include but not be limited to lidocaine, mexiletin, anti-arrhythmics of class I (*e.g.*, propafenone, flecainide); anti-arrhythmics of class II, including beta blockers receptor: metoprolol, esmolol, propranolol, etc.; anti-arrhythmics class III such as amiodarone and sotalol; anti-arrhythmics class IV such as diltiazem, verapamil, etc. The drug eluting balloon can also provide a slew of therapeutic agents to treat the border zone including angiogenic, arterogenic and antioxidant agents.

The foregoing disclosure of the exemplary embodiments of the present invention has been presented for purposes of illustration and description. It is not intended to be exhaustive or to limit the invention to the precise forms disclosed. Many variations and modifications of the embodiments described herein will be apparent to one of ordinary skill in the art in light of the above disclosure. The scope of the invention is to be defined only by the claims appended hereto, and by their equivalents.

Further, in describing representative embodiments of the present invention, the specification may have presented the method and/or process of the present invention as a particular sequence of steps. However, to the extent that the method or process does not rely on the particular order of steps set forth herein, the method or process should not be limited to the particular sequence of steps described. As one of ordinary skill in the art would appreciate, other sequences of steps may be possible. Therefore, the particular order of the steps set forth in the specification should not be construed as limitations on the claims. In addition, the claims directed to the

method and/or process of the present invention should not be limited to the performance of their steps in the order written, and one skilled in the art can readily appreciate that the sequences may be varied and still remain within the spirit and scope of the present invention.

WHAT IS CLAIMED IS:

1. A device for controlling a volume of a chamber of a heart, the device comprising:

a metallic pouch positioned within the chamber of the heart; and

a magnet positioned outside of the chamber and in communication with the metallic pouch to decrease available blood volume of the chamber.

2. The device of claim 1, wherein the chamber is a ventricle.

3. The device of claim 1, wherein the chamber is an atrium.

4. The device of claim 1, wherein the metallic pouch is shaped to fit within a particular position within the chamber.

5. The device of claim 1, wherein the metallic pouch includes barbs to grip onto an interior surface of the chamber.

6. The device of claim 1, wherein the magnet is included within a sheath.

7. The device of claim 6, wherein the sheath is shaped to be placed on a pericardial surface of the heart to promote magnetic communication with the metallic pouch such that a sufficient holding force is created to withstand any natural movement of the heart.

8. The device of claim 1, wherein the metallic patch and the magnet are placed in position using a percutaneous catheter.

9. The device of claim 1, wherein the metallic pouch contains metallic beads.

10. The device of claim 1, wherein the metallic pouch contains metallic strips.

11. The device of claim 1, wherein the metallic pouch contains metal embedded within material composing the pouch.

12. The device of claim 11, wherein the material composing the pouch is polymeric.

13. The device of claim 1, wherein the metallic pouch is coated with one or more anti-arrhythmic agents.

14. A device for controlling a volume of a chamber of a heart, the device comprising:

a pouch positioned within a heart chamber to controls its volume; and

a sheath positioned outside of the chamber;

wherein the pouch and the sheath are in magnetic communication with one another.

15. The device of claim 14, wherein the pouch contains metallic beads.

16. The device of claim 14, wherein the pouch contains metallic strips.

17. The device of claim 14, wherein the pouch contains metal embedded within material composing the pouch.

18. The device of claim 17, wherein the material composing the pouch is polymeric.

19. The device of claim 14, wherein the pouch is coated with one or more anti-arrhythmic agents.

20. A method for magnetic tissue support, the method comprising:

introducing a metallic body on one side of a tissue that needs support; and

placing a magnet on the opposite side of the tissue that needs support so that the magnet and the metallic body form an attractive force across a body of the tissue that needs support, thereby providing support to the tissue.

