US 20070112244A1

(19) United States

(12) Patent Application Publication (10) Pub. No.: US 2007/0112244 A1

McCarthy et al. (43) Pub. Date: May 17, 2007

(54) METHODS AND DEVICES FOR IMPROVING CARDIAC FUNCTION IN HEARTS

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(21) Appl. No.: **11/404,092**

(22) Filed: **Apr. 14, 2006**

Related U.S. Application Data

(63) Continuation of application No. 11/316,341, filed on Dec. 23, 2005, which is a continuation of application No. 10/136,446, filed on May 2, 2002, now Pat. No. 7,189,199, which is a continuation of application No. 09/422,328, filed on Oct. 21, 1999, now Pat. No. 6,406,420, which is a continuation-in-part of application No. 09/124,286, filed on Jul. 29, 1998, now Pat. No. 6,045,497, which is a continuation-in-part of application No. 08/933,456, filed on Sep. 18, 1997, now Pat. No. 5,961,440, which is a continuation-in-part of application No. 08/778,277, filed on Jan. 2, 1997, now Pat. No. 6,050,936.

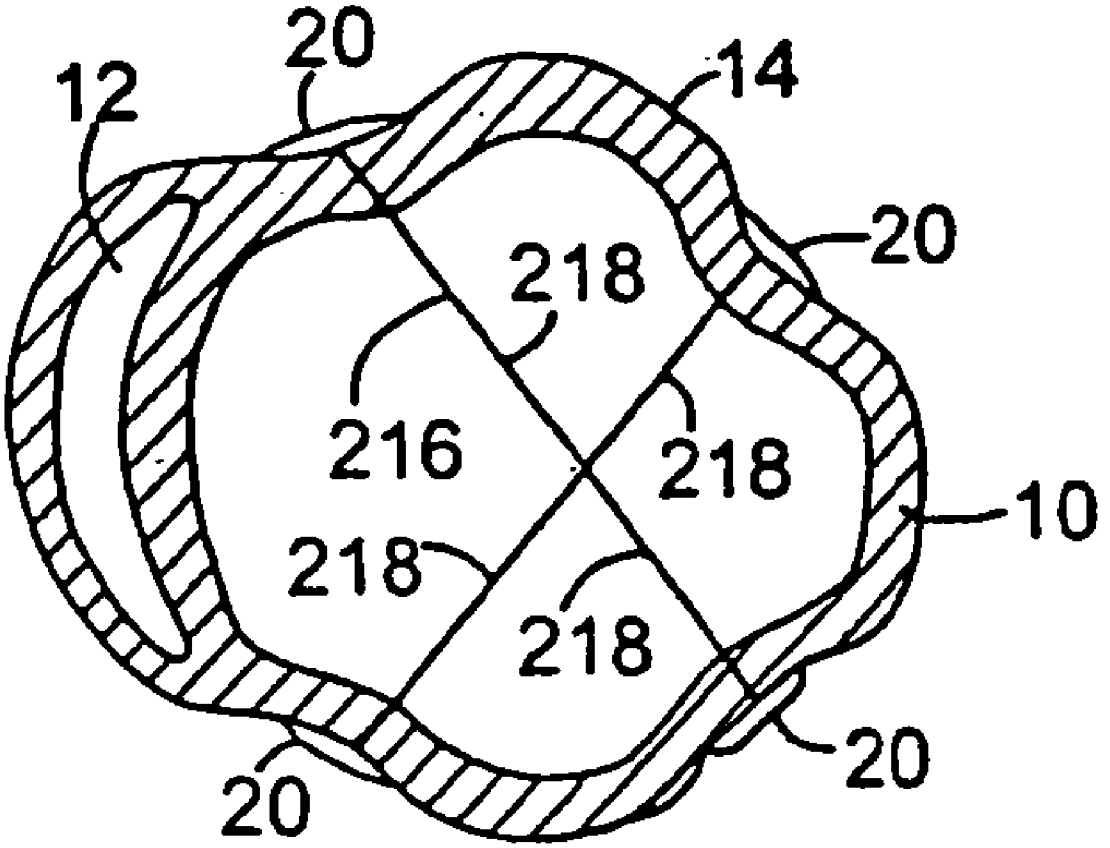
Publication Classification

(51) **Int. Cl.**
A61F 13/64 (2006.01)

(52) **U.S. Cl.** **600/16**; 600/37

(57) **ABSTRACT**

Various methods and devices are disclosed for improving cardiac function in hearts having zones of infarcted (akinetic) and aneurysmal (dyskinetic) tissue regions. The methods and devices reduce the radius of curvature in walls of the heart proximal infarcted and aneurysmal regions to reduce wall stress and improve pumping efficiency. The inventive methods and related devices include splinting of the chamber wall proximal the infarcted region and various other devices and methods including suture and patch techniques.



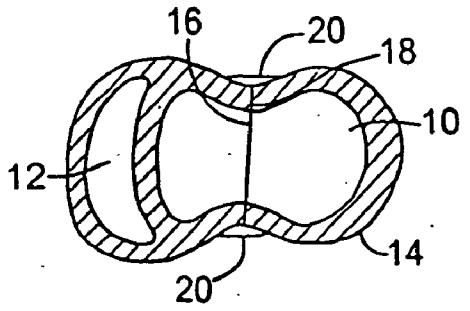


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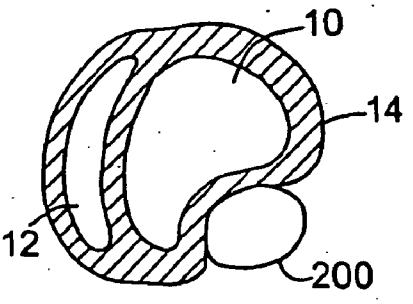


FIG.2

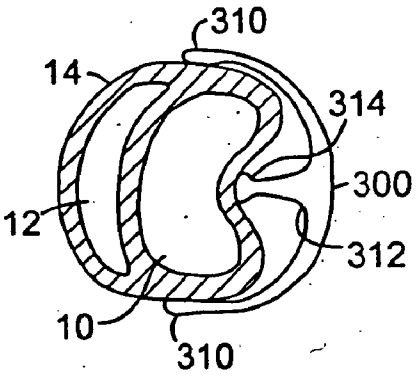


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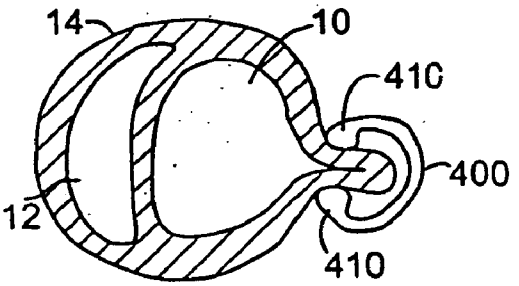


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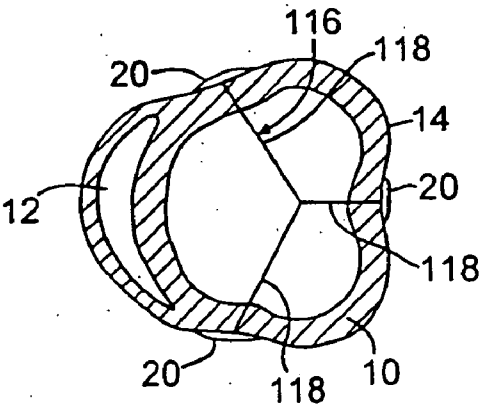


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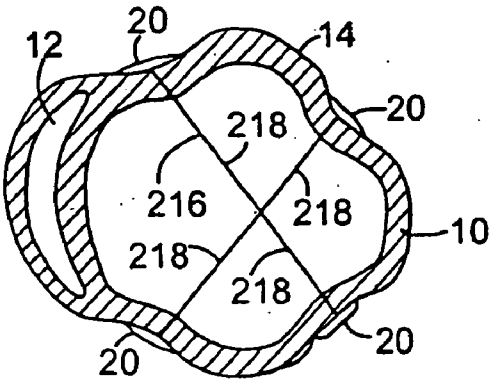


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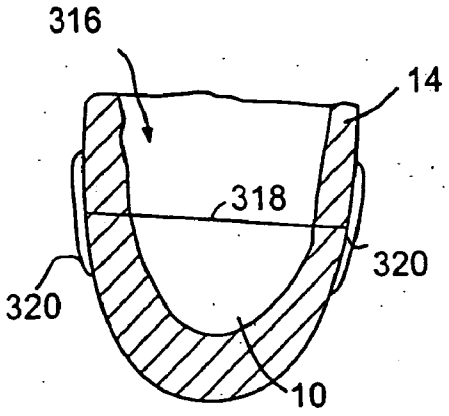


FIG. 7

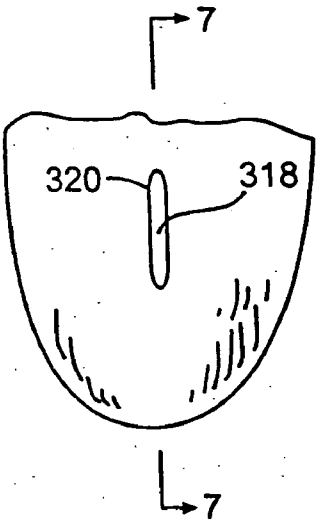


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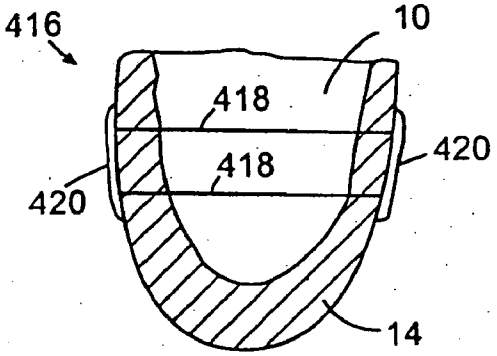


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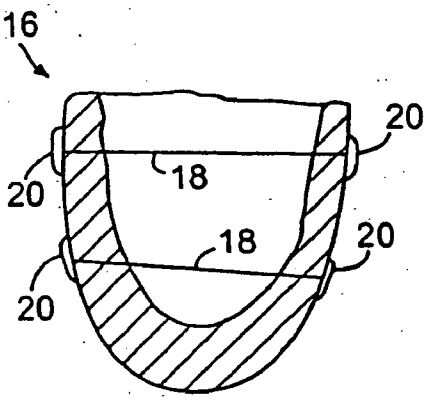


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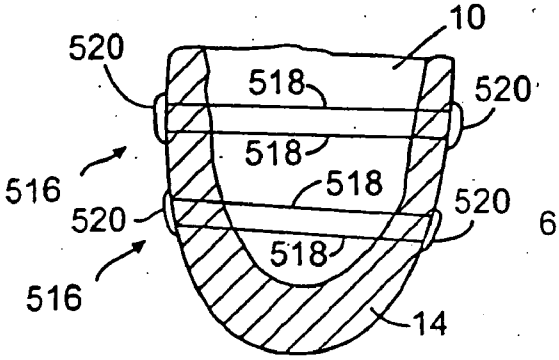


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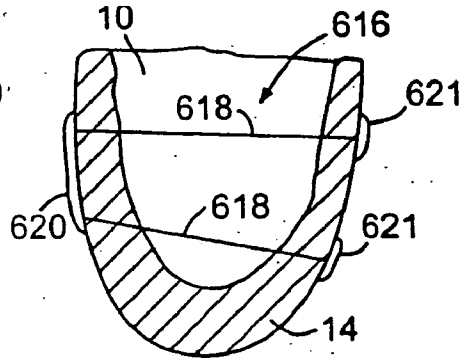


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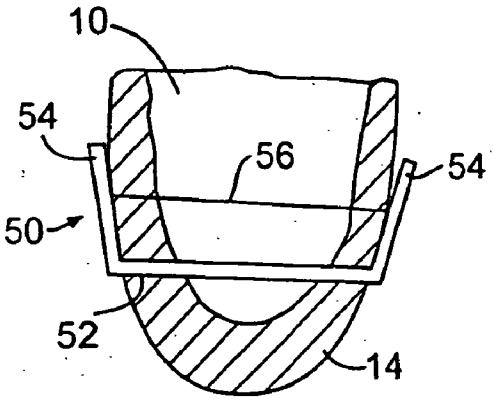


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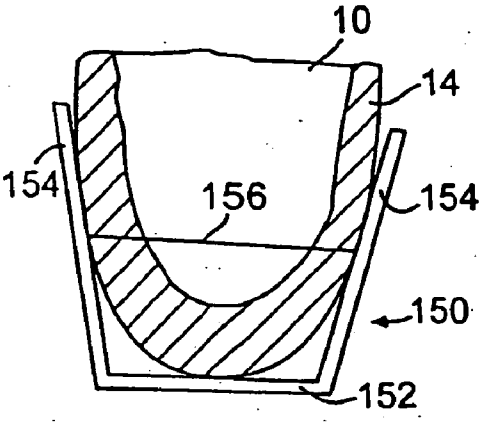


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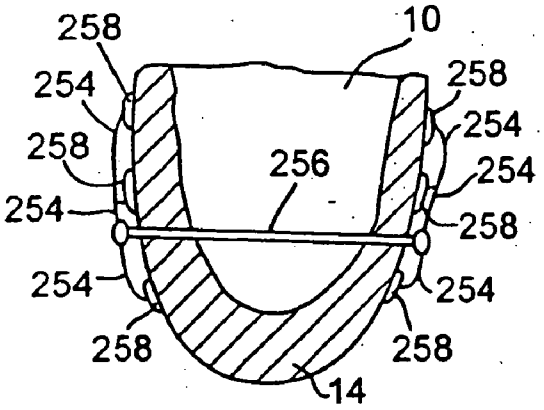


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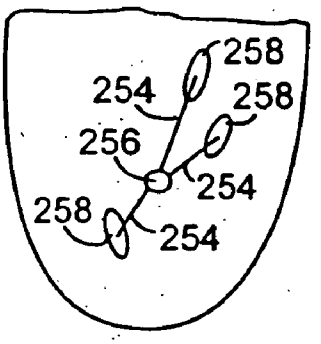


FIG. 16

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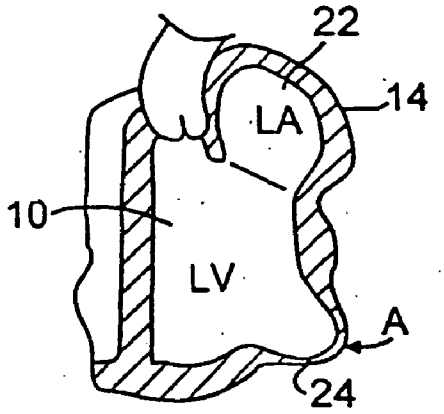


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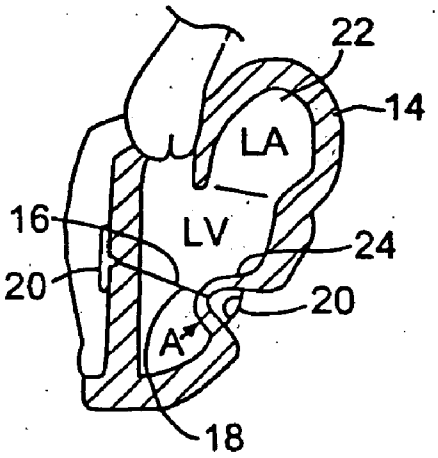
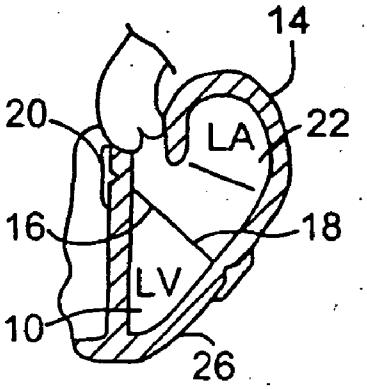


FIG. 19



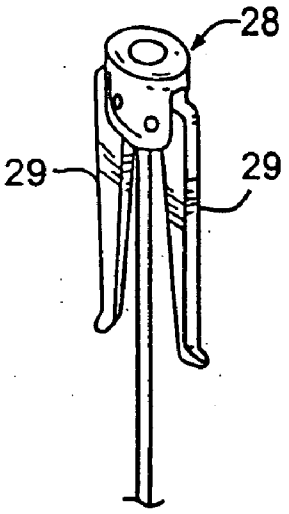


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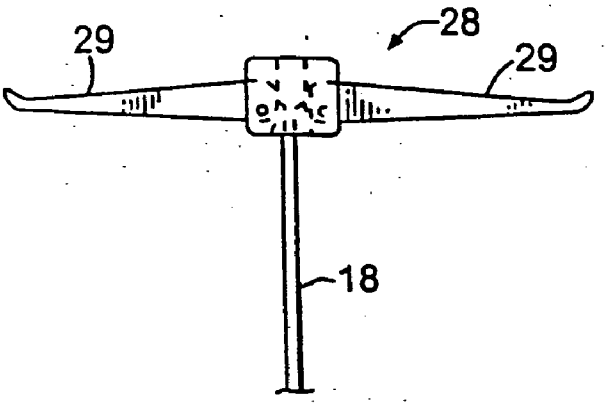


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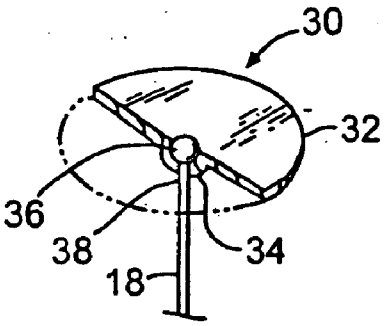


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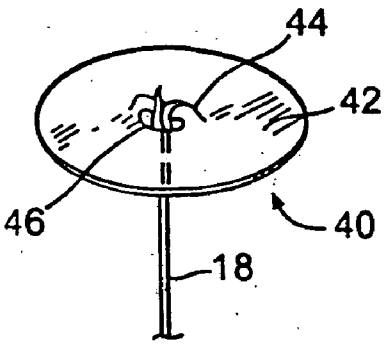


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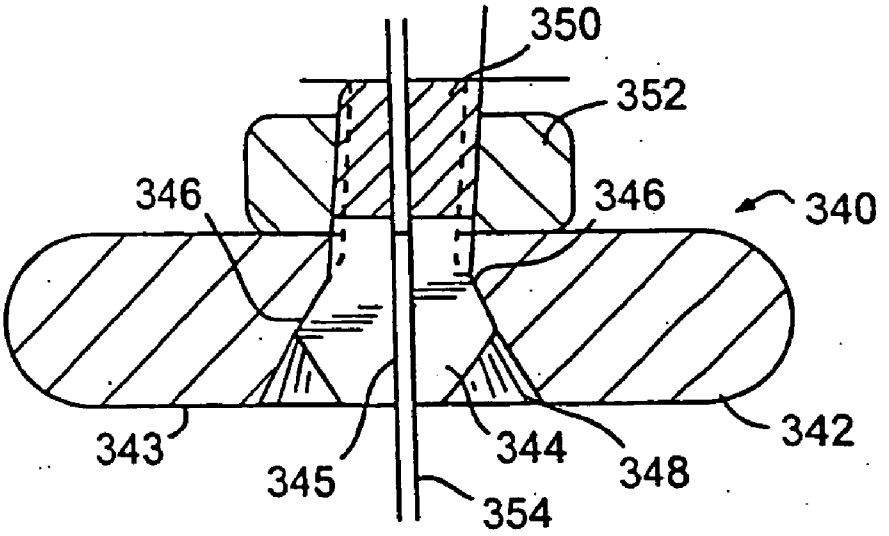

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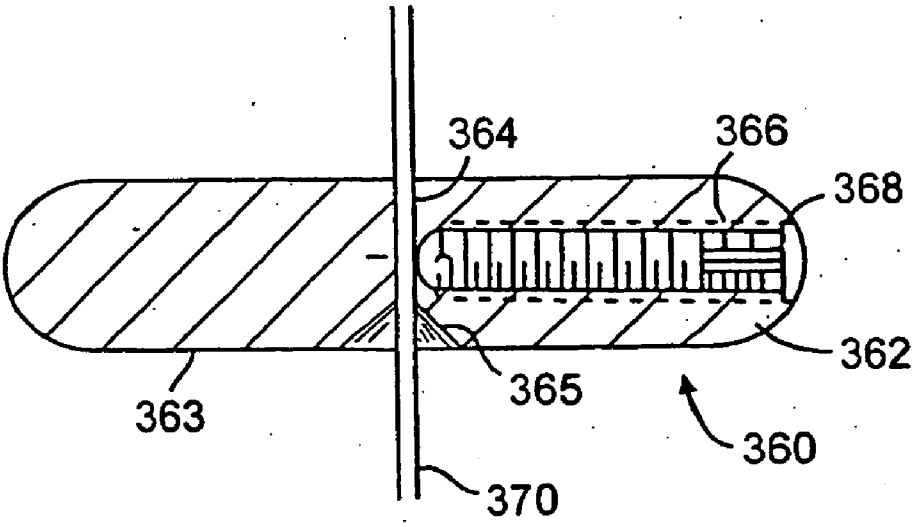

