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Argenta et al.

(54) **APPARATUS AND METHOD FOR CARDIAC TISSUE MODULATION BY TOPICAL APPLICATION OF VACUUM TO MINIMIZE CELL DEATH AND DAMAGE**

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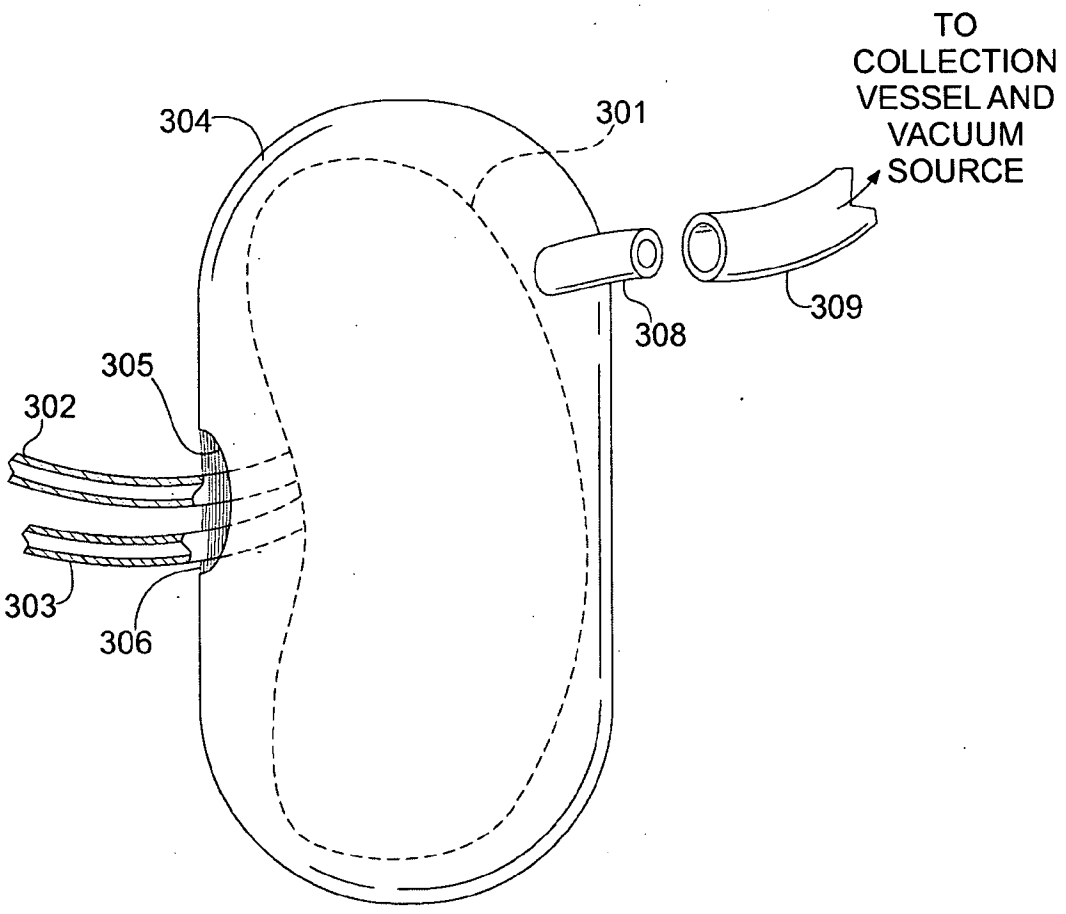
(57) **ABSTRACT**

A method and apparatus are provided for treating cardiac tissue to modulate ischemic heart tissue with topical sub-atmospheric pressure to minimize cell death and damage.



