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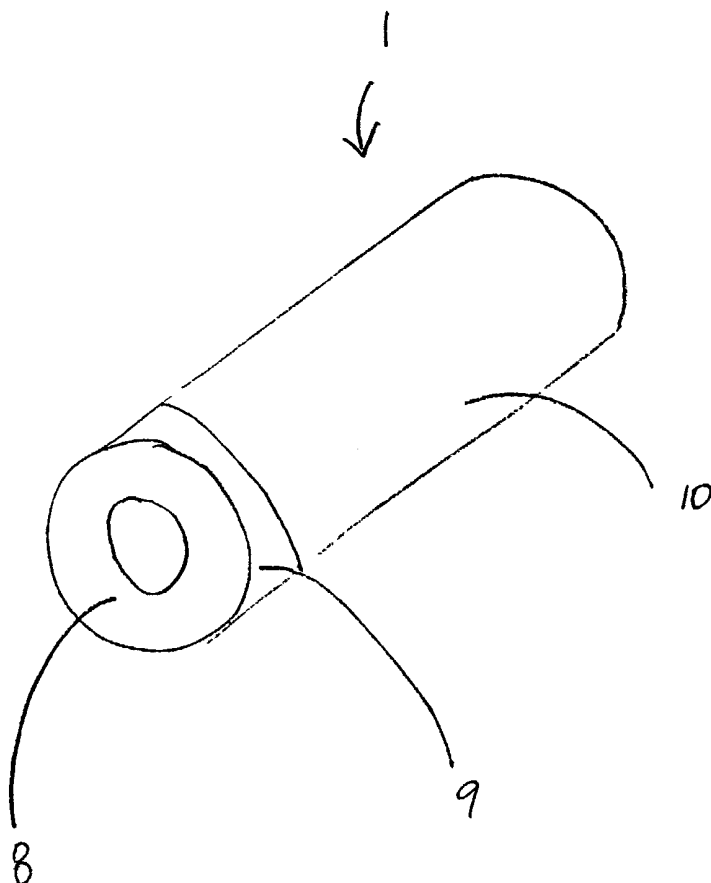
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(54) Title: PULMONARY VEIN STENT AND METHOD FOR USE



(57) Abstract: Ablation fo the pulmonary veins causes damage to the tissue which may affect the viability of the tissue. By placing a stent (1), a vascular endoprosthesis, within a target pulmonary vein (4), it is possible to protect the functionality of the veins after the ablation procedure. Placement of a stent (1), endoprosthesis or mere circuit interrupting structure into a target pulmonary vein, without ablation, prevents aberrant electrical activity in the pulmonary veins from interfering with the electrical activity of the left atrium. The stent (1), endoprosthesis or circuit interrupting structure may also be coated or comprised of a drug-eluting compound (8), loaded with a drug which inhibits arrhythmia.

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PULMONARY VEIN STENT AND METHOD FOR USE

Peter Altman

Related Patent Application

5 This patent application claims priority to provisional patent application 60/159,343, filed September 13, 1999.

Field of the Inventions

10 The inventions described below relate to the field of implantable medical devices. Specifically the invention relates to endoluminally placed prosthesis known as stents. More specifically, the invention relates to the placement of stents in the pulmonary veins which have advantages for the treatment of atrial fibrillation.

Background of the Inventions

15 Atrial fibrillation (AF) is a form of heart disease that afflicts millions of people. It is a condition in which the normal contraction of the heart is interrupted, primarily by abnormal and uncontrolled action of the atria of the heart. The heart has four chambers: the right atrium, right
20 ventricle, the left ventricle, and the left atrium. The right atrium pumps de-oxygenated blood from the vena cava to the right ventricle, which pumps the blood to the lungs, necessary for return flow of de-oxygenated blood from the body. The right atrium contracts to squeeze blood into the right
25 ventricle, and expands to suck blood from the vena cava. The left atrium pumps oxygenated blood from the pulmonary veins (returning from the lungs), necessary for flow of oxygenated