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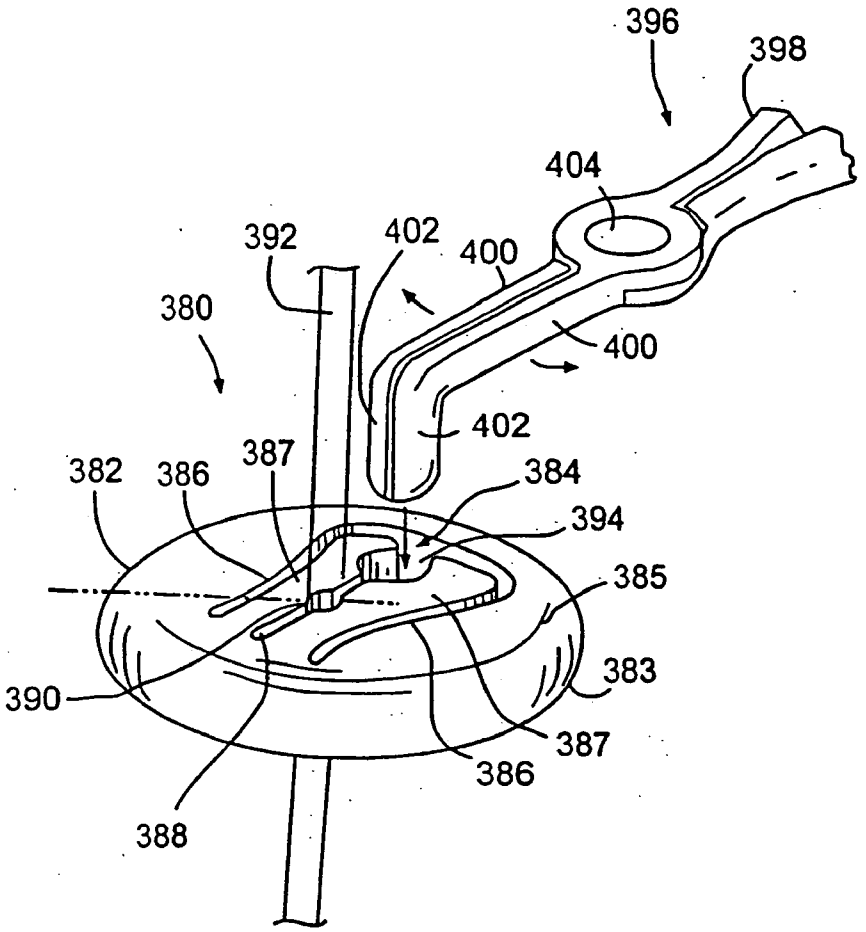

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FIG. 26

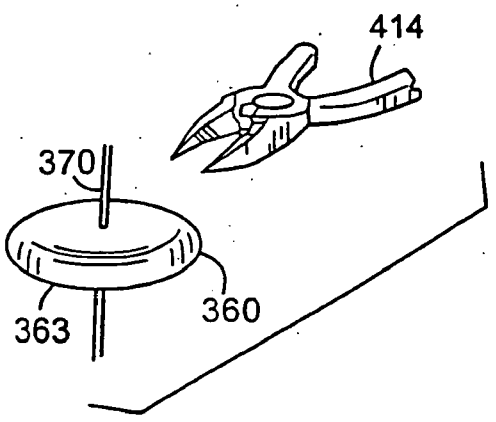

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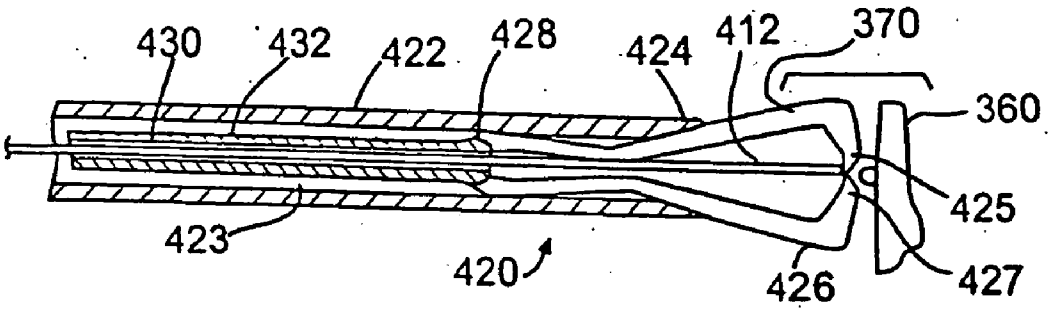

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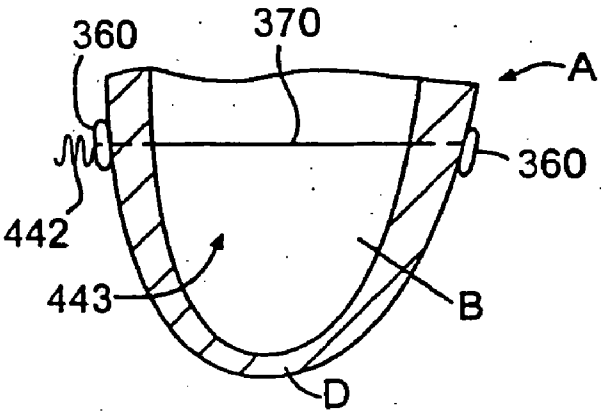

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FIG. 29

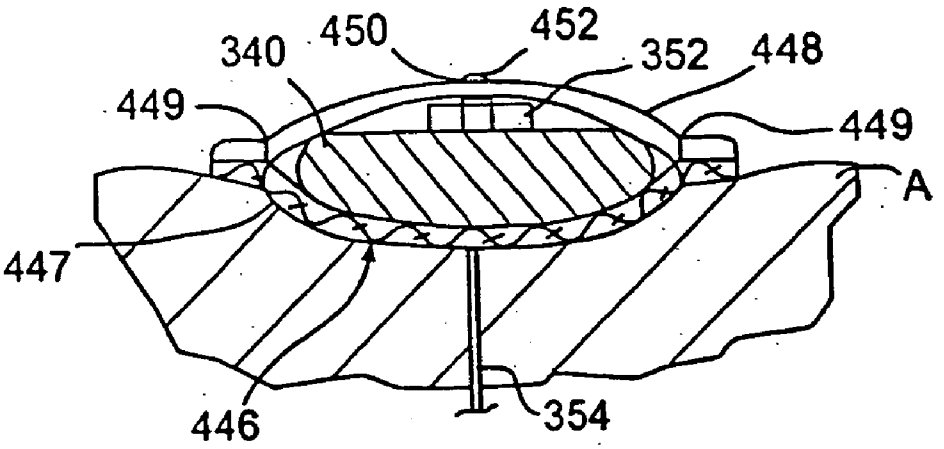


FIG. 30

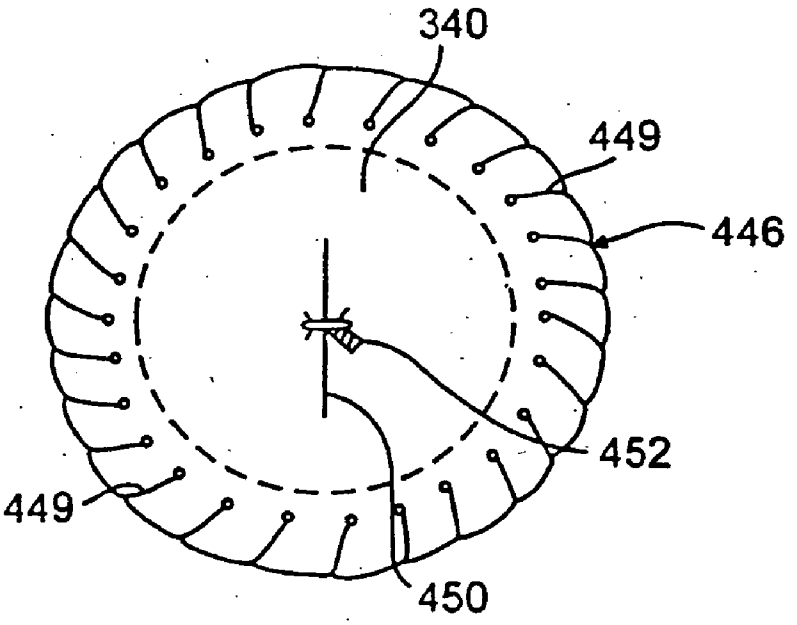


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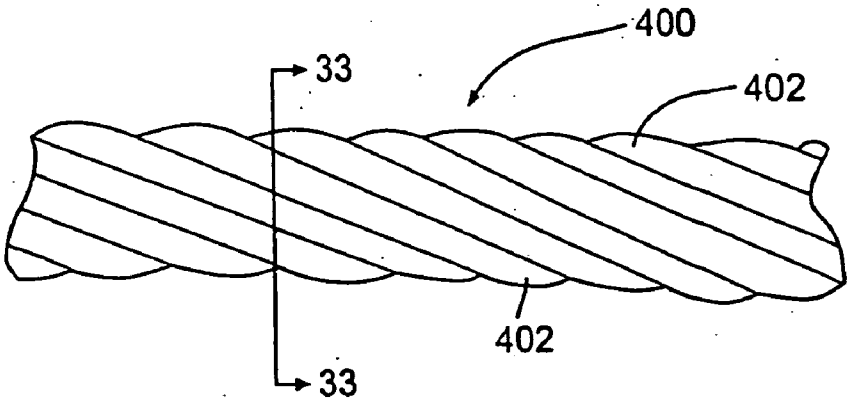


FIG. 32

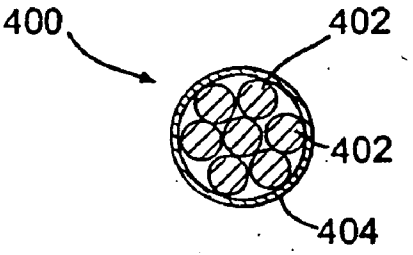


FIG. 33

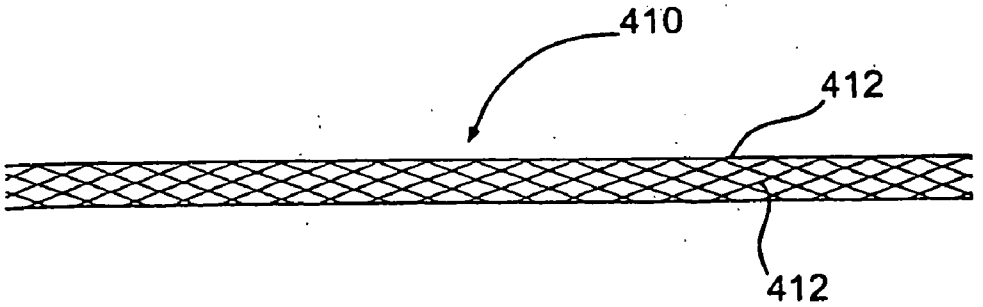


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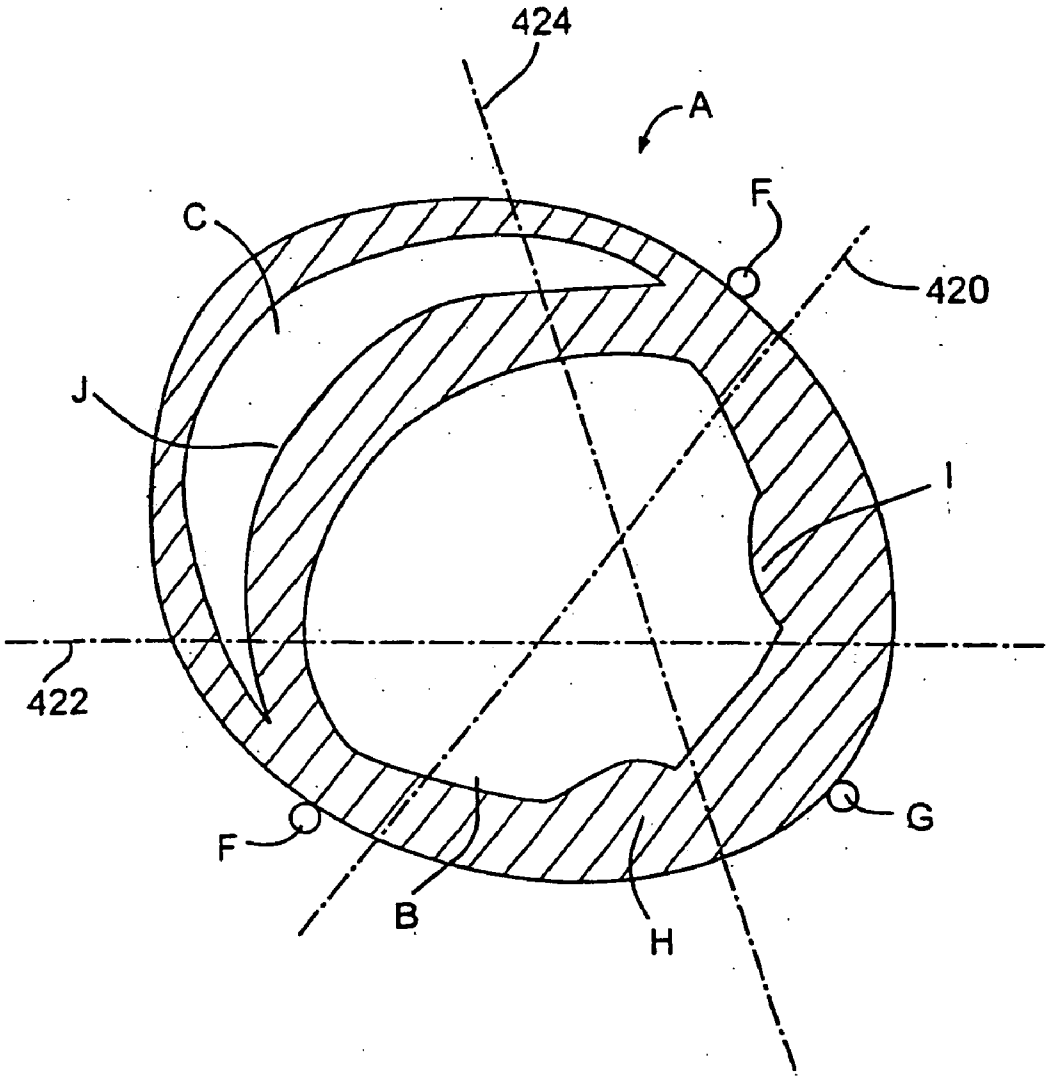


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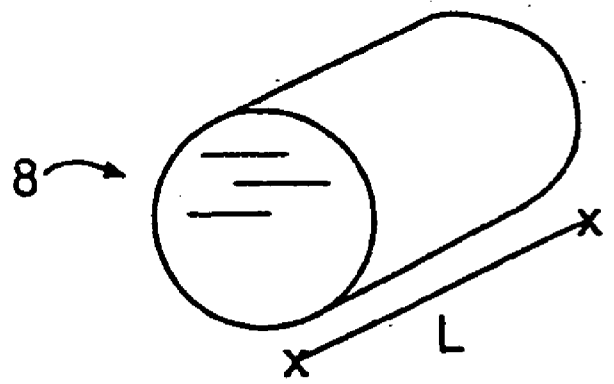


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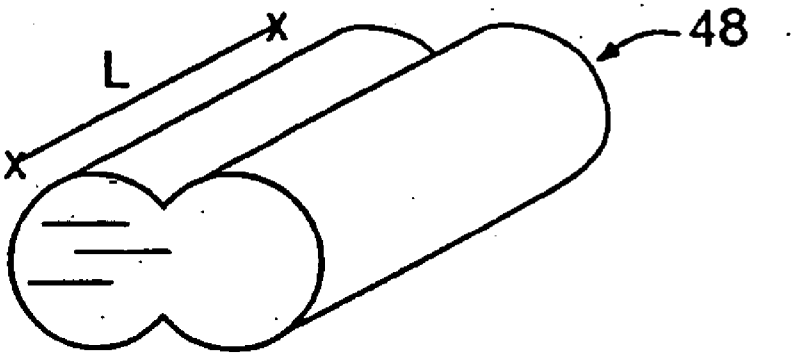


FIG. 37

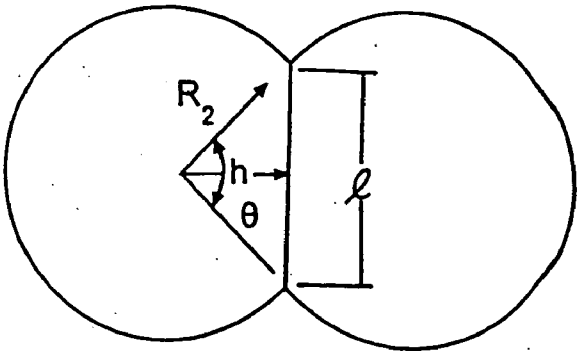


FIG. 38

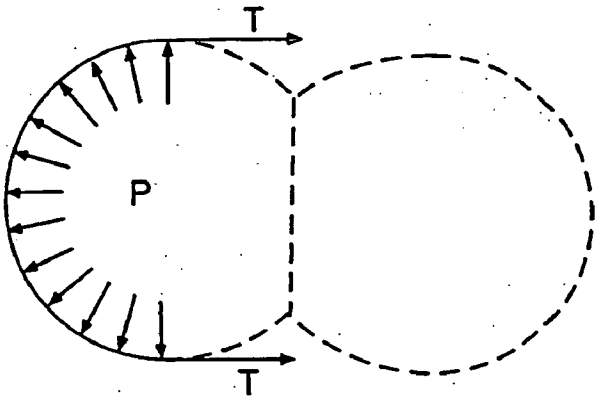


FIG. 39

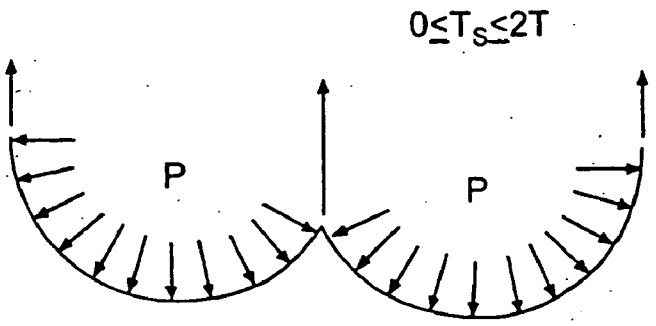


FIG. 40

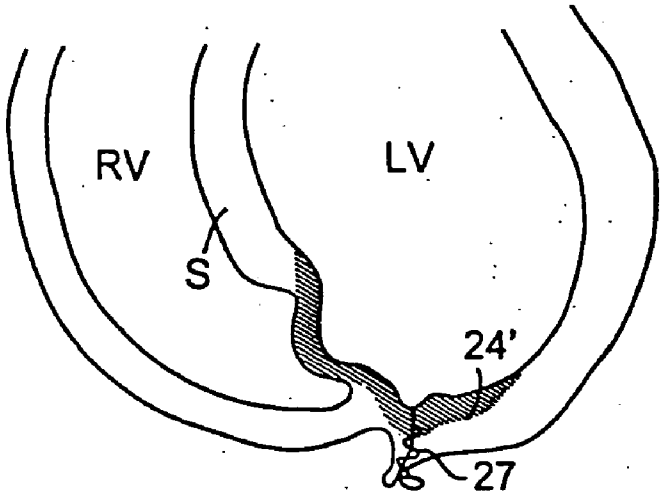


FIG. 41
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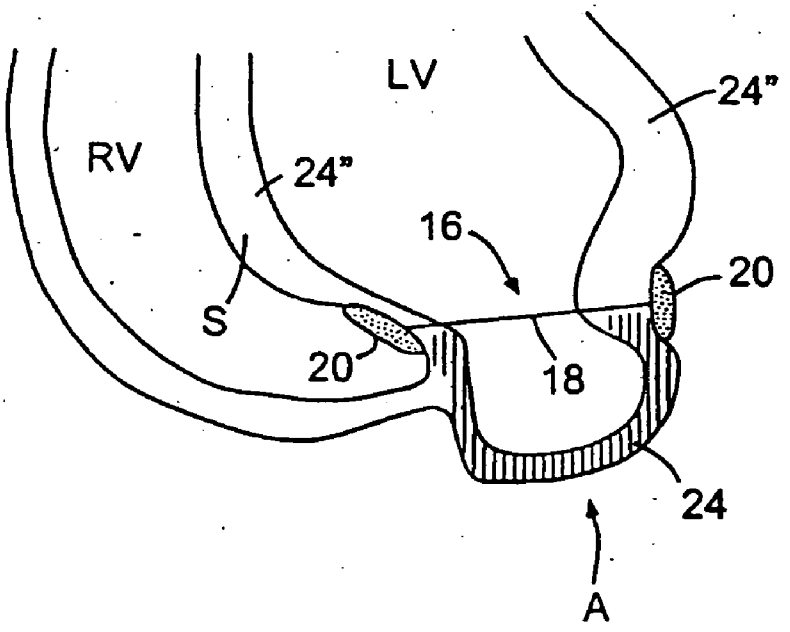