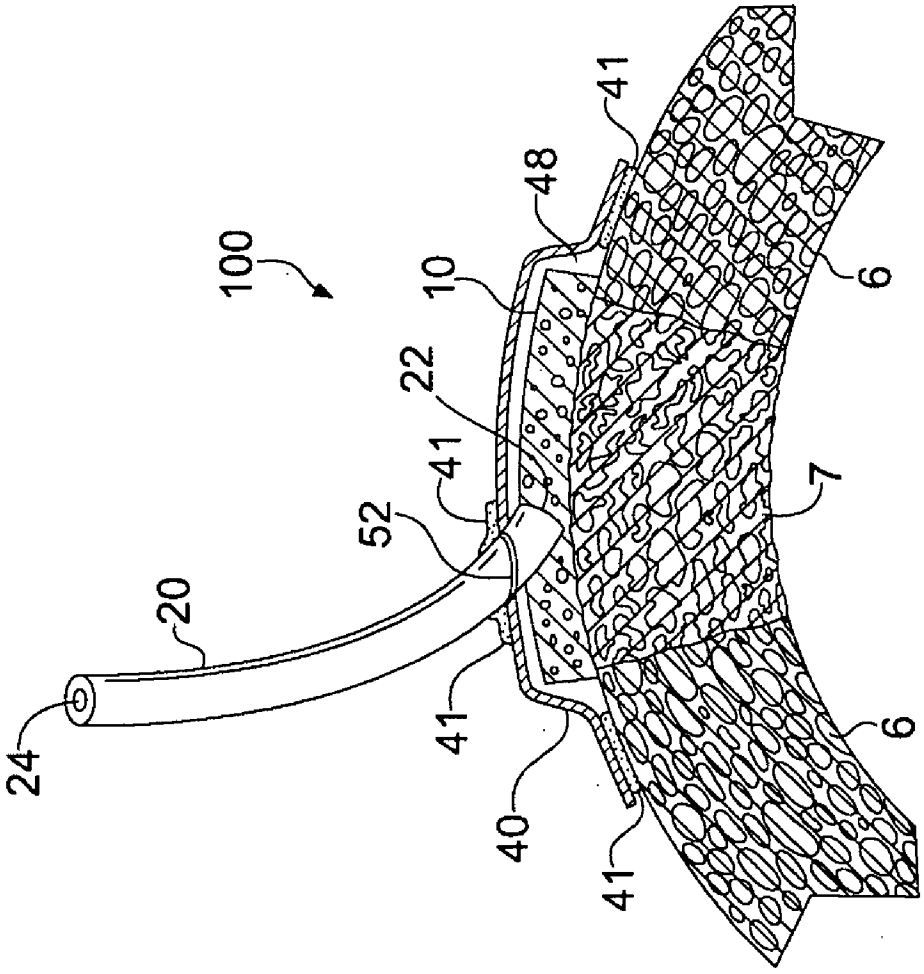


Fig. 1

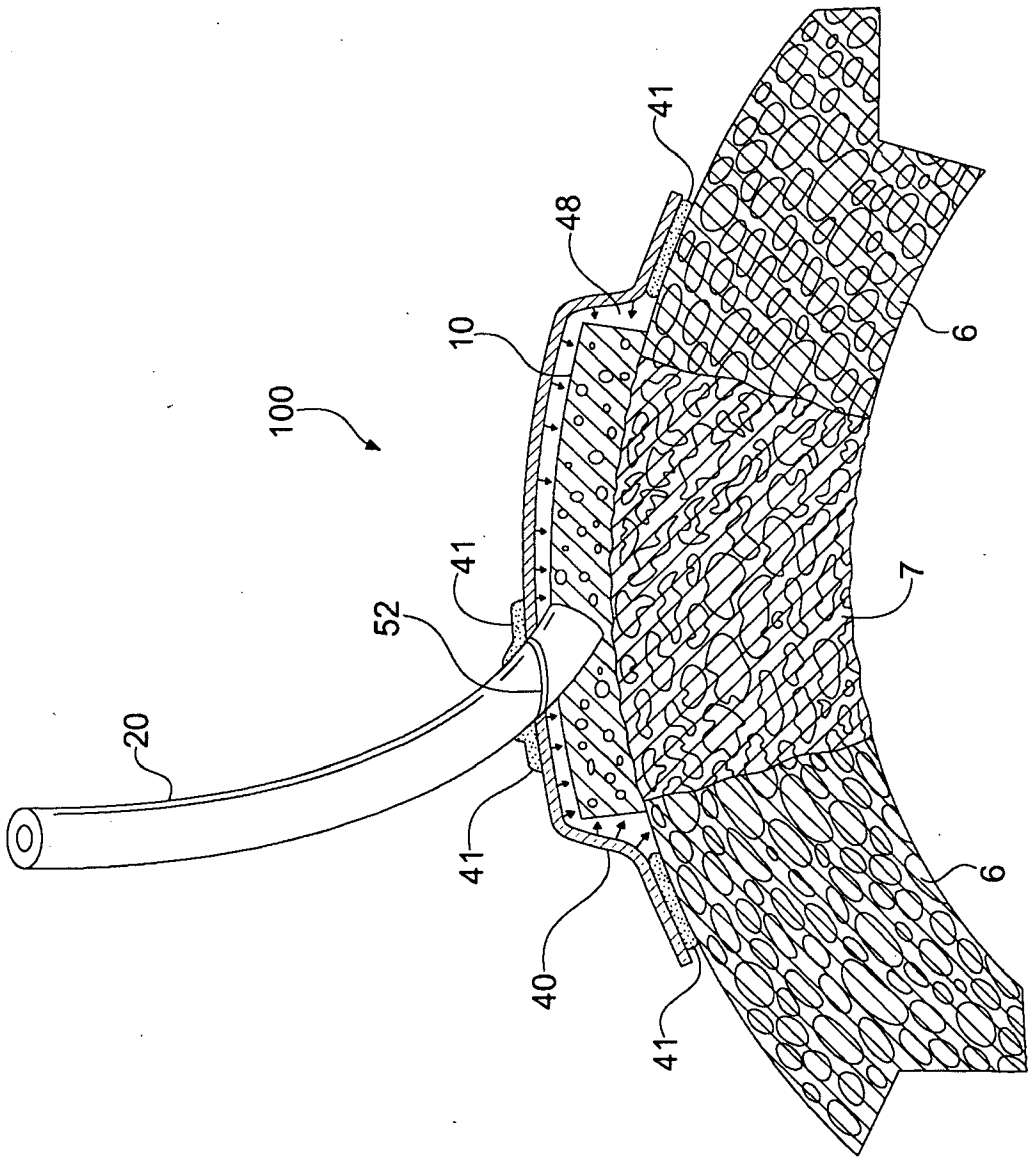


Fig. 2

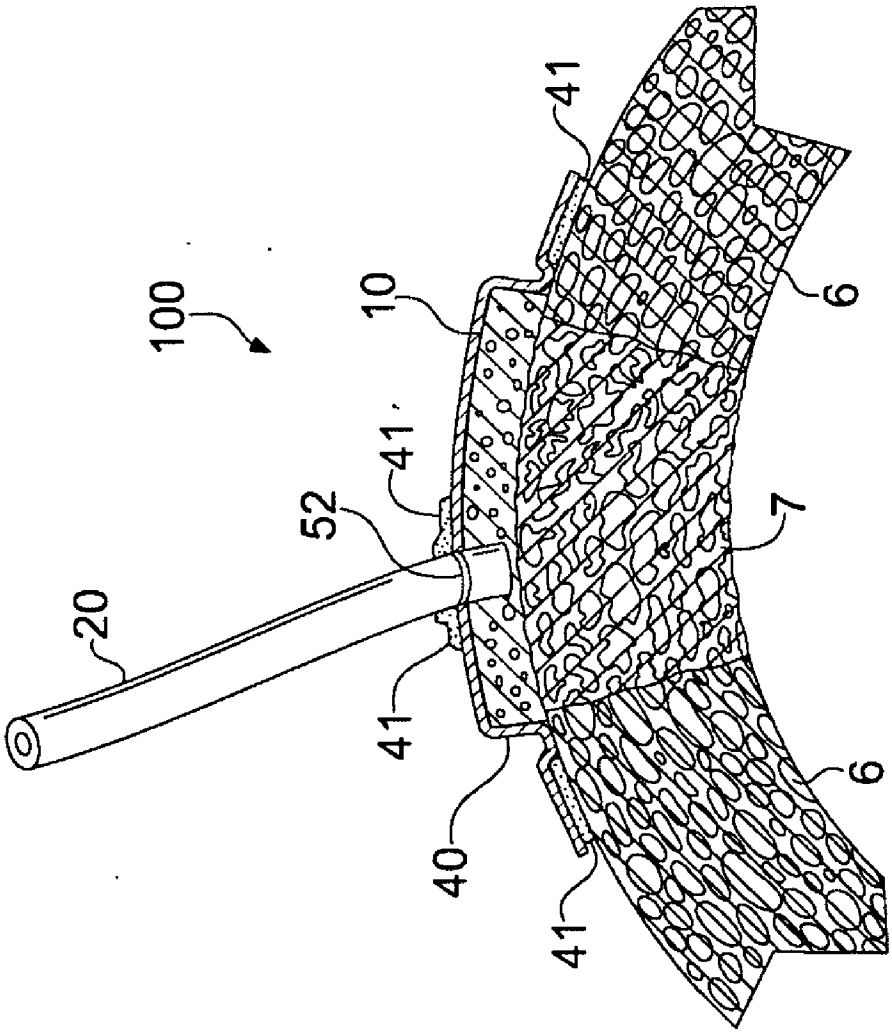


Fig. 3

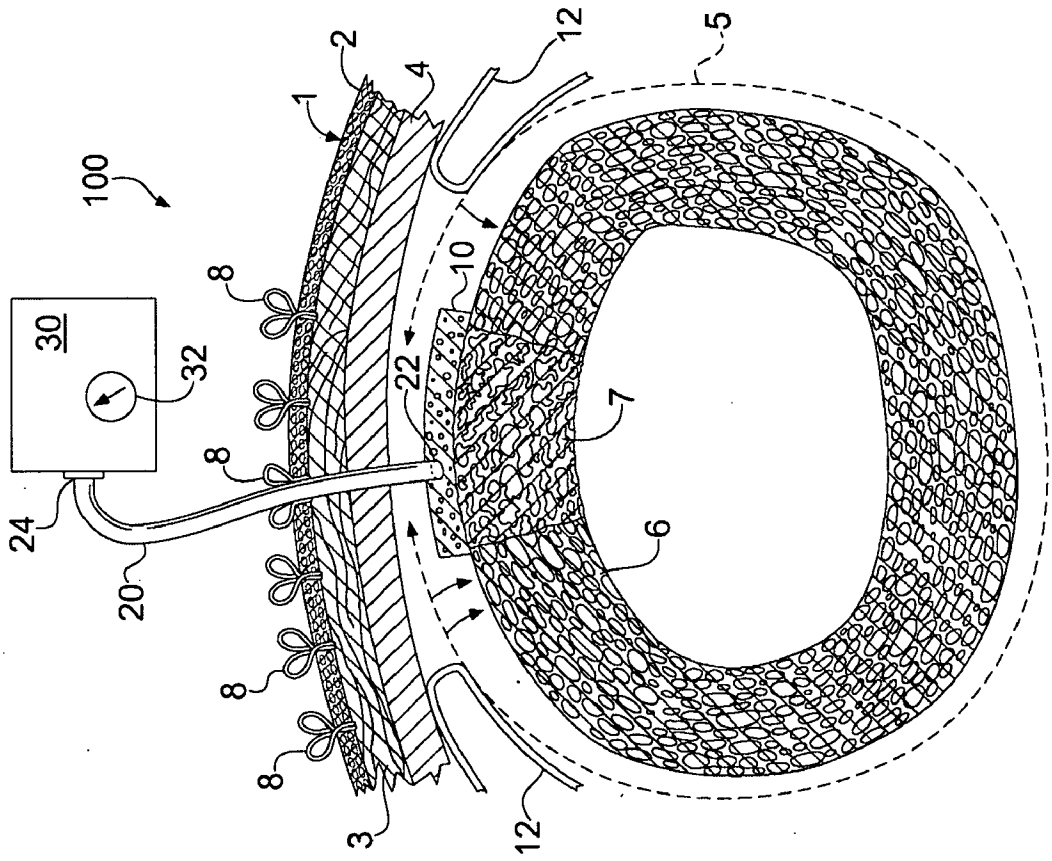
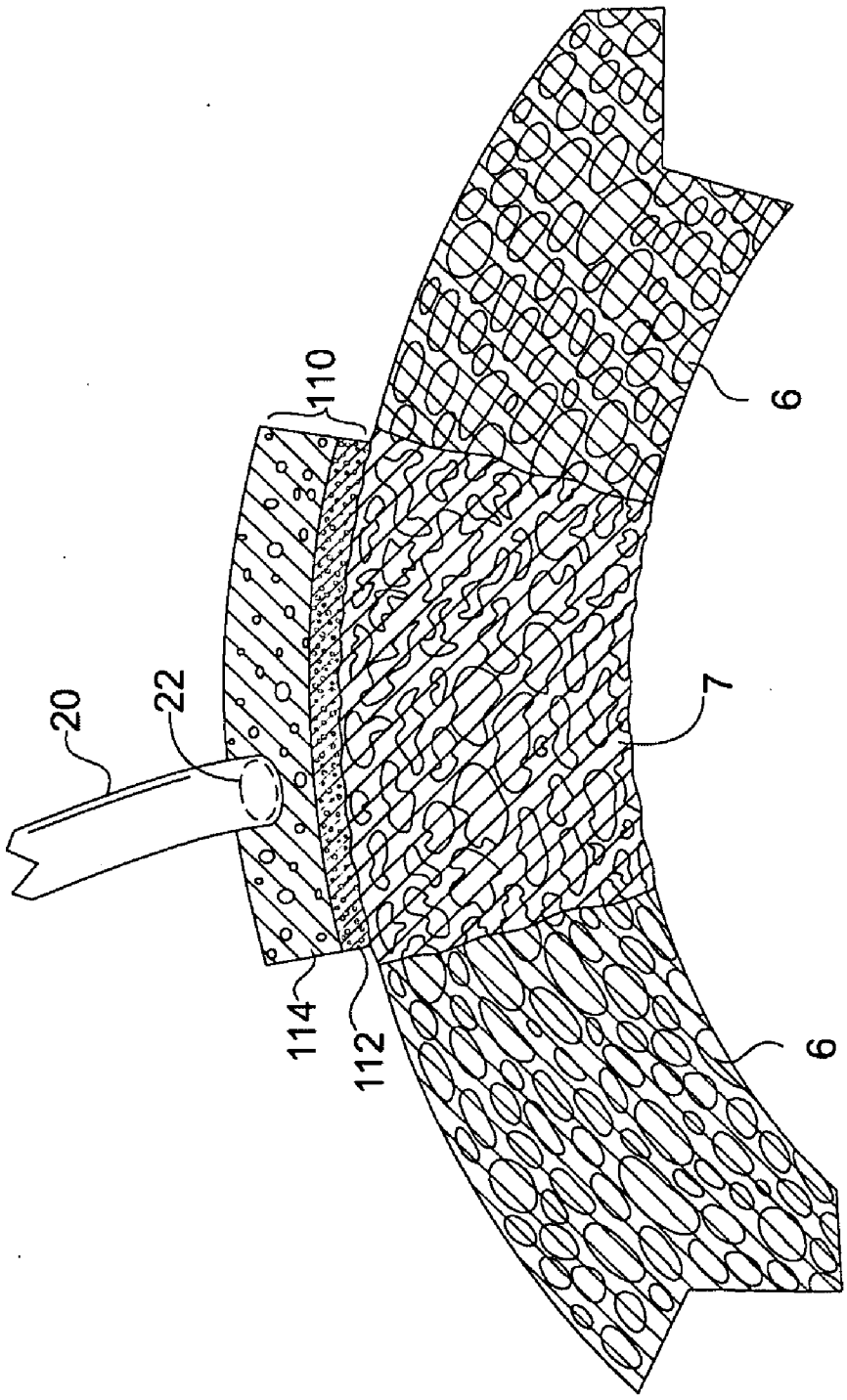


Fig. 4



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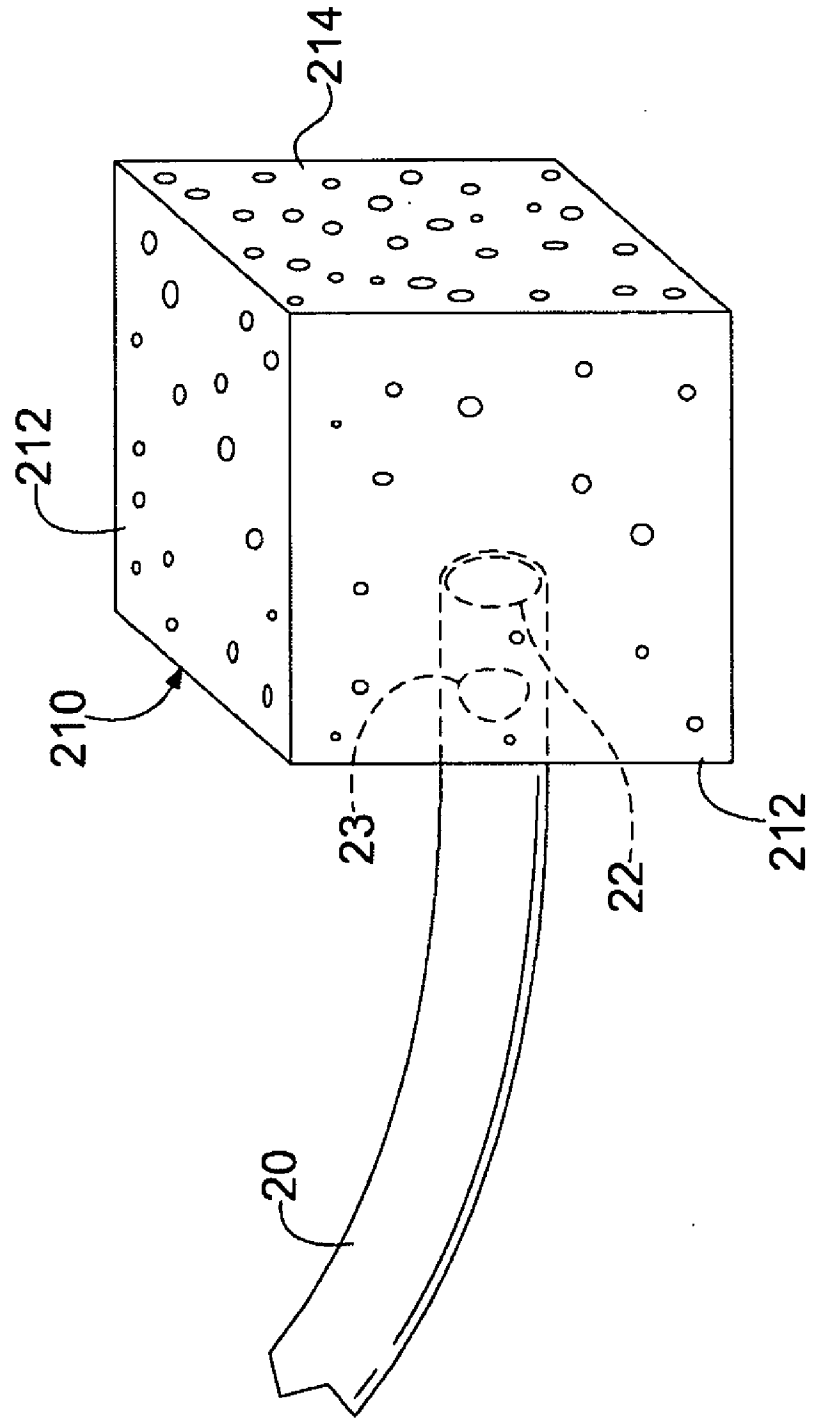