blood from the lungs. The left atrium contracts to squeeze blood into the left ventricle, which then pumps the blood into the aorta and thence to the entire body, and expands to suck blood from the pulmonary veins. The contractions of the atria
5 normally occur in a controlled sequence with the contractions of the other chambers of the heart. When the left atrium or the right atrium fails to contract, contracts out of sequence, or contracts ineffectively, blood flow within the heart is disrupted. The disruption of the normal rhythm of contraction
10 is referred to as an arrhythmia. The arrhythmia, known as atrial fibrillation, can cause weakness of the heart due to reduced ventricular filling and reduced cardiac output, stroke due to clot formation in a poorly contracting atria, which may lead to brain damage and death, and even life threatening
15 ventricular arrhythmias.

There is a broad spectrum of situations which fall under the broad heading of AF. For example, in older patients where there is substantial heterogeneity in the conduction within the atrial tissue, the patient is said to have the tissue
20 substrate for AF such that any trigger will result in maintaining AF. In younger patients, the tissue may have more homogeneous conduction and be less likely to have sustained AF. In the younger patient it may be the often reoccurrence of a premature depolarizing tissue which acts as a trigger
25 that causes the clinical manifestation of problematic episodes of AF. Clearly, there is a continuous spectrum of degrees of triggered AF and conduction heterogeneity which acts as a substrate for this arrhythmia, and it is appropriate that a number of medical therapies are being developed to treat this
30 disease.

Atrial fibrillation may be treated with an atrial defibrillator. Atrial defibrillators are typically

implantable electrical therapy devices which deliver
defibrillating energy to the atrium to terminate arrhythmias.
They sense the electrical activity of the atrium and deliver
an electrical shock to the atrium when the electrical activity
5 indicates that the atrium is in fibrillation. Electrical
defibrillation has two major problems: the therapy causes
substantial pain, and has the potential to initiate a life
threatening ventricular arrhythmias. The pain associated with
the electrical shock is severe and unacceptable for many
10 patients. Unlike electrical ventricular defibrillators, where
the patient loses consciousness prior to receiving therapy,
the patient who suffers an atrial arrhythmia is conscious and
alert when the device delivers electrical therapy.

The potential for inappropriate induction of ventricular
15 fibrillation by the shock intended to defibrillate the atrium
exists. The induction of ventricular fibrillation has great
potential to result in death in just a few minutes if no
intervening therapy is provided. Even with careful algorithms
to deliver shocks to the periods in the ventricular
20 contraction cycle when the heart is not susceptible to shock
induced ventricular fibrillation, the risk of setting off a
ventricular fibrillation remains substantial.

Doctors have treated atrial fibrillation with drugs
injected intravenously or administered orally. Recent
25 literature describes the potential for the delivery of drugs
to the heart on demand to terminate arrhythmias. The concept
has been suggested for use in the atrium to treat atrial
fibrillation. Arzbaeher, Pharmacologic Atrial Defibrillator
and Method, U.S. Patent 5,527,344 (Jun. 18, 1996) describes a
30 pharmacological atrial defibrillator and method for
automatically delivering a defibrillating drug into the
bloodstream of a patient upon detection of atrial arrhythmias

in order to terminate the atrial arrhythmias. Arzbaecher teaches that unspecified defibrillating drugs should be injected into the bloodstream with a large initial dose followed by delivery of a continuous smaller dose (this is the
5 "two-compartment pharmacokinetic model" discussed in the Arzbaecher patent). By delivering agents to a blood vessel and maintaining a therapeutic level of drugs in the blood stream, Arzbaecher requires systemic effects to be achieved in order to terminate atrial arrhythmias. In other words, if
10 drugs injected according to Arzbaecher are to have any effective concentrations within the heart, then a large amount must be injected in the blood stream to ensure that an adequate dose is delivered to the affected area of the heart. While the drugs are in the blood stream, they are available
15 throughout the body to cause side effects on all other organs.