FIG. 42

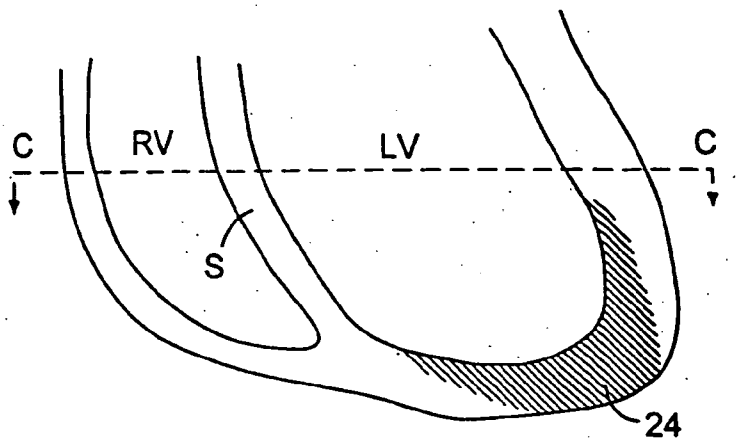


FIG. 43A

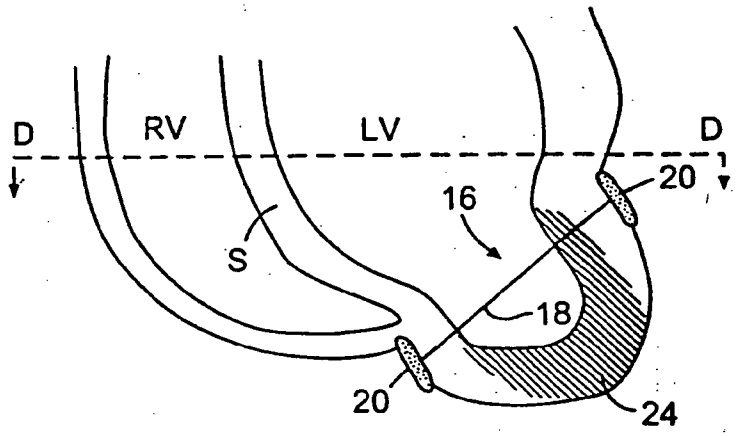


FIG. 43B

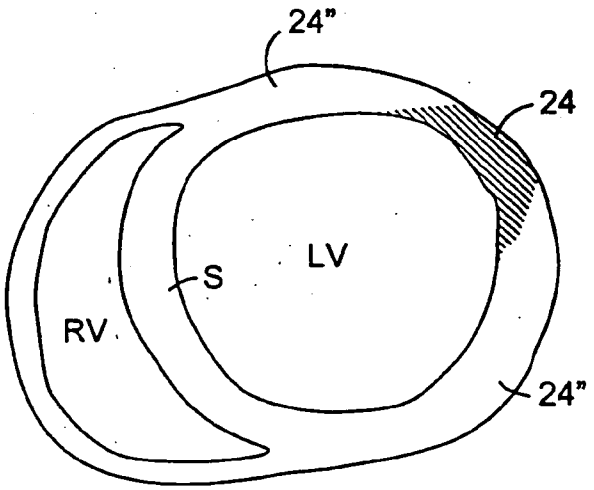


FIG. 43C

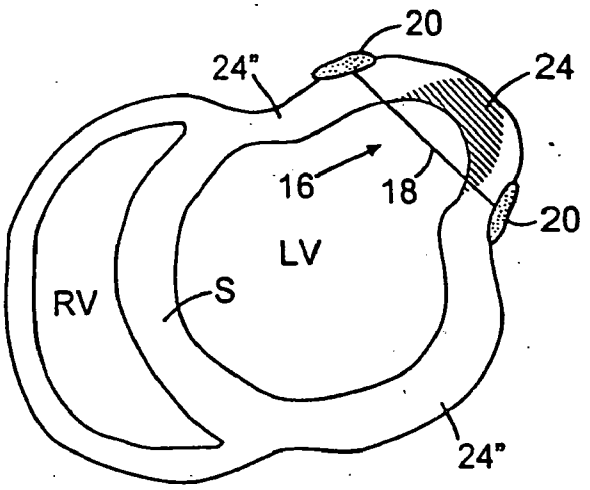


FIG. 43D

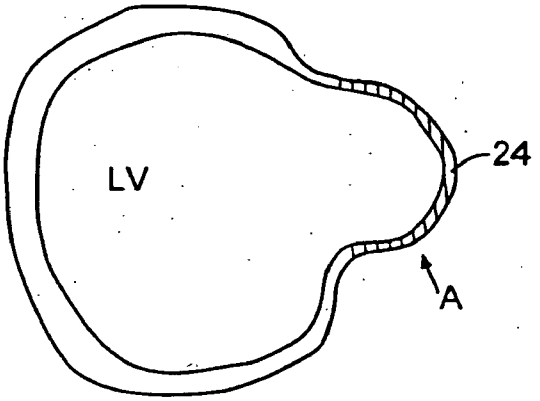


FIG. 44A

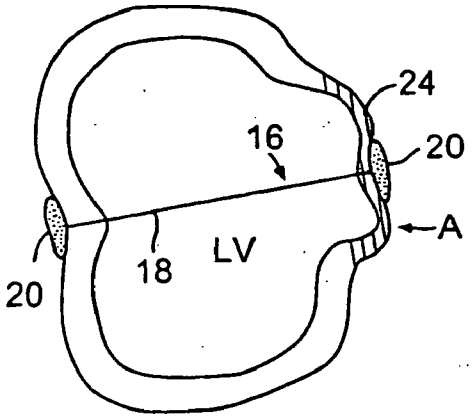


FIG. 44B

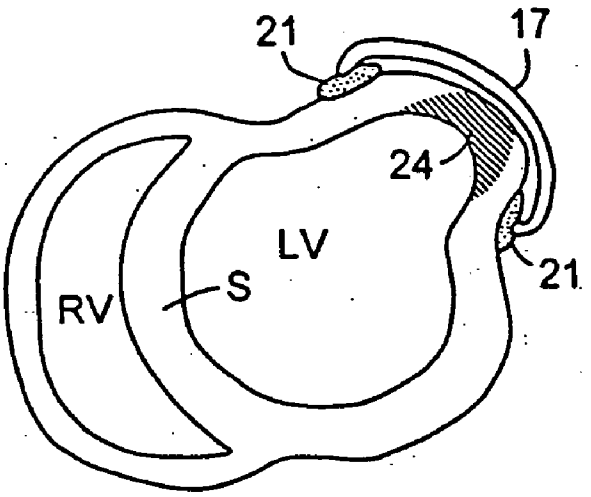


FIG. 45

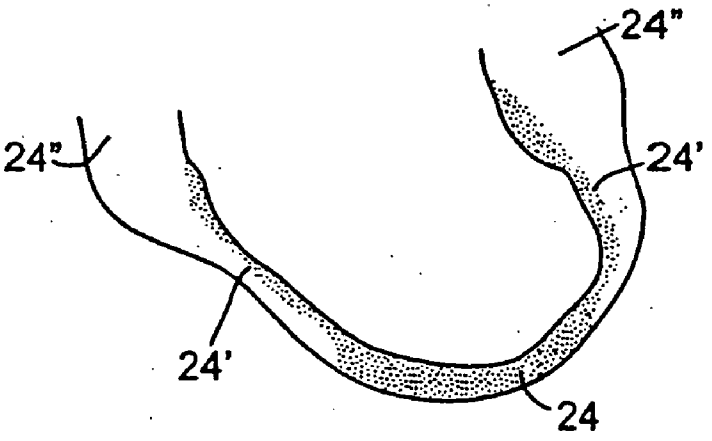


FIG. 46A

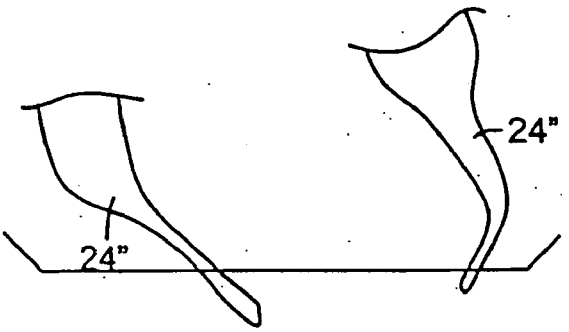


FIG. 46B

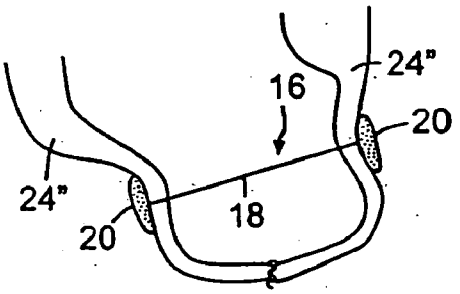


FIG. 46C

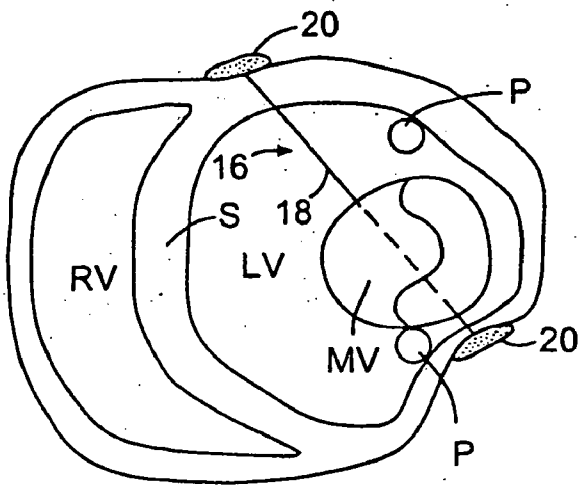


FIG. 47

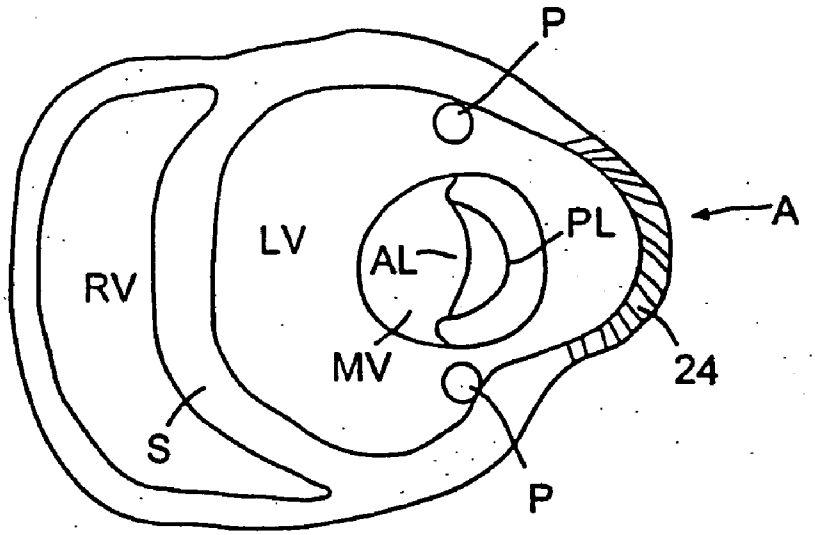


FIG. 48A

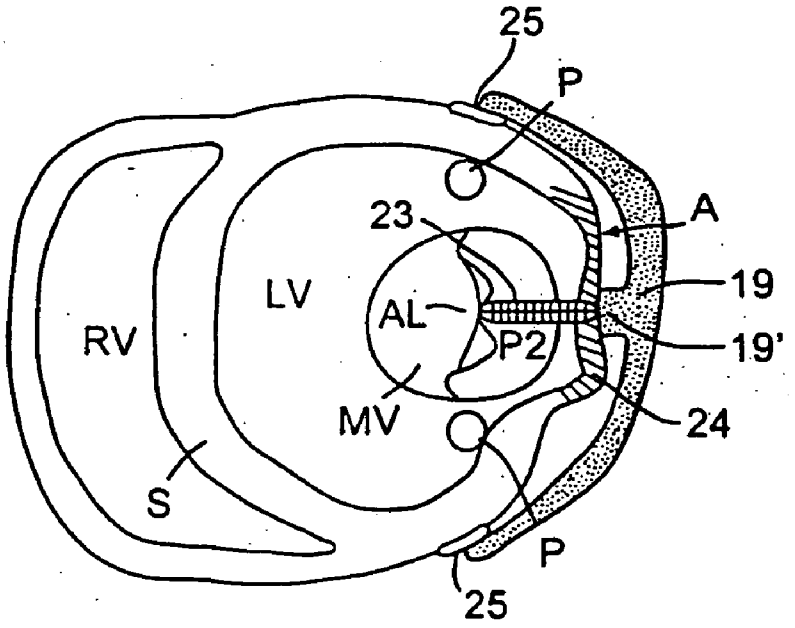


FIG. 48B

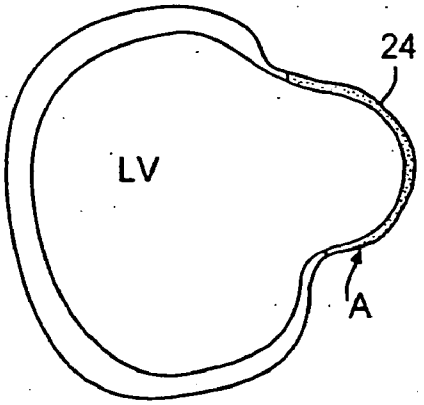


FIG. 49A

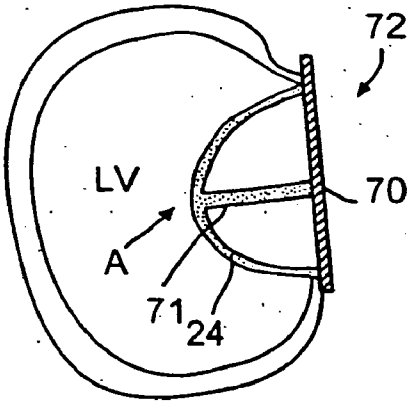


FIG. 49B

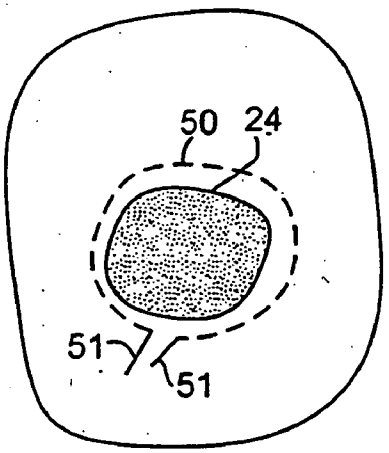


FIG. 50A

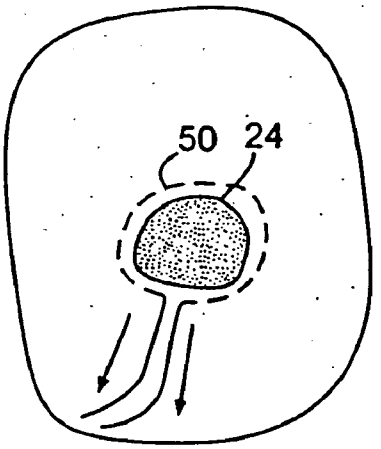


FIG. 50B

FIG. 51A

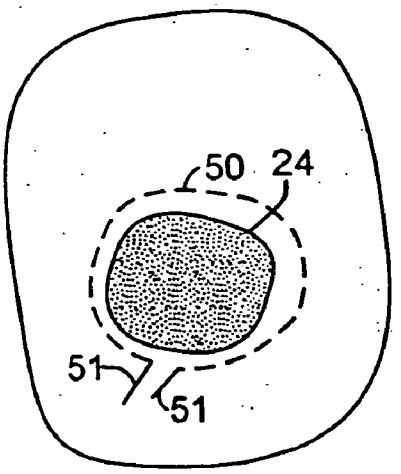


FIG. 51B

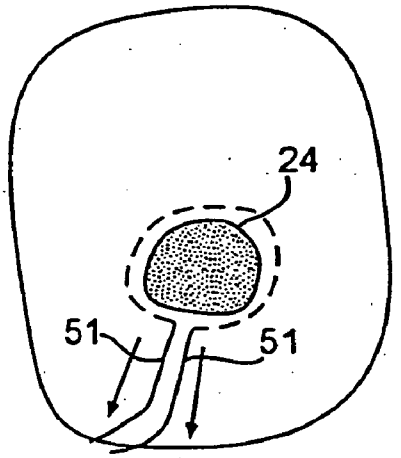
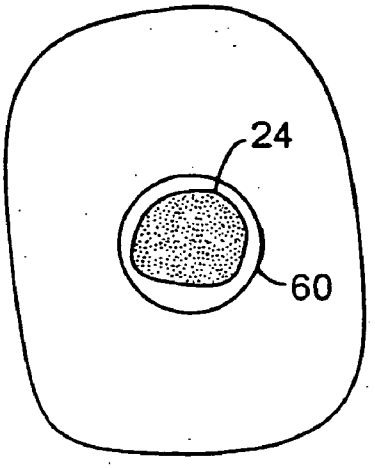