Fig. 6

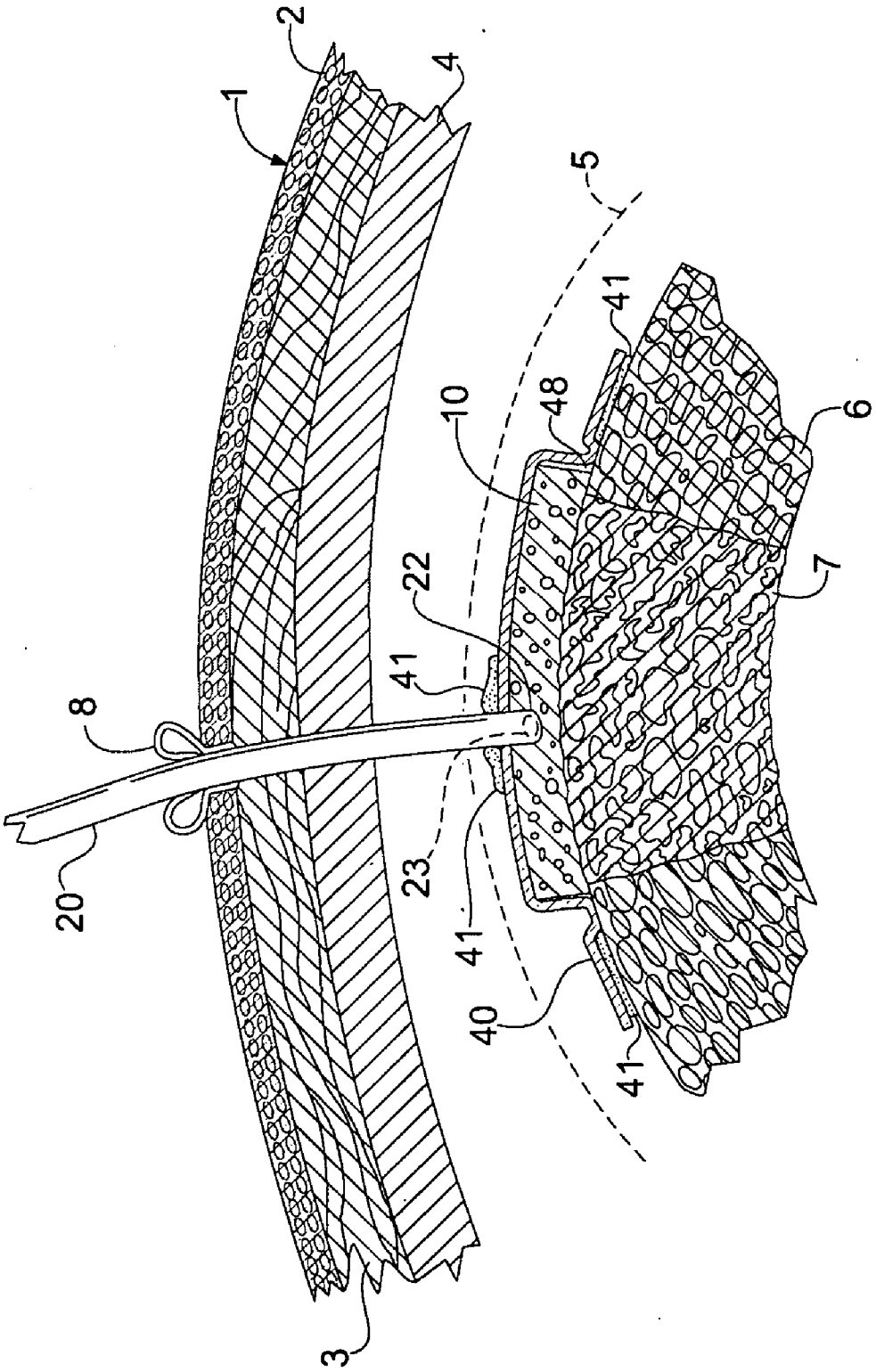

Fig. 7

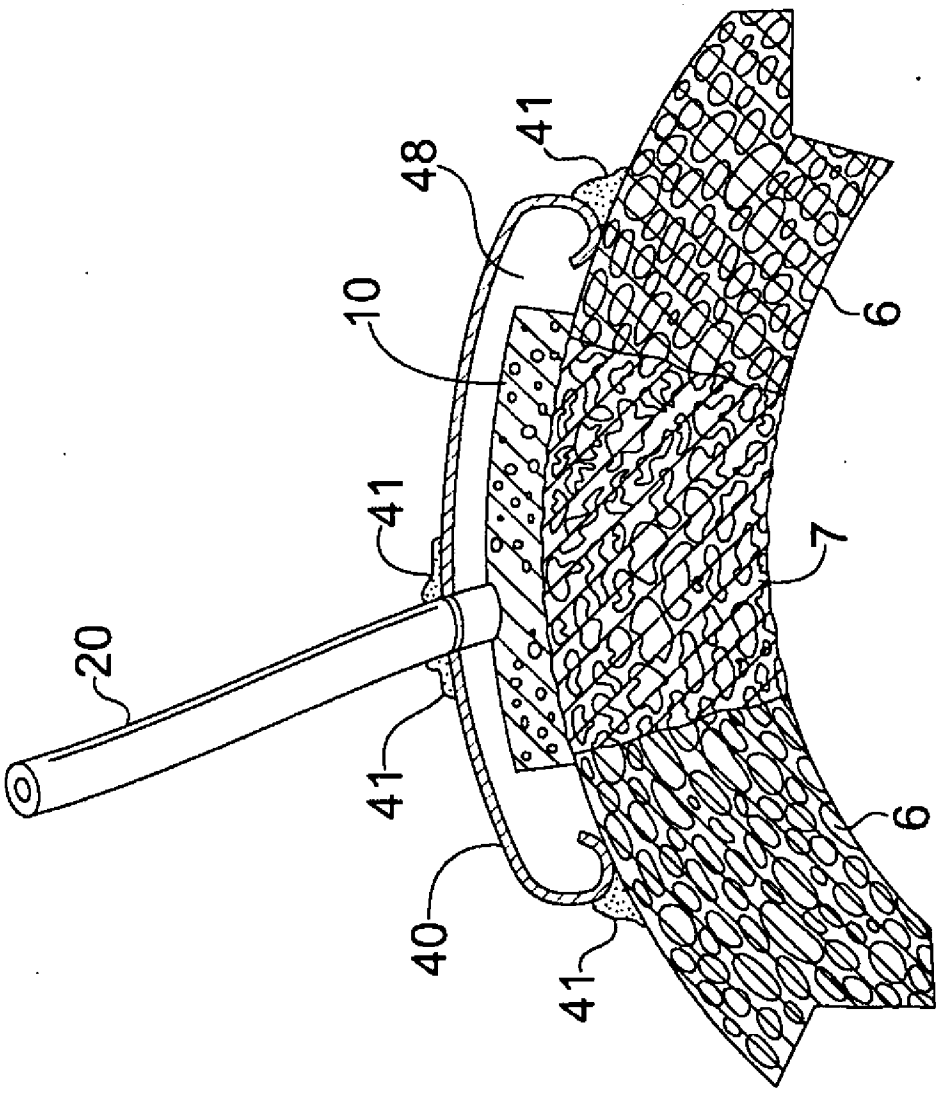

Fig. 8

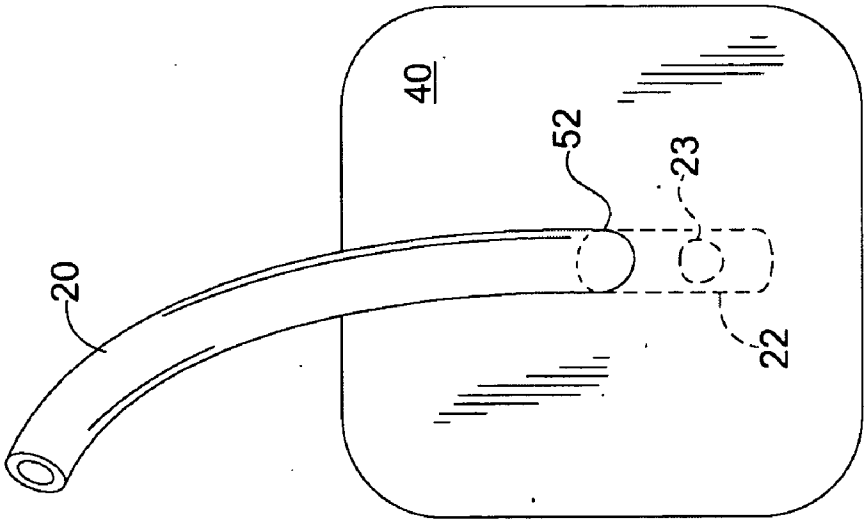

Fig. 10

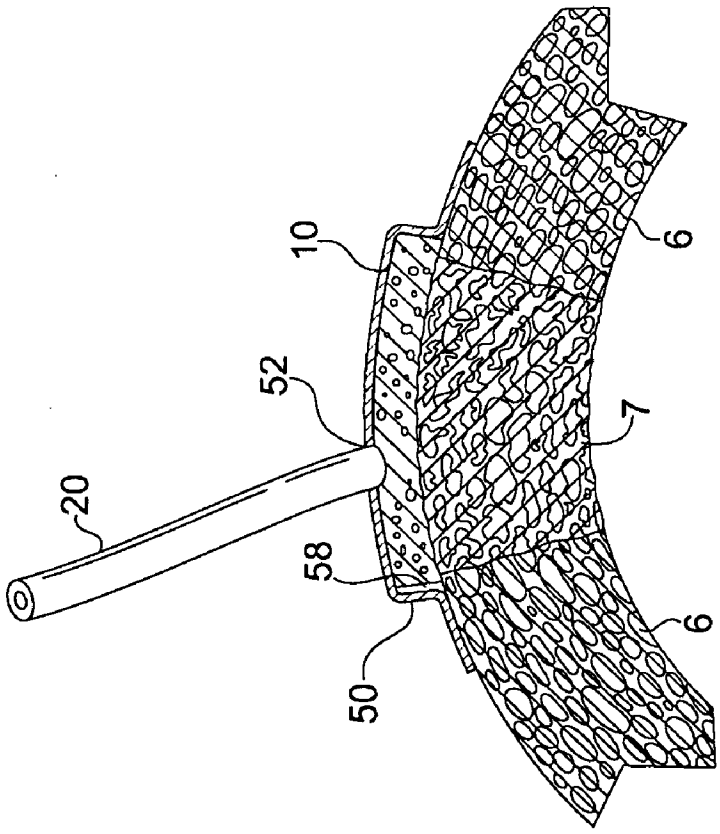

Fig. 9

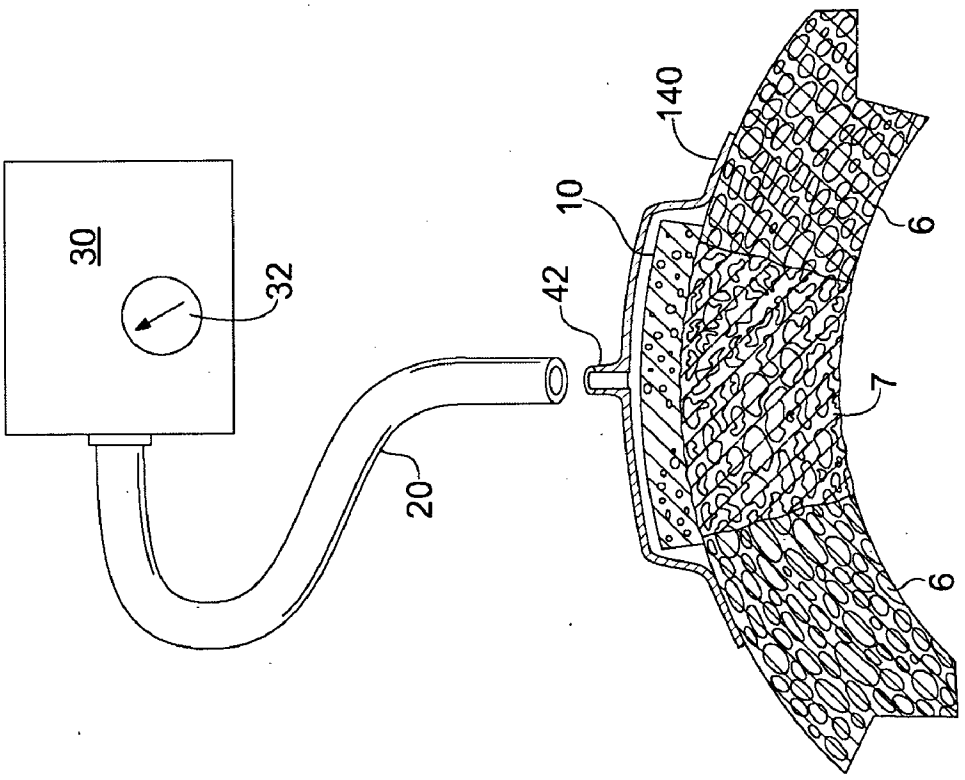

Fig. 11

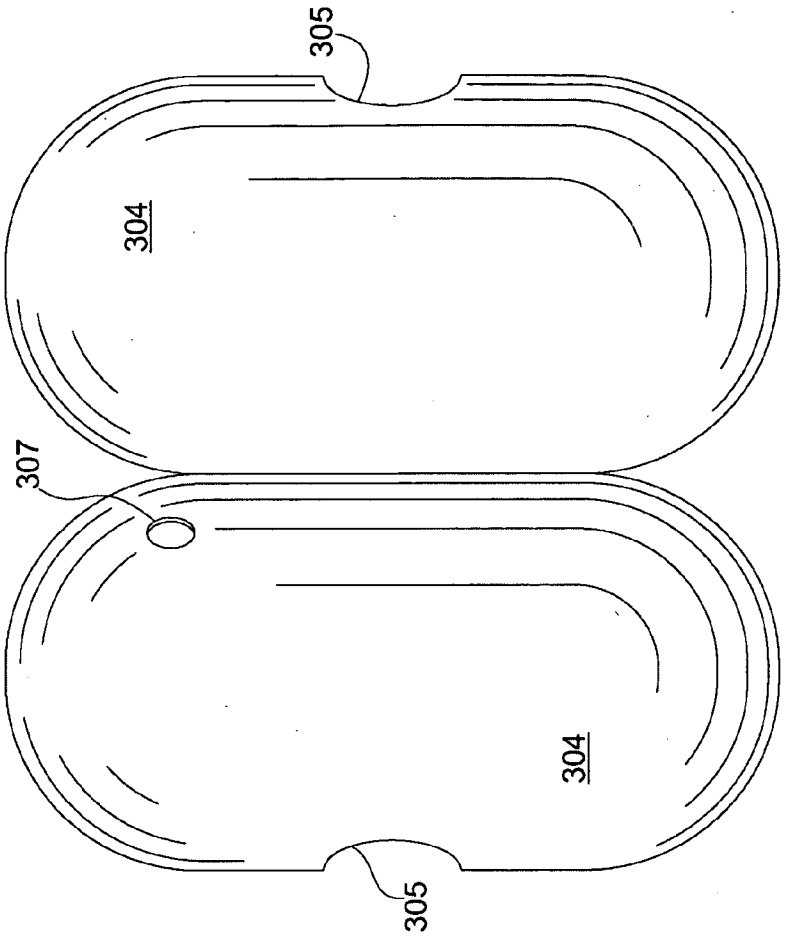

Fig. 13

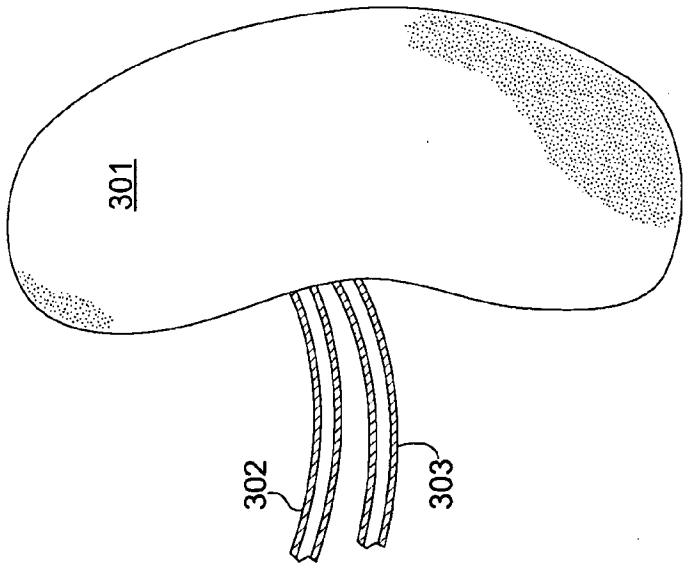

Fig. 12

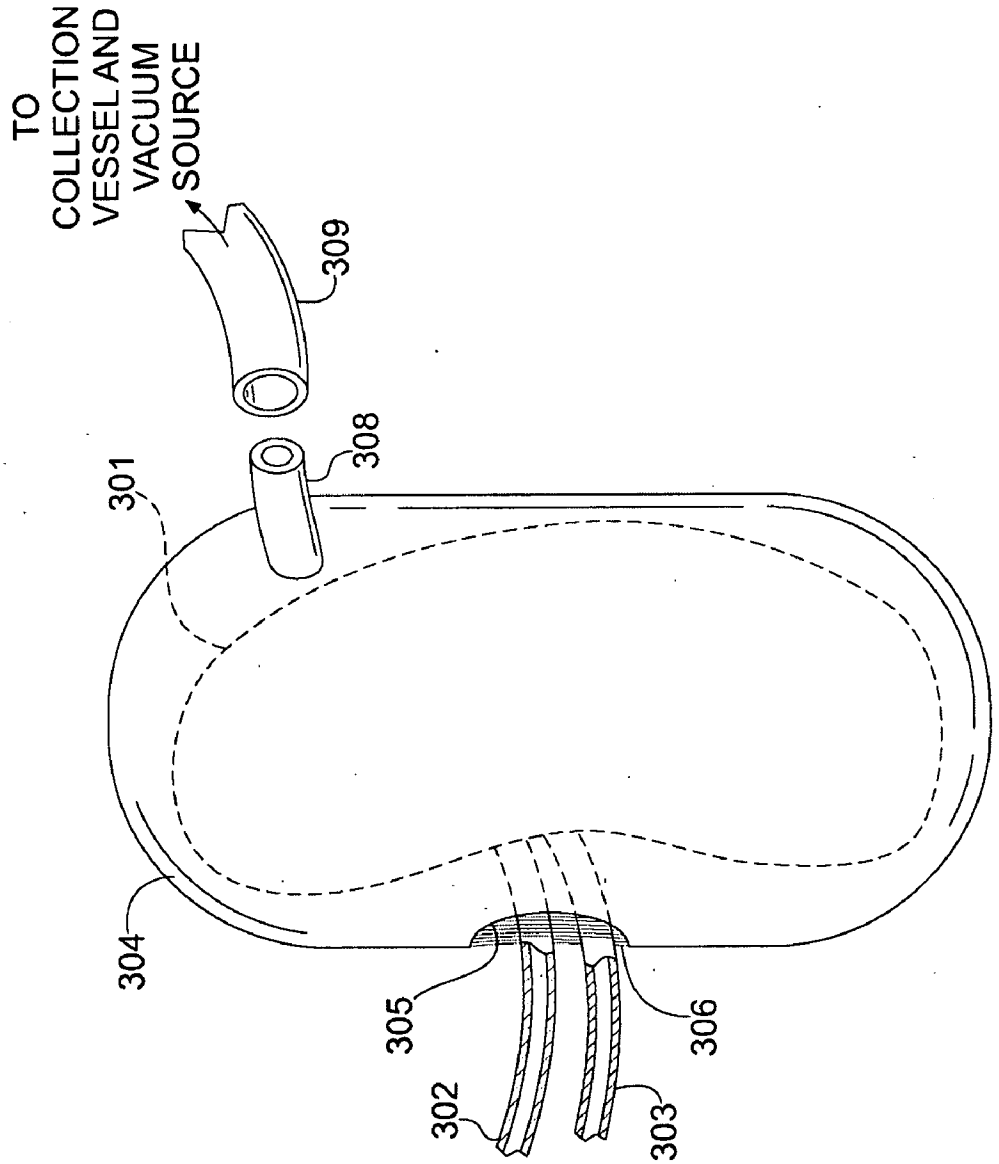
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Fig. 14

APPARATUS AND METHOD FOR CARDIAC TISSUE MODULATION BY TOPICAL APPLICATION OF VACUUM TO MINIMIZE CELL DEATH AND DAMAGE

RELATED APPLICATIONS

[0001] The present application claims the benefit of priority of U.S. Provisional Application 61/088,558, filed on Aug. 13, 2008 and U.S. Provisional Application No. 61/081,997, filed on Jul. 18, 2008, the entire contents of which are incorporated herein by reference.

FIELD OF THE INVENTION

[0002] The present invention relates generally to a method and apparatus for treating cardiac tissue, and more particularly, but not exclusively, to modulating ischemic and reperfused heart tissue with topical sub-atmospheric pressure to minimize cell death and damage.

BACKGROUND OF THE INVENTION

[0003] Myocardial ischemia occurs when a portion of the heart does not receive sufficient oxygen and energy substrates to meet its demand. This usually occurs because of a blockage in the artery due to either atherosclerotic plaque or thrombus formation. In a myocardial infarction there is an area of injury where the cells, because of lack of blood flow, will die immediately. There is a layer adjacent where there is impaired blood flow that is equivalent to the zone of stasis and there is a more peripheral unaffected zone. Unfortunately the infarcted heart will attempt to increase rate of contracture and overall work to compensate for areas of the heart that are not functioning adequately. Consequentially the areas that are in the "zone of stasis" are called upon to do more work which will increase the energy requirements placed upon them and will subsequently result in further progression of death. If left untreated, this ischemia will lead to an expanding zone of infarction that may eventually extend transmurally across the thickness of the ventricle.