There are several disadvantages to the transient introduction of systemic drug levels by an implantable device. Systemic effects resulting from such delivery may result in detrimental effects to ventricular cardiac conduction. These
20 detrimental effects could be life threatening, and several studies suggest that use of these drugs actually leads to higher mortality. The large amount of drugs required for systemic delivery of therapeutic doses demands a larger, less comfortable device than smaller dosages would allow. The
25 large quantity of drug in the implantable reservoir of such a system is potentially more dangerous if it develops a leak or is ruptured. Such a large single dosage requires a reservoir that requires frequent follow-ups for refilling post therapy by a clinician. Lastly, the large quantity of drugs required
30 to obtain therapeutic levels in the entire body may cost substantially more than that required to treat a specific site within the heart.

Atrial fibrillation can be treated by atrial ablation. There are two general approaches for providing ablative therapy to the heart for the treatment of atrial fibrillation. These shall be called the long linear ablative lesion approach
5 and the focal ablation approach.

In the long linear lesion approach, the heart tissue is killed along a linear pathway. The cardiac electrophysiologist does this to segment the heart into regions which are too small to sustain atrial fibrillation.
10 Such an approach is very similar to performing the Maze procedure using radiofrequency, microwave, and ultrasound ablative energy sources on the end of catheters. In the Maze procedure, a number of incisions are made with a scalpel in an attempt to terminate inappropriate accessory pathways.

In the focal ablation approach, the heart tissue is killed at a single site. The cardiac electrophysiologist attempts to ablate the region of the heart that prematurely depolarizes, and which has been described as acting as a trigger for the initiation of atrial fibrillation. Recently,
15 work in ablating regions at the junction of the pulmonary veins and the left atrium has been performed. Such ablations remove the possibility of triggers for AF initiating within the pulmonary veins, or at the region near the junction of the veins with the left atrial tissue. Such ablations may also
20 remove disturbances introduced into the conduction pathway by the heterogeneity of the junction region anatomy.
25

Summary

Focal ablation of the region within or adjacent to the pulmonary vein to terminate atrial fibrillation with different
30 energy transfer techniques such as cryoablation, RF ablation, laser ablation, ultrasound ablation, and microwave ablation

causes damage to the tissue which may affect the viability of the tissue. By placing a stent, a vascular endoprosthesis, within a target pulmonary vein it is possible to protect the functionality of the veins after the ablation procedure.

5 In some cases, placement of a stent, endoprosthesis or mere circuit interrupting structure into the pulmonary veins prevents aberrant electrical activity in the pulmonary veins from interfering with the electrical activity of the left atrium and thereby eliminate the need for destructive
10 ablation. The stent, endoprosthesis or circuit interrupting structure may be coated or comprised of a drug-eluting compound, loaded with a drug which inhibits arrhythmia. Embodiments include a new indication for maintaining vessel patency after a destructive ablation procedure, and the use of
15 a stent both with and without local drug delivery to disrupt the electrical contribution of the pulmonary veins to atrial electrical activity leading to the induction and maintenance of atrial fibrillation.

Brief Description of The Drawings

20 Figure 1 is a cross-sectional view of the left atrium with the stent deployed into the target pulmonary vein.

 Figure 2 is a schematic of a drug elution stent.

 Figure 3 is a schematic of a drug elution stent where only a narrow band of the stent is covered with a drug eluting
25 compound.

 Figure 4 is a schematic of a drug elution stent having a uniform coating of drug eluting compound and a coating covering a substantial portion of the stent such that the drug is eluted from a narrow band of the stent.

Figure 5 is an isometric view of the left atrium having a stent deployed into the ostium of the target pulmonary vein, wherein the drug-eluting band is proximate the left atrium.