FIG. 51C



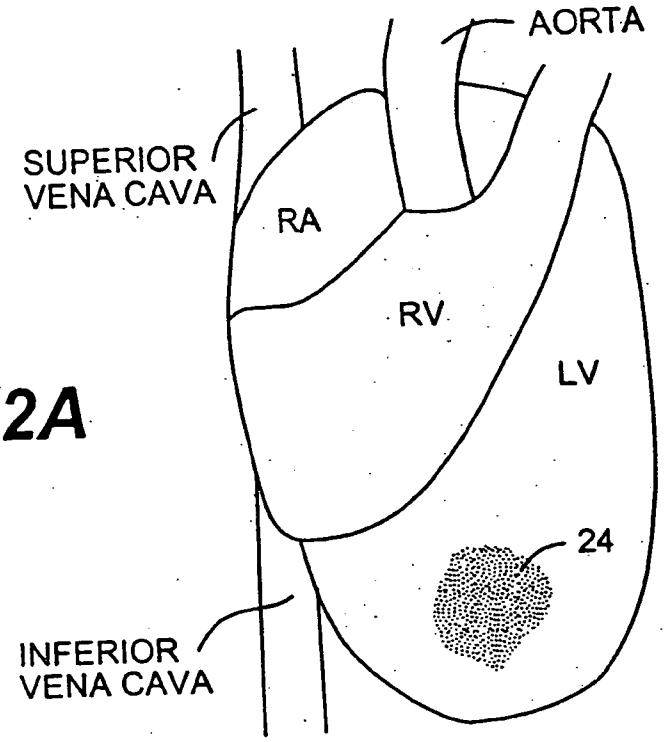


FIG. 52A

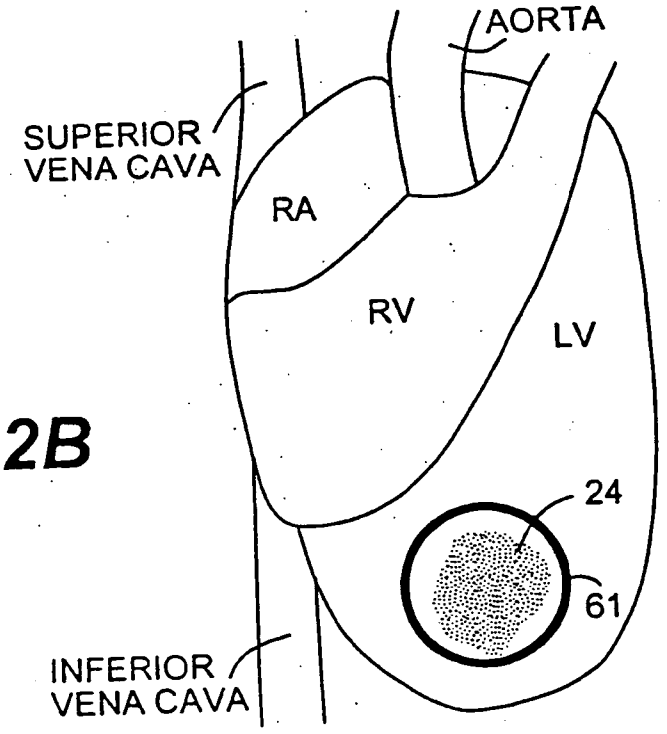


FIG. 52B

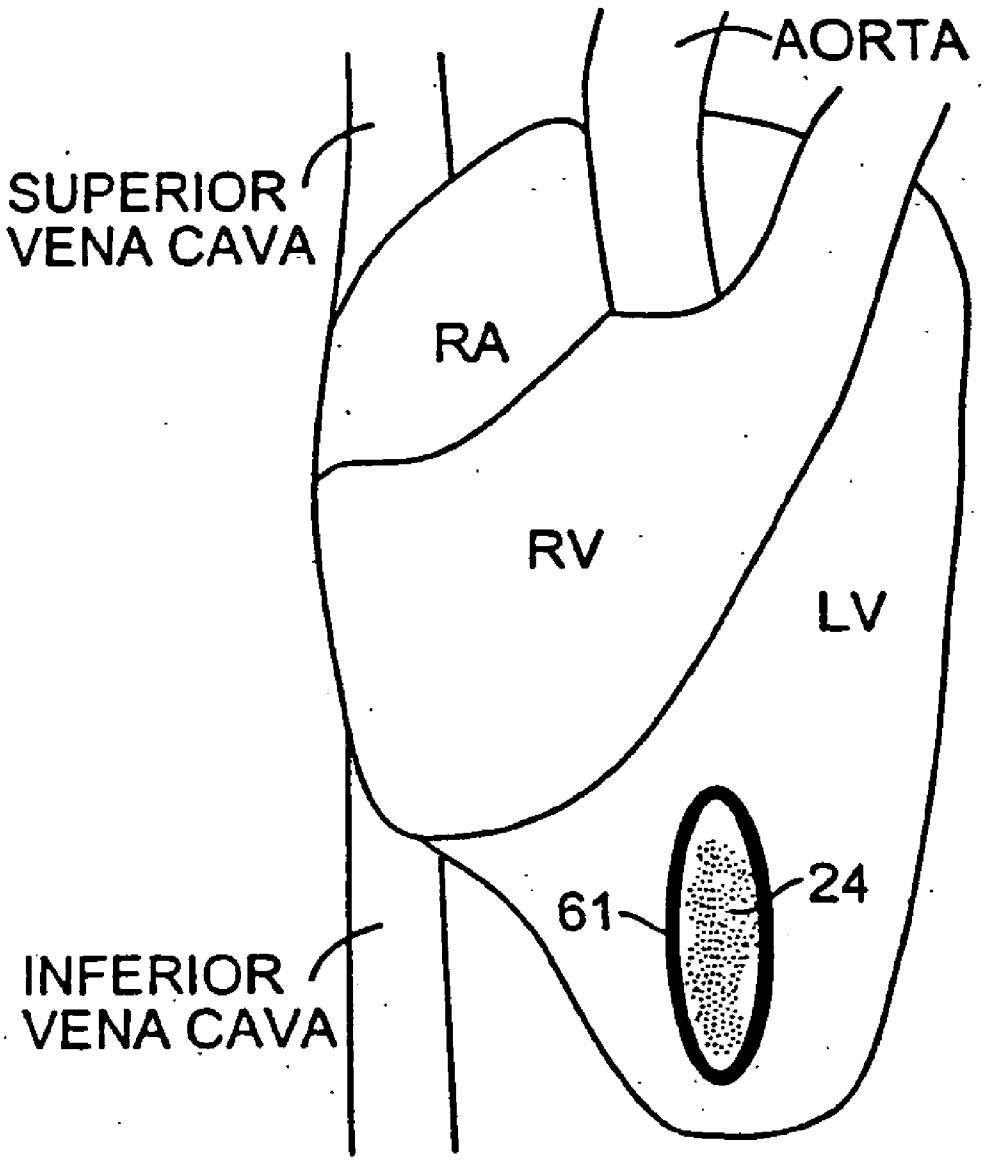


FIG. 52C

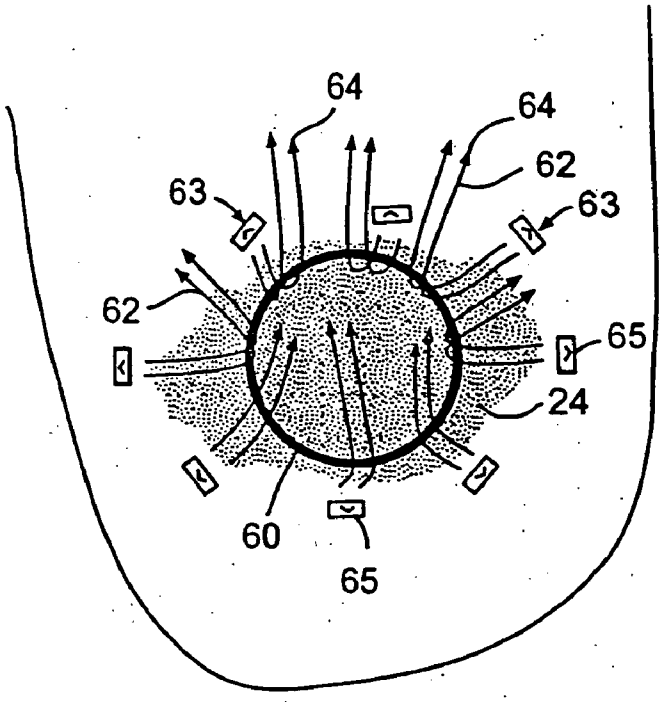


FIG. 53A

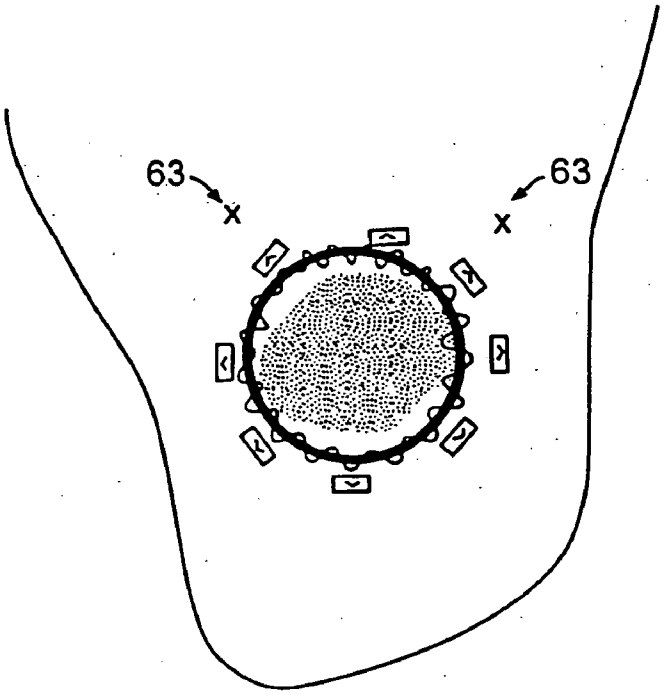


FIG. 53B

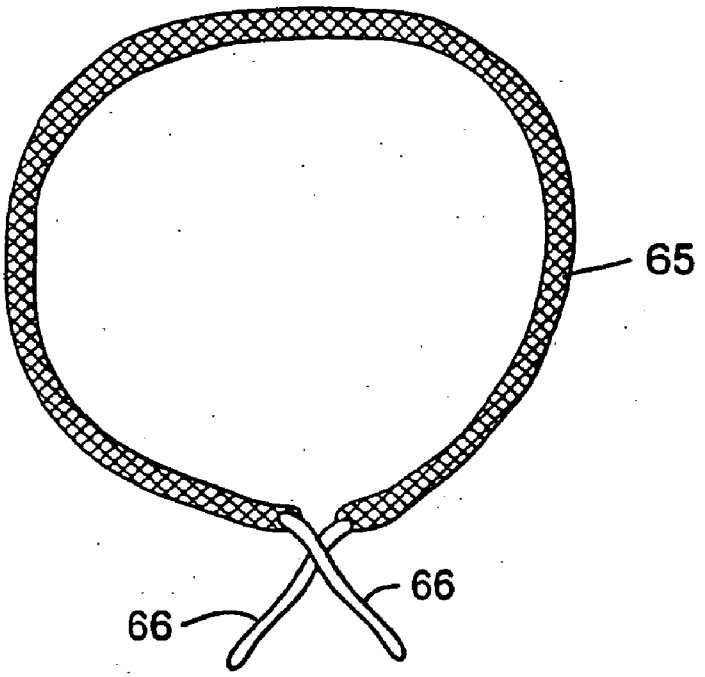


FIG. 54

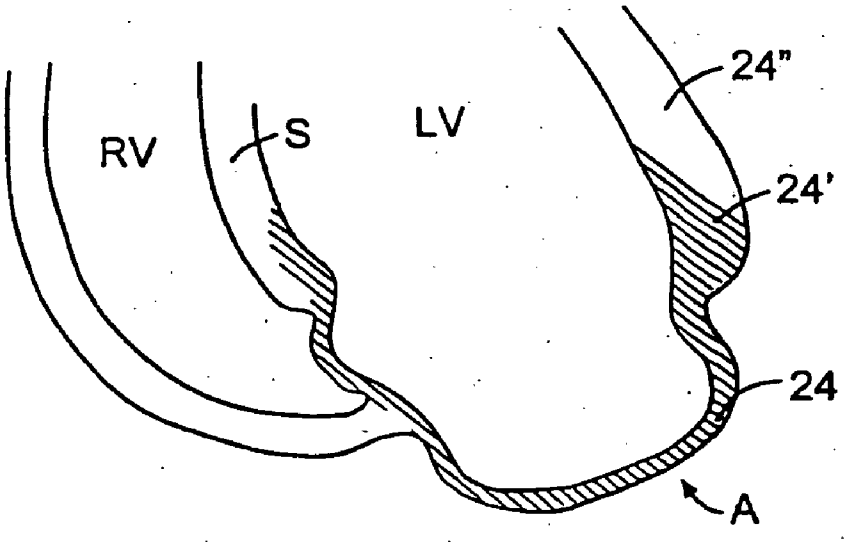


FIG. 55

METHODS AND DEVICES FOR IMPROVING CARDIAC FUNCTION IN HEARTS

CROSS REFERENCE TO RELATED APPLICATIONS

[0001] This application is a continuation-in-part of U.S. patent application Ser. No. 09/124,286 filed Jul. 29, 1998, now pending, which is a continuation-in-part of U.S. patent application Ser. No. 08/933,456 filed Sep. 18, 1997, now pending, which is a continuation-in-part of U.S. patent application Ser. No. 08/778,277, filed Jan. 2, 1997, now pending.

FIELD OF THE INVENTION

[0002] The present invention pertains to the field of apparatus for treatment of a failing heart. In particular, the apparatus and its related methods of the present invention is directed toward reducing the wall stress in the failing heart. The present invention further includes methods and devices for improving cardiac function in hearts having discrete zones of infarcted tissue. Such methods and devices reduce the radius of curvature and/or alter the geometry or shape of the infarcted tissue and adjacent regions to thereby reduce wall stress on the heart and improve the heart's pumping performance.

BACKGROUND OF THE INVENTION

[0003] The syndrome of heart failure is a common course for the progression of many forms of heart disease. Heart failure may be considered to be the condition in which an abnormality of cardiac function is responsible for the inability of the heart to pump blood at a rate commensurate with the requirements of the metabolizing tissues, or can do so only at an abnormally elevated filling pressure. There are many specific disease processes that can lead to heart failure. Typically these processes result in dilatation of the left ventricular chamber. Etiologies that can lead to this form of failure include idiopathic, valvular, viral, and ischemic cardiomyopathies.

[0004] The process of ventricular dilatation is generally the result of chronic volume overload or specific damage to the myocardium. In a normal heart that is exposed to long term increased cardiac output requirements, for example, that of an athlete, there is an adaptive process of slight ventricular dilation and muscle myocyte hypertrophy. In this way, the heart fully compensates for the increased cardiac output requirements. With damage to the myocardium or chronic volume overload, however, there are increased requirements put on the contracting myocardium to such a level that this compensated state is never achieved and the heart continues to dilate.

[0005] The basic problem with a large dilated left ventricle is that there is a significant increase in wall tension and/or stress both during diastolic filling and during systolic contraction. In a normal heart, the adaptation of muscle hypertrophy (thickening) and ventricular dilatation maintain a fairly constant wall tension for systolic contraction. However, in a failing heart, the ongoing dilatation is greater than the hypertrophy and the result is a rising wall tension requirement for systolic contraction. This is felt to be an ongoing insult to the muscle myocyte resulting in further-muscle damage. The increase in wall stress is also true for diastolic filling. Additionally, because of the lack of cardiac output, there is generally a rise in ventricular filling pressure from several physiologic mechanisms. Moreover, in diastole there is both a diameter increase and a pressure increase over normal, both contributing to higher wall stress levels. The increase in diastolic wall stress is felt to be the primary contributor to ongoing dilatation of the chamber.

[0006] Prior treatments for heart failure associated with such dilatation fall into three general categories. The first being pharmacological, for example, diuretics and ACE inhibitors. The second being assist systems, for example, pumps. Finally, surgical treatments have been experimented with, which are described in more detail below.

[0007] With respect to pharmacological treatments, diuretics have been used to reduce the workload of the heart by reducing blood volume and preload. Clinically; preload is defined in several ways including left ventricular end diastolic pressure (LVEDP), or indirectly by left ventricular end diastolic volume (LVEDV). Physiologically, the preferred definition is the length of stretch of the sarcomere at end diastole. Diuretics reduce extra cellular fluid which builds in congestive heart failure patients increasing preload conditions. Nitrates, arteriolar vasodilators, angiotensin converting enzyme (ACE) inhibitors have been used to treat heart failure through the reduction of cardiac workload by reducing afterload. Afterload may be defined as the tension or stress required in the wall of the ventricle during ejection. Inotropes function to increase cardiac output by increasing the force and speed of cardiac muscle contraction. These drug therapies offer some beneficial effects but do not stop the progression of the disease.

[0008] Assist devices include mechanical pumps. Mechanical pumps reduce the load on the heart by performing all or part of the pumping function normally done by the heart. Currently, mechanical pumps are used to sustain the patient while a donor heart for transplantation becomes available for the patient.

[0009] There are at least four surgical procedures for treatment of heart failure associated with dilatation: 1) heart transplantation; 2) dynamic cardiomyoplasty; 3) the Batista partial left ventriculectomy; and 4) the Jatene and Dor procedures for ischemic cardiomyopathy, discussed in more detail below. Heart transplantation has serious limitations including restricted availability of organs and adverse effects of immunosuppressive therapies required following heart transplantation. Cardiomyoplasty involves wrapping the heart with skeletal muscle and electrically stimulating the muscle to contract synchronously with the heart in order to help the pumping function of the heart. The Batista partial left ventriculectomy surgically remodels the left ventricle by removing a segment of the muscular wall. This procedure reduces the diameter of the dilated heart, which in turn reduces the loading of the heart. However, this extremely invasive procedure reduces muscle mass of the heart.

[0010] One form of heart failure, ischemic cardiomyopathy, results from the formation of one or more zones of ischemia, or infarction, of the myocardium. Infarction occurs when blood supply to the heart tissue has been obstructed resulting in a region of tissue that loses its ability to contract (referred to as infarcted tissue). The presence of infarcted tissue may lead to three conditions in the heart causing cardiac malfunction. These conditions are ventricu-

lar aneurysms (ventricular dyskinesia), non-aneurysmal ischemic or infarcted myocardium (ventricular akinesia), and mitral regurgitation.

[0011] Ventricular aneurysms typically result from a transmural myocardial infarction, frequently due to the occlusion of the left anterior descending artery (LAD). This results in a transmural infarcted region of the apical portion of the left ventricle and anterior septal. A ventricular aneurysm is formed when the infarction weakens the heart wall to such an extent that the tissue stretches and thins, causing the left ventricular wall to expand during systole (dyskinesia). FIG. **55** illustrates a ventricular aneurysm A occurring in the apical region of left ventricle LV. As shown by the shaded region in FIG. **55**, aneurysm A includes infarcted tissue **24** that results in a reduced wall thickness when compared to adjacent non-infarcted wall regions, as shown by the unshaded regions in FIG. **55**. FIG. **55** also shows the septal wall S partially infarcted, again shown by the shaded region. The ventricular aneurysm also may be dyskinetic, meaning that when the ventricle contracts, the aneurysm further dilates, or bulges, outward. The infarcted region of the septal wall S also may be particularly dyskinetic, especially in the case of the infarcted tissue having progressed to an aneurysm.

[0012] The bulge resulting from an aneurysm can have several serious effects on the heart and its performance that can lead to in both morbidity and mortality. For example, because the bulge creates a geometric abnormality as well as a region of non-contracting tissue, thrombosis is more likely to occur in that region. Thrombosis is the formation of a blood clot, or thrombus, that can cause other medical complications, such as a stroke. An ischemic stroke is a blockage of blood flow to the brain that occurs when the thrombus breaks free and is ejected out of the ventricle.

[0013] Another serious effect this bulging can have is the denigration of the heart's pumping function. The aneurysmal bulge creates problems with pumping function in at least three ways. First, the infarcted tissue does not contribute to the pumping of the ventricle because it does not contract (akinesia). To account for this loss of pumping, remaining portions of the ventricle wall may contract more to maintain cardiac output. If the infarcted region thins and progresses to an aneurysm (dyskinesia), this effect is further exacerbated by the aneurysm expanding with a portion of the blood from the ventricular contraction. This further increases the contractile requirement of the remaining functional myocardium.

[0014] Second, the aneurysmal bulge alters the geometry of the entire ventricular chamber. Thus the ventricle develops a larger radius of curvature, which directly applies more tension to the heart wall, as characterized by LaPlace's law.

[0015] Third, over time, the above two conditions lead the functional muscle of the ventricle to work harder than normal. This can lead to continued dilatation of the ventricle, increasing tension in the walls of the heart, with increased myocardial oxygen requirement and further progressing heart failure.

[0016] Non-aneurysmal ischemic or infarcted myocardium (akinesia) occurs when a major coronary artery is occluded and results in infarction in the myocardial tissue, but without a bulging aneurysm. In a manner similar to an aneurysm, the akinetic ischemic or infarcted zone ceases to participate in the ventricular contraction. This results in the functioning, contractile myocardium needing to contract more to make up for the lack of contraction of the akinetic zone. Typically, the result is the entire ventricle increasing in size, which increases wall stress. Again, since the functioning myocardium must work harder, continuing progression of heart failure can occur.

[0017] Mitral regurgitation also may result from infarcted tissue, depending on the region of the ventricle that has become infarcted or aneurysmal and any subsequent overall ventricular dilation. Mitral regurgitation is a condition whereby blood leaks through the mitral valve due to an improper positioning of the valve structures that causes it not to close entirely. If the infarcted or aneurysmal region is located in the vicinity of the mitral valve, geometric abnormalities may cause the mitral valve to alter its normal position and dimension, and may lead to annular dilatation and the development of mitral regurgitation.

[0018] Typical treatments of infarcted tissue, and ventricular aneurysms in particular, include a variety of open surgical procedures. In the case of a ventricular aneurysm, traditionally, a "linear" aneurysmectomy is performed. This procedure involves the removal of aneurysmal portions of the anterior wall along with any thrombus that may exist. FIG. **41** illustrates the result of a conventional surgical method when an aneurysm occurs in the distal left ventricle. According to this method, the region of aneurysmal scar tissue that extends through the entire thickness of the chamber wall (transmural infarction) is removed by incision and the remaining border zone regions **24'** (i.e., regions where infarcted tissue meets non-infarcted muscle) are sewn together with a suture **27**. In a linear aneurysm repair procedure, the ventricular septal wall S that is infarcted is left untouched. Additionally, the septal wall generally remains untouched because simple excision and suturing does not involve excluding or cutting the septal wall. Usually, only those wall portions having infarcted tissue through their thickness (transmural infarcted) are removed while the portions having infarcted tissue only on an inner wall (endocardial infarcted) are left in place. The term border zone refers to this region of endocardial infarction. This surgical procedure results in some infarcted tissue regions remaining in the heart chamber, particularly any infarcted tissue in the septal wall. The effects of the remaining non-contractile tissue stresses the remaining contractile tissue because this contractile tissue must "make up" for the non-contracting and often dyskinetic tissue. Over time, these effects can continue to lead to progression of heart failure.

[0019] These procedures have to be performed with the patient on cardiopulmonary bypass. The heart also may be stopped in order to perform the surgery. Any thrombus inside the ventricle is removed. Clinical results of this traditional surgical procedure have been mixed with respect to improvement in cardiac function.

[0020] Newer surgical approaches include the "Dor" and "Jatene" procedures. In the "Dor" procedure, the aneurysm is removed and an endocardial patch is placed to cover the dyskinetic septal wall portion of the aneurysm. In this manner, at least the portion of stroke volume "lost" to dyskinesia is restored. In the "Jatene" technique, a purse string suture is placed at the base of the aneurysm. The

infarcted septal wall is circumferentially reduced by inbrication with sutures. The result is that most of the aneurysmal tissue is excluded from the ventricle. These procedures address the infarcted septal wall, generally left untouched in the traditional linear aneurysmectomy, by either exclusion or by the use of a surgical patch. These newer techniques are also used in cases of non-aneurysmal infarctions (akinesia). In these cases, the exclusion or elimination of the infarcted region reduces the size and therefore the radius of the chamber, thereby lowering wall stress.

[0021] These various described techniques for treating infarcted and aneurysmal tissue regions in the heart wall suffer from limitations and drawbacks. For instance, many of the surgical techniques involve invasive incisions in the heart wall which can be traumatic and risky to patients. Also, while these procedures attempt to improve cardiac function by removal of the aneurysm or infarcted tissue, they only minimally reduce the wall stress of the remaining contractile ventricle. Furthermore, patients typically undergo cardiopulmonary bypass and/or their heart is stopped during many of these surgeries.