[0004] Limiting the degree of infarction resulting from myocardial ischemia is paramount to improving both short- and long-term outcomes in patients. Therefore, in order to salvage this myocardial tissue, timely reperfusion (re-establishment of coronary blood flow) of the tissue must take place. The amount of salvageable tissue within an ischemic zone is dependent on the timeliness of reperfusion. While reperfusion halts the ischemic processes by delivering oxygen and nutrients (including energy substrates), this process also rapidly sets into motion a series of events and cascades that exacerbates injury, extending the area of necrosis beyond that encountered during ischemia alone. Much of this reperfusion injury appears to be inflammatory in nature, but inappropriately directed against host tissues instead of foreign substances. Being able to reduce this reperfusion injury allows for the salvage of the greatest amount of myocardium.

[0005] Reperfusion injury manifests itself in a number of ways, including myocardial dysfunction (myocardial stunning), arrhythmias, and a collection of events that result in lethal reperfusion injury. Currently, there are effective pharmacologic therapies to treat reperfusion arrhythmias, and myocardial stunning will generally resolve by itself given time, leaving the mediators of lethal reperfusion injury as the logical targets in an attempt to preserve ischemic-reperfused, but viable tissue.

[0006] There are a large number of potential mediators of lethal reperfusion injury including calcium overload, oxygen radicals, changes in osmotic gradients (and subsequent cell swelling), the mitochondrial permeability transition pore, and inflammation (itself a complex set of cascades and mediators including complement activation, leukocyte infiltration and pro-inflammatory cytokines and mediators). In addition, the cardioprotective effects of selective inhibition of any and all of these phenomenon, including antioxidants, sodium-hydrogen exchange inhibitors, anti-inflammatory agents (including adenosine, adhesion molecule antibodies and complement inhibitors) in animal models of myocardial ischemia-reperfusion are known. However, very few have demonstrated any degree of clinical success in people, likely due to the fact that these therapeutics act selectively at a single point within a cascade of events, or on a single facet of a very complex and multifaceted process. Thus, though the application of negative (or sub-atmospheric) pressure therapy to wounded cutaneous and subcutaneous tissue demonstrates an increased rate of healing compared to traditional methods (as set forth in U.S. Pat. Nos. 5,645,081, 5,636,643, 7,198,046, and 7,216,651, as well as US Published Application Nos. 2003/0225347, 2004/0039391, and 2004/0122434, the contents of which are incorporated herein by reference), there remains a need in the art for devices and methods for treating myocardial ischemia. In these type wounds of cutaneous and subcutaneous wounds the screen/dressing can often be easily and non-invasively changed at routine, pre-determined intervals without significant disruption to the healing tissues. However, when techniques are used to treat tissues or organs in which the overlying skin is intact, the overlying skin must be surgically disrupted by the deliberate creation of a wound through the overlying tissue to expose the tissue or organ that was originally injured. The overlying, originally healthy tissues which were disrupted to expose the injured tissue can be sutured closed over top of the injured tissue. This allows for negative pressure treatment of the wounded tissues with restoration of the suprawound tissues. Current commercially available embodiments of negative pressure dressings and cover are not biodegradable or bioresorbable. This lack of biodegradability/bioresorbability necessitates re-opening of the sutured incision, removal of the dressing and cover, placement of a new dressing and cover, and again suturing the incision closed. This sequence would have to be repeated until the original wounded tissue is healed, with one final re-opening of the incision to remove the dressing and cover. Every time the incision is opened to change or remove the dressing and cover, it increases the risk that the site will become infected.

SUMMARY OF THE INVENTION

[0007] The present invention relates to devices and methods for treating damaged heart tissue, such as myocardial infarction in the ischemic or early reperfusion phase, by treatment with sub-atmospheric (or negative) pressure. Treatment with the devices and methods of the present invention may salvage cells in the zone of stasis and thereby decrease the size of the infarct. Such treatment would be especially efficacious in endstage myocardial disease where bypass or stenting would not be possible. The treatment would also be useful as an adjunct to ECMO (extracorporeal membrane oxygenation) for resting the heart, following cardiac arrest, in situations with left main artery lesions, etc.

[0008] An exemplary negative pressure therapy device of the present invention may include a vacuum dressing, e.g., porous material, for placement over the tissue to be treated. The vacuum dressing may be bio-incorporable in nature so that a second stage for removal would not be required. (As used herein the term "bio-incorporable" is defined to describe a material that may be left in the patient indefinitely and is capable of being remodeled, resorbed, dissolved, and/or otherwise assimilated or modified.) The device of the present invention may also include a bio-incorporable overlay cover for placement over the vacuum dressing to form a sealed enclosure in which sub-atmospheric pressure may be provided and maintained to the vacuum dressing and the tissue to be treated. The overlay cover may be adherent to the dressing and extend beyond the vacuum dressing to permit attachment of the overlay cover to surrounding non-damaged heart tissue. The overlay cover may be gelatinous in nature to contour to the heart and may be sufficiently pliable so as not to interfere with cardiac function. The overlay cover may be secured to the myocardium with fibrin glue, mini-staples, or sutures.

[0009] In use, the device of the present invention may be placed thoracoscopically over the area of muscle that has infarcted and over the adjacent zone of stasis. The device may be placed through a small incision made in the chest wall and perforated through the pericardium. The vacuum dressing may be collapsible in structure such that it can be rolled up or folded so as to be small enough for insertion through a thoracoscope tube. The epicardium may be perforated with a CO_2 or similar laser or other cutting instrument to expose the underlying ischemic myocardium. The vacuum dressing may then be placed directly over this ischemic area. The overlay cover may also be placed and secured to surrounding heart tissue endoscopically as well. A vacuum tube, e.g., a small catheter, may then be introduced so that the distal end of the vacuum tube is in gaseous communication with the enclosure under the overlay cover to supply sub-atmospheric pressure to the enclosure and the tissue to be treated. The other end of the vacuum tube may then be placed in gaseous communication with a vacuum source to produce sub-atmospheric pressure, and the vacuum source may be activated to supply the sub-atmospheric pressure to effect negative pressure therapy of the damaged heart tissue. In addition, the sub-atmospheric pressure may be supplied intermittently at a rate that is matched to the heart rate.

[0010] The present invention may also provide delayed treatment of myocardial infarction where there is already a stable zone of myocardial cell death. Again through an endoscope and a small incision in the chest wall, a bio-incorporable vacuum dressing may be placed on the area that is infarcted. Again, exposure of the myocardium involved and adjacent myocardium may be required and provided with a CO_2 or similar cutting device to perforate the epicardium. The vacuum dressing may be modified so that a lattice of myocardial or peripheral muscle cells may be incorporated within it. The vacuum dressing may also incorporate a small catheter with the ability to reinfuse additional myocardial cells, pleuripotent progenitor cells, or peripheral muscle cells at subsequent serial times. In areas where there is near complete cell death or there is little or no contraction of the muscle cells in the damaged cardiac tissue, new contractile cells could be seeded to replace and restore the contractile function of the damaged cardiac tissue. Initially, peripheral muscle or peripheral muscle cells grown from culture could be used. These cells have a finite life cycle and would be expected to fatigue over time. The myocardium could be biopsied at the time of the treatment of the initial treatment and myocardial cells removed and cultured to create a larger mass of viable of cells. The harvested myocardial cells could be maintained in culture and used for later periodic infusion to develop a myocardial patch that would cover the area of previous infarction. Also, progenitor cells could be harvested and immediately infused to the area of damaged cardiac tissue, or they could be grown in culture and periodically infused to the area of damaged cardiac tissue with the expectation that they would develop into cardiac myocytes. Over time the introduced cells would be induced to undergo mitosis or self-replication thus increasing the functional mass of the heart. The ability to progressively add cells that would be progressively vascularized is a major step in regenerative medicine where presently only a sheet of cells can be expected to survive.

[0011] More specifically, in one of its aspects the present invention provides a method for treating damaged cardiac tissue using sub-atmospheric pressure. The method comprises placing a porous material in direct or indirect contact with the damaged cardiac tissue to provide gaseous communication between one or more pores of the porous material and the damaged cardiac tissue. The porous material may comprise at least one of an electrospun material, a cast material, an open-cell foam, or a printed material. Alternatively or additionally, the porous material may comprise a bio-incorporable material. The porous material may include, for example, collagen, chitosan, polycaprolactone, polyglycolic acid, polylactic acid, and combinations thereof. In addition, the porous material may be a polyvinyl alcohol foam which may be disposed in direct contact with the damaged cardiac tissue.

[0012] The porous material may be sealed in situ over the damaged cardiac tissue to provide a region about the damaged cardiac tissue for maintaining sub-atmospheric pressure at the damaged cardiac tissue. The porous material may be operably connected with a vacuum source for producing sub-atmospheric pressure at the damaged cardiac tissue, and the vacuum source activated to provide sub-atmospheric pressure at the damaged cardiac tissue. The sub-atmospheric pressure may be maintained at the damaged cardiac tissue for a time sufficient to reduce edema (thus restoring contractility and compliance), decrease interstitial pressure, remove inflammatory mediators, remove inflammatory amplifiers, modulate intracellular mediators, increase reperfusion and microvascular flow, decrease microvascular plugging, and/or decrease retention of inflammatory cells within the damaged cardiac tissue. Micro and macro deformation of the cardiac tissue being treated would increase vasculoneogenesis or the formation of new blood vessels in the ischemic tissue. This would increase the survivability of the cardiocytes and ultimately improve function of the ischemic portion of the heart. In addition, macro and micro deformation of small arterioles already existing in the heart would result in their physical reorientation into the areas of ischemic tissue, thus increasing perfusion and ultimately function.

[0013] For example, the sub-atmospheric pressure may be maintained at about 25-125 mm Hg below atmospheric pressure. The method may also include locating a cover, such as a bio-incorporable cover, over damaged cardiac tissue and sealing the cover to tissue proximate the damaged cardiac tissue, e.g., to non-damaged cardiac tissue, for maintaining sub-atmospheric pressure at the damaged cardiac tissue. The cover may be provided in the form of a self-adhesive sheet

which may be located over the damaged cardiac tissue. In such a case, the step of sealing the cover may include adhesively sealing and adhering the self-adhesive sheet to tissue surrounding the damaged cardiac tissue to form a seal between the sheet and tissue surrounding the damaged cardiac tissue.

[0014] In another of its aspects the present invention provides an apparatus for treating damaged cardiac tissue. The apparatus includes a porous material for treating damaged cardiac tissue having a pore structure configured to permit gaseous communication between one or more pores of the porous material and the cardiac tissue to be treated. The porous material may include at least one of an electrospun material, a cast material, and a printed material. Alternatively or additionally, the porous material may comprise a bio-incorporable material. In such instances, it may also be beneficial for the porous material to be formulated in such a manner that the outer edges of the porous material would be resorbed or degraded more quickly than the inner portion. The rate of removal (resorption/degradation) of the porous material could be matched to the rate of formation of new tissue. One way to control the rate of degradation or resorption is by varying the number of crosslinks introduced into the porous material.

[0015] The apparatus may also include a vacuum source for producing sub-atmospheric pressure; the vacuum source may be disposed in gaseous communication with the porous material for distributing the sub-atmospheric pressure to the cardiac tissue. The porous material may have, at least at a selected surface of the porous material, pores sufficiently small to prevent the growth of tissue therein. In addition, the porous material may have, at least at a selected surface of the porous material, a pore size smaller than the size of fibroblasts and cardiac cells, and may have a pore size at a location other than the selected surface that is larger than that of fibroblasts and cardiac cells. The pore size of the porous material may be large enough to allow movement of proteins the size of albumin therethrough. Also, the porous material may include at least one surface that is sealed to prevent the transmission of sub-atmospheric pressure therethrough. The apparatus may also include a cover, such as a bio-incorporable cover, configured to cover the damaged cardiac tissue to maintain sub-atmospheric pressure under the cover at the damaged cardiac tissue.

[0016] The bio-incorporable porous material and/or cover may be constructed from synthetic materials such as polyglycolic acid, polylactic acid, or poly-o-citrate, or they can be constructed of naturally occurring molecules such as collagen, elastin, or proteoglycans. Combinations of synthetic molecules, combinations of naturally occurring molecules, or combinations of synthetic with naturally occurring molecules can be used to optimize the material properties of the porous material and cover.

[0017] An example of a material which may be used to fabricate the porous material is polycaprolactone (PCL). In one exemplary formulation, polycaprolactone is mixed with sodium chloride (1 part caprolactone to 10 parts sodium chloride) and placed in a sufficient volume of chloroform to dissolve the components. The solution is poured into an appropriately sized and shaped container and allowed to dry for twelve hours. The sodium chloride is then leached out in water.

[0018] A second exemplary cast formulation for the porous material is chitosan, 1.33% (weight/volume) in 2% acetic acid. The solution (20 ml) is poured into an appropriately sized container and frozen for 2 hours at −70° C., then transferred to a lyophylizer and vacuum applied for 24 hours. The freeze dried dressing is then crosslinked with 2.5 to 5% glutaraldehyde vapor for 12 to 24 hours.

[0019] Thus, the present invention provides devices and methods for minimizing the progression of pathologic processes, minimizing the disruption of physiological cardiac integrity, and minimizing the interference with cardiac blood flow and nutrition and increasing revascularization of ischemic areas of the heart by vascular neogenesis and reorientation of existing vessels. By decreasing cardiac edema and interstitial pressure the risk of cardiac cell death and compromise may be minimized. In addition, the present invention facilitates the removal of mediators, degradation products, and toxins that enhance the inflammatory and pathophysiological response in the damaged cardiac tissue.

BRIEF DESCRIPTION OF THE DRAWINGS

[0020] The foregoing summary and the following detailed description of the preferred embodiments of the present invention will be best understood when read in conjunction with the appended drawings, in which:

[0021] FIG. **1** schematically illustrates a partial cross-sectional view of an exemplary configuration of an apparatus of the present invention in situ prior to the application of sub-atmospheric pressure;

[0022] FIG. **2** schematically illustrates the partial cross-sectional view of FIG. **1** as a sub-atmospheric pressure is being applied;

[0023] FIG. **3** schematically illustrates the partial cross-sectional view of FIG. **1** after sub-atmospheric pressure has been applied;

[0024] FIG. **4** schematically represents a cross-sectional view of an exemplary configuration of the present invention in situ in which the tissues overlying the heart have been closed around the tube to create a space capable of maintaining a vacuum so no overlay cover is required;

[0025] FIG. **5** schematically represents a partial cross-sectional view of the apparatus of the present invention in situ in which the porous material is layered with a smaller pore layer adjacent to the damaged tissue and a layer with larger pores above the smaller pore layer;

[0026] FIG. **6** schematically represents a view of an exemplary configuration of a porous material of the present invention in which only one side of the porous material is open and not sealed;

[0027] FIG. **7** schematically represents a cross-sectional view of an exemplary configuration of the present invention in which an overlay cover has been placed over the porous material and potential leaks sealed with fibrin glue;

[0028] FIG. **8** schematically represents a partial cross-sectional view of an exemplary configuration of the present invention in which the edges of the overlay cover have been turned under;

[0029] FIG. **9** schematically represents a cross-sectional view of an exemplary configuration of the present invention in which the overlay cover is self adhesive;

[0030] FIG. **10** schematically represents an exemplary configuration of the cover of the present invention in which the tube passes through the overlay cover;

[0031] FIG. **11** schematically represents a partial cross-sectional view of the vacuum tube attaching to the overlay cover;

[0032] FIG. **12** schematically represents a kidney, with artery and vein;

[0033] FIG. **13** schematically represents an open clamshell or bi-valve chamber for application of sub-atmospheric pressure; and

[0034] FIG. **14** schematically represents a kidney disposed within the chamber of FIG. **13**.