Detailed Description of the Inventions

5 In Figure 1 the stent **1** has been deployed, by the catheter **2**, into the ostium **3** of the target pulmonary vein **4**. The stent alone acts to isolate the electrical impulses of the pulmonary veins from the atrial conduction tissue. The stent, endoprosthesis or circuit interrupting structure is placed
10 into the ostium of the target pulmonary veins. The term "stent" is being used in its broad sense as is meant to encompass a stent, endoprosthesis or a circuit interrupting structure. The stent, when fabricated of a material with desirable electrical properties (for example, metal, which has
15 a low resistivity) may comprise the circuit interrupting structure. To place the stent in the target pulmonary vein, access to the left atrium **5** is first gained by percutaneous insertion of a catheter into the left atrium. To accomplish this, a needle catheter is placed through the venous system
20 into the right atrium, and then penetrates the fossa ovalis **6** (the atrial septum) to gain access to the left atrium. Access to the right atrium (not shown) can be through the femoral vein in the thigh and the inferior vena cava, or may be through the subclavian vein, brachial vein or cephalic vein,
25 etc., in the shoulder and arm, and then through the superior vena cava. A catheter sheath **7**, such as a guiding catheter, is advanced over the needle catheter and is inserted into the left atrium. The needle catheter is then removed, and a stent delivery catheter is inserted with a stent loaded on the
30 distal end of the catheter. The distal end of the catheter is then maneuvered into the left atrium and into the target pulmonary veins. Location of the stent is confirmed and the

stent is expanded or released, and expanded to the point where it securely engages the wall of the pulmonary vein. If necessary, the stent may be pressed into the wall of the pulmonary vein, and may have fastening appendages such as
5 barbs, to more securely attach to the pulmonary vein wall.

As previously noted, placement of the stent alone can act to isolate the target pulmonary vein from the atrial conduction and excitation process. The primary advantage of this method is that no tissue is damaged. The ramifications
10 regarding the viability of the pulmonary veins long term is obvious. Less obvious is the fact that the non-damaging procedure can be reversed, if desired. This stent device may operate by acting as an electrically insulative barrier to an electrical signal, a capacitively coupled short across the
15 region of tissue in question, an averager that reduces the effective signal of the myocardial region in question, a substrate for the delivery of pharmacological agents to alter the electrical excitation and conduction properties of the local tissue region, or any combination of these mechanisms.
20 The preferred embodiment incorporates local delivery of therapeutic agents to a region equivalent to the lumen wall on the outer wall of the stent.

Where placement of the stent is accomplished without an accompanying ablation procedure, the device may be fabricated
25 in various forms. A nonconductive embodiment of any of the different possible geometries of the device will act as an insulative barrier preventing conduction through the device, and acting similarly to a region of necrotic tissue created by ablation. There is one fundamental difference: the device
30 disclosed here changes the cellular conductivity locally without destroying tissue.

The conductive embodiments of the device act as a short across the pulmonary vein region which may introduce a triggered premature depolarization of cells. By electrically connecting the tissue around the arrhythmogenic site, the
5 cells on either side of a conductive device will be coupled capacitively to the device and therefore to each other.

Metals are very efficient conductors of electrons, but not for ions. On the other hand, aqueous electrolyte solutions are ionic conductors and are hostile to electrons.
10 Consequently, at the interface between a metal and an aqueous electrolyte solution, there is a mismatch in the type of charge carrier used. In the absence of a chemical mechanism to convert one type of charge into the other, the interface behaves as a capacitance: a change in the electronic charge
15 density on the metal side is accompanied by a compensating change in ionic charge density on the solution side, so that electroneutrality is maintained.

The two types of charges can come very close to each other spatially without the possibility of neutralizing each
20 other. This gives rise to an interfacial capacitance. [de Levie, Robert: The Admittance of the Interface between a Metal Electrode and an Aqueous Electrolyte Solution: Some Problems and Pitfalls, pp337-347 Annals of Biomedical Engineering, Special Issue]. Typically the interface between a metal and
25 tissue is modeled as a resistor and a capacitor in parallel; at low currents the impedance associated with the capacitive leg of the circuit is small and the impedance associated with the resistive leg is large. Thus, any electrical discharge from electrically active tissue on the pulmonary vein side of
30 the stent, or underlying the stent, will be hindered in its transmission to the atrium thereby limiting its ability to initiate an atrial fibrillation in the left atrium. Different

biocompatible metals such as Platinum Iridium Alloys, MP35N, Titanium, nitinol, and Stainless Steels may be selected for different capacitive and resistive effects.