21. The method of claim 20, wherein the tissue is part of a heart.

22. The method of claim 21, wherein the tissue includes a ventricular wall.

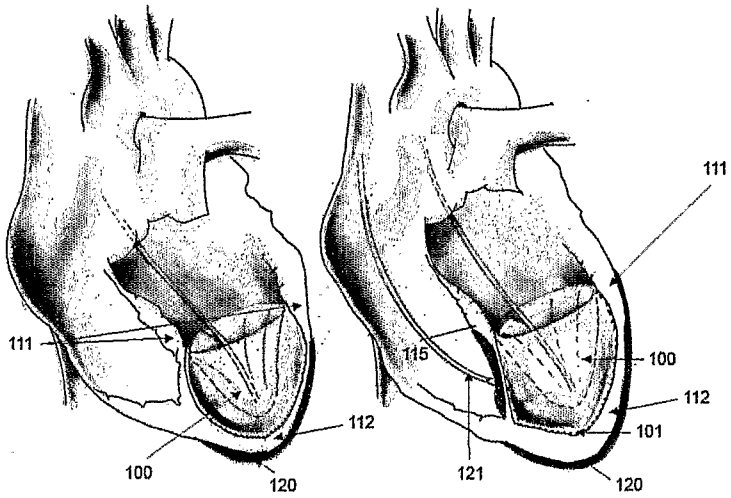

Fig. 1A

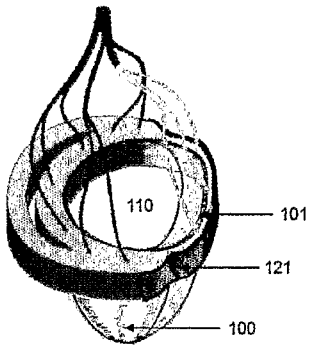

Fig. 1B