DETAILED DESCRIPTION OF THE INVENTION

[0035] Referring now to the figures, wherein like elements are numbered alike throughout, the present invention relates to devices and methods that use sub-atmospheric (or negative) pressure for treating damaged cardiac tissue, where "damaged" tissue is defined to include tissue that is injured, compromised, or in any other way impaired, such as damage due to trauma, disease, infection, surgical complication, or other pathologic process, for example. More specifically, the devices and methods of the present invention can effect treatment of myocardial infarction.

[0036] An exemplary configuration of a sub-atmospheric cardiac treatment device **100** of the present invention may include a vacuum source **30** for supplying sub-atmospheric pressure via a tube **20** to a porous material **10**, such as a bio-incorporable porous material, disposed in direct or indirect contact with the damaged cardiac tissue **7**, FIGS. **1-4**. As used here, "indirect contact" is defined to mean placement of an intermediate material for transmitting sub-atmospheric pressure in contact with both the damaged cardiac tissue **7** and the porous material **10**. In this regard, the porous material **10** may be structured to deliver and distribute sub-atmospheric pressure to the damaged cardiac tissue **7**. Alternatively, the porous material **10** may be comprised of a material that needs to be removed after sub-atmospheric therapy is given, which could require a second surgery. The cardiac treatment device **100** may be applied to a patient by locating a porous material **10** in contact with the damaged cardiac tissue **7** to provide gaseous communication between one or more pores of the porous material **10** and the damaged cardiac tissue **7**. A tube **20** may be connected to the porous material **10** at a distal end **22** of the tube **20**, and the porous material **10** may be sealed in situ by sutures **8** in the skin **1** and subcutaneous tissues **2** to provide a region about the damaged cardiac tissue **7** for maintaining sub-atmospheric pressure, FIG. **4**. The proximal end **24** of the tube **20** may be attached to a vacuum source **30** to operably connect the porous material **10** to the vacuum source **30** for producing sub-atmospheric pressure at the damaged cardiac tissue **7** upon activation of the vacuum source **30**. Optionally, an overlay cover **40**, such as a bio-incorporable overlay cover **40**, may be located over the damaged cardiac tissue **7** and sealed proximate the damaged cardiac tissue **7** to maintain sub-atmospheric pressure at the damaged cardiac tissue **7**.

[0037] Turning to FIGS. **1-4** in greater detail, an exemplary configuration of a sub-atmospheric pressure cardiac treatment device **100** of the present invention is illustrated in partial cross-section with the porous material **10** in contact with the damaged cardiac tissue **7**. An overlay cover **40** covers the porous material **10** and may extend onto healthy cardiac tissue **6** creating an enclosed space **48**. An adhesive **41**, such as fibrin glue or other material, may be placed between the overlay cover **10** and the healthy cardiac tissue **6**. The adhesive **41** may also or alternatively be placed around the periphery of the overlay cover **10** to prevent leaks, and may also be placed around a passthrough **52** where the tube exits from the overlay cover **10** to prevent leaks. FIG. **1** depicts the device **100** prior to application of sub-atmospheric pressure. FIG. **2** depicts the device **100** as sub-atmospheric pressure is being applied, and the enclosed space **48** decreases in volume as fluid and gas are evacuated from the enclosed space **48** and the overlay cover **40** conforms to the porous material **10**. FIG. **3** depicts the device **100** after sub-atmospheric pressure has been applied, with the overlay cover **40** conforming to the shape of the porous material **10**.

[0038] Turning to FIG. **4** specifically, an exemplary configuration of a sub-atmospheric cardiac treatment device **100** of the present invention is illustrated in situ in a patient with surrounding tissues shown in partial cross-section. The tissues illustrated include the skin **1** and subcutaneous tissue **2**, muscle **3**, bone **4**, pericardium **5**, healthy non-damaged cardiac tissue **6**, the damaged cardiac tissue **7**, and the pleural tissues **12**. To provide access to the damaged cardiac tissue **7**, a portion of the pericardium **5** may be missing due to surgical dissection or injury. A porous material **10**, such as an open-cell collagen material, may be placed in the subcutaneous space in contact (direct or indirect) with the cardiac tissue **7** to be treated with sub-atmospheric pressure to decrease edema and interstitial pressure, oxygen radicals, inflammatory mediators, and other molecules which may adversely affect cellular resuscitation or viability within the damaged cardiac tissues to improve physiologic function, for example. The distal end **22** of the tube **20** may connect to the porous material **10** and the tube **20** may exit the body through an incision. The tube **20** may have one or more fenestrations **23** in that portion of the tube **20** in contact with the porous material **10**, FIG. **6**. The tissues between the cardiac tissue **7** up to and including the skin **1** are closed with, for example sutures **8**, to create an airtight seal capable of maintaining a vacuum. When sub-atmospheric pressure is applied, the edges of the incised tissues **1-5** are drawn together and the pleural tissues **12** are drawn toward the porous material to help maintain the vacuum. The proximal end of the tube **24** may be connected to a vacuum source **30** and the level of sub-atmospheric pressure controlled by a controller **32**. The vacuum source **30** may include a canister to collect any fluid removed.

[0039] The cover **40** may serve to further confine the region about the damaged cardiac tissue **7** at which sub-atmospheric pressure is maintained. That is, as illustrated in FIGS. **1-3**, **7-9**, the cover **40**, **50** provides an enclosed space/region **48**, **58** about the damaged cardiac tissue **7** under the cover **40**, **50**, which can serve to isolate the tissues exterior to the cover **40**, **50** from exposure to the sub-atmospheric pressure applied to the damaged cardiac tissue **7**. In contrast, as illustrated in FIG. **4**, in the absence of an overlay cover, sub-atmospheric pressure delivered to the porous material **10** and damaged cardiac tissue **7** may draw the surrounding tissues, such as the pericardium **5** and pleural tissues **12**, inward towards the tube **20** and porous material **10** along the directions of the arrows shown in FIG. **4**. In this regard the stretched and/or moved tissues, such as pericardium **5** and pleural tissues **12** can help to confine the applied sub-atmospheric pressure to a region between the pericardium **5** and the damaged cardiac tissue **7**. In addition the covers **40**, **50** may further protect the damaged cardiac tissue **7** from exogenous infection and contamination beyond the protection already afforded by the porous material **10** and sutured skin **1** and subcutaneous tissue **2**. Likewise, the covers **40**, **50** may further protect the damaged cardiac tissue **7** from the spread of infections from the surrounding tissues (such as cardiac abscesses and mediastinitis).

[0040] To assist in maintaining the sub-atmospheric pressure at the damaged cardiac tissue **7**, a flexible overlay cover **40** (FIG. **7**), or a self adhesive flexible overlay cover **50** (FIG. **9**) may be provided over the damaged cardiac tissue **7** to provide a region **48**, **58** about the damaged cardiac tissue **7** where sub-atmospheric pressure may be maintained, FIGS. **7**, **8**. Specifically, with reference to FIGS. **7**, **8**, and **9**, an overlay cover **40**, **50** may be provided over the damaged cardiac tissue **7** and porous material **10** by adhering the cover **40**, **50** to cardiac tissues proximate the damaged cardiac tissue **7** to define an enclosed region **48**, **58** about the damaged cardiac tissue **7** and porous material **10**. For instance, the cover **40** may be glued to cardiac tissue using an adhesive **41**, such as a fibrin glue. The adhesive **41** may comprise an auto-polymerizing glue and/or may desirably include a filler to provide the adhesive **41** with sufficient bulk to permit the adhesive **41** to conform to the shapes of the potentially irregular surfaces which the adhesive **41** contacts. The adhesive **41** may be provided as a separate component or as a portion of the cover **40**. For the flexible overlay cover **40**, an outside edge or border of the flexible overlay cover **40** may either be rolled away from (or laid flat on) the non-damaged cardiac tissue **6** or rolled under (or toward) the damaged cardiac tissue **7**, FIGS. **7**, **8**. The adhesive **41** may be placed between the edge of the overlay cover **40** and the healthy cardiac tissue **6** to promote an airtight seal. The adhesive **41** may also be placed around the tube **20** where it exits through the overlay cover **40**. Alternatively, a self-adhesive flexible overlay cover **50** may be curled out away from the damaged cardiac tissue **7** so that the underside of the cover **50** (that side facing the porous material **10**) may then contact with the surrounding non-damaged cardiac tissue **6**, FIG. **9**.

[0041] In addition to an open-cell collagen material, the porous material **10** may also include a polyglycolic and/or polylactic acid material, a synthetic polymer, a flexible sheet-like mesh, an open-cell polymer foam, a foam section, a porous sheet, a polyvinyl alcohol foam, a polyethylene and/or polyester material, or other suitable materials which may be fabricated by electrospinning, casting, or printing, for example. Such materials include a solution of chitosan (1.33% weight/volume in 2% acetic acid, 20 ml total volume) which may be poured into an appropriately sized mold. The solution is then frozen for 2 hours at −70° C., and then transferred to the lyophylizer and vacuum applied for 24 hours. The dressing may be cross-linked by 2.5%-5% glutaraldehyde vapor for 12-24 hours to provide a cast porous material.

[0042] Additionally, the porous material **10** may be made by casting polycaprolactone (PCL). Polycaprolactone may be mixed with sodium chloride (1 part caprolactone to 10 parts sodium chloride) and placed in a sufficient volume of chloroform to dissolve the components. A desired amount, e.g., 8 ml, of the solution may be poured into an appropriately sized and shaped container and allowed to dry for twelve hours. The sodium chloride may then be leached out in water for 24 hours.

[0043] The overlay cover **40** may also be bio-incorporable and may consist of an electrospun mixture of Type I collagen and poly 1,8-octanediol citrate (POC) (80%:20% weight/weight). The solution concentration may be 15% dissolved in hexafluoro-2 proponal (HFP) with a total volume of 9.5 ml. The solution may then be ejected from a syringe through an 18 gauge needle at a flow rate of 1-3 ml/hour. The voltage may be 25 KV with a working distance of 20-25 cm. The film may then be heat polymerized at 80° C. for 48 hours (of 90° C. for 96 hours) and cross-linked in 2.5%-10% glutaraldehyde vapor for 24 hours.

[0044] It is also possible to use electrospun materials for the porous material **10** and cast materials for the overlay cover **40**. One example of a formulation and method for making an electrospun porous material **10** is a combination of collagen Type I:chondroitin-6-sulfate (CS):poly 1,8-octanediol citrate (POC) in a ratio of 76%:4%:20%: by weight. Two solvents were utilized for the collagen/CS/POC. The CS was dissolved in water and the collagen and POC were dissolved in 2,2,2-trifluoroethanol (TFE). A 20% water/80% TFE solution (volume/volume) solution was then used. For electrospinning, the solution containing the collagen:CS:POC mixture was placed in a 3 ml syringe fitted to an 18 Ga needle. A syringe pump (New Era Pump Systems, Wantaugh, N.Y.) was used to feed the solution into the needle tip at a rate of 2.0 ml/hr. A voltage of 10-20 kV was provided by a high voltage power supply (HV Power Supply, Gamma High Voltage Research, Ormond Beach. FL) and was applied between the needle (anode) and the grounded collector (cathode) with a distance of 15-25 cm. The dressings were then cross-linked with glutaraldehyde (Grade II, 25% solution) and heat polymerized (80° C.) for 48 hours. It is also possible to electrospin collagen Type I dressings starting with an initial concentration of 80 mg/ml of collagen in 1,1,1,3,3,3-hexafluoro-2-propanol (HFP), then use the same electrospinning conditions as the collagen:CS:POC combination.

[0045] Examples of cast overlay cover formulas include the use of 1,8 poly (octanediol) citrate (POC) or other combinations of diol citrates, which could be 1,6 hexanediol or 1,10 decanediol, for example. To make the cast overlay cover **40**, equimolar amounts of anhydrous citric acid and the diol of choice may be combined in a round bottom flask. (As an example: 38.4 g citric acid and 29.2 g octanediol). The solution may be heated in an oil bath for 10 min at 165° C. until melted, then continued to be heated at 140° C. for 45 min. The polymer may be used in this form although unreacted monomers are also present. To remove the unreacted monomer, equivolume amounts of polymer and 100% acetone may be added to a flask and shaken until the polymer is completely dissolved, then poured into an appropriately shaped mold. The acetone may be evaporated overnight in a chemical hood at room temperature. The films may be polymerized at 80° C. for 36 hr and then 18 hr at 110° C.

[0046] Alternatively, to cast overlay covers **40** of chitosan, a solution of 2% acetic acid in water may be added to 1% chitosan weight/volume. (For example 400 μl acetic acid may be added to 20 ml water, then 200 mg chitosan added.) Films may be prepared by pouring the mixture directly into the mold and allowing the solution to dry overnight. Cast overlay covers **40** of poly L (lactic acid) or poly D,L (co-glycolic lactic acid) dissolved in chloroform can also be made by pouring the solution into molds and evaporating the solvent (chloroform) off.

[0047] An additional method for creating porous materials **10** and overlay covers **40** is to use thermal inkjet printing technologies. Bio-incorporable materials such as collagen, elastin, hyaluronic acid, alginates, and polylactic/polyglycolic acid co-polymers may be printed. As examples, Type I collagen (Elastin Products Co., Owensville, Mo.) dissolved in 0.05% acetic acid, then diluted to 1 mg/ml in water can be printed, as can sodium alginate (Dharma Trading Co., San Raphael, Calif.) 1 mg/ml in water. A mixture of Type I col-

lagen (2.86 mg/ml in 0.05% acetic acid) and polylactic/polyglycolic acid (PURAC America, Blair, Nebr.) (14.29 mg/ml in tetraglycol (Sigma Aldrich, St. Louis Mo.)) can also be printed. Hardware from a Hewlett Packard 660c printer can be attached to a platform for which the height can be adjusted for printing in layers. With minimal changes to the hardware, no software changes need to be made.

[0048] Turning to FIG. **5**, the porous material **10** may comprise layers, with the layer **112** closest to the damaged cardiac tissue containing pores sufficiently small at the interface between the porous material **110** and the damaged cardiac tissue **7** to prevent the growth of tissue therein, e.g., a pore size smaller than the size of fibroblasts and cardiac cells. Otherwise the porous material **110** may stick to the damaged cardiac tissue **7** and cause bleeding or trauma, and potentially even disruption of the ventricular wall when the porous material **110** is removed. Additionally, growth of tissues into the porous material **110** may result in eventual erosion through the ventricular wall or pleural tissues with continual movement and rubbing of the porous material **110** against these tissues if the porous material **110** is left in the patient. Further, growth of tissues into the porous material **110** may result in non-contractible scar formation within the porous material or potential calcification of tissues within the porous material **110** if the porous material **110** is left within the patient. In addition, the pore size at the interface between the porous material **10**, **110** and the damaged cardiac tissue **7** may be sufficiently small so as to avoid the excessive production of granulation or scar tissue at the damaged cardiac tissue **7** which may interfere with the physiologic function of the heart. At the same time, the pore size of the porous material **10**, **110** may be large enough to allow movement of proteins the size of albumin therethrough to permit undesirable compounds to be removed, such as mediators, degradation products, and toxins.