5 If pulmonary vein ablation is to be performed as the primary therapy and the stent is to be used to maintain pulmonary vein patency after ablation, then the necessary ablation catheter is passed through the catheter sheath into the left atrium to gain access to the pulmonary veins and perform the ablation prior to stent placement. The ablation
10 catheter may be placed within the opening of the pulmonary veins or adjacent to the pulmonary veins to produce lesions using radiofrequency, ultrasound, microwave, laser, cryogenic or other energy transfer means as well as chemical means to kill the tissue locally. The damaged pulmonary vein or veins
15 are now at risk for stenosis and stenting the pulmonary vein avoids the resulting pulmonary hypertension or occlusion. Stenting is performed by removing the ablation catheter of choice, and inserting a stenting catheter. The stenting catheter may use balloon expandable, self-expanding, or other
20 types of expanding stents to maintain the patency of the pulmonary vein.

After one or more stents are placed in the pulmonary veins, with one or more catheters swapped out through the catheter sheath, the catheter sheath is removed, and the
25 patient is closed. In most cases the opening through the fossa ovalis is very small and it heals up on its own. However, it is conceivable that a repair may be required in some patients using catheter techniques developed for closing septal defects.

30 The stent may be coated with a drug delivery compound or it may be partially made of a drug delivery compound. The stent is placed such that it delivers a sustained release of

an antiarrhythmic drug which affects conduction locally. There are a number of viable pharmacologic therapies available. Drugs that predominantly affect slow pathway conduction include digitalis, calcium channel blockers, and beta-blockers. Drugs that predominantly prolong refractoriness, or time before a heart cell can be activated, produce conduction block in either the fast pathway or in accessory AV connections including the class IA antiarrhythmic agents (quinidine, procainimide, and disopyrimide) or class IC drugs (flecainide and propafenone). The class III antiarrhythmic agents (sotalol or amiodorone) prolong refractoriness and delay or block conduction over fast or slow pathways as well as in accessory AV connections. Temporary blockade of slow pathway conduction is usually achieved by intravenous administration of adenosine or verapamil. [Scheinman, Melvin: Supraventricular Tachycardia: Drug Therapy Versus Catheter Ablation, Clinical Cardiology Vol. 17, Supp. II -11-II-15 (1994)]. Other agents such as encainide, diltiazem, and nickel chloride are also available.

As in Figures 2, 3, and 4, the drugs may be placed on the stent 1 in a drug eluting coating 8. The drug eluting coating may be placed uniformly over the outer wall of the stent as in Figure 2 or they may be placed in a narrow circumferential band on the proximal end of the stent as in Figure 3. This band creates a short longitudinal segment of the stent which is capable of eluting drugs into the surrounding pulmonary vein. The remaining longitudinal segment of the stent is not covered with a drug-eluting compound. The stent is placed into the target pulmonary vein 4 such that the drug-eluting band is on the end of the stent nearest the left atrium after placement, as in Figure 5.

As shown in Figure 4, the drugs may be placed uniformly over or within the entire stent, while only a small band **9** is permitted to elute drugs outward into the pulmonary vein wall. This is accomplished by having a coating **10** covering a

5 substantial longitudinal portion of the stent which prevents elution of the drug, thus leaving a small circumferential band which allows elution of the drug into the target pulmonary vein. The coating impedes the elution of drugs from the portion of the stent which is covers. This coating may a thin
10 layer of polyurethane or any other biocompatible coating impervious to the drug to be eluted. Having a small region **9** through which the drugs are eluted increases the ratio of drug volume to elution surface area. In other words, the large amount of drug-eluting compound can only be eluted through the
15 small surface area, thus, increasing the ratio of drug volume to elution surface area. Where the stent is made of a polymer or hydrogel, the entire stent material may be loaded with the eluting drug.

The methods and devices described above can be
20 accomplished with many embodiments of stents. Additionally, many stent materials and drug compounds may be substituted for the materials and drugs described. Thus, while the preferred embodiments of the devices and methods have been described in reference to the environment in which they were developed,
25 they are merely illustrative of the principles of the inventions. Other embodiments and configurations may be devised without departing from the spirit of the inventions and the scope of the appended claims.

I claim:

1. A method of treating a pulmonary vein, said method comprising the step of:

deploying a stent into the pulmonary vein.