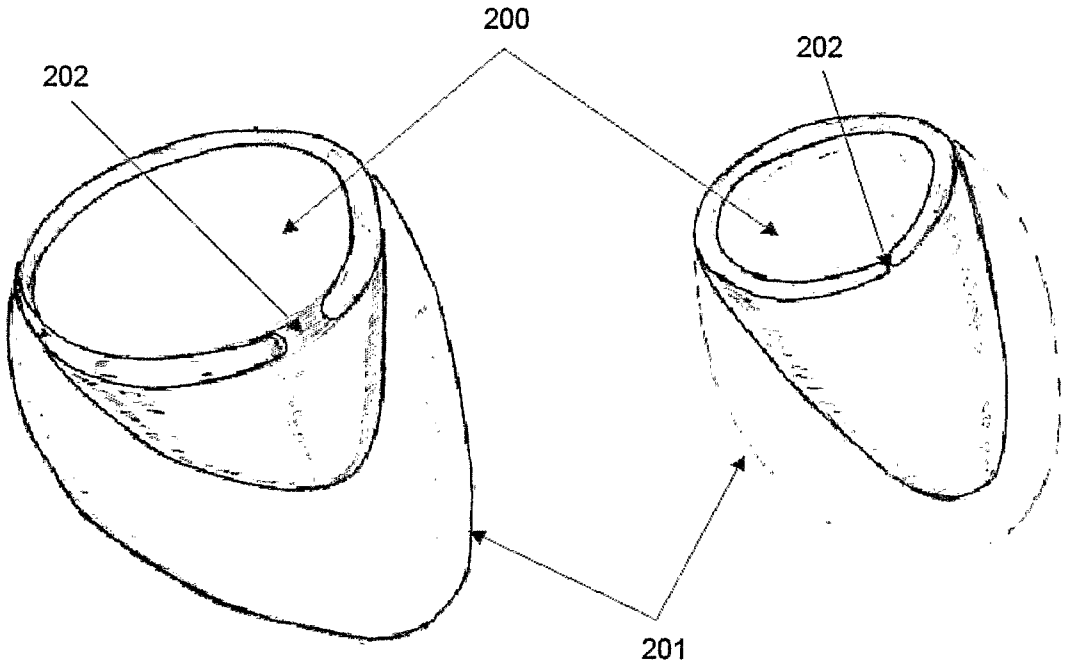

Fig. 2

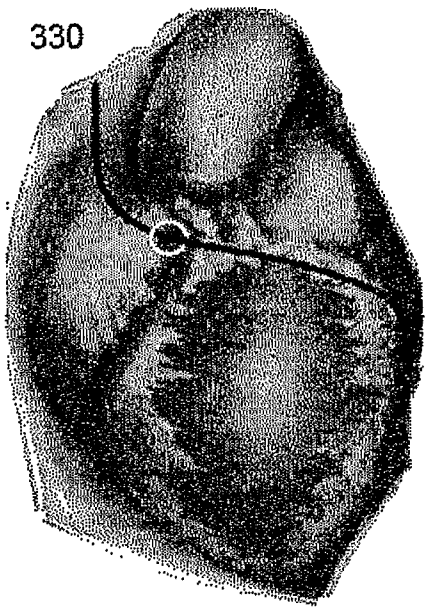

Fig. 3A

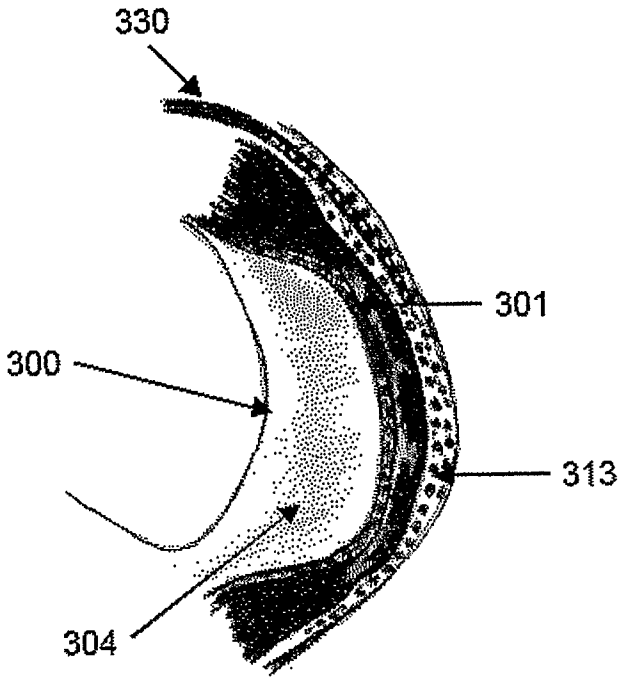

Fig. 3B