[0049] The porous material **10**, **110** may, however, have a larger pore size (e.g., larger than that of fibroblasts and cardiac cells) interior to the porous material **10**, **110** or at any other location of the porous material **10** that is not in contact with cardiac tissue **7**. For example, the porous material **110** may comprise a multi-layer structure with a non-ingrowth layer **112** having a sufficiently small pore size to prevent the growth of tissue therein for placement at the cardiac tissue **7**, and may have an additional layer **114** of a different material that has a relatively larger pore size in contact with the non-ingrowth layer **112**.

[0050] Alternatively, as depicted in FIG. **6**, the porous material **210** may be homogeneous in composition and/or morphology. At a location away from the interface with the damaged cardiac tissue, the porous material **210** may have a pore size sufficiently large to promote the formation of granulation tissue at other tissues in the spaces surrounding the damaged cardiac tissue, such as promotion of granulation tissue in areas where cardiac disruption has occurred. In addition, the porous material **210** may have a configuration in which one or more sides or surfaces **212** of the porous material **210** are sealed to prevent the transmission of sub-atmospheric pressure through such a sealed surface **212**, while at the same time having at least one surface **214** through which sub-atmospheric pressure may be transmitted. Such a configuration of the porous material **210** can present preferential treatment of tissue on one side of the porous material **210** while not treating tissue on the other side. For instance, the damaged cardiac tissue could be treated with the non-sealed interface on one side **214** of the porous material **210**.

[0051] In addition, the porous material **10** may comprise a non-metallic material so that an MRI can be performed while the porous material **10** is in situ. The porous material **10** may also comprise a material that is sufficiently compliant so that it does not interfere with cardiac function. At the same time, the porous material **10** may comprise a material that is sufficiently firm so that the porous material **10** does not collapse so much as to create a pull on, or distortion of, the cardiac tissue **6**, **7** that might interfere with cardiac function.

[0052] Turning to FIG. **7**, to deliver sub-atmospheric pressure to the porous material **10** for distribution to the damaged cardiac tissue **7**, a tube **20** may be connected directly or indirectly in gaseous communication with the porous material **10** at the distal end **22** of the tube **20**. For example, the distal end **22** of the tube **20** may be embedded in the porous material **10** or may be placed over the porous material **10**. The distal end **22** of the tube **20** may also include one or more fenestrations **23** to assist in delivering the sub-atmospheric pressure to the porous material **10** and the damaged cardiac tissue **7**. The tube **20** may extend through an opening in the skin **1** and subcutaneous tissue **2** which may be secured about the tube **20** with a suture **8** to assist in providing a seal about the tube **20**. The proximal end **24** of the tube **20** may be operably connected to a vacuum source **30** (e.g., The V.A.C., Model 30015B, Kinetic Concepts, Inc., San Antonio, Tex.) to provide sub-atmospheric pressure that is transmitted via the tube **20** to the porous material **10** and the damaged cardiac tissue **7**.

[0053] The vacuum source **30** may include a controller **32** to regulate the production of sub-atmospheric pressure. For instance, the vacuum source **30** may be configured to produce sub-atmospheric pressure continuously or intermittently; e.g., the vacuum source **30** may cycle on and off to provide alternating periods of production and non-production of sub-atmospheric pressure. The duty cycle between production and non-production may be between 1 to 10 (on/off) and 10 to 1 (on/off). In addition, intermittent sub-atmospheric pressure may be applied by a periodic or cyclical waveform, such as a sine wave, or may be cycled after initial treatment to mimic a more physiologic state, such as the heart rate. The sub-atmospheric pressure may also be cycled on-off as-needed as determined by monitoring of the pressure in the damaged cardiac tissue **7**. In general, the vacuum source **30** may be configured to deliver sub-atmospheric pressure between atmospheric pressure and 200 mm Hg below atmospheric pressure to minimize the chance that the sub-atmospheric pressure may result in reduction in localized blood flow due to either constriction of capillaries and small vessels or due to congestion (hyperemia) within the damaged cardiac tissue **7** or otherwise be deleterious to the damaged cardiac tissue **7**. The application of such a sub-atmospheric pressure can operate to remove edema from the damaged cardiac tissue **7**, thus preserving cardiac function to increase the probability of recovery and survival in a more physiologically preserved state.

[0054] Turning to FIG. **10**, sub-atmospheric pressure may be delivered under the cover **50** by cooperation between the cover **50** and the tube **20**. Specifically, the flexible overlay cover **40** (or self-adhesive flexible overlay cover **50**) may include a passthrough **52** through which the distal end **22** of the tube **20** passes to provide gaseous communication

between the tube **20** and the space under the flexible overlay cover **40** over the damaged cardiac tissue.

[0055] In another of its aspects, the present invention also provides a method for treating damaged cardiac tissue using sub-atmospheric pressure with, by way of example, the devices illustrated in FIGS. **1-4**. In particular, the method may comprise locating a porous material **10** proximate the damaged cardiac tissue **7** to provide gaseous communication between one or more pores of the porous material **10** and the damaged cardiac tissue **7**. The porous material **10** may be sealed in situ proximate the damaged cardiac tissue **7** to provide a region about the damaged cardiac tissue **7** for maintaining sub-atmospheric pressure at the damaged cardiac tissue **7**. In this regard, the muscles **3**, and bone **4** may be loosely re-approximated over top of the porous material **10** with the tube **20** exiting through the skin **1** and subcutaneous tissue **2** and the skin **1** and subcutaneous tissue **2** sutured closed. A further airtight dressing may optionally be placed over the suture site to promote an airtight seal. The porous material **10** may be operably connected with a vacuum source **30** for producing sub-atmospheric pressure at the damaged cardiac tissue **7**, and the vacuum source **30** activated to provide sub-atmospheric pressure at the damaged cardiac tissue **7**. For example, the sub-atmospheric pressure may be maintained at about 25 to 125 mm Hg below atmospheric pressure. The sub-atmospheric pressure may be maintained at the damaged cardiac tissue **7** for a time sufficient to decrease edema at the damaged cardiac tissue **7**. In addition, the sub-atmospheric pressure may be maintained at the damaged cardiac tissue **7** for a time sufficient to prepare the cardiac tissue **7** to achieve a stage of healing and diminution of edema and inflammatory mediators or amplifiers. The method may be used for at least 2 hours, or can be used for many days. At the end of the vacuum treatment, the sutures **8** may be removed and the skin **1**, subcutaneous tissue **2**, muscles **3** and bone **4** re-opened. The porous material **10** may then be removed and the skin **1**, subcutaneous tissue **2**, and/or muscles **3** re-sutured closed.

[0056] The method may also include locating an overlay cover **40**, **50**, such as a bio-incorporable cover **40**, **50**, over the damaged cardiac tissue **7** and sealing the overlay cover **40**, **50** to tissue proximate the damaged cardiac tissue **7** for maintaining sub-atmospheric pressure at the damaged cardiac tissue **7**. The step of sealing the overlay cover **40**, **50** to tissue surrounding the damaged cardiac tissue **7** may comprise adhesively sealing and adhering the overlay cover **40**, **50** to tissue surrounding the damaged cardiac tissue **7**. The overlay cover **50** may be provided in the form of a self-adhesive sheet **50** which may be located over the damaged cardiac tissue **7**. In such a case, the step of sealing the overlay cover **50** may include adhesively sealing and adhering the self-adhesive overlay cover **50** to non-damaged cardiac tissue **6** surrounding the damaged cardiac tissue **7** to form a seal between the overlay cover **50** and the non-damaged cardiac tissue **6** surrounding the damaged cardiac tissue **7**. In addition, the step of operably connecting a vacuum source **30** in gaseous communication with the porous material **10** may comprise connecting the vacuum source **30** to the tube **20** which attaches to the vacuum port **42** of the cover **140** FIG. **11**.

[0057] In still another aspect of the present invention, in addition to injured tissues and organs, the devices and methods may also be used to increase the size and function of diseased or damaged organs. For example, the size of a partially functioning kidney may be increased to a size sufficient to return the total filtering capacity to normal levels, FIGS. **12-14**, such as the increase in size of the remaining kidney **301** as is observed in patients who only have one functioning kidney **301**. In such a situation, a rigid or semi-rigid bi-valved enclosure **304** with an opening **305** for the vascular pedicle may be placed around the kidney **301**. When the bi-valved enclosure **304** is closed, the area where the two halves meet creates an air tight seal. The vascular pedicle enters (artery **302**) and exits (vein **303**) through the opening **305**. Fibrin glue **306** or other biocompatible sealant may be placed around the artery **302** and vein **303** at the site of the opening **305** to create an airtight seal. The enclosure **304** may include a second opening **305** or a nipple **308**. A tube **309** may be inserted through the second opening **305** or attached to the nipple **308**. The tube **309** may exit through the skin, be connected to a collection vessel, and then connected to a vacuum source. A controlled vacuum of up to 125 mm Hg sub-atmospheric pressure may be applied either intermittently, with an 'on' time of up to five minutes and an 'off' time of up to 10 minutes. Alternatively, the vacuum may be applied in a periodic or cyclical manner, such as a sine wave, in which the absolute value of the lower (closest to atmospheric pressure) values of the applied vacuum are less than the diastolic blood pressure to allow blood to flow out of the treated organ. The time in which the applied vacuum is greater (in absolute value) than the diastolic blood pressure may be up to five minutes, with the time in which the applied vacuum is lower (in absolute value) than the diastolic blood pressure may be up to ten minutes. The technique is continued until the treated organ has either reached the desired level of function or fills the container. As an additional example, this device and technique may similarly be used on lobes of the liver or for increasing the size of the pancreas.

EXAMPLES

Example 1

[0058] The porcine heart has anatomy similar to that of humans with the main vasculature consisting of the right and left coronary arteries. The left main coronary artery splits into the circumflex coronary artery and the left anterior descending (LAD) coronary artery. The LAD runs down along the anterior septum and perfuses the anterior portion of the left ventricle with diagonal branches. For these studies, a porcine model of ischemia-reperfusion was used that included the temporary ligation of 2-3 diagonal branches of the LAD in order to create an ischemic area on the anterior portion of the heart. These coronary arteries were occluded for 75 minutes and then reperfused for 3 hours to allow for ischemia/reperfusion injury to develop. The negative pressure therapy was applied only during the reperfusion phase of the experiments to simulate a clinically relevant treatment window.

[0059] To begin the study, the animals were sedated and transported to the operating room. The first 13 animals had the heart exposed through a thoracotomy, all subsequent animals had the heart exposed through a sternotomy. The 2-3 diagonal branches of the LAD were ligated (occluded with suture) in order to create an ischemic area on the anterior portion of the heart. These coronary arteries were occluded for 75 minutes and then reperfused for 3 hours to allow for reperfusion injury to develop. The negative pressure therapy was applied only during the reperfusion phase of the experiments to simulate a clinically relevant treatment window. Five control animals were created from the first 13 animals of the study.

[0060] Following successful completion of control animals to validate the study design, the subsequent 5 successful (sternotomy) animals had negative pressure therapy treatment to the ischemic area of the heart for 3 hours during the reperfusion time. For the first 5 successfully treated animals, the vacuum dressing included use of a polyvinyl alcohol porous material (Versafoam, KCl, San Antonio Tex.), cut to approximately 1 mm thickness and trimmed to match the ischemic area. The evacuation tube was either embedded into a slit cut into the porous material (2 animals), or was sutured to the outer surface of the porous material (3 animals). This vacuum dressing was then covered with a biologically derived overlay cover. These biological coverings included: 1 animal treated with E•Z DERM™ (Non-perforated porcine biosynthetic wound dressing, Brennen Medical, St. Paul, Minn.); 1 animal treated with bovine pericardium; and 3 animals treated with AlloDerm® (human dermis) (LifeCell). The overlay covers were attached to the heart by three means: suturing, fibrin glue, and self sealing due to a relatively large 'apron' of the cover material around the periphery of the vacuum dressing. The evacuation tube exited from under the edge of the 'apron' of the overlay covers. The fibrin glue was used in conjunction with suturing and also with spot sealing for the self sealing application (at wrinkles, where the evacuation tube exited, etc.). Negative pressure of 125 mm Hg (i.e., 125 mm Hg below atmospheric) was then applied for 3 hours during the reperfusion period using The V.A.C., Model 30015B, Kinetic Concepts, Inc., San Antonio, Tex.

[0061] To determine the effects of ischemia/reperfusion, the sutures were re-tied at the end of the 3 hour reperfusion period. Blue dye (patent blue, Sigma-Aldrich Inc, St. Louis, Mo.) was injected into the right atrium. This stained the areas of the heart that were normally perfused. The left ventricle was dissected free from the rest of the heart and weighed (LV in Table). The area of ischemia (non-blue area) was further dissected from the left ventricle. The blue area of the left ventricle was then weighed (Blue in Table). The ischemic area (non-blue tissue) was then stained with a dye (2,3,5-triphenyltetrazolium chloride, Sigma-Aldrich Inc., St Louis Mo.) which stains live cells red. The red areas were dissected from the area of ischemia and were weighed (Red in Table), leaving areas of pale dead tissue (area of necrosis—AN in Table), and these pale tissue samples were weighed (Pale in Table). The combined Red and Pale areas constitute the area at risk (AAR in Table). The AN/AAR is the size of the infarct (percent of tissue that died during the ischemia/reperfusion time periods).

[0062] The results for the 5 control animals were:

TABLE 1

Control Animals

	Blue	Red	Pale (AN)	LV	AAR	AAR/LV (%)	AN/AAR (%)
Animal 1	75.6	5.85	2.18	83.63	8.03	9.60	27.15
Animal 2	90.5	10.63	2.44	103.57	13.07	12.62	18.67
Animal 3	85.39	12.16	4.26	101.81	16.42	16.13	25.94
Animal 4	92.45	8.17	3.47	104.09	11.64	11.18	29.81
Animal 5	81.24	9.86	4.34	95.44	14.20	14.88	30.56
			Mean	97.71	12.67	12.88	26.43
			Std Dev	8.59	3.13	2.66	4.73
			N	5.00	5.00	5.00	5.00
			Std Err	3.84	1.40	1.19	2.12

[0063] The results for the 5 treated animals were:

TABLE 2

−125 mmHg Treated Animals

Group	Blue	Red	Pale	LV	AAR	AAR/LV (%)	AN/AAR (%)
Animal 1	73.06	10.31	1.23	84.60	11.54	13.64	10.66
Animal 2	73.2	5.9	0.61	79.71	6.51	8.17	9.37
Animal 3	75	11.15	2.05	88.20	13.20	14.97	15.53
Animal 4	54.1	4.85	0.52	59.47	5.37	9.03	9.68
Animal 5	62.12	8.63	1.42	72.17	10.05	13.93	14.13
			Mean	76.83	9.33	11.95	11.87
			Std Dev	11.41	3.32	3.11	2.78
			N	5.00	5.00	5.00	5.00
			Std Err	5.10	1.48	1.39	1.24

[0064] Thus, the mean sizes of the infarct (AN/AAR; percent of tissue that died during the ischemia/reperfusion time period) for the control and treated animals were:

[0065] Control 26.43+/−2.12% (mean+/−SEM) (n=5)

[0066] Treated 11.87+/−1.24% (mean+/−SEM) (n=5),

with T-test results of $P<0.001$ for infarct size and $P<0.625$ for area at risk.