SUMMARY OF THE INVENTION

[0022] The advantages and purpose of the invention will be set forth in part in the description which follows, and in part will be obvious from the description, or may be learned by practice of the invention. The advantages and purpose of the invention will be realized and attained by means of the elements and combinations particularly pointed out in the appended claims.

[0023] Due to the drawbacks and limitations of the previous techniques for treating dilated, infarcted, and aneurysmal tissue in hearts, there exists a need for alternative methods and devices that are less invasive, pose less risk to the patient, and are likely to prove more clinically effective. The present invention provides improvements in these areas over the existing techniques.

[0024] One aspect of the present invention pertains to a non-pharmacological, passive apparatus and method for the treatment of a failing heart due to dilatation. The device is configured to reduce the tension in the heart wall. It is believed to reverse, stop or slow the disease process of a failing heart as it reduces the energy consumption of the failing heart, decreases isovolumetric contraction, increases isotonic contraction (sarcomere shortening), which in turn increases stroke volume. The device reduces wall tension during-diastole and systole.

[0025] These apparatus of the present invention which reduce heart wall stress by changing chamber wall geometry can be referred to as "splints". Splints can be grouped as either "full cycle splints," which engage the heart to produce a chamber shape change throughout the cardiac cycle, or "restrictive splints," which do not engage the heart wall at end systole to produce a chamber shape change.

[0026] In one embodiment, the apparatus includes a tension member for drawing at least two walls of the heart chamber toward each other to reduce the radius or area of the heart chamber in at least one cross sectional plane. The tension member has anchoring members disposed at opposite ends for engagement with the heart or chamber wall.

[0027] In another embodiment, the apparatus includes a compression member for drawing at least two walls of a heart chamber toward each other. In one embodiment, the compression member includes a balloon. In another embodiment of the apparatus, a frame is provided for supporting the compression member.

[0028] Yet another embodiment of the invention includes a clamp having two ends biased toward one another for drawing at least two walls of a heart chamber toward each other. The clamp includes at least two ends having atraumatic anchoring members disposed thereon for engagement with the heart or chamber wall.

[0029] In yet another embodiment, a heart wall tension reduction apparatus is provided which includes a first tension member having two oppositely disposed ends and first and second elongate anchor members. A second tension member can be provided. One of the elongate anchors may be substituted for by two smaller anchors.

[0030] In an alternate embodiment of the heart wall tension reduction apparatus, an elongate compression member can be provided. First and second elongate lever members preferably extend from opposite ends of the compression member. A tension member extends between the first and second lever members.

[0031] The compression member of the above embodiment can be disposed exterior to, or internally of the heart. The tension member extends through the chamber or chambers to bias the lever members toward the heart.

[0032] In yet another embodiment of a heart wall tension reduction apparatus in accordance with the present invention, a rigid elongate frame member is provided. The frame member can extend through one or more chambers of the heart. One or more cantilever members can be disposed at opposite ends of the frame member. Each cantilever member includes at least one atraumatic pad disposed thereon. The atraumatic pads disposed at opposite ends of the frame member can be biased toward each other to compress the heart chamber.

[0033] One method of placing a heart wall tension apparatus or splint on a human heart includes the step of extending a hollow needle through at least one chamber of the heart such that each end of the needle is external to the chamber. A flexible leader is connected to a first end of a tension member. A second end of the tension member is connected to an atraumatic pad. The leader is advanced through the needle from one end of the needle to the other. The leader is further advanced until the second end of the tension member is proximate the heart and the first end of the tension member is external to the heart. A second atraumatic pad is connected to the first end of the tension member such that the first and second atraumatic pads engage the heart.

[0034] Yet another method of placing a heart wall tension apparatus on a heart includes the step of extending a needle having a flexible tension member releasably connected thereto through at least one chamber of the heart such that opposite ends of the tension member are external to the chamber and exposed on opposite sides of the chamber. The needle is removed from the tension member. Then first and second atraumatic pads are connected to the tension member at opposite ends of the tension member.

[0035] In the treatment of heart failure due to infarcted tissue, possibly including an aneurysm as well, another

aspect of the invention involves placing the splint relative to the infarcted or aneurysmal zone, and, in a preferred embodiment, diametrically across the infarcted or aneurysmal zone, to decrease the stress on the infarcted tissue and adjacent border zone tissue. An alternative to diametric placement of the splint includes placing one atraumatic anchor member of the splint at the center of the infarcted or aneurysmal region, extending the splint across the entire heart chamber, and placing the second atraumatic anchor member on the opposite chamber wall. In the case of infarcted or aneurysmal tissue in the vicinity of the mitral valve, an aspect of the present invention includes a method of placing the splint adjacent but below the mitral valve to draw the papillary muscles together or the walls of the valve seat together. It is also envisioned to use the splint both as the sole device for treating infarcted tissue and aneurysms or in combination with the surgical techniques described earlier.

[0036] An external splint, using a compression member, also may be used to treat a heart having infarcted or aneurysmal tissue. The compression member is placed entirely exterior to the heart and positioned so as to result in similar effects as discussed above with reference to the splint.

[0037] Other inventive methods and devices to treat infarcted tissue and aneurysms include a variety of patching and suturing methods and related devices. Each of these methods and related devices reduces the radius of curvature of the infarcted wall region and adjacent regions and contains the infarcted region to stop further progression.

[0038] A further aspect of the invention involves the identification of aneurysmal and infarcted regions using any one or more of a variety of devices and methods. These devices and methods, which will be described more specifically herein, include a bipolar electrode, liquid dye injection and tracing, fiber optics, MRI, and ultrasound. These devices can be used to distinguish between healthy and infarcted heart tissue.

[0039] In accordance with the purposes of the invention as embodied and broadly described herein, methods and related devices for treating a heart having infarcted tissue in one of its chambers are disclosed. In a preferred embodiment of the invention, a method for treating a heart having a zone of infarcted tissue in its chamber includes deforming a wall of the chamber that includes the infarcted tissue such that a radius of curvature of the wall is reduced.

[0040] In another preferred embodiment of the present invention, the method involves providing at least one tension member having two ends and an anchor on each end. The tension member is positioned transverse to the chamber to reduce the radius of curvature of the wall of the chamber that includes the infarcted tissue and/or to draw the walls containing the infarcted tissue together.

[0041] In another preferred embodiment, the present invention involves positioning a tension member having anchors on each of its ends transverse to the heart chamber so that the infarcted tissue is drawn toward an interior of the heart chamber. The anchors are placed exterior to the chamber.

[0042] In yet another preferred embodiment, the present invention includes positioning a compression member having a first end and a second end, each having anchor members around an exterior of the heart. The compression member is positioned so as to surround the chamber with infarcted tissue and to reduce the radius of curvature of the portion of the heart wall that has the infarcted tissue.

[0043] In accordance with another preferred embodiment, a method of treating a heart having infarcted tissue in one of its chambers involves epicardial suturing around the perimeter of a region of infarcted tissue and pulling free ends of the suture to draw the infarcted tissue region together. The suture is then secured to hold the infarcted tissue together. This suture also may be employed in combination with a myocardial patch or a substantially rigid enclosure member, both of which represent other preferred embodiments of the present invention.

[0044] In accordance with another preferred embodiment of the present invention, a method of treating a heart having infarcted tissue in one of its chambers involves positioning an enclosure member around a zone of infarcted tissue. During the positioning, the enclosure member has a first configuration. After positioning, the enclosure member is then secured to a wall of the heart and the enclosure member reconfigures to a second configuration. Upon reconfiguration to the second configuration, the radius of curvature of the portion of the heart wall including the infarcted tissue reduces.

[0045] In accordance with yet another preferred embodiment of the present invention, an apparatus for treating a heart having a zone of infarcted tissue in one of its chambers is provided. The apparatus includes an enclosure member adapted to assume a first configuration during placement of the enclosure member around an infarcted tissue zone. The enclosure member further is adapted to assume a second configuration after securing the enclosure member to a heart wall surrounding the chamber. The second configuration draws the infarcted tissue toward a center of the enclosure member and reduces the radius of curvature of the heart wall.

[0046] In accordance with another preferred embodiment of the present invention, a device for treating a heart having infarcted tissue in one of its chambers is provided. The device includes a patch adapted to be attached to the heart, with a substantially elongated member secured to the patch. When the patch is placed over the infarcted or aneurysmal tissue region, the elongated member tends to push the infarcted tissue region toward an interior of the heart chamber.

[0047] In accordance with yet another preferred embodiment of the present invention, a plurality of sutures are attached at one end to points on a chamber wall proximate to an infarcted tissue region and the sutures are extended up through a space defined by an enclosure member to draw the infarcted tissue together and through the enclosure member. The other ends of the sutures are then attached to points on the wall of the chamber to hold the tissue in place.

[0048] It is to be understood that both the foregoing general description and the following detailed description are exemplary and explanatory only and are not restrictive of the invention, as claimed.

BRIEF DESCRIPTION OF THE DRAWINGS

[0049] The accompanying drawings, which are incorporated in and constitute a part of this specification, illustrate

several embodiments of the invention and, together with the description, serve to explain the principles of the invention. In the drawings,

[0050] FIG. **1** is a transverse cross-section of the left and right ventricles of a human heart showing the placement of a splint in accordance with the present invention;

[0051] FIG. **2** is a transverse cross-section of the left and right ventricles of a human heart showing the placement of a balloon device in accordance with the present invention;

[0052] FIG. **3** is a transverse cross-section of the left and right ventricles of a human heart showing the placement of an external compression frame structure in accordance with the present invention;

[0053] FIG. **4** is a transverse cross-section of the left and right ventricles of a human heart showing a clamp in accordance with the present invention;

[0054] FIG. **5** is a transverse cross-section of the left and right ventricles of a human heart showing a three tension member version of the splint of FIG. **1**;

[0055] FIG. **6** is a transverse cross-section of the left and right ventricles of a human heart showing a two tension member version of the splint shown in FIG. **1**;

[0056] FIG. **7** is a vertical cross-sectional view of the left ventricle of a human heart showing an alternate version of the splint in accordance with the present invention;

[0057] FIG. **8** is an end of the splint shown in FIG. **7**;

[0058] FIG. **9** is a vertical cross-sectional view of a chamber of a human heart showing another alternative embodiment of the splint in accordance with the present invention;

[0059] FIG. **10** is a vertical cross-section of a chamber of a human heart showing another alternative configuration of splints in accordance with the present invention;

[0060] FIG. **11** is a vertical cross-sectional view of a chamber of a human heart showing another embodiment of a splint in accordance with the present invention;

[0061] FIG. **12** is a vertical cross-sectional view of a chamber of a human heart showing another embodiment of the splint in accordance with the present invention;

[0062] FIG. **13** is a vertical cross-sectional view of a chamber of a human heart showing a compression member version of the splint in accordance with the present invention;

[0063] FIG. **14** is a vertical cross-sectional view of a chamber of a human heart showing another version of the splint shown in FIG. **13**;

[0064] FIG. **15** is a vertical cross-sectional view of a chamber of a human heart showing a frame member version of the splint in accordance with the present invention;

[0065] FIG. **16** is an end view of the splint of FIG. **15**;

[0066] FIG. **17** is a vertical cross-section of the left ventricle and atrium, the left ventricle having aneurysmal scar tissue;

[0067] FIG. **18** is a vertical cross-section of the heart of FIG. **17** showing the splint of FIG. **1** drawing the aneurysmal scar tissue toward the opposite wall of the left ventricle;

[0068] FIG. **19** is a vertical cross-section of the left ventricle and atrium of a human heart showing a version of the splint of FIG. **1** having an elongate anchor bar;

[0069] FIG. **20** is a side view of an undeployed hinged anchor member;

[0070] FIG. **21** is a side view of a deployed hinged anchor member of FIG. **10**;

[0071] FIG. **22** is a cross-sectional view of an captured ball anchor member;

[0072] FIG. **23** is a perspective view of a cross bar anchor member;

[0073] FIG. **24** is a cross sectional view of an alternate anchor pad;

[0074] FIG. **25** is a cross sectional view of an alternate anchor pad;

[0075] FIG. **26** is a perspective view of yet another alternate embodiment of an anchor pad including an anchor pad loosening device;

[0076] FIG. **27** is a perspective view of a tension member clip;

[0077] FIG. **28** is a cross sectional view of an alternate embodiment of a tension member clip;

[0078] FIG. **29** is a cross sectional view of a heart including a tension member having a heat set end;

[0079] FIG. **30** is a cross sectional view of the pad including an envelope;

[0080] FIG. **31** shows the envelope of FIG. **30**;

[0081] FIG. **32** is a side view of a multifilament twisted cable;

[0082] FIG. **33** is a cross sectional of the cable of FIG. **32**;

[0083] FIG. **34** is a side of a multifilament braided tension member;

[0084] FIG. **35** is a schematic generally horizontal cross sectional view of the heart showing preferred tension member alignments;

[0085] FIG. **36** is a idealized cylindrical model of a left ventricle of a human heart;

[0086] FIG. **37** is a splinted model of the left ventricle of FIG. **14**;

[0087] FIG. **38** is a transverse cross-sectional view of FIG. **15** showing various modeling parameters;

[0088] FIG. **39** is a transverse cross-section of the splinted left ventricle of FIG. **15** showing a hypothetical force distribution;

[0089] FIG. **40** is a second transverse cross-sectional view of the model left ventricle of FIG. **15** showing a hypothetical force distribution;

[0090] FIG. **41** is a transverse, partial cross-section of left and right ventricles showing a traditional surgical method of treating infarcted tissue regions;

[0091] FIG. **42** is a transverse, partial cross-sectional view of left and right ventricles with an infarcted or aneurysmal region in the apical portion of the left ventricle and a splint according to an embodiment of the invention placed diametrically across the region;

[0092] FIGS. **43***a*-**43***b* are long axis cross-sectional views of left and right ventricles showing a region of infarcted tissue in a portion of the basal left ventricle and a splint according to an embodiment of the invention placed across the infarcted region;

[0093] FIGS. **43***c*-**43***d* are short axis cross-sectional views of the heart in FIGS. **43***a*-**43***b* shown from the perspective of lines c-c and d-d, respectively;

[0094] FIGS. **44***a*-**44***b* are short axis cross-sectional views of the left ventricle having an aneurysm and a placement of a splint according to an embodiment of the present invention with respect to the aneurysm to treat the heart;

[0095] FIG. **45** is a short axis cross-sectional view of the right and left ventricles having an infarcted region like that shown in FIG. **43***c* and an external splint device according to an embodiment of the present invention placed to treat the infarction;

[0096] FIGS. **46***a*-**46***c* show transverse, partial cross-sectional views of a left ventricle having an infarcted region, illustrating the combined inventive method of surgical removal of the infarcted tissue and placement of a splint according to an embodiment of the present invention;

[0097] FIG. **47** is a short axis cross-sectional view of the left and right ventricles including a view of the mitral valve with an infarcted or aneurysmal region in a portion of the basal left ventricle and a splint according to an embodiment of the invention placed in the vicinity of the mitral valve;

[0098] FIGS. **48***a*-**48***b* are short axis cross-sectional views of the left and right ventricles and a view of the mitral valve with an aneurysmal region in a portion of the basal left ventricle and an external device according to an embodiment of the invention placed in the vicinity of the mitral valve and aneurysm;

[0099] FIGS. **49***a*-**49***b* are transverse cross-sections of a left ventricle having an aneurysmal region and the placement of a staked patch according to an embodiment of the present invention;

[0100] FIGS. **50***a*-**50***b* are planar views of an infarcted or aneurysmal tissue region with a purse-string suture according to an embodiment of the present invention;

[0101] FIGS. **51***a*-**51***c* are planar views of an infarcted or aneurysmal tissue region and a purse-string suture and enclosure member according to an embodiment of the present invention;

[0102] FIGS. **52***a*-**52***c* are planar exterior views of the heart with an infarcted region of tissue in the left ventricle and an enclosure member according to an embodiment of the invention, showing one configuration during application of the member and a second configuration after application of the member;

[0103] FIG. **53***a*-**53***b* are planar views of an infarcted or aneurysmal tissue region and placement of a tie enclosure according to an embodiment of the present invention;

[0104] FIG. **54** is a planar view of yet another embodiment of an enclosure member according to an embodiment of the present invention; and

[0105] FIG. **55** is a transverse, partial cross-section of left and right ventricles with an aneurysmal region located at an apical portion of the left ventricle and infarcted tissue along the septal wall.

DESCRIPTION OF THE PREFERRED EMBODIMENTS

[0106] The various aspects of the invention to be discussed herein generally pertain to devices and methods for treating heart conditions, including, for example, dilatation and infarction, including infarction causing aneurysms. For the purposes of providing clarity and consistency throughout the remaining description of the invention, the following terms have the general definitions set forth below.

[0107] "infarction" or "infarcted": refers to myocardium (also described as tissue or muscle) that has lost its ability to contract as a result of cellular necrosis, this term can include, for example, aneurysmal tissue and scar tissue that replaces the necrotic cellular muscle tissue;

[0108] "aneurysm" or "aneurysmal": refers to infarcted myocardium that is dyskinetic with respect to surrounding portions of the myocardium;

[0109] "contractile": refers to myocardium that is not infarcted and has generally retained contractile potential, though this muscle tissue may not be fully contracting given other conditions, e.g. too much wall stress; and

[0110] "border zone": refers to chamber wall that has a region of infarcted tissue and a region of contractile tissue through its thickness.

[0111] These definitions are generally consistent with the accepted definitions recognized by those skilled in the art.

[0112] The devices of the present invention operate passively in that, once placed in the heart, they do not require an active stimulus either mechanical, electrical, or otherwise, to function. The devices alter the shape or geometry of the heart, both locally and globally, and increase the heart's efficiency by their placement with respect to the heart. That is, the heart experiences an increased pumping efficiency through an alteration in its shape or geometry and concomitant reduction in stress on the heart walls.

[0113] The inventive devices and methods offer numerous advantages over the existing treatments for various heart conditions. The devices are relatively easy to manufacture and use, and the related surgical techniques for their implementation do not require the invasive procedures of current surgical techniques. For instance, the surgical technique does not necessarily require removing portions of the heart tissue, opening the heart chamber, or stopping the heart. For these reasons, the surgical techniques of the present invention are also less risky to the patient than other techniques.

[0114] The devices and methods of the present invention used to treat infarcted tissue and aneurysms also are likely to be more effective than prior devices. As will be described, the inventive devices alter the shape or geometry of the

chamber, either globally or locally, and reduce the radius of curvature of the chamber wall, resulting in lower stresses in the heart wall. Moreover, with many of the inventive devices there is no need to open the heart chamber to deploy the device, even when the device is deployed on the septal wall. These methods and devices also could be used in conjunction with coronary artery bypass grafting (CABG). In CABG surgery, the use of the inventive methods and related devices allow for quickly reducing stress on the myocardium, which may save "stunned" tissue, i.e., tissue that is being starved of nutrients carried with the blood flow, that otherwise may not be recoverable after a certain time period. Also, the inventive methods and device may hinder further progression or dilation of scarred, non-contractile tissue.

[0115] The disclosed inventive methods and related devices involve geometric reshaping of the heart. In certain aspects of the inventive methods and related devices, substantially the entire chamber geometry is altered to return the heart to a more normal configuration. FIGS. **36** through **40**, which will be described in further detail later, show a model of this geometric reshaping, which includes a reduction in radius of curvature of the chamber walls. Prior to reshaping the chamber geometry, the heart walls experience high stress due to a combination of both the relatively large increased diameter of the chamber and the thinning of the chamber wall. Geometric reshaping according to the present invention reduces the stress in the walls of the heart chamber to increase the heart's pumping efficiency, as well as to stop further dilatation of the heart.

[0116] Other aspects of the inventive methods and devices involve geometric reshaping a particular area of the chamber and/or reducing the radius of curvature of the chamber wall in that area. When portions of the heart wall form a bulge due to an aneurysm, the radius of much of the heart chamber changes. This increases stress on the heart walls. Additionally, the healthy regions of the heart work harder to pump in order to make up pumping volume due to lost contractility in the infarcted tissue region. Together, these effects limit the pumping effectiveness of the heart and can contribute to further degradation of the heart. Geometrically reshaping the area of the aneurysm by, for example, reducing the radius of curvature of the wall, lowers stress on the wall regions in that vicinity and improves pumping function. In addition, the geometric reshaping permits the scar tissue to heal in a more organized fashion and reduces progression of the scar tissue into other areas and further aneurysmal bulging.

[0117] Although many of the methods and devices are discussed below in connection with their use in the left ventricle of the heart, these methods and devices may be used in other chambers of the heart for similar purposes. One of ordinary skill in the art would understand that the use of the devices and methods described herein would be substantially the same if employed in other chambers of the heart. The left ventricle has been selected for illustrative purposes because a large number of the disorders that the present invention treats occur in the left ventricle.

[0118] Reference will now be made in detail to the present preferred embodiments of the invention, examples of which are illustrated in the accompanying drawings. Wherever possible, the same reference numbers will be used throughout the drawings to refer to the same or like parts.