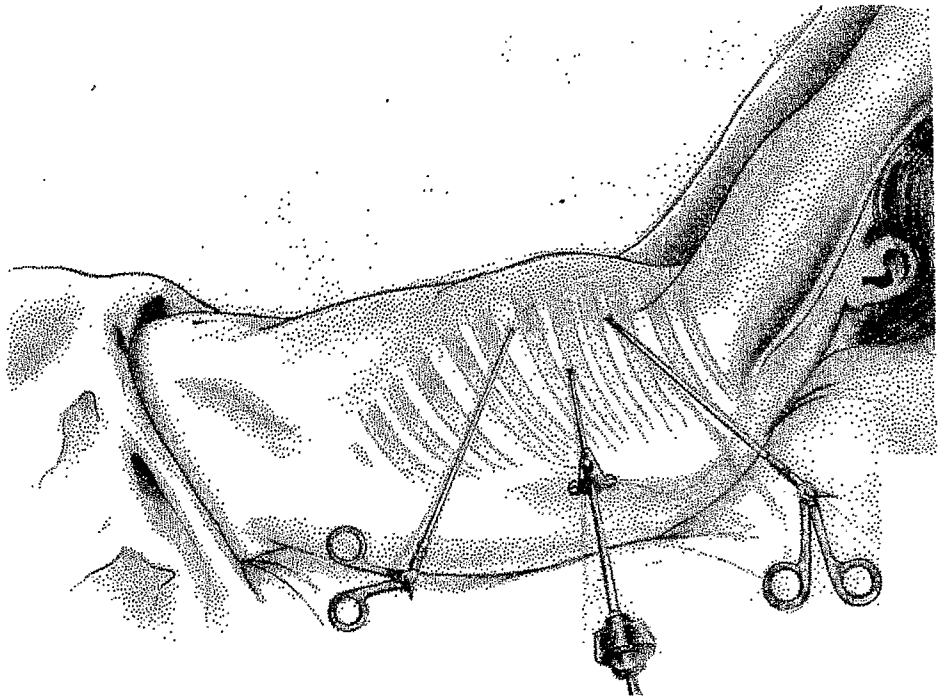

Fig. 4A

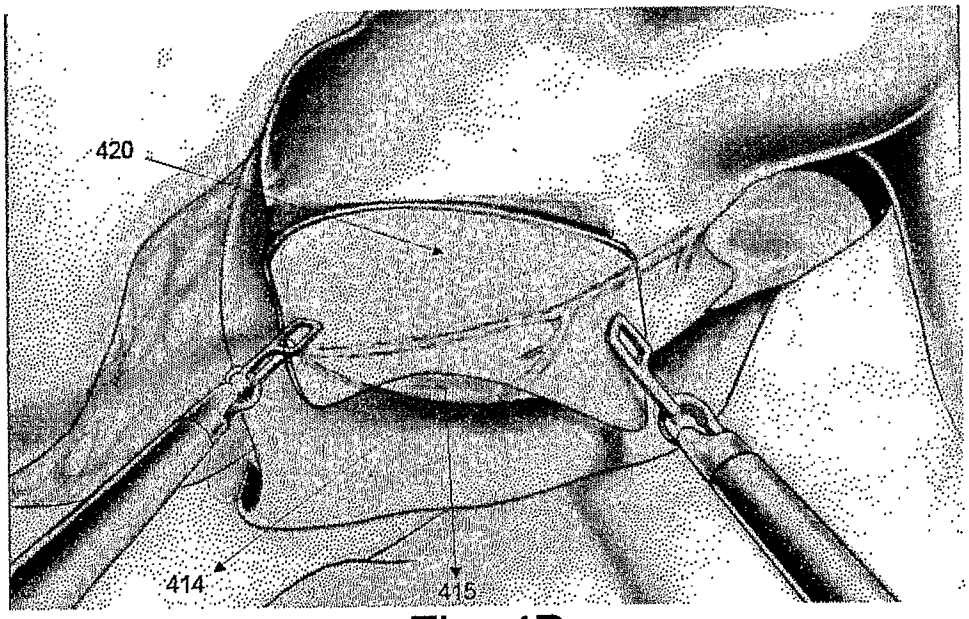

Fig. 4B

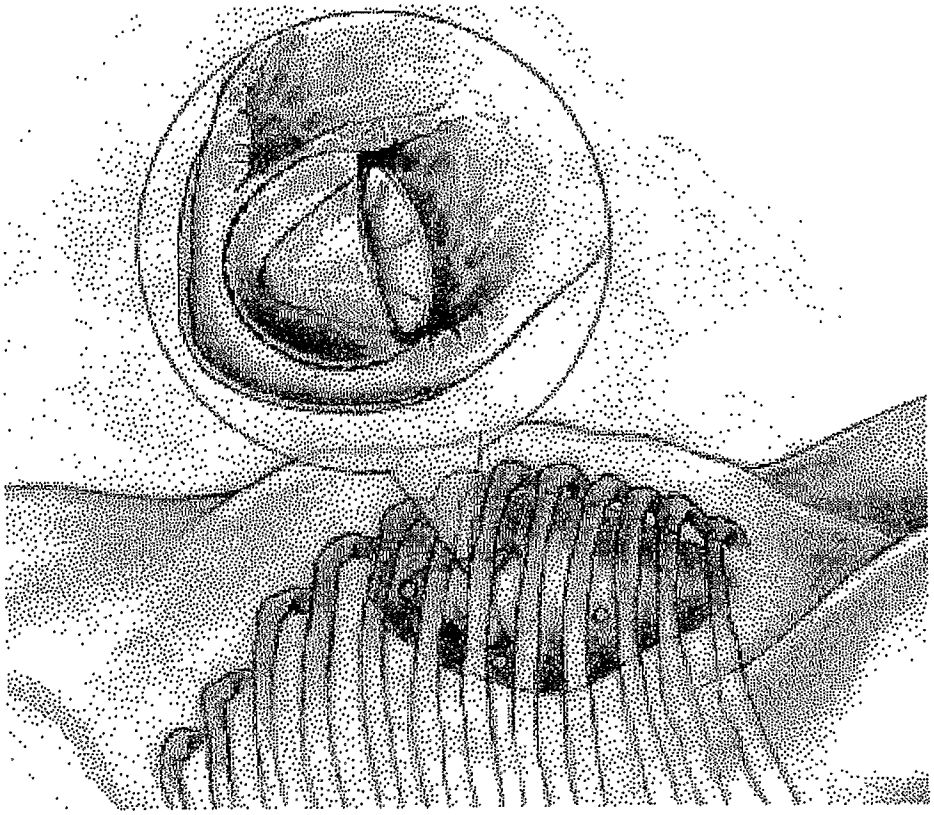