Example 2

[0067] Another experiment was conducted using 50 mm Hg vacuum for treatment for comparison to original control animals from Example 1 above. The surgical technique in this experiment was similar to that used for those of Example 1. These animals were sedated and prepped for surgery. The heart was exposed through a midline sternotomy. Branches of the left anterior descending artery were ligated for 75 minutes. A polyvinyl alcohol vacuum dressing was placed over the ischemic area and an AlloDerm® cover was placed over the vacuum dressing and sealed into place with a combination of sutures and fibrin glue. Negative pressure of 50 mm Hg was applied for 3 hours. At the end of this time the heart was stained for area of risk, removed and then counter stained for area of necrosis. The infarct size results for these five, 50 mm Hg negative pressure therapy animals were significantly smaller ($P<0.001$) than for the control animals. The infarct size for the 50 mm Hg treated animals was smaller than the infarct size for the 125 mm Hg treated animals, but was not significantly smaller.

Group	AAR/LV	(%) AN/AAR
Control	12.9 ± 1.2	26.4 ± 2.1
50 mmHg negative pressure	11.8 ± 2.0	9.3 ± 1.8**
125 mmHg negative pressure	11.9 ± 1.4	11.9 ± 1.2**

**p < 0.001 compared to Control animals

[0068] The mean arterial pressure and heart rate of animals in all three groups (control, −125 mm Hg, −50 mm Hg) were comparable during the course of these experiments.

[0069] Fifteen micron neutron-activated microspheres (BioPAL, Inc, Worcester, Mass.) were injected into the left atrium at baseline, end of ischemia, 30 minutes into reperfusion and at 180 minutes of reperfusion (end of the experiment). A reference sample of arterial blood was simulta-

neously drawn from the femoral artery at a rate of 7 mL per minute for ninety seconds. Following infarct sizing procedures, tissue samples from the non-ischemic (blue tissue), ischemic non-necrotic (red tissue), and ischemic necrotic areas (pale tissue) were collected and sent to the manufacturer for blood flow analysis (BioPAL, Inc., Worchester, Mass.). Blood flow was calculated as [(FR×CPMT)/CPMR)/tissue weight in grams, where FR=reference sample flow rate (7 mL/min), CPMT=counts per minute in tissue samples and CPMR=counts per minute in the reference blood sample. Blood flow is reported as mL/min/gram tissue.

[0070] Analysis of blood flow reveals that both treated groups had similar baseline blood flows in all 3 regions. In the normally perfused non-ischemic zone, blood flow remained relatively constant throughout the experiment with no significant group or time related differences. (Table 3) In the ischemic, non-necrotic (red) and ischemic, necrotic zones (pale), ischemia was characterized by an equivalent and nearly complete loss of blood flow among all three groups. These zones also exhibited normal reactive hyperemia (30 minutes after reperfusion), and blood flow that returned approximated baseline flow levels by the end of the 3 hour reperfusion time. (Table 4).

TABLE 3

Blood flow (ml/minute/gram tissue) from microsphere analysis

Baseline

	Control			−125 mmHg			−50 mmHg		
Animal	blue	Red	Pale	blue	Red	Pale	blue	Red	Pale
1	—	—	—	0.36	0.328	0.333	0.596	1.1	0.77
2	1.072	0.709	0.716	0.308	0.401	0.448	0.474	0.321	0.551
3	0.378	0.347	0.505	0.392	0.411	0.353	0.531	0.444	0.422
4	0.577	0.729	0.599	0.643	1.32	0.82	0.625	0.629	0.699
5	0.376	0.495	0.412	0.423	0.687	0.482	0.393	0.57	0.596
Mean	0.603	0.57	0.558	0.4252	0.629	0.487	0.524	0.613	0.608
SD	0.33	0.18	0.13	0.13	0.41	0.20	0.09	0.30	0.13
N	4	4	4	5	5	5	5	5	5
SEM	0.16	0.09	0.07	0.06	0.18	0.09	0.04	0.13	0.06

During Occlusion

	Control			−125 mmHg			−50 mmHg		
Animal	Blue	Red	pale	blue	Red	pale	blue	Red	pale
1	—	—	—	0.345	0.065	0.012	0.387	0.056	0.025
2	1.031	0.073	0.0255	0.335	0.064	0.029	0.352	0.008	0.029
3	0.3	0.016	0.022	1.196	0.06	0.051	0.714	0.024	0.041
4	0.428	0.129	0.017	0.454	0.084	0.071	0.494	0.038	0.035
5	0.4	0.024	0.011	0.509	0.054	0.029	0.441	0.037	0.1
Mean	0.540	0.061	0.0189	0.568	0.065	0.038	0.478	0.033	0.046
SD	0.33	0.05	0.01	0.36	0.01	0.02	0.14	0.02	0.03
N	4	4	4	5	5	5	5	5	5
SEM	0.17	0.03	0.00	0.16	0.01	0.01	0.06	0.01	0.01

Reperfusion 30 minutes

	Control			−125 mmHg			−50 mmHg		
Animal	blue	red	pale	blue	Red	pale	blue	red	pale
1	—	—	—	0.379	1.341	1.022	0.441	1.355	2.361
2	1.102	1.522	1.872	0.37	0.559	0.692	0.402	0.628	0.708
3	0.348	0.54	0.286	0.298	0.878	0.6	0.741	1.699	1.626
4	0.439	1.054	1.225	1.439	0.909	1.288	0.603	1.126	1.477
5	0.496	1.272	1.4	—	—	—	0.676	1.866	1.147
Mean	0.596	1.097	1.196	0.622	0.922	0.901	0.573	1.335	1.464
SD	0.34	0.42	0.67	0.55	0.32	0.32	0.15	0.49	0.61
N	4	4	4	4	4	4	5	5	5
SEM	0.17	0.21	0.33	0.27	0.16	0.16	0.07	0.22	0.27

Reperfusion 180 minutes

	Control			−125 mmHg			−50 mmHg		
Animal	blue	red	pale	blue	Red	Pale	blue	red	Pale
1	—	—	—	0.404	0.367	0.795	0.467	0.385	0.837
2	1.102	1.522	1.872	0.291	0.365	0.6	0.593	0.186	0.649
3	0.348	0.54	0.286	0.38	0.303	0.515	0.804	0.649	0.699
4	0.439	1.054	1.225	0.513	0.449	0.845	0.912	0.803	0.946

TABLE 3-continued

Blood flow (ml/minute/gram tissue) from microsphere analysis									
5	0.496	1.272	1.4	0.53	0.477	0.76	0.483	0.471	0.495
Mean	0.596	1.097	1.196	0.424	0.392	0.703	0.652	0.499	0.725
SD	0.34	0.42	0.67	0.10	0.07	0.14	0.20	0.24	0.17
N	4	4	4	5	5	5	5	5	5
SEM	0.17	0.21	0.33	0.04	0.03	0.06	0.09	0.11	0.08

TABLE 4

Regional Myocardial blood flow (mL/min/100 g tissue)									
	Control			−50 mmHg			−125 mmHg		
	Blue	Red	Pale	Blue	Red	Pale	Blue	Red	Pale
Baseline	0.60 ± 0.16	0.57 ± 0.09	0.56 ± 0.07	0.52 ± 0.04	0.61 ± 0.13	0.61 ± 0.06	0.43 ± 0.06	0.63 ± 0.18	0.49 ± 0.09
Occlusion	0.54 ± 0.17	0.06 ± 0.03†	0.02 ± 0.00†	0.48 ± 0.06	0.03 ± 0.01†	0.05 ± 0.01†	0.57 ± 0.16	0.07 ± 0.01†	0.04 ± 0.01
R30	0.60 ± 0.17	1.10 ± 0.21	1.2 ± 0.33†	0.57 ± 0.07	1.33 ± 0.22†	1.46 ± 0.27*†	0.62 ± 0.27	0.92 ± 0.16†	0.90 ± 0.16
R180	0.41 ± 0.04	1.39 ± 0.35†	0.95 ± 0.16	0.65 ± 0.09	0.50 ± 0.11	0.73 ± 0.08	0.42 ± 0.04	0.39 ± 0.03	0.70 ± 0.06*

Regional myocardial blood flow was determined in 3 regions of the heart: 1) non-ischemic left ventricle; 2) ischemic, non-necrotic left ventricle; 3) necrotic left ventricle.
*$p < 0.05$ vs Control within a time period and within tissue area;
†$p < 0.05$ vs. Baseline within group and tissue area.

Example 3

[0071] A subsequent study was performed to examine resorbable vacuum dressings and overlay covers. One animal was sedated, prepared for surgery as described, and the heart exposed through a mid-line sternotomy. Branches of the LAD were ligated for 90 minutes. The dressing was prepared by freeze drying. A solution of chitosan (1.33% weight/volume in 2% acetic acid, 20 ml total volume) was poured into an appropriately sized mold. The solution was frozen for 2 hours at −70° C., then transferred to the lyophylizer for 24 hours. The dressing was cross-linked by 2.5% glutaraldehyde vapor for 12 hours to provide a porous material. The overlay cover was an electrospun mixture of Type I collagen and poly 1,8-octanediol citrate (POC) (80%:20% weight/weight). The solution concentration was 15% dissolved in hexafluoro-20proponal (HFIP) with a total volume of 9.5 ml. The solution was ejected from a syringe through an 18 gauge needle at a flow rate of 3 ml/hour. The voltage was 25 KV with a working distance of 25 cm. The film was then heat polymerized at 80° C. for 48 hours and cross-linked in 2.5% glutaraldehyde vapor for 24 hours. The overlay cover was able to maintain the vacuum for the duration of the experiment. However, the vacuum dressing did not distribute the vacuum equally throughout the dressing due to collapse and flow of the material under vacuum.

Example 4

[0072] A further study was performed to test variations of the overlay cover. Three animals were sedated and the heart exposed through a midline sternotomy. No infarct was created in this study of materials. The overlay cover was created similar to Example 3, but with variations, including changes in voltage, flow rate, and concentration of glutaraldehyde vapor for cross-linking. For these animals, the porous material vacuum dressing was formed from a solution of 80% Type I collagen/20% POC, 12% total concentration in 8.5 ml HFIP was used. The flow rate was 2 ml/hour, with the fluid ejected through an 18 gauge needle at 35 KV with a working distance of 25 cm. The film was heat polymerized at 80° C. for 48 hours, then cross-linked with exposure to 5% glutaraldehyde vapor for 24 hours. The evacuation tube was sutured to a thin polyvinyl alcohol dressing. The dressing was placed over a portion of the left ventricle and tacked in place with 2-4 sutures. The overlay cover was placed over the dressing and fibrin glue was placed around the edges of the overlay cover to insure a vacuum seal. 50 mm Hg was applied continuously to the dressing. For two animals a small air leak developed after approximately 2.5 hours, the source of the leak was not identified despite a diligent search for the source. The source of the leak could have been at the site of a wrinkle in the overlay cover, a tail of the suture material could have punctured a hole in the overlay cover, fluid collecting in the pericardial sack could have 'floated' a small portion of the cover off the heart tissue, etc. For the third animal, the negative pressure was maintained for the duration of the study (4 hours application of negative pressure).

Example 5

[0073] Two animals were used to test the dressing. The surgical technique was similar to that used above. These animals were sedated, prepped for surgery and the heart exposed through an midline sternotomy. Branches of the left anterior descending artery were ligated for 75 minutes. A dressing was made by casting polycaprolactone (PCL). Polycaprolactone was mixed with sodium chloride (1 part caprolactone to 10 parts sodium chloride) and placed in a sufficient volume of chloroform to dissolve the components. 8 ml of the solution was poured into an appropriately sized and shaped container and allowed to dry for twelve hours. The sodium chloride was then leached out in water for 24 hours. The dressing was cut to the size of the ischemic area. The evacuation tube was sutured to the dressing and the dressing placed over the ischemic area and tacked into place. At the end of the 75 minutes of ischemia the tissue was reperfused. The dressing was covered with AlloDerm® and fibrin glue was placed

around the edges of the AlloDerm®. 50 mm Hg vacuum was applied for 3 hours. At the end of this time the heart was stained for area of risk, removed and then counter stained for area of necrosis as described for Examples 1 and 2. For the first animal, the area at risk (ischemic area, AAR) was fairly small at 7.9% of the left ventricle (LV). The infarct size (area of necrosis divided by area at risk (AN/AAR×100%) was very small at 2.6% of the area at risk. For the second animal, the area at risk was larger at 14.3% (AAR/LV), with an infarct size (AN/AAR) of 11.52%.

[0074] These and other advantages of the present invention will be apparent to those skilled in the art from the foregoing specification. Accordingly, it will be recognized by those skilled in the art that changes or modifications may be made to the above-described embodiments without departing from the broad inventive concepts of the invention. It should therefore be understood that this invention is not limited to the particular embodiments described herein, but is intended to include all changes and modifications that are within the scope and spirit of the invention as set forth in the claims.

1. A method for treating damaged cardiac tissue using sub-atmospheric pressure, comprising:

i. placing a porous material proximate the damaged cardiac tissue to provide gaseous communication between one or more pores of the porous material and the damaged cardiac tissue, the porous material comprising at least one of an electrospun material, a cast material, an open-cell foam, and a printed material;

ii. sealing the porous material in situ over the damaged cardiac tissue to provide a region about the damaged cardiac tissue for maintaining sub-atmospheric pressure at the damaged cardiac tissue;

iii. operably connecting a vacuum source in gaseous communication with the porous material for producing sub-atmospheric pressure at the damaged cardiac tissue; and

iv. activating the vacuum source to provide sub-atmospheric pressure at the damaged cardiac tissue.

2. The method for treating damaged cardiac tissue according to claim **1**, wherein the porous material comprises a bio-incorporable material.

3. The method for treating damaged cardiac tissue according to claim **2**, wherein the rate of bio-incorporation of the dressing is higher at the periphery of the dressing than at the center of the dressing.

4. The method for treating damaged cardiac tissue according to claim **1**, wherein the porous material comprises a polyethylene, polyurethane and/or polyester material.

5. The method for treating damaged cardiac tissue according to claim **1**, wherein the step of placing a porous material proximate the damaged cardiac tissue comprises placing a polyvinyl alcohol foam in direct contact with the damaged cardiac tissue.

6. A method for treating damaged cardiac tissue using sub-atmospheric pressure, comprising:

i. placing a porous bio-incorporable material proximate the damaged cardiac tissue to provide gaseous communication between one or more pores of the porous material and the damaged cardiac tissue;

ii. sealing the porous material in situ over the damaged cardiac tissue to provide a region about the damaged cardiac tissue for maintaining sub-atmospheric pressure at the damaged cardiac tissue;

iii. operably connecting a vacuum source in gaseous communication with the porous material for producing sub-atmospheric pressure at the damaged cardiac tissue; and

iv. activating the vacuum source to provide sub-atmospheric pressure at the damaged cardiac tissue.