5

2. The method of claim 1, further comprising the step of eluting drugs into the pulmonary vein by the stent.

3. A method of using a stent in a pulmonary vein having an ostium, said method comprising the step of:

deploying the stent into the ostium of the pulmonary vein.

4. A method of using a stent in a pulmonary vein, said method comprising the steps of:

inserting a stent delivery catheter into the pulmonary vein; and

deploying the stent into the pulmonary vein.

5. A method of using a stent in a pulmonary vein, said method comprising the step of:

deploying the stent into the pulmonary vein, the stent being composed of a material having a low resistivity.

6. A method of using a stent in a pulmonary vein, said method comprising the step of:

deploying the stent into the target pulmonary vein, the stent being composed of a non-conductive material.

5

7. A method of using a stent in a target pulmonary vein, said method comprising the step of:

deploying a metal stent into the target pulmonary vein.

10 8. The method of claim 7, wherein the metal is selected from the group consisting of Platinum Iridium Alloys, MP35N, Titanium, nitinol, and Stainless Steel.

9. A drug delivery stent comprising:

15 a stent; and

a drug-eluting coating on the stent, said drug eluting coating comprising a drug;

the stent having a first longitudinal region through which the drug is eluted and a second longitudinal region through which the drug is not eluted.

20

10. The stent of claim 9, wherein the drug is an antiarrhythmic drug.

11. A drug delivery stent comprising:

a stent;

a drug-eluting compound uniformly covering the stent; and

a small longitudinal region of the drug-eluting compound

5 being capable of eluting the drug.

12. The stent of claim 11, wherein the drug is an antiarrhythmic drug.

10 13. A drug delivery stent comprising:

a stent;

a drug incorporated into the stent; and

a coating covering a substantial longitudinal portion of the stent, said coating inhibiting elution of the drug

15 from the stent in said longitudinal portion, the drug being eluted from the portion of the stent not covered by the coating.

14. The stent of claim 13, wherein the drug is an antiarrhythmic drug.

20

15. A drug delivery stent comprising:

a stent;

a drug-eluting compound, uniformly covering the stent, the drug-eluting compound comprising a drug; and

5 a coating covering a first longitudinal portion of the drug-eluting compound, said coating impeding the elution of drugs from the stent, whereby the drug is eluted from the portion of the drug-eluting compound not covered by the coating.

16. The stent of claim 15, wherein the drug is an antiarrhythmic drug.

10 17. A stent for use in the pulmonary vein of a patient exhibiting aberrant electrical activity within a pulmonary vein, said pulmonary vein communicating through an ostium to the atrium of the heart, said stent having a proximal end and a distal end, said distal end adapted for insertion into the pulmonary vein, said proximal end adapted for insertion into
15 ostium, said stent comprising a region on the proximal end thereof which comprises a material capable of effecting the electrical activity of the pulmonary vein.

18. The stent of claim 17 wherein the stent is comprised of a
20 metal.

19. The stent of claim 17 wherein the stent is comprised of a drug eluting polymer loaded with an antiarrhythmic drug.

20. The stent of claim 19 wherein the distal end of the stent is covered with a material inhibiting elution of the drug from
25 the distal end of the stent.