Fig. 4C

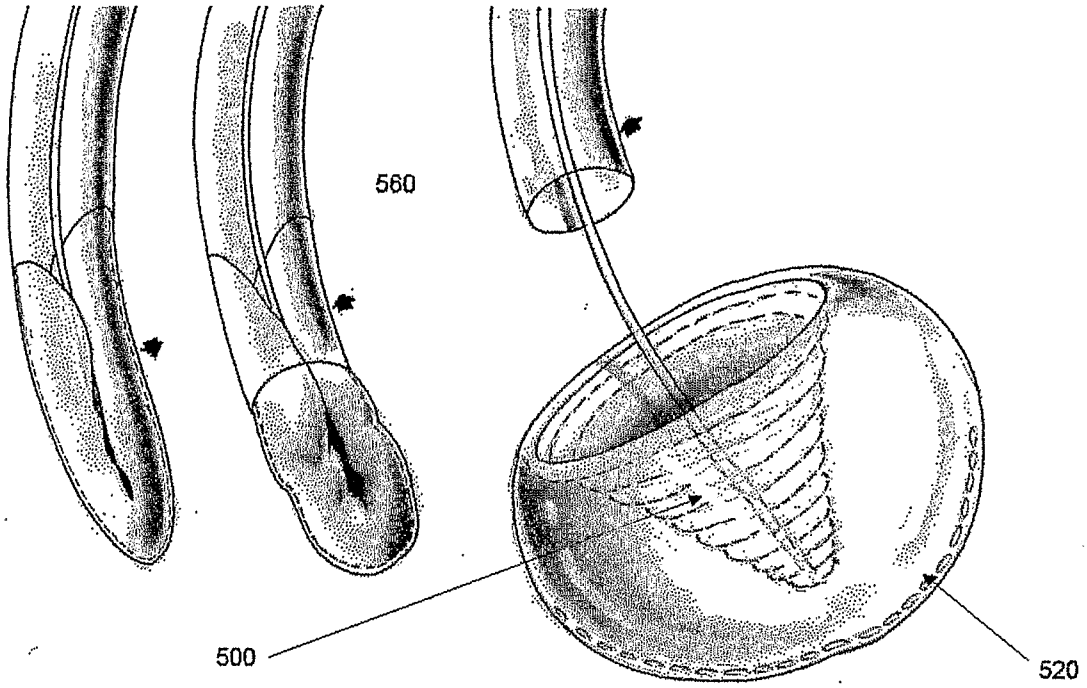

Fig. 5

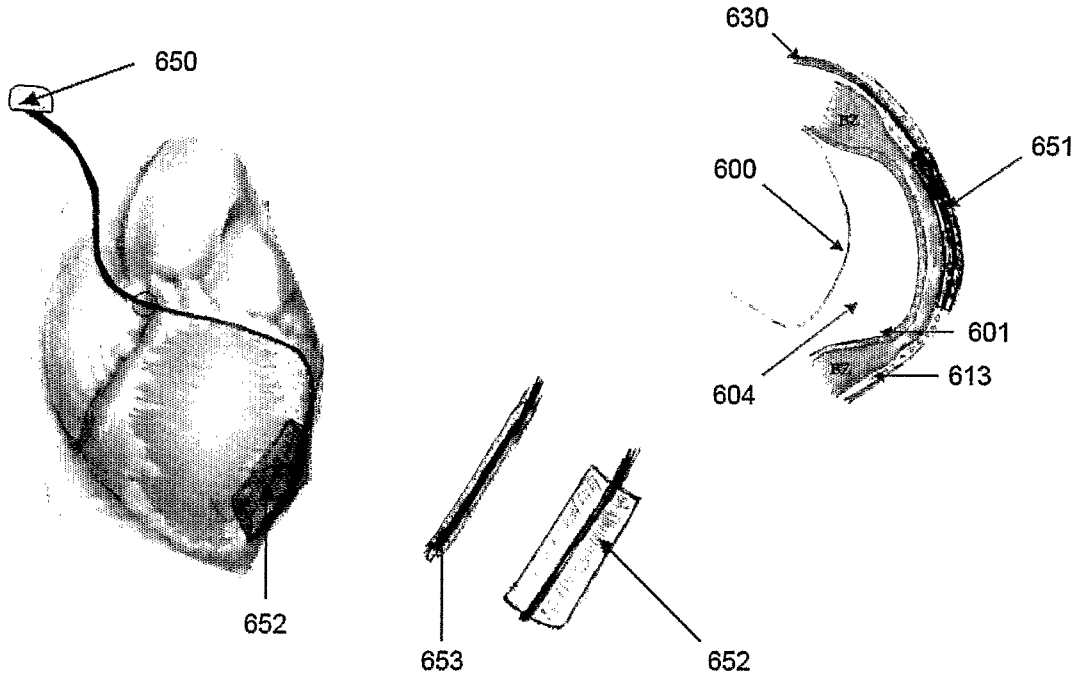

Fig. 6