[0119] FIG. **1** shows a transverse cross-section of a left ventricle **10** and a right ventricle **12** of a human heart **14**. Extending through the left ventricle is a splint **16** including a tension member **18** and oppositely disposed anchors **20**. Splint **16**, as shown in FIG. **1**, has been positioned to draw opposite walls of left ventricle **10** toward each other to reduce the "radius" of the left ventricular cross-section or the cross-sectional area thereof to reduce left ventricular wall stresses. It should be understood that although the splint **16** and the alternative devices disclosed herein are described in relation to the left ventricle of a human heart, these devices could also be used to reduce the radius or cross-sectional area of the other chambers of a human heart in transverse or vertical directions, or at an angle between the transverse and vertical.

[0120] Those apparatus of the present invention which reduce heart wall stress by changing chamber wall geometry can be referred to as "splints". "Full cycle splints" engage the heart to produce a chamber shape change throughout the cardiac cycle. "Restrictive splints" do not engage the heart wall at end systole to produce a chamber shape change.

[0121] FIG. **2** discloses an alternate embodiment of the present invention, wherein a balloon **200** is deployed adjacent the left ventricle. The size and degree of inflation of the balloon can be varied to reduce the radius or cross-sectional area of left ventricle **10** of heart **14**.

[0122] FIG. **3** shows yet another alternative embodiment of the present invention deployed with respect to left ventricle **10** of human heart **14**. Here a compression frame structure **300** is engaged with heart **14** at atraumatic anchor pads **310**. A compression member **312** having an atraumatic surface **314** presses against a wall of left ventricle **10** to reduce the radius or cross-sectional area thereof.

[0123] FIG. **4** is a transverse cross-sectional view of human heart **14** showing yet another embodiment of the present invention. In this case a clamp **400** having atraumatic anchor pads **410** biased toward each other is shown disposed on a wall of left ventricle **10**. Here the radius or cross-sectional area of left ventricle **10** is reduced by clamping off the portion of the wall between pads **410**. Pads **410** can be biased toward each other and/or can be held together by a locking device.

[0124] Each of the various embodiments of the present invention disclosed in FIGS. **1-4** can be made from materials which can remain implanted in the human body indefinitely. Such biocompatible materials are well-known to those skilled in the art of clinical medical devices.

[0125] FIG. **5** shows an alternate embodiment of the splint of FIG. **1** referred to in FIG. **5** by the numeral **116**. The embodiment **116** shown in FIG. **5** includes three tension members **118** as opposed to a single tension member **18** as shown in FIG. **1**. FIG. **6** shows-yet another embodiment of the splint **216** having four tension members **218**. It is anticipated that in some patients, the disease process of the failing heart may be so advanced that three, four or more tension members may be desirable to reduce the heart wall stresses more substantially than possible with a single tension member as shown in FIG. **1**.

[0126] FIG. **7** is a partial vertical cross-section of human heart **14** showing left ventricle **10**. In FIG. **7**, another splint embodiment **316** is shown having a tension member **318** extending through left ventricle **10**. On opposite ends of

tension member **318** are disposed elongate anchors or pads **320**. FIG. **8** is an end view of tension member **318** showing elongate anchor **320**.

[**0127**] FIG. **9** shows another embodiment of a splint **416** disposed in a partial vertical cross-section of human heart **14**. Splint **416** includes two elongate anchors or pads **420** similar to those shown in FIGS. **7** and **8**. In FIG. **9**, however, two tension members **418** extend through left ventricle **10** to interconnect anchors **420** on opposite sides of heart **14**.

[**0128**] FIG. **10** is a vertical cross section of heart **14** showing left ventricle **10**. In this case, two splints **16** are disposed through left ventricle **10** and vertically spaced from each other to resemble the configuration of FIG. **9**.

[**0129**] FIG. **11** is a vertical cross-sectional view of the left ventricle of heart **14**. Two alternate embodiment splints **516** are shown extending through left ventricle **10**. Each splint **516** includes two tension members **518** interconnecting two anchors or pads **520**.

[**0130**] FIG. **12** is yet another vertical cross-sectional view of left ventricle **10** of heart **14**. An alternate embodiment **616** of the splint is shown extending through left ventricle **10**. Splint **616** includes an elongate anchor pad **620** and two shorter anchors or pads **621**. Splint **616** includes two tension members **618**. Each tension member **618** extends between anchors **620** and respective anchors **621**.

[**0131**] FIG. **13** is a vertical cross sectional view of left ventricle **10** of heart **14**. A splint **50** is shown disposed on heart **14**. Splint **50** includes a compression member **52** shown extending through left ventricle **10**. Opposite ends of compression member **52** are disposed exterior to left ventricle **10**. Lever members **54** extend from each end of compression member **52** upwardly along the exterior surface of ventricle **10**. A tension member **56** extends between lever members **54** to bias lever members **54** toward heart **14** to compress chamber **10**. Compression member **52** should be substantially rigid, but lever members **54** and to some degree compression member **52** should be flexible enough to allow tension member **56** to bias lever members **54** toward heart **14**. Alternately, lever members **54** could be hinged to compression member **52** such that lever members **54** could pivot about the hinge when biased toward heart **14** by tension member **56**.

[**0132**] FIG. **14** shows an alternate embodiment **156** of the splint shown in FIG. **13**. In this case lever members **154** are longer than members **54** as compression member **152** of splint **150** has been disposed to the exterior of left ventricle **10**.

[**0133**] FIG. **15** is a vertical cross sectional view of left ventricle **10** of heart **14**. An alternate embodiment **250** of the splint is shown on heart **14**. A preferably relatively rigid frame member **256** extends through ventricle **10**. Disposed on opposite ends of frame **256** are cantilever member **254**. Disposed on cantilever members **254** are atraumatic pads **258**. Cantilever members **254** can be positioned along frame member **256** such that atraumatic pads **258** press against heart **14** to compress chamber **10**. FIG. **16** is an end view of frame member **256** showing cantilever members **254** and pads **258**.

[**0134**] It should be understood that each of the embodiments described above should be formed from suitable biocompatible materials known to those skilled in the art. The tension members can be formed from flexible or relatively more rigid material. The compression members and frame member should be formed from generally rigid material which may flex under load, but generally hold its shape.

[**0135**] As will be described in more detail herein, FIG. **17** is a partial vertical cross-section of human heart **14** showing left ventricle **10** and left atrium **22**. As shown in FIG. **17**, heart **14** includes a region of scar tissue **24** associated with an aneurysm or ischemia. As shown in FIG. **17**, the scar tissue **24** increases the radius or cross-sectional area of left ventricle **10** in the region affected by the scar tissue. Such an increase in the radius or cross-sectional area of the left ventricle will result in greater wall stresses on the walls of the left ventricle.

[**0136**] FIG. **18** is a vertical cross-sectional view of the heart **14** as shown in FIG. **17**, wherein a splint **16** has been placed to draw the scar tissue **24** toward an opposite wall of left ventricle **10**. As a consequence of placing splint **16**, the radius or cross-sectional area of the left ventricle affected by the scar tissue **24** is reduced. The reduction of this radius or cross-sectional area results in reduction in the wall stress in the left ventricular wall and thus improves heart pumping efficiency.

[**0137**] FIG. **19** is a vertical cross-sectional view of left ventricle **10** and left atrium **22** of heart **14** in which a splint **16** has been placed. As shown in FIG. **19**, splint **16** includes an alternative anchor **26**. The anchor **20** is preferably an elongate member having a length as shown in FIG. **19** substantially greater than its width (not shown). Anchor bar **26** might be used to reduce the radius or cross-sectional area of the left ventricle in an instance where there is generalized enlargement of left ventricle **10** such as in idiopathic dilated cardiomyopathy. In such an instance, bar anchor **26** can distribute forces more widely than anchor **20**.

[**0138**] FIGS. **20** and **21** are side views of a hinged anchor **28** which could be substituted for anchors **20** in undeployed and deployed positions respectively. Anchor **28** as shown in FIG. **20** includes two legs similar to bar anchor **26**. Hinged anchor **28** could include additional legs and the length of those legs could be varied to distribute the force over the surface of the heart wall. In addition there could be webbing between each of the legs to give anchor **28** an umbrella-like appearance. Preferably the webbing would be disposed on the surface of the legs which would be in contact with the heart wall.

[**0139**] FIG. **22** is a cross-sectional view of a capture ball anchor **30**. Capture ball anchor **30** can be used in place of anchor **20**. Capture ball anchor **30** includes a disk portion **32** to distribute the force of the anchor on the heart wall, and a recess **34** for receiving a ball **36** affixed to an end of tension member **18**. Disk **32** and recess **34** include a side groove which allows tension member **38** to be passed from an outside edge of disk **32** into recess **34**. Ball **36** can then be advanced into recess **34** by drawing tension member **18** through an opening **38** in recess **34** opposite disk **32**.

[**0140**] FIG. **23** is a perspective view of a cross bar anchor **40**. The cross bar anchor **40** can be used in place of anchors **20**. The anchor **40** preferably includes a disk or pad portion **42** having a cross bar **44** extending over an opening **46** in pad **42**. Tension member **18** can be extended through opening **46** and tied to cross bar **42** as shown.

[0141] FIG. **24** is a cross sectional view of an alternate embodiment of anchor pad **340** in accordance with the present invention. Anchor pad **340** preferably includes a disc shaped pad portion **342**. Disc-shaped pad portion **342** includes side **343**, which in use is disposed toward the heart. A conical aperture **348** having sloping sides **346** extends through pad **342**. Collet **344** is disposed within orifice **348**. A threaded portion **350** of collet **344** extends from orifice **348** opposite side **343**, nut **352** is threaded over threaded portion **350**. Lumen **345** extends through collet **344**. A tension member **354** is shown extending through lumen **345**. Lumen **345** has a diameter such that when nut **352** is not tightened on threaded portion **350**, tension member **354** can slide freely through lumen **345**. When nut **352** is tightened, it draws collet **344** away from side **343**. Collet **344** is then pinched between walls **346** of orifice **348**. When collet **344** is pinched, the size of lumen **345** is reduced such that tension member **354** can no longer move freely within lumen **345**, fixing the position of pad **340** on tension member **354**.

[0142] FIG. **25** is a cross sectional view of an alternate embodiment of an anchor pad **360** in accordance with the present invention. Anchor pad **360** includes a generally disc-shaped pad portion **362**. Pad **362** includes a side **363** which when the pad is in use, is disposed toward the heart. A tension member lumen **364** extends through pad **362**. Lumen **364** preferably has a generally conical shaped portion **365** disposed toward side **363**. Tension member **370** is shown disposed through lumen **364** in FIG. **25**. Pad **362** includes a threaded passage **366** extending from an edge of pad **362** to lumen **364**. A set screw **368** is threaded into passage **366**. Set screw **368** can be tightened to engage tension member **370** to fix the position of anchor pad **360**. When set screw **368** is not tightened, the size of lumen **364** is preferably large enough that anchor pad **360** can slide relatively freely over tension member **370**.

[0143] FIG. **26** is a perspective view of yet another embodiment of anchor pad **380** in accordance with the present invention. Anchor pad **380** preferably includes a generally disc-shaped pad portion **382** having a first side **383** which in use would be disposed toward the heart and a second side **385**. Pad **382** as well as pads **342** and **362** are preferably formed from a metal such as stainless steel alloys or titanium alloys.

[0144] A tension member fastener **384** is formed in pad **382** by cutting a series of grooves and apertures through pad **382** from side **385** to side **383**. A first groove **386** has a generally horseshoe shape. Second groove **388** extends between opposite portions of horseshoe shaped groove **386** to form two oppositely disposed cantilever members **387**. A relatively large aperture **394** is formed between cantilever members **387** proximate their free ends. A second and smaller aperture **390** is formed closer to the fixed ends of cantilever members **387**. Tension member **392** is shown extending through aperture **390**.

[0145] As shown in FIG. **26**, tension member **392** is clamped between cantilever members **387** such that the location of pad **382** is fixed along tension member **392**. Pad **382** can be released by using a spreading device **396** to spread cantilever members **387** apart. Spreading device **396** includes handle **398** to spreading arms **400** each having a finger **402**. Fingers **402** can be placed within aperture **394** then aims **400** and fingers **402** can be spread apart by pivoting them around a pin **404** such that cantilevers **387** are spread apart and pad **382** can move freely along tension member **392**. It can be appreciated that although spreader **396** is shown extending transversely from tension member **392**, it could also be configured such that fingers **402** do not curve transversely from arms **400** and thus spreader **396** could be disposed parallel to tension member **392**. This would be particularly desirable in a situation where anchor pad **380** was being placed through a port or window during a less invasive splint implantation procedure. It can be appreciated that cantilever members **387** can be held apart such that pad **380** can be moved along tension member **392** by placement of a temporary wedge or pin in groove **388**. For example, grooves **388** may include an additional small aperture disposed between aperture **390** and aperture **394** into which a pin could be placed to hold open members **387**. When it is desired to fix the position of anchor pad **380** on tension member **392**, device **396** could be used to spread cantilever members **387** to remove the pin. The cantilever members could then be released to engage tension member **392**. Aperture **390** of pad **380** can also include a conical portion disposed toward side **383** such as conical portion **365** of pad **360**.

[0146] Cantilever arms **384** are preferably configured such that they do not stress tension member **392** beyond its elastic limit. It can also be appreciated that the force developed by cantilever members **387** impinging on tension member **392** is operator independent and defined by the geometry and material characteristics of members **387**.

[0147] FIG. **27** is a perspective view of an anchor pad **360** having a tension member **370** extending therethrough. After pad **360** is secured to tension member **370**, that portion of tension member **370** which extends from the side of anchor pad **360** opposite side **363** is preferably removed. This can be accomplished by trimming tension member **370** with wire cutter **414** or scissors. Although anchor pad **360** is used here to illustrate trimming tension member **370**, it can be appreciated that in each of the embodiments disclosed herein there may be an excess portion of tension member extending from an anchor, which is preferably removed or trimmed.

[0148] FIG. **28** is a cross sectional view of an alternate embodiment **420** of a tension member cutter. Device **420** includes an elongate outer tube **422** having a distal end **424**. Tube **424** defines a lumen **423** through which extends a second tube **430** having a distal end **428**. Extending distally from distal end **428** are two cutting arms **424** and **426** which are shown partially withdrawn into lumen **423** and transversely restrained by distal end **424** of outer tube **422**. When unrestrained by distal end **424**, arms **424** and **426** are biased apart. Each arm **424** and **426** has a cutting element **425** and **427**, respectively. Elements **425** and **427** are shown in contact with each other in FIG. **28**. A tension member **370** extends between arms **424** and through lumen **432** of inner tube **430**. A representative anchor pad **360** is disposed adjacent elements **425** and **427**. Device **420** of FIG. **28** is particularly useful when trimming excess tension member using less invasive techniques as it can be readily advanced over a tension member through a port or window trocar.

[0149] FIG. **29** is a vertical cross sectional view of left ventricle B of heart A. A transventricular splint **443** including a tension member **370** and anchor pads **360** are shown disposed on heart A. To the left of heart A as shown in the

figure is a coiled portion **442** of tension member **470**. As an alternative to trimming an excess length of tension member, tension member **370** could be formed from a shape memory alloy such that portion **442** could be preset to assume a coil shape when warmed to near body temperature.

[0150] Once the length of the tension member has been adjusted, the anchors are secured in place along the tension member and the excess length of tension member removed if desired, the anchor or anchor pads are preferably secured in place on the heart. The anchor or anchor pads are secured such that relatively movement between the anchors or anchor pads and the heart is limited to reduce abrasion of the heart wall. To secure the anchor or anchor pads to heart A, a biocompatible adhesive could be placed between the pad and the heart to adhere the pad to the heart. Alternately, apertures could be provided in the pad such that sutures could be extended through the apertures and into the heart to secure the pad. In addition to sutures, the pad could include threaded apertures into which anchor screws could be advanced through the pad and into the heart wall to secure the pad to the heart.

[0151] FIG. **30** illustrates yet another alternative approach to securing the anchors or anchor pads to the heart surface. FIG. **30** is a cross sectional view of an anchor pad **340** disposed on heart A. Anchor pad **340** is disposed within an envelope **446**. Envelope **446** includes a bottom layer **447** disposed between anchor pad **340** and heart A and a top layer **448** disposed on the opposite side of anchor pad **340**. Layers **347** and **340** are held together by sutures **449**. Bottom layer **447** is preferably a mesh or expanded PTFE which has a pore size or intranodial dimension sufficient to promote tissue ingrowth. The pore size is preferably between about 10 and about 100 microns and more preferably, between about 20 and about 40 microns. With respect to expanded PTFE, the intranodial dimension is preferably between about 10 to about 100 microns and more preferably between about 20 to about 40 microns. The top material could also be expanded PTFE or the like having a pore size which preferably does not promote ingrowth and thus resists adhesion to surrounding tissue. As an alternative embodiment, the pores could be formed directly in the pad surface.

[0152] Envelope **446** would preferably be placed around pad **340** prior to placing pad **340** on tension member **354**. A window **450** can be provided to provide access to nut **352** to secure pads to tension member **354**. After tightening nut **352**, window **450** can be closed by suture **452**. FIG. **31** is a top view of pad **340** and envelope **446** of FIG. **30**. It can be appreciated that a similar envelope can be placed around the various anchor pads disclosed herein. The location of the window may have to vary, however, to provide access to the respective means for securing the anchor pads to the tension member.

[0153] The splints of the present invention can be implanted acutely or chronically. When the splints are implanted chronically, it is particularly important that the tension member or members be highly fatigue resistant. Typical materials for the tension member can include, among other biocompatible materials, stainless steel, titanium alloys, NiTi alloys such as Nitinol or elgiloy. In a preferred embodiment, the tension member is a wire having a diameter of between 0.005 to 0.035 inches in diameter or, more preferably, between 0.01 and 0.02 inches in diameter and, most preferably, about 0.014 inches in diameter. The length of the tension member between the pads is preferably about 0.6 to 4 inches, and more preferably, between about 1 to 3 inches and, most preferably, about 2 inches. To improve the fatigue resistance of the metallic tension members, their surface can be electro-polished, buffed or shot peened. Drawing or annealing of the metal will also improve fatigue resistance.

[0154] The tension member, in a preferred embodiment, articulates with respect to the anchor pad to reduce bending of the tension member at the pad. This can be accomplished by a ball and socket joint shown in FIG. **22**, for example. The tension member itself can be made more flexible or bendable by providing a multi-filament tension member such as a braided or twisted wire cable tension member. A multifiber filament structure of numerous smaller wires can then easily, while reducing the stress level on any individual wire as compared to a solid wire of the same diameter as the multifilament bundle. Such a mufti-filament tension member can be made from biocompatible materials such as, but not limited to, stainless steel, Nitinol, titanium alloys, LCP (liquid crystal polymer), Spectra™ fiber, kevlar fiber, or carbon fiber. In a preferred embodiment, the multi-filament structure is coated or covered to substantially seal the multi-filament structure. Coatings such as silicone, urethane or PTFE are preferred.

[0155] FIG. **32** is a side view of multifilament twisted cable **400**. Cable **400** includes a plurality of wires or filaments **402** twisted about the longitudinal axis of cable **400**. FIG. **33** is a transverse cross sectional view of cable **400**. In FIG. **33**, cable **400** includes a surrounding coating **404** not shown in FIG. **32**.

[0156] FIG. **34** is a side view of a braided multifilament tension member **410**. Tension member **410** includes a plurality of filaments or wires **412**. It can be appreciated that numerous braiding patterns are known to those skilled in the art of multifilament members. It is anticipated that in a preferred embodiment, braided member **410** can have an optional core of fibers running parallel to an elongate axis of tension member **410**. In yet another preferred embodiment, tension member **410** could have a solid wire core extending parallel to and along the longitudinal axis of tension member **410**.

[0157] The tension members and anchors or anchor pads are preferably bio-resistant, i.e., resistant to physiologic attack. To improve bio-resistance, tension member and/or anchors or anchor pads can be coated with carbon material such as glass, pyrolytic carbon, diamond or graphite, zirconium nitrate or oxide. Roughened or porous urethanes, silicone or polymer coatings or sheaths can be used to promote tissue ingrowth to create a biological seal. Hydrophilic and albumin coatings can also be used. Drugs incorporated into a binder coating can also be used to reduce biological attack on the splint and irritation of tissue by the splint. Such drugs include heparin, coumadin, anti-inflammatory steroid or ASA-aspirin. The oxide layer of the underlying metal could also be optimized to improve bioresistance. This is particularly true for stainless steel, titanium, or nickel titanium on which an oxide layer can be formed by heating the component to improve biocompatibility. Further coatings include calcium hydroxy appetite, beta tricalcium phosphate and aluminum oxide can be

applied to the tension member. The tension member and/or pad or anchor pad can at least be, in part, formed from titanium to enhance electronegativity.

[0158] The anchors or anchor pads and, particularly the tension members are biocompatible, preferably antithrombogenic and made to prevent hemolysis. The coatings used to enhance bio-resistance described above can generally be used to improve biocompatibility. Since the tension member is exposed to significant blood flows through the left ventricle, in a preferred embodiment, the tension member has a generally small size and shape elliptical cross sectional shape to reduce turbulence or drag over the tension member. If such elliptical, transverse cross section tension member were used, it can be appreciated that the narrow end would be preferably oriented toward the direction of blood flow. It is also desirable to select a tension member material and shape which would not vibrate at resonant-frequency under the influence of blood flow.