7. The method for treating damaged cardiac tissue according to claim **6**, wherein the rate of bio-incorporation of the dressing is higher at the periphery of the dressing than at the center of the dressing.

8. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises myocardial, peripheral muscle cells, or combinations thereof.

9. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the step of operably connecting a vacuum source comprises connecting a tube between the vacuum source and the porous material, the tube having a distal end in contact with the porous material.

10. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the step of placing a porous material proximate the damaged cardiac tissue comprises placing the porous material in direct contact with the damaged cardiac tissue.

11. The method for treating damaged cardiac tissue according to claim **10**, wherein the porous material comprises an open-cell foam.

12. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the step of placing a porous material proximate the damaged cardiac tissue comprises placing the porous material in indirect contact with the damaged cardiac tissue by locating a porous intermediate material between the porous material and the damaged heart tissue, with the porous intermediate material disposed in contact with both the porous material and the damaged heart tissue.

13. The method for treating damaged cardiac tissue according to claim **12**, wherein the porous material comprises an open-cell foam.

14. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the step of sealing porous material in situ comprises locating a cover over damaged cardiac tissue and sealing the cover to cardiac tissue proximate the damaged cardiac tissue for maintaining sub-atmospheric pressure at the damaged cardiac tissue.

15. The method for treating damaged cardiac tissue according to claim **14**, wherein the cover comprises a vacuum port for receiving sub-atmospheric pressure from the vacuum source, and wherein the step of operably connecting a vacuum source in gaseous communication with the porous material comprises connecting the vacuum source with the vacuum port.

16. The method for treating damaged cardiac tissue according to claim **14**, wherein the step of sealing the cover to tissue surrounding the damaged cardiac tissue comprises adhesively sealing and adhering the cover to cardiac tissue surrounding the damaged cardiac tissue.

17. The method for treating damaged cardiac tissue according to claim **14**, wherein the step of locating a cover comprises locating a self-adhesive sheet over the damaged cardiac tissue, and wherein the step of sealing the cover comprises adhesively sealing and adhering the self-adhesive sheet to cardiac tissue surrounding the damaged cardiac tissue to form a seal between the sheet and surrounding cardiac tissue.

18. The method for treating damaged cardiac tissue according to claim **14**, wherein the step of locating a cover comprises locating a bio-incorporable cover over the damaged cardiac tissue.

19. The method for treating damaged cardiac tissue according to claim **14**, wherein the cover comprises an electrospun material.

20. The method for treating damaged cardiac tissue according to claim **14**, wherein the cover comprises a cast material.

21. The method for treating damaged cardiac tissue according to claim **14**, wherein the cover comprises collagen.

22. The method for treating damaged cardiac tissue according to claim **19**, wherein the cover comprises collagen.

23. The method for treating damaged cardiac tissue according to claim **14**, wherein the cover comprises a diol citrate.

24. The method for treating damaged cardiac tissue according to claim **19** wherein the cover comprises a diol citrate.

25. The method for treating damaged cardiac tissue according to claim **14**, wherein the cover comprises poly 1,8-octanediol citrate.

26. The method for treating damaged cardiac tissue according to claim **19**, wherein the cover comprises poly 1,8-octanediol citrate.

27. The method for treating damaged cardiac tissue according to claim **14**, wherein the cover comprises chitosan.

28. The method for treating damaged cardiac tissue according to claim **19**, wherein the cover comprises chitosan.

29. The method for treating damaged cardiac tissue according to claim **14**, wherein the cover comprises polylactic acid.

30. The method for treating damaged cardiac tissue according to claim **19**, wherein the cover comprises polylactic acid.

31. The method for treating damaged cardiac tissue according to claim **1** or **6**, comprising maintaining the sub-atmospheric pressure at the damaged cardiac tissue for a time sufficient to decrease edema in the damaged cardiac tissue.

32. The method for treating damaged cardiac tissue according to claim **1** or **6**, comprising maintaining the sub-atmospheric pressure at the damaged cardiac tissue for a time sufficient to decrease mediators, degradation products, and/or toxins that enhance the inflammatory and pathophysiological response in the damaged cardiac tissue.

33. The method for treating damaged cardiac tissue according to claim **1** or **6**, comprising maintaining a sub-atmospheric pressure of about 25 mm Hg below atmospheric pressure at the damaged cardiac tissue.

34. The method for treating damaged cardiac tissue according to claim **1** or **6**, comprising maintaining sub-atmospheric pressure of between about 25 and 125 mm Hg below atmospheric pressure at the damaged cardiac tissue.

35. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the step of placing a porous material comprises placing a porous material having pores sufficiently small to prevent the ingrowth of tissue therein.

36. The method for treating damaged cardiac tissue according to claim **35**, wherein the step of placing a porous material comprises placing a porous material having a pore size smaller than the size of fibroblasts.

37. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises collagen.

38. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises chitosan.

39. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises polycaprolactone.

40. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises a polyglycolic and/or polylactic acid.

41. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises a porous, open-cell collagen material.

42. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises a porous synthetic polymer material.

43. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises at least one of a porous sheet and a flexible, sheet-like mesh.

44. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises two or more layers, with the layer closest to the damaged cardiac tissue containing pores sufficiently small at the interface between the porous material and the damaged cardiac tissue to prevent the growth of tissue therein.

45. The method for treating damaged cardiac tissue according to claim **44**, wherein the porous material comprises a pore size sufficiently large to promote the formation of granulation tissue at other tissues in the spaces surrounding the damaged cardiac tissue.

46. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises pores sufficiently small at the interface between the porous material and the damaged cardiac tissue to prevent the growth of tissue therein.

47. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material comprises a pore size large enough to allow movement of proteins the size of albumin therethrough to permit undesirable compounds to be removed.

48. The method for treating damaged cardiac tissue according to claim **1** or **6**, wherein the porous material is sealed to prevent the transmission of sub-atmospheric pressure on all surfaces but one.

49. The method for treating damaged cardiac tissue according to claim **1** or **6**, comprising infusing peripheral muscle cells into the damaged cardiac tissue.

50. The method for treating damaged cardiac tissue according to claim **1** or **6**, comprising infusing myocardial cells into the damaged cardiac tissue.

51. The method for treating damaged cardiac tissue according to claim **1** or **6**, comprising infusing pleuripotent progenitor cells into the damaged cardiac tissue.

52. An apparatus for treating damaged cardiac tissue, comprising:

a porous material for treating damaged cardiac tissue having a pore structure configured to permit gaseous communication between one or more pores of the porous material and the cardiac tissue to be treated, the porous material comprising at least one of an electrospun material, a cast material, and a printed material; and

a vacuum source for producing sub-atmospheric pressure disposed in gaseous communication with the porous material for distributing the sub-atmospheric pressure to the cardiac tissue to be treated.

53. The apparatus according to claim **52**, wherein the porous material comprises a bio-incorporable material.

54. The apparatus according to claim **53**, wherein the rate of bio-incorporation of the dressing is higher at the periphery of the dressing than at the center of the dressing.

55. The apparatus according to claim **52**, wherein the porous material comprises a polyethylene, polyurethane, and/or polyester material.

56. An apparatus for treating damaged cardiac tissue, comprising:

a porous bio-incorporable material for treating damaged cardiac tissue having a pore structure configured to permit gaseous communication between one or more pores of the porous material and the cardiac tissue to be treated; and

a vacuum source for producing sub-atmospheric pressure disposed in gaseous communication with the porous material for distributing the sub-atmospheric pressure to the cardiac tissue to be treated.

57. The apparatus according to claim **56**, wherein the porous material comprises an open-cell foam.

58. The apparatus according to claim **56**, wherein the rate of bio-incorporation of the dressing is higher at the periphery of the dressing than at the center of the dressing.

59. The apparatus according to claim **52** or **56**, where in the porous material comprises myocardial, peripheral muscle cells, or combinations thereof.

60. The apparatus according to claim **52** or **56**, comprising a porous intermediate material for contacting the damaged heart tissue, the porous intermediate material disposed below and in contact with the porous material.

61. The apparatus according to claim **52** or **56**, comprising a cover for placement over the damaged cardiac tissue for sealing engagement with cardiac tissue proximate the damaged cardiac tissue for maintaining sub-atmospheric pressure at the damaged cardiac tissue.

62. The apparatus according to claim **61**, wherein the cover comprises a vacuum port disposed in gaseous communication with the vacuum source for receiving sub-atmospheric pressure from the vacuum source.

63. The apparatus according to claim **61**, wherein the cover comprises an adhesive sealing for adhering and sealing the cover to cardiac tissue surrounding the damaged cardiac tissue.

64. The apparatus according to claim **61**, wherein the cover comprises a self-adhesive sheet.

65. The apparatus according to claim **61**, wherein the cover comprises a bio-incorporable material.

66. The apparatus according to claim **65**, wherein the cover comprises an electrospun material.

67. The apparatus according to claim **65**, wherein the cover comprises a cast material.

68. The apparatus according to claim **61**, wherein the cover comprises an electrospun material.

69. The apparatus according to claim **61**, wherein the cover comprises a cast material.

70. The apparatus according to claim **61**, wherein the cover comprises collagen.

71. The apparatus according to claim **68**, wherein the cover comprises collagen.

72. The apparatus according to claim **61**, wherein the cover comprises a diol citrate.

73. The apparatus according to claim **68**, wherein the cover comprises a diol citrate.

74. The apparatus according to claim **61**, wherein the cover comprises poly 1,8-octanediol citrate.

75. The apparatus according to claim **68**, wherein the cover comprises poly 1,8-octanediol citrate.

76. The apparatus according to claim **61**, wherein the cover comprises chitosan.

77. The apparatus according to claim **68**, wherein the cover comprises chitosan.

78. The apparatus according to claim **61**, wherein the cover comprises polylactic acid.

79. The apparatus according to claim **68**, wherein the cover comprises polylactic acid.

80. The apparatus according to claim **52** or **56**, wherein the vacuum source is configured to maintain a sub-atmospheric pressure of about 50 mm Hg below atmospheric pressure at the damaged cardiac tissue.

81. The apparatus according to claim **52** or **56**, wherein the vacuum source is configured to maintain sub-atmospheric pressure of between about 50 and 125 mm Hg below atmospheric pressure at the damaged cardiac tissue.

82. The apparatus according to claim **52** or **56**, wherein the porous material comprises pores sufficiently small to prevent the ingrowth of tissue therein.

83. The apparatus according to claim **82**, wherein the porous material comprises a pore size smaller than the size of fibroblasts.

84. The apparatus according to claim **52** or **56**, wherein the porous material comprises collagen.

85. The apparatus according to claim **52** or **56**, wherein the porous material comprises chitosan.

86. The apparatus according to claim **52** or **56**, wherein the porous material comprises polycaprolactone.

87. The apparatus according to claim **52** or **56**, wherein the porous material comprises a polyglycolic and/or polylactic acid.

88. The apparatus according to claim **52** or **56**, wherein the porous material comprises a porous, open-cell collagen material.

89. The apparatus according to claim **52** or **56**, wherein the porous material comprises a porous synthetic polymer material.

90. The apparatus according to claim **52** or **56**, wherein the porous material comprises at least one of a porous sheet and a flexible, sheet-like mesh.

91. The apparatus according to claim **52** or **56**, wherein the porous material comprises two or more layers, with the layer closest to the damaged cardiac tissue containing pores sufficiently small at the interface between the porous material and the damaged cardiac tissue to prevent the growth of tissue therein.

92. The apparatus according to claim **91**, wherein the porous material comprises a pore size sufficiently large to promote the formation of granulation tissue at a selected surface of the porous material.

93. The apparatus according to claim **52** or **56**, wherein the porous material comprises pores sufficiently small at a surface of the porous material for placement proximate the damaged cardiac tissue to prevent the growth of tissue therein.

94. The apparatus according to claim **52** or **56**, wherein the porous material comprises a pore size large enough to allow movement of proteins the size of albumin therethrough to permit undesirable compounds to be removed.

95. The apparatus according to claim **52** or **56**, wherein the porous material is sealed to prevent the transmission of sub-atmospheric pressure through all surfaces but one.

96. The apparatus according to claim **52** or **56**, wherein the vacuum source comprises a vacuum pump.

97. A degradable or resorbable vacuum appliance for treating injured or diseased tissues in a body, comprising

a dressing configured to be implanted in the body, the dressing having a void structure configured to permit the transmission of sub-atmospheric pressure therethrough; and

a bio-incorporable cover configured to be implanted in the body to cover and enclose the dressing to provide a chamber about the dressing in which sub-atmospheric pressure may be maintained.

98. The vacuum appliance according to claim **97**, wherein the dressing comprises a bio-incorporable material.

99. The vacuum appliance according to claim **98**, wherein the rate of bio-incorporation of the dressing is higher at the periphery of the dressing than at the center of the dressing.

100. The vacuum appliance according to claim **97**, wherein the dressing comprises an electrospun or cast material, or combinations thereof.

101. The vacuum appliance according to claim **97**, wherein the dressing comprises synthetic molecules.

102. The vacuum appliance according to claim **97**, wherein the dressing comprises naturally occurring molecules.

103. The vacuum appliance according to claim **97**, wherein the dressing comprises a combination of synthetic and naturally occurring molecules.

104. The vacuum appliance according to claim **97**, wherein the cover comprises an electrospun or cast material.

105. The vacuum appliance according to claim **97**, wherein the cover comprises synthetic molecules.

106. The vacuum appliance according to claim **97**, wherein the cover comprises naturally occurring molecules.

107. The vacuum appliance according to claim **97**, wherein the cover comprises a combination of synthetic and naturally occurring molecules.

108. The vacuum appliance according to claim **97**, comprising an evacuation tube in gaseous communication with the dressing.

109. A method for treating an organ, comprising enclosing the organ in an air-tight chamber, applying sub-atmospheric pressure to the organ, and maintaining the sub-atmospheric pressure for a sufficient time to increase the function of the organ.

110. A method for treating an organ, comprising enclosing the organ in an air-tight chamber, applying sub-atmospheric pressure to the organ, and maintaining the sub-atmospheric pressure for a sufficient time to increase the size of the organ.

111. A method for treating an organ according to claim **110**, wherein the chamber is larger than the organ and wherein the sub-atmospheric pressure is maintained until the organ has increased in size to fill the chamber.

112. A method for treating an organ according to claim **109** or **110**, wherein the sub-atmospheric pressure is applied intermittently to the chamber.

113. A method for treating an organ according to claim **112**, wherein the time in which the absolute value of the applied sub-atmospheric pressure is greater than diastolic blood pressure is less than five minutes.

114. A method for treating an organ according to claim **112**, wherein the time in which the absolute value of the applied sub-atmospheric pressure is less than the diastolic blood pressure is less than ten minutes.

115. An apparatus for treating an organ, comprising an air-tight chamber configured to surround and contain the organ, a vacuum source operably connected to the chamber for applying and maintaining sub-atmospheric pressure to the organ.

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