1 stent

2 catheter

3 ostium

4 target pulmonary vein

5 left atrium

6 atrial septum

7 catheter sheath

8 drug-eluting coating

9 small band

10 coating

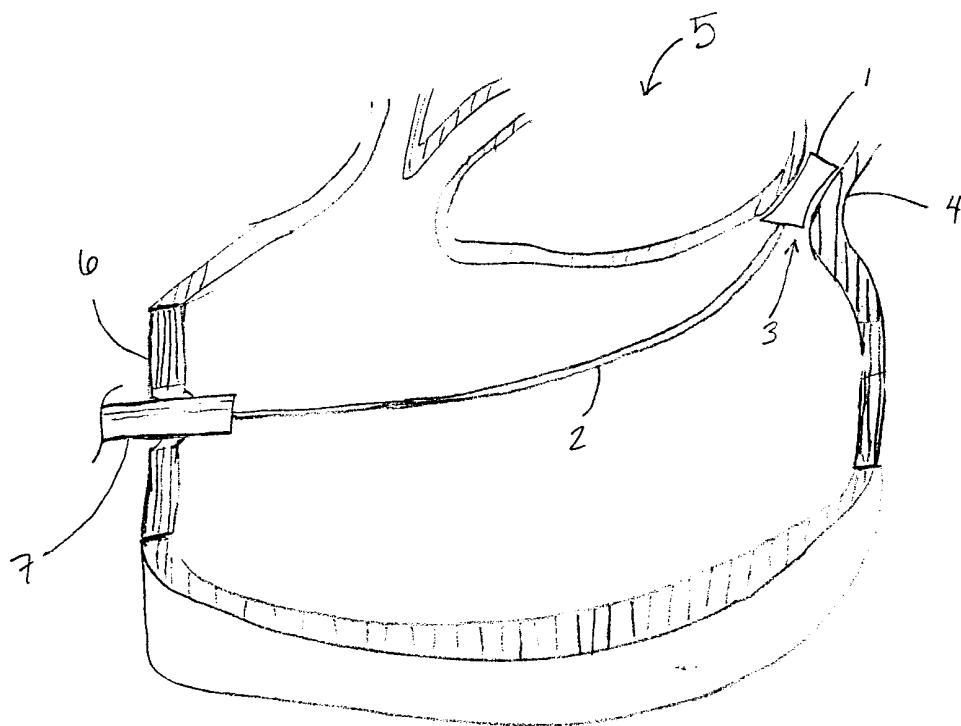


Fig. 1

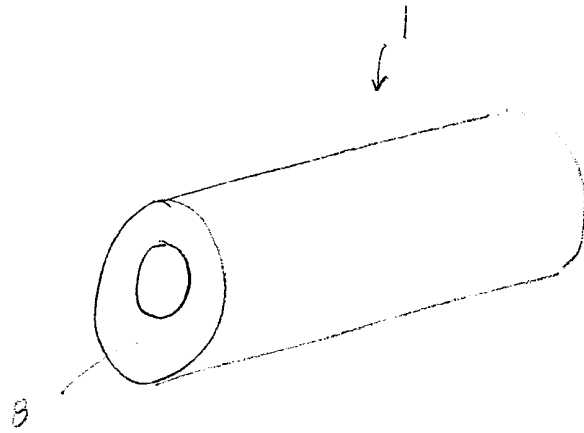


Fig. 2

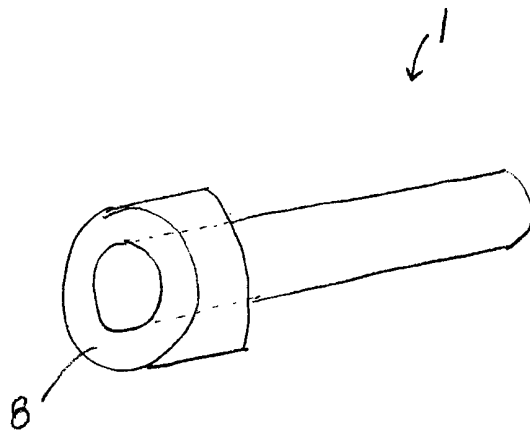


Fig. 3

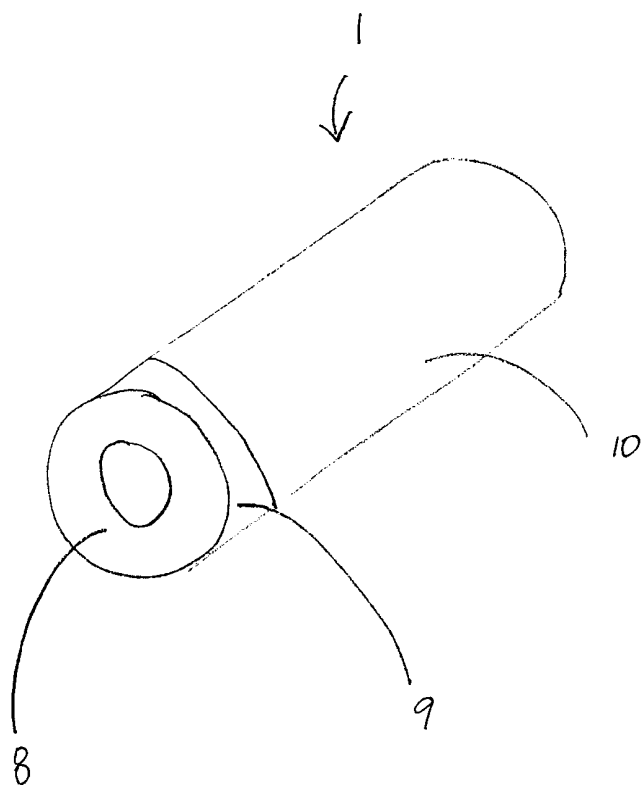


Fig. 4

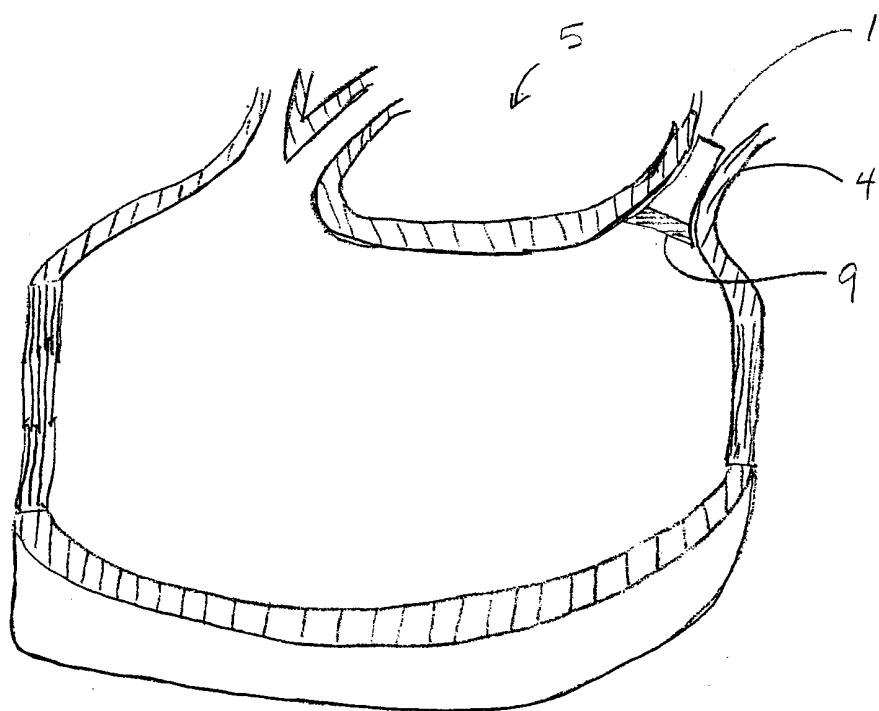


Fig. 5

INTERNATIONAL SEARCH REPORT

International application No.

PCT/US00/28488

A. CLASSIFICATION OF SUBJECT MATTER

IPC(7) : A61F 2/06
US CL : 623/1.42, 1.11

According to International Patent Classification (IPC) or to both national classification and IPC

B. FIELDS SEARCHED

Minimum documentation searched (classification system followed by classification symbols)
U.S. : 623/1.42, 1.11, 1.15, 1.43

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched
NONE

Electronic data base consulted during the international search (name of data base and, where practicable, search terms used)
NONE

C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category *	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
X	US 5,725,567 A (WOLFF et al.) 10 March 1998, column 1, lines 54-64.	1-20
A	US 5,769,883 A (BUSCEMI et al.) 23 June 1998.	1-20
A	US 5,891,108 A (LEONE et al.) 06 April 1999.	1-20



Further documents are listed in the continuation of Box C.



See patent family annex.

<p>* Special categories of cited documents:</p>		
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"O" document referring to an oral disclosure, use, exhibition or other means	"&"	document member of the same patent family
"P" document published prior to the international filing date but later than the priority date claimed		

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