[0159] Where the tension member passes through the heart wall, various approaches can be taken to reduce or prevent bleeding. For example, the surface of the anchor or anchor pad and/or tension member in contact with the heart wall can be coated or include an ingrowth inducing covering such as collagen, dacron, expanded PTFE or a roughened/porous surface. A clotting inducing substance may also be bound to the tension member and/or anchor or anchor pads, such as avitene or collagen. It is also contemplated that the portion of the heart wall where the tension member passes through could be cauterized. In a preferred embodiment, the tissue can be cauterized by heating the tension member. A glue such as cyanoacrylate can also be disposed between the tension member and the heart wall to reduce or prevent bleeding from the heart wall. Mechanical means such as an O-ring or compression fitting could also be disposed between the heart wall and the tension member to reduce bleeding. A purse string suture can be placed on the heart, around the tension member adjacent the pad as well.

[0160] The tension member is preferably flexible enough to allow for changing interface conditions between the heart and the splint; and alternating pad orientation throughout the cardiac cycle. The flexibility should be sufficient enough to avoid injury to the heart or bleeding. It is also preferable that if the heart were to contract sufficiently enough to put the tension member in compression that it would readily buckle. Buckling could be promoted by providing a ribbon shaped tension member, chain link tension member, thin wire tension member, bent tension member or mufti-filament tension member.

[0161] The tension member is preferably radiopaque, echo cardiographic visible, or MRI compatible or includes a marker which is radiopaque, echo visible, or MRI compatible. The preferred locations for markers would-include the center of the tension member and at the ends of the tension member disposed at the heart walls. The radiopaque markers could be gold or platinum or other biocompatible metal or heavy metal filled polymeric sleeves. With respect to echo compatible or MRI compatible tension members or markers, the tension or marker are preferably non-interfering or visible. Having radiopaque echo compatible or MRI compatible tension members or markers is particularly desirable for follow-up, non-invasive monitoring of the tension member after implantation. The presence of the tension member can be visualized and the distance between two or more markers measured. Integrity of the tension member can be confirmed as well.

[0162] In a preferred embodiment, the tension member is not conductive to the action potential of muscle. This can be accomplished by insulating the tension member, anchor and/or anchor pad interface or fabricating the tension member anchor and/or anchor pad from a non-conductive metal such as titanium.

[0163] In addition to monitoring the performance of the tension member by visualization techniques such as fluoroscopy or echo imagery, sensors can advantageously be incorporated into the splints. For example, a strain gauge can be disposed on a tension member to monitor the loading on the member in use. Strain can be related to load as known to those skilled in the art by developing a stress/strain relationship for a given tension member. The strain gauge can be connected by a biocompatible lead to a conventional monitoring device. A pressure gauge formed from, for example, piezo electric material can also be disposed on the tension member to monitor filling pressures or muscle contractility.

[0164] In a preferred embodiment, a tension member can be slidably enclosed within a tube. If the tension member were to fail, the tube would contain the tension member therein.

[0165] It is anticipated that the tension member could be connected to a pacing lead. In such an instance, if the tension member were conductive, pacing signals could be conveyed along the tension member from one heart wall to another.

[0166] In use, the various embodiments of the present invention are placed in or adjacent the human heart to reduce the radius or cross-section area of at least one chamber of the heart. This is done to reduce wall stress or tension in the heart or chamber wall to slow, stop or reverse failure of the heart. In the case of the splint **16** shown in FIG. **1**, a cannula can be used to pierce both walls of the heart and one end of the splint can be advanced through the cannula from one side of the heart to the opposite side where an anchor can be affixed or deployed. Likewise, an anchor is affixed or deployed at the opposite end of splint **16**. Additional methods for splint placement are, described in more detail in U.S. patent application Ser. No. 09/123,977, filed on Jul. 29, 1998 and entitled "Transventricular Implant Tools and Devices" and incorporated herein by reference.

[0167] It can be appreciated that the methods described above to advance the tension members through the ventricles can be repeated to advance the desired number of tension members through the ventricle for a particular configuration. The length of the tension members can be determined-based upon the size and condition of the patient's heart. It should also be noted that although the left ventricle has been referred to here for illustrative purposes, that the apparatus and methods of this invention can also be used to splint multiple chambers of a patient's heart as well as the right ventricle or either atrium.

[0168] FIG. **35** is a schematic view of generally horizontal cross section of heart A including left ventricle B and right ventricle C. Also shown are left anterior descending artery E, posterior descending artery F, obtuse marginal artery G, postero-medial papillary muscle H and antero-lateral papil-

lary muscle **1**. Shown in FIG. **35** are three generally horizontal preferred alignments for tension member placement for the splints of the present invention when used for the purpose of treating ventricular dilatation. These alignments generally met three goals of splint positioning including good bisection of the left ventricle, avoidance of major coronary vessels and avoidance of valve apparatus including chordae leaflets and papillary muscles. Alignment **420** can be referred to as the anterior/posterior (AP) position. Alignment **422** can be referred as the posterior septal/lateral wall (PSL) position. Alignment **424** can be referred to as the anterior septal/lateral wall (ASL) position.

[**0169**] It can be appreciated that the alignments shown are illustrative only and that the alignments may be shifted or rotated about a vertical axis generally disposed through the left ventricle and still avoid the major coronary vessels and papillary muscles. When the alignment passes through a substantial portion of right ventricle C, it may be desirable to dispose not only two pads on the exterior of the heart at opposite ends of a tension member, but also a third pad within right ventricle C on septal J. The spacing between the third pad and the pad disposed outside the heart proximate left ventricle B preferably defines the shape change of left ventricle B. This will allow the spacing of the third pad relative to the pad disposed outside the heart proximate-right ventricle C to define a shape change if any of right ventricle C in view of the spacing between those pads. With the alignments as shown in FIG. **35**, the third pad will be unnecessary. It is likely, however, that with alignments **422** and **424** in order to achieve the desired shape change of left ventricle B, the exterior pad of the wall proximate the right ventricle C will be drawn into contact with septal J. This will consequently somewhat reduce the volume of right ventricle C.

[**0170**] FIG. **36** is a view of a cylinder or idealized heart chamber **48** which is used to illustrate the reduction of wall stress in a heart chamber as a result of deployment of the splint in accordance with the present invention. The model used herein and the calculations related to this model are intended merely to illustrate the mechanism by which wall stress is reduced in the heart chamber. No effort is made herein to quantify the actual reduction which would be realized in any particular in vivo application.

[**0171**] FIG. **37** is a view of the idealized heart chamber **48** of FIG. **36** wherein the chamber has been splinted along its length L such that a "figure eight" cross-section has been formed along the length thereof. It should be noted that the perimeter of the circular transverse cross-section of the chamber in FIG. **36** is equal to the perimeter of the figure eight transverse cross-section of FIG. **37**. For purposes of this model, opposite lobes of the figure in cross-section are assumed to be mirror images.

[**0172**] FIG. **38** shows various parameters of the FIG. **1** cross-section of the splinted idealized heart chamber of FIG. **37**. Where l is the length of the splint between opposite walls of the chamber, R_2 is the radius of each lobe, θ is the angle between the two radii of one lobe which extends to opposite ends of the portion of the splint within chamber **48** and h is the height of the triangle formed by the two radii and the portion of the splint within the chamber **48** (R_1 is the radius of the cylinder of FIG. **36**). These various parameters are related as follows:

$$h=R_2\,\mathrm{COS}(\theta/2)$$

$$l=2R_2\,\mathrm{SIN}(\theta/2)$$

$$R_2=R_1\pi/(2\eta-\theta)$$

[**0173**] From these relationships, the area of the figure eight cross-section can be calculated by:

$$A_2=2\pi(R_2)^2(1-\theta/2\pi)+hl$$

[**0174**] Where chamber **48** is unsplinted as shown in FIG. **36** A_1 the original cross-sectional area of the cylinder is equal to A_2 where θ=180°°, h=**0** and l=$2R_2$. Volume equals A_2 times length L and circumferential wall tension equals pressure within the chamber times R_2 times the length L of the chamber.

[**0175**] Thus, for example, with an original cylindrical radius of four centimeters and a pressure within the chamber of **140** mm of mercury, the wall tension T in the walls of the cylinder is 104.4 newtons. When a 3.84 cm splint is placed as shown in FIGS. **37** and **38** such that l=3.84 cm, the wall tension T is 77.33 newtons.

[**0176**] FIGS. **39** and **40** show a hypothetical distribution of wall tension T and pressure P for the figure eight cross-section. As θ goes from 180° to 0°, tension T_s, in the splint goes from 0 to a 2 T load where the chamber walls carry a T load. In yet another example, assuming that the chamber length L is a constant 10 cm, the original radius R_1, is **4** cm, at a 140 mmHg the tension in the walls is 74.7 N. If a 4.5 cm splint is placed such that l=4.5 cm, the wall tension will then be 52.8 N.

[**0177**] When a splint is actually placed on the heart, along an alignment such as those shown in FIG. **35**, the length l between the two pads as measured along the tension member is preferably 0.4 to about 0.8 and more preferably between about 0.5 to about 0.7 and most preferably about 0.6 times the distance along the length of the tension member at end diastole if the pads were not secured to the tension member and provided no resistance to expansion of the heart. A more detailed discussion of tension member length can be found in U.S. patent application Ser. No. 09/123,977, filed on Jul. 29, 1998 and entitled "Transventricular Implant Tools and Devices" which is incorporated herein by reference.

[**0178**] As mentioned earlier, FIG. **17** is a partial vertical cross-section of human heart **14** showing left ventricle **10** and left atrium **22**. As shown in FIG. **17**, heart **14** includes a region of scar tissue **24** associated with an aneurysm. The aneurysmal scar tissue **24** increases the radius or cross-sectional area of left ventricle **10** in the region affected by the scar tissue. Such an increase in the radius or cross-sectional area of the left ventricle will result in greater wall stresses on the walls of the left ventricle, especially those walls adjacent to the aneurysm.

[**0179**] In addition to the various uses of the splint to treat ventricular dilatation as heretofore discussed, the inventive splint also can be used to treat infarcted tissue or aneurysms occurring on the heart wall, as illustrated by FIGS. **18** and **42-44**, **46**, and **47**. These figures show various placements of a splint to treat infarcted tissue or aneurysms. It is to be understood that variations of these placements that have similar effects are within the scope of this invention.

[**0180**] FIG. **42** illustrates a method for placing a splint of the present invention to treat a heart with infarcted tissue, including an aneurysm. The particular aneurysm A shown in

FIG. **42** affects the ventricular septal wall. FIG. **42** shows a partial transverse cross-section of a human heart having an aneurysm A (shown by shading) in the left ventricle wall. It is contemplated that the methods and devices of this invention also apply to treatment of hearts with akinetic scar tissue that has not progressed past an infarcted stage and into an aneurysmal stage, in which case there would be little or no bulging of the heart wall. Such a condition is shown in FIGS. **43***a*-**43***d* to be described shortly. In FIG. **42**, splint **16** is placed diametrically across aneurysm A to lessen the load carried by the transmural infarcted tissue **24** forming aneurysm A, as well as any adjacent border zone tissue that may be present. The border zone (although not shown in FIG. **42**) is the portion of the heart wall which has a mix of contractile tissue **24"** and infarcted tissue **24**. Anchors **20** of splint **16** are located on the outside of the chamber walls and are placed generally adjacent to the portions of the chamber wall that transition from infarcted myocardium **24** to regions of contractile myocardium **24"**. Tension member **18** extends through the heart chamber with each of its ends connecting to opposing anchors **20**. Anchors **20**, especially when used to anchor splint **16** on septal wall S, can be of the self-deploying type disclosed in co-pending application U.S. Ser. No. 09/123,977, filed Jul. 29, 1998, entitled "Transventricular Implant Tools and Devices," the complete disclosure of which is incorporated herein by reference.

[**0181**] Splint **16** reduces the radius of curvature of the aneurysmal region A and the adjacent regions of the chamber wall. By reducing the radius of curvature in these regions, contractile regions **24"** of the myocardium that were under high stress due to geometric abnormalities associated with an aneurysmal region A are relieved from that high stress, thereby resulting in increased pumping ability upon contraction. Even if the infarcted tissue has not led to bulging of the heart wall, reducing the radius of curvature helps to reduce some of the stress in the adjacent contractile myocardium. By increasing the pumping ability of the contractile myocardium **24"**, the heart can more easily pump the required blood flow output, helping to offset the pumping lost by the infarcted muscle **24**. Those regions of the chamber wall that have only endocardial infarcted tissue, that is border zone regions **24'**, likely will experience an increase in their ability to contract and contribute to pumping.

[**0182**] It is also contemplated to use more than one splint, and splints having different lengths, to optimize the reduction in the radius of curvature of infarcted and aneurysmal regions and adjacent regions. Another contemplated mode of the invention includes closing off the infarcted or aneurysmal region completely by shortening the splint so that the walls adjacent the anchors contact each other. Closing off the infarcted or aneurysmal tissue from the rest of the heart chamber in this way renders this tissue completely non-functional with respect to contributing to the pumping. Shortening the splint to achieve contact of the heart walls may also reduce the risk of embolic thrombus because no blood would be expected to flow into the excluded region. Additionally, the need to remove any thrombus already adhered to the heart wall may be unnecessary because the thrombus would have no way of escaping back into the heart chamber to cause stroke or other malfunctions. If the infarcted or aneurysmal tissue extends to the septal wall of the chamber, the splint would be placed across the chamber so as to exclude the non-contractile tissue of the septal wall as well.

[**0183**] As described earlier, FIG. **18** is a vertical cross-sectional view of heart **14** as shown in FIG. **17**. FIG. **18** depicts another method of the present invention, wherein splint **16** is placed to draw aneurysm A toward an opposite wall of left ventricle **10**. An anchor **20** of splint **16** is placed on the outside wall of heart **14**, approximately at the center of the infarcted tissue **24** forming aneurysm A. Tension member **18**, connected to this anchor, is then extended across the chamber of the heart to the opposite wall and connected to another anchor **20** placed on the outside chamber wall to secure splint **16**. The radius or cross-sectional area of the left ventricle affected by the infarcted tissue **24** is thereby reduced. The reduction of this radius or cross-sectional area results in reduction in the stress in the left ventricular wall and thus improves heart pumping efficiency. Furthermore, infarcted tissue **24** is supported by anchor **20** of the splint to prevent any additional bulging of the wall or progression of the infarcted tissue to other areas of the myocardium.

[**0184**] Bringing infarcted tissue **24** into the chamber, as shown in FIG. **18**, likely reduces the risk of thrombosis due to contact between the endocardial infarcted tissue and circulating blood flow occurring in these regions of the chamber. Clots are less likely to form on tissue that is subject to an active flow of blood. The forces associated with such flow diminish stagnation points that allow clots to form and adhere to the wall more readily.

[**0185**] FIGS. **43***a*-**43***d* depict the use of splint **16** to treat a heart chamber having a discrete zone of akinetic infarcted myocardium **24** that has not yet developed into an aneurysm. FIGS. **43***a* and **43***b* depict a long axis (or essentially vertical) cross-sectional view of the heart, with FIG. **43***b* showing the placement of splint **16**. FIGS. **43***c* and **43***d* show the heart in short axis (or essentially horizontal) cross-section, with placement of splint **16** shown in FIG. **43***d*. In FIGS. **43***b* and **43***d*, splint **16** is shown treating a heart including infarcted tissue **24** that does not affect septal wall S. Thus, both anchors **20** are placed on exterior wall portions of left ventricle LV. However, it is contemplated that a splint could be used to alter the geometry of the chamber in cases in which an infarcted region does affect septal wall S.

[**0186**] By utilizing splint **16**, the radius of curvature, particularly with respect to the short axis direction, is reduced. This reduction of curvature facilitates the pumping ability of any portions near the infarcted region **24** that have some contractile potential by reducing wall stress in the region. By allowing this once marginally contractile tissue to increase its contractile ability, the heart improves its ejection fraction, cardiac reserve, and muscle contractibility. Additionally, the remainder of the ventricle also experiences a reduced radius of curvature, as shown in FIGS. **43***b* and **43***d*, further facilitating the contractile ability of the entire ventricle. By improving the contracting ability of the entire chamber, the heart has the ability to account for the lost pumping ability of the infarcted myocardium **24**. Left untreated, this condition causes the rest of the ventricle to attempt to contract more, ultimately over-working and weakening the heart to a greater degree.

[**0187**] FIGS. **44***a*-**44***b* show a cross-sectional view of the left ventricle with an aneurysmal region A. As shown in FIG.

44*b*, splint **16** can also be placed such that one anchor **20** engages approximately the center of the bulge formed by aneurysm A. Tension member **18** is then extended through the center of aneurysm A and across left ventricle LV to anchor splint **16** to a point on the surrounding heart wall substantially opposite to aneurysm A. This positioning of splint **16** tends to bring the aneurysmal bulge in line with the normal curvature of the heart wall. By this placement of splint **16**, it is expected that greater blood flow would occur in the region of aneurysmal tissue A, potentially reducing thrombus formation.

[0188] FIG. **45** shows the use of a completely external device to treat a heart having a zone of infarcted tissue **24**. In the embodiment shown in FIG. **45**, the external splint device is an external frame generally in the form of a clamp **17** with anchor pads **21** on each end of the clamp. The clamp **17** is configured to exert a compressive force on the heart wall to cause shape change of the heart chamber. Other external splint devices, in addition to clamp **17**, that are contemplated for use in treating a heart having infarcted myocardium are disclosed in co-pending U.S. patent application Ser. No. 09/157,486, filed Sep. 21, 1998, and entitled "External Stress Reduction Device and Method," the complete disclosure of which is incorporated herein by reference. In addition to the benefits described above with respect to using a splint to treat a heart having infarcted tissue, external splint devices such as that shown in FIG. **45** include the potential further advantage of significantly reducing the possibility of thrombus formation resulting from surfaces of devices that contact blood flowing through the chamber.

[0189] Another use for splint **16** in treating heart chambers having infarcted or aneurysmal tissue is shown with reference to FIGS. **46***a* through **46***c*. This method combines the use of splint **16** with the traditional surgical technique in which the infarcted tissue is removed. By using the techniques described later for identifying and distinguishing between healthy tissue and infarcted tissue, all of the infarcted myocardium **24** shown in FIG. **46***a* can be excised from the chamber, as shown in FIG. **46***b*. The separated portions of the chamber walls will then be sutured back together. Splint **16** is then placed diametrically transverse to the portion of heart chamber walls **24"** that have been partially excised, as shown in FIG. **46***c*. Anchors **20** are placed between the portions of the chamber wall from which infarcted tissue **24** was removed and portions of the chamber wall that contained contractile tissue throughout its thickness. Because only contractile tissue regions **24"** would remain after this procedure, as opposed to conventional surgery which leaves some of the infarcted tissue occurring in border zone regions in place, contractile function, and thus cardiac function, likely would improve. Splint **16** reduces the radius of curvature in those thinner regions of the walls that remain after the surgery, allowing them to produce stronger contractions and relieving stress in those wall portions.

[0190] If the infarcted tissue is not removed, as shown in FIG. **42**, the border zone cannot contribute significantly to the pumping function, for the contractile muscle must contract against the stiffness of the infarcted muscle. But, with the infarcted tissue removed, the thin contractile section of myocardium shown in FIG. **46***c* can be made to contract and contribute to pumping, particularly since splint **16** has reduced stress enough to allow the thin region of tissue to exert the required pressure to pump. In addition to combining the surgical excision of the infarcted tissue with the use of a splint of the present invention, an external device, like clamp **17** shown in FIG. **45**, can also be combined with the surgical technique. Such an external device would be placed with respect to the heart chamber walls such that its anchors are disposed in the approximate locations as anchors **20** on splint **16**, and the device between the anchors could extend around the portions of the walls that have been sutured together. It is contemplated that the splint **16** could be used in hearts where the aneurysm involves the septal wall of the ventricle. Similar to the splinting shown in FIG. **42**, one or both anchor pads could be self-deploying.

[0191] Another use of the splint to treat infarcted or aneurysmal tissue near the base of a mitral valve MV is shown in FIG. **47**. In this embodiment, the splint is placed such that its anchors **20** are diametrically opposed to one another across an aneurysm, or generally dilated annular region, located in a portion of the basal left ventricle in the vicinity of mitral valve MV. Anchors **20** are placed on the outer wall of left ventricle LV with tension member **18** of splint **16** drawn through the heart chamber and diametrically across the infarcted or aneurysmal region. The dotted line of tension member **18** shown in FIG. **47** illustrates that splint **16** lies below mitral valve MV. Placing the splint in this manner results in the papillary muscles P of mitral valve MV or the leaflets of mitral valve MV being drawn together and reduces the risk of mitral regurgitation. In addition, splint **16** reduces the radius of curvature of the aneurysmal or infarcted region. As previously described, this reduction lowers the stress in the heart wall, improving pumping effectiveness. FIG. **47** shows the placement of splint **16** such that both anchors are disposed on external walls of the left ventricle. It should be noted that the splint also could be placed so that one anchor is disposed on septal wall S, in which case a self-deploying anchor preferably would be used. Also, tension member **16** may be made to curve between anchors **20** in order to avoid damaging internal structures of the ventricle.