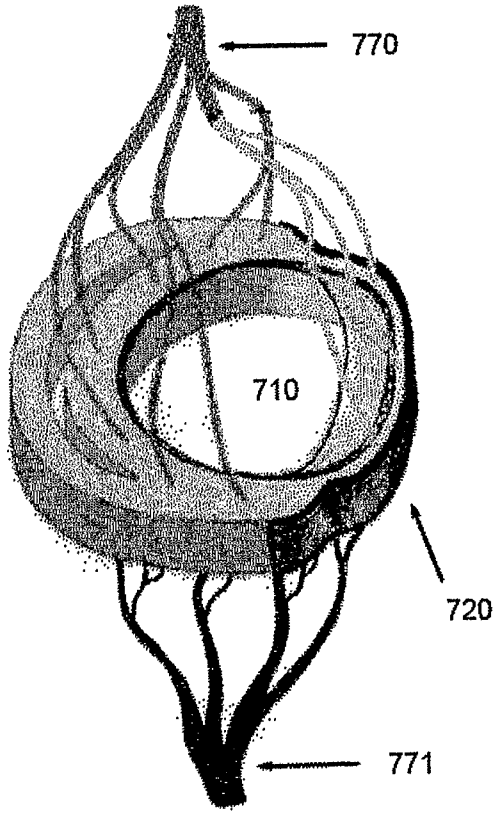

Fig. 7

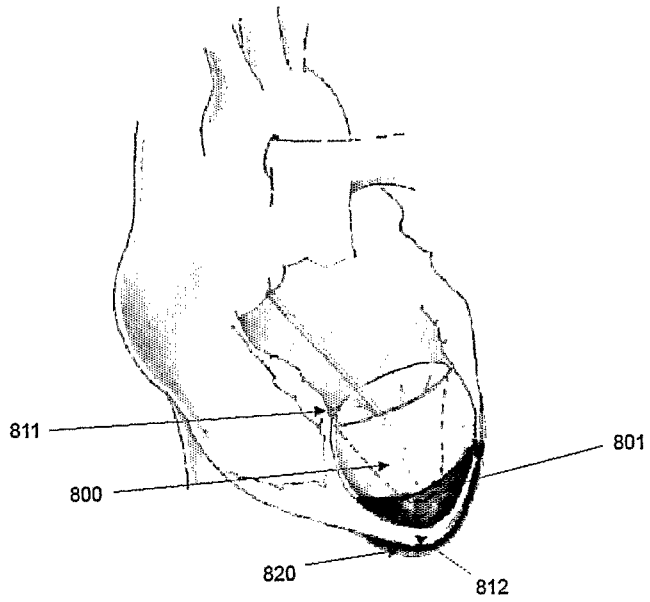

Fig. 8

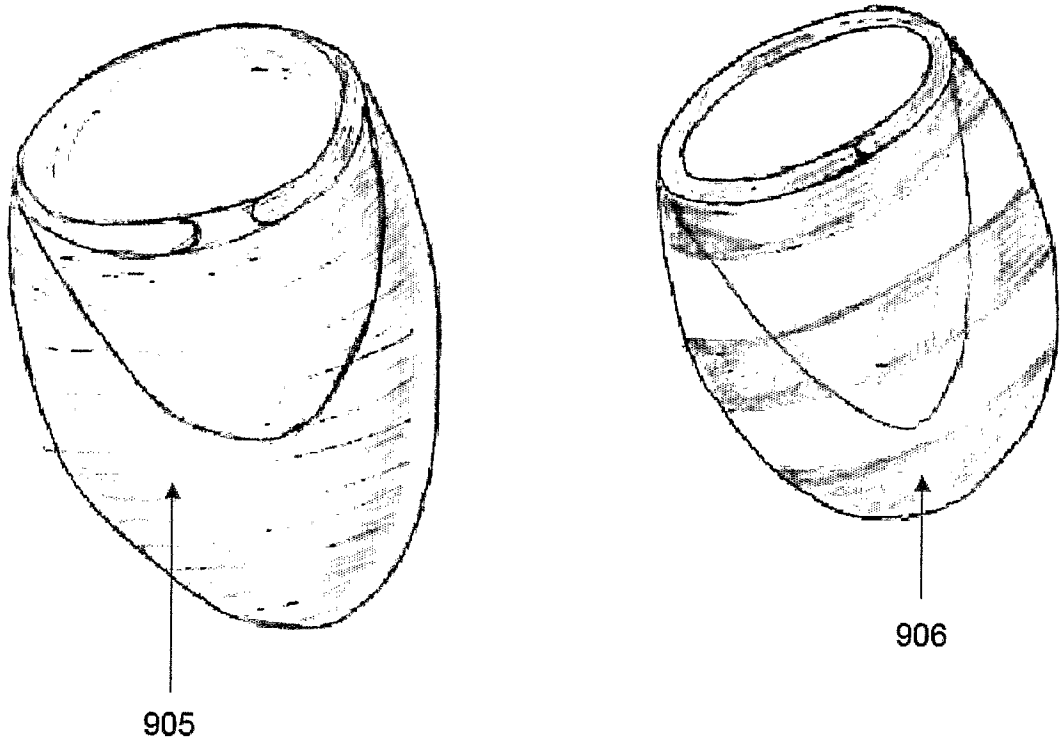

Fig. 9