[0192] FIGS. **48***a*-**48***b* show the use of an external splint device to treat a heart having an aneurysm A in the vicinity of mitral valve MV. As a result of the aneurysm, the anterior leaflet AL and posterior leaflet PL of mitral valve MV have separated causing mitral regurgitation, as shown in FIG. **48***a*. FIG. **48***b* shows the placement of an external splint device having a clamp portion **19** connecting two anchors **25**. It is contemplated that the external splint device used to treat this particular heart condition may also include a protrusion **19'** as part of clamp portion **19**. In placing clamp **19** on the heart, protrusion **19'** engages aneurysm A so as to push aneurysm A toward a center of the ventricle. Additionally, a stabilizing bar **23** could be attached to the end of protrusion **19'**. Stabilizing bar **23** is configured to extend through the heart chamber wall and anchor to the edge of posterior leaflet PL of mitral valve MV to bring the leaflet in close proximity to anterior leaflet AL. Such a stabilizing bar may be made of a semi-rigid, or rigid, material such as implantable metals or other suitable material. While FIGS. **48** and **49** show the use of various splints associated with an aneurysm in the vicinity of the mitral valve, it is contemplated to utilize splints also in the case where there is no true aneurysm, but an area of infarcted tissue near the mitral valve.

[0193] The present invention also includes myocardial patches and related methods used to treat aneurysms. FIGS. **49***a*-**49***b* illustrate one preferred embodiment of the invention. FIG. **49***a* shows left ventricle LV with an aneurysm A in a portion of the chamber wall. FIG. **49***b* illustrates placement of a staked patch **72** according to an embodiment of the present invention used to treat aneurysmal bulge A. Staked patch **72** includes a patch **70**, made of Dacron or PTFE for example, and an elongated member **71** secured to the patch. When staked patch **72** is secured into position (by sutures or the like) over the surface of aneurysmal bulge A, elongated member **71** pushes on the aneurysmal tissue region. Staked patch **72** pushes bulge A into the heart chamber. This pushing likely will result in an even further reduction in the stresses experienced by the adjacent heart wall due to the reduction in the radius of curvature resulting from drawing adjacent regions in toward each other when the patch is in place. Such further reduction in stress would tend to promote a more organized healing of the scar tissue region and prevent progression of the scar into other healthy myocardium. Additionally, by pushing the bulge into the chamber space, thrombosis is less likely to occur because of the active blood flow past the surface of the bulge.

[0194] As shown in FIG. **49***b*, staked patch **72** has a single stake **71** secured to the patch in a substantially perpendicular direction. However, it is contemplated that several stakes could be secured to the patch depending on the size of the affected tissue area to be treated. Additionally, stakes could be secured in various orientations, including skewed orientations, relative to the patch. The stakes have different sizes in order to optimize the degree and direction in which the bulge is pushed in, especially when the bulge has a non-uniform surface configuration. The stakes may be rigid or semi-rigid and may be manufactured from implantable metals and polymers, or other suitable materials of similar characteristics.

[0195] A three-dimensional patch also is contemplated by the present invention. Such a patch could be inflatable or solid. The patch would consist of an essentially flat surface configured to lie flush with the epicardial surface and a bulging surface that would engage with the aneurysmal region to push the aneurysm into the chamber in a manner similar to the stake described above. A suture ring may be placed around the perimeter of the flat surface to secure the patch into place on the heart.

[0196] A further embodiment of the present invention is a shrinkable patch for treating aneurysms. For example, such a patch may be made of heat-shrinkable material and applied via sutures to the aneurysmal tissue region while the tissue is in a relaxed state. Gentle heat, such as that produced by, for example, a hot air gun, an infrared heating lamp, or other similar heating mechanisms, would then be applied to shrink the patch. This shrinking also will cause the size of the affected area to be decreased by being pulled in tightly with the shrinking patch, thereby reducing the radius of curvature of the adjacent myocardium. The patch may be made of any suitable material compatible with the human body and having heat-shrinking characteristics. Examples of such a material include oriented polyethylene, oriented polypropylene, and a woven Dacron polyester of partially-oriented yarn. Partially-oriented yarn is capable of significant longitudinal shrinkage when heated. Additionally, targeting and heating certain areas yields a non-uniform shrinking that more precisely tailors the resulting configuration of the patch and the affected tissue region.

[0197] Another embodiment of the present invention, which may be used either alone or in combination with a patch, is a purse-string suture **50**, as shown in FIGS. **50***a*-**50***b*. In this embodiment, a suture **50** is placed to encircle the affected tissue area **24**, as shown in FIG. **50***a*. Suture **50** has free ends **51** that are pulled to draw in suture **50** and reduce the perimeter of the affected tissue, as shown in FIG. **50***b*. Free ends **51** are then secured so as to keep the gathered tissue region in place. The securing can be accomplished by tying free ends **51** to one another or by some like means. By gathering the infarcted tissue together and reducing the perimeter, tension in the walls adjacent to the infarcted tissue **24** decreases due to the reduction in radius of the wall in that region. Thus, improved contractile function of the adjacent tissue is expected. Purse-string suture **50** could be drawn in to such an extent that the outer walls of the perimeter of infarcted tissue **24** contact each other. Drawing the infarcted tissue **24** to this extent cuts off the tissue completely from the rest of the heart chamber and renders it non-functional. In addition to the purse-string suture **50**, a patch may be applied to support any bulging tissue area that remains after application of suture **50**, as well as provide a more secure means of maintaining the gathered portions of the myocardium.

[0198] FIGS. **51***a*-**51***c* illustrate a further embodiment of the present invention, the combination of a purse-string suture and an enclosure member **60**. In this embodiment, purse-string suture **50** is applied to infarcted or aneurysmal tissue **24** as described above with reference to FIGS. **50***a*-**50***b*. After the suture is pulled tight to gather the affected tissue region in, an enclosure member **60** is secured into place. As shown in FIG. **51***c*, enclosure member **60** is placed around the gathered tissue **24** so that substantially all of the infarcted tissue is contained inside enclosure member **60**. In a preferred form of the invention, sutures are used to secure the enclosure member **60** into place, however other securing means are also contemplated. Enclosure member **60** preferably is made from a substantially rigid material, for example stainless steel, semi-rigid material, or any other material exhibiting like characteristics such as, for example, polyamide imide, titanium, or ultra high molecular weight polyethelene, in order to carry the load created by the gathered infarcted tissue. Because of its relative rigidity, enclosure member **60** withstands the load of the gathered tissue better than sutures alone and contains the progression of the infarcted tissue. Although FIG. **51***c* shows a ring as enclosure member **60**, other shapes may be used to serve the inventive purposes. Overall, regardless of the shape of enclosure member **60**, its perimeter should be selected to have approximately the same shape as the perimeter of the infarcted tissue region once it has been gathered together by purse-string suture **50**.

[0199] Another enclosure device is shown in FIGS. **52***a*-**52***c*. FIG. **52***a* shows a region **24** of infarcted myocardium on left ventricle LV. Other heart structure shown in these figures includes the right ventricle RV, the right atrium RA, the superior and inferior vena cavas, and the aorta. The inventive enclosure member **61** of FIGS. **52***a*-**52***c* is configured to change from a circular shape to an elliptical or oval shape. Such an enclosure member can be fabricated from a shape memory alloy, such as nitinol, or other similar suitable

material, and processed such that it has a circular shape at a temperature below its transformation temperature, which is approximately equal to body temperature. Upon reaching the transformation temperature, the ring, comprised of the shape memory material, alters its configuration to the shape of an ellipse or oval, or other suitable configurations. During surgical application, the temperature of the heart is below body temperature and therefore ring **61** remains in the circular shape during application of ring **61** to the heart, as shown in FIG. **52***b*. After installation of the ring is complete, the heart reaches normal body temperature, transforming ring **61** into the elliptical or oval shape, as shown in FIG. **52***c*. This shape transformation deforms the infarcted region of tissue on the heart wall to change the shape and/or size of the heart chamber, relieve stress on the heart walls, and improve overall contractile function of the heart. In a preferred embodiment, infarcted region **24** deforms in a short axis direction, as shown in FIG. **52***c*.

[**0200**] Alternatively, enclosure member **61** can be formed of a spring metal, such as, for example, high tensile strength stainless steel. When such a material is used, the enclosure member is initially processed into the elliptical or oval configuration. The enclosure member is then attached around a circular polymer, or other suitable material, sheet to form and maintain a circular shape during attachment of the ring to the ventricle. Once attached, the circular polymer sheet is removed, causing the enclosure member to spring back to its original elliptical shape.

[**0201**] Other embodiments of the present invention include enclosure members that deform non-uniformly, either by having a non-uniform configuration at the shape memory transformation temperature, or at the initial processing shape of the spring metal. It also should be noted that enclosure member **61** can take on any suitable shape both before application and after, depending on such factors as, for example, the particular infarcted region to be treated and the desired final radius of curvature.

[**0202**] FIGS. **53***.a*-**53***b* illustrate a tie enclosure, a further embodiment of the invention and a variation on the use of the enclosure member. The tie enclosure includes a plurality of sutures **62** having free ends **64**. Sutures **62** secure around the perimeter of the infarcted or aneurysmal tissue **24**, as shown in FIG. **53***a*. Sutures **62** are secured at points **63** using pledgets **65**. Enclosure member **60** is then placed over the affected tissue region and free ends **64** of the plurality of sutures **62** are extended through enclosure member **60** to pull sutures **62** through enclosure member **60** as well. By pulling on free ends **64** of sutures **62**, the infarcted or aneurysmal tissue region **24** is again gathered and its perimeter reduced. The tissue is drawn so that pledgets **65** ultimately are adjacent enclosure member **60**. The tissue, attached to the sutures, is drawn through enclosure member **60**, and once in place, enclosure member **60** can be sutured to the myocardium. Free ends **64** of sutures **62** are drawn until pledgets **65** are brought close to enclosure member **60**. Sutures **62** are then tied to the enclosure member **60**. Additionally, enclosure member **60** may be directly sutured to the myocardium, as shown in FIG. **53***b*. Again, drawing in the aneurysmal tissue region reduces stress on the chamber walls, hinders the progression of the infarction, and results in a more uniform scar formation, and reduces the radius of curvature, and therefore the stress, in the region of the chamber adjacent the infarction, as well as more global reduction of radius of curvature.

[**0203**] The tie enclosure also allows for non-uniform drawing in of the affected tissue region by using a plurality of sutures of varying lengths. Thus, the tie enclosure allows for particular regions of the aneurysm or infarction to be targeted and drawn in while others are left intact or drawn in to a lesser degree. This allows for a more precise change in geometrical configuration that may be necessary due to non-uniformities existing in the initial geometry of the infarcted or aneurysmal region. Enclosure member **60** also may be made of a flexible material so as to allow enclosure member **60** to take on a non-uniform configuration when sutures **62** are drawn and secured.

[**0204**] As discussed previously, enclosure member **60** may be made of a rigid, semi-rigid, or flexible material, depending on the particular application for which the device will be used. The shape of enclosure member **60** can be that of a ring as shown in FIGS. **53***a*-**53***b* or can be another shape, as long as the perimeter of enclosure member **60** is less than or equal to that of the drawn in tissue region.

[**0205**] Using enclosure member **60** and sutures **62** in the manner described with respect to FIGS. **53***a*-**53***b*, wider zones of infarcted or aneurysmal tissue preferentially may be drawn further toward enclosure member **60**, thereby maximizing the radius reduction in the direction of the widest dimension. This enables portions of the ventricle that are the most affected by the infarction to experience the greatest radius reduction and therefore the greatest stress relief.

[**0206**] Yet another form of an enclosure member contemplated by the present invention is illustrated in FIG. **54**. Enclosure member **65** in FIG. **54** takes the form of a braided ring with a lumen through which tightening cords **66** extend. Enclosure member **65** can be sutured into place surrounding an infarcted zone of tissue, with the sutures passing through enclosure member **65** only and not through tightening cords **66**. Once member **65** is secured around the infarcted tissue, tightening cords **66** can be pulled to draw in enclosure member **65**, causing the infarcted tissue region to be drawn together to thereby reduce the radius of curvature of the heart wall in that location. Enclosure member **65** may be made of a relatively flexible material that is either atraumatic itself or is wrapped in an atraumatic material such as Dacron or PTFE for example. Other materials that encompass these characteristics are considered to be within the scope of this invention as well.

[**0207**] In treating infarcted tissue and aneurysms with the various inventive methods and devices discussed above, it may be necessary to identify and distinguish between the infarcted tissue regions of the chamber wall and the contractile tissue regions of the chamber wall. Thus, a further aspect of the invention consists of the use of various devices for performing such identification in order to achieve precise placement of the inventive devices, including the splints, sutures, patches, and rings. The identification devices can be used either endocardially, epicardially, or transcardially, depending on which treatment procedure is being performed.

[**0208**] One such method of identifying infarcted tissue regions from contractile tissue regions involves the use of a

bipolar electrode. Using the electrode, differences in impedance sensed by the electrode will indicate regions of infarcted versus contractile tissue.

[0209] Another method involves the use of fiber optics to distinguish infarcted from contractile regions of tissue. In this case, the fiber optics sense either density or color differences in transmitted or reflected light. Such differences would indicate whether a region contained infarcted tissue or contractile tissue. For instance, a known intensity of light could be directed toward a tissue region, with a known nominal intensity transmitting through contractile tissue regions. Intensities of the transmitted light through the chamber wall could then be measured. Upon sensing a decrease in intensity from the nominal value, the infarcted region could be pinpointed. Border zones also could be sensed in this way by looking for a gradation in transmission differences from the nominal value to a lowest value.

[0210] Another method to locate regions of infarcted versus contractile tissue can be employed during the surgical procedure itself. This method involves the injection of radioactive media, such as in a thallium scan, with "real-time" imaging of the radiation during the surgery through the use of a Geiger counter contact probe. Higher observed radiation densities indicate regions of perfused tissue. Yet another injection method uses a visible dye injected into coronary vessels to identify contractile perfused tissue. In this identification technique, the dye travels only to contractile tissue, not to infarcted or scarred tissue regions. Finally TEE, or transesophageal echo ultrasounds may be used to detect regions of infarcted tissue.

[0211] Aside from those listed above, other methods are contemplated to locate infarcted tissue regions. For example, the surgeon may use his fingers to probe the outer surface of the chamber wall and feel for differences in the tissue regions.

[0212] All of the inventive passive devices to be implanted in the heart may be made of biocompatible material that can remain in the human body indefinitely. Any surface engaging portions of the heart should be atraumatic in order to avoid tissue damage.

[0213] It will be understood that this disclosure, in many respects, is only illustrative. Changes may be made in details, particularly in matters of shape, size, material, number and arrangement of parts without exceeding the scope of the invention. Accordingly, the scope of the invention is as defined in the language of the appended claims.

[0214] Other embodiments of the invention will be apparent to those skilled in the art from consideration of the specification and practice of the invention disclosed herein. It is intended that the specification and examples be considered as exemplary only, with a true scope and spirit of the invention being indicated by the following claims.

1-77. (canceled)

78. A method of direct localized therapeutic treatment of a heart, the method comprising:

identifying a target region of the myocardium of the heart; and

applying a self-anchoring mechanical device directly to a portion of the myocardial tissue of the identified target region,

whereby motion of the myocardial tissue of the identified target region is at least one of: restricted and limited.

79-83. (canceled)

84. A method of treating a heart having experienced a myocardial infarction, the method comprising:

identifying and locating at least one of: non-ischemic tissue, infarcted tissue, peri-infarcted tissue and ischemic tissue in the myocardium of the heart; and

placing a self-anchoring mechanical device in at least one of the non-ischemic tissue, infarcted tissue, peri-infarcted tissue and ischemic tissue to restrict or limit motion in at least one of the infarcted tissue, peri-infarcted tissue and ischemic tissue.

85-98. (canceled)

99. A method of treating a heart with abnormal heart wall motion, the method comprising:

identifying and locating at least one of: non-ischemic tissue, infarcted tissue, peri-infarcted tissue and ischemic tissue in the myocardium of the heart; and

placing a self-anchoring mechanical device in at least one of the non-ischemic tissue, infarcted tissue, peri-infarcted tissue and ischemic tissue to restrict or limit motion in at least one of the infarcted tissue, peri-infarcted tissue and ischemic tissue.

100-101. (canceled)

102. A device for placement in a myocardium of a heart, the device comprising:

means for attaching to the myocardium at two or more attachment locations; and

means for restricting, to substantially a predetermined value, a distance between at least two attachment locations.

103-109. (canceled)

110. A device for reducing stress in myocardial tissue, comprising:

a substantially tubular body having proximal and distal portions;

a first expanding anchor located at the proximal portion; and

a second expanding anchor located at the distal portion, wherein the tubular body comprises a lumen having a proximal opening in the proximal portion and a distal opening in the distal portion.

111-121. (canceled)

122. A method of direct localized therapeutic treatment of a heart, the method comprising:

identifying a target region of the myocardium of the heart; and

applying a self-anchoring mechanical device directly to a portion of the myocardial tissue of the identified target region to limit substantial lengthening of myocytes in the target region.

123-126. (canceled)

127. A method of treating mitral valvular regurgitation in a human heart, the method comprising:

providing a self-anchoring mechanical device;

identifying a target region of a myocardium of the heart; and

positioning the self-anchoring mechanical device within a portion of the identified target region so as to adjust a position of a base of at least one papillary muscle coupled to a mitral valve.

128-138. (canceled)

139. A method of treating mitral valvular regurgitation, the method comprising:

providing a restraining device having a distal anchor and a proximal anchor;

embedding the distal anchor of the device in heart tissue; and

embedding the proximal anchor in the heart tissue, wherein the restraining device is substantially positioned in a mid-wall of the heart and oriented to adjust a position of a base of at least one papillary muscle coupled to a mitral valve.

140-145. (canceled)

146. A method of treating a heart, the method comprising:

providing a restraining device having a distal anchor and a proximal anchor;

embedding the distal anchor of the device in heart tissue; and

embedding the proximal anchor in the heart tissue, wherein the restraining device is substantially positioned in a mid-wall of the heart.

147-153. (canceled)

154. A method for direct localized therapeutic treatment of myocardial tissue in heart having a pathological condition comprising the steps of:

a. identifying a target region of the myocardium;

b. applying a device to at least a portion of the myocardial tissue of the target region substantially identified in step (a) to physically modify the mechanical properties of said tissue and limit motion in the peri-infarct or infarct region.

155. The method of claim 154, wherein geometry and wall stress of the infarct or peri-infarct tissue is altered.

156. The method of claim 155, wherein the infarct region is excluded or shrunk.

157. The method of claim 156, wherein expansion of the infarct or peri-infarct tissue during systole is limited.

158. The method of claim 154, wherein the infarct or peri-infarct tissue is physically modified by at least one of stiffening, restraining, or constraining.

159. The method of claim 154, wherein a device is placed through the myocardium to restrict motion in the peri-infarct or infarct regions.

160. The method of claim 154, wherein the modified mechanical properties include an increase in systolic performance.

161. The method of claim 160, wherein the modified mechanical properties includes substantially no decrease in global diastolic performance.

162. The method of claim 154, wherein the device is utilized without external stimulation.

163. The method of claim 154, wherein the target region includes a myocardial infarct.

164. The method of claim 154, wherein the target region at least in part underlies the papillary muscles associated with a cardiac valve.

165. The method of claim 164, wherein said cardiac valve is the mitral valve.

166. The method of claim 154, wherein the device is a tubular structure positioned between at least first and second anchors.

167. The method of claim 166, wherein the tubular structure includes slots configured to provide lateral flexibility.

168. The method of claim 167, wherein the tubular structure is configured to provide lateral and longitudinal flexibility.

169. The method of claim 168, wherein the tubular structure is configured to provide lateral shortening and limited lateral lengthening.

170. The method of claim 166, wherein the at least first and second anchors are end portions of the tubular structure.

171. The method of claim 154, wherein said applying step includes employing a guide catheter.

172. The method of claim 171, wherein the guide catheter is inserted through the femoral artery, threaded through the aorta and inserted retrograde through the aortic valve into the left ventricle.

173. The method of claim 172, wherein the guide catheter is placed against the endocardial surface of the left ventricle.

174. The method of claim 173, further comprising:

placing a puncture device through the guide catheter to make a puncture into the endocardium.

175. The method of claim 174, further comprising:

inserting a guidewire until it is located distal to the myocardial infarct and the distal peri-infarct region.

176. The method of claim 175, further comprising:

introducing the delivery device over the guidewire to the desired location in the myocardium.

177. The method of claim 171, wherein said catheter is employed percutaneously.

178. The method of claim 154, wherein said applying step is preceded by performing a minithoractomy.

179. The method of claim 154, wherein said applying step includes applying material intramyocardially.

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