DEVICES

Chronic Implantation of Intravascular Cardioverter Defibrillator in a Canine Model: Device Stability, Vascular Patency, and Anchor Histology

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Introduction: *A percutaneously placed implantable intravascular defibrillator (PICD) has been developed with a right ventricular (RV) single-coil lead and titanium electrodes in the superior vena cava (SVC) and the inferior vena cava (IVC). This study evaluated implant techniques, device stability, and anchor histology of the PICD over 9 months in a canine model.*

Methods: *Twenty-four hounds (wt = 30–55 kg) were anesthetized and a custom sheath introduced into the right femoral vein. The PICD was advanced over a wire and positioned with the titanium electrodes (cathodes) in the SVC and the IVC. A nitinol anchor secured the device in the jugular. The RV lead was positioned in the RV apex and screwed into place. The catheters, wires, and sheath were removed with an average implant time of 14 minutes. In one group of animals (n = 13), serial venograms were performed at 7 days, 14 days, and 28 days. In a second group (n = 6) and third group (n = 5), venograms were also performed at 90 days and 270 days, respectively. Six canines were sacrificed and anchor histologic examination done at 90 days.*

Results: *All implants were successful with no surgical complications observed. Devices (N = 24) remained appropriately positioned with no anchor migration. Histology at 90 days showed 98% endothelialization of the anchor. Venograms revealed patent IVC and jugular veins in all animals at every time point examined.*

Conclusions: *The PICD can be rapidly and chronically implanted in animals. Long-term intravascular defibrillator placement is feasible in a canine model. (PACE 2013; 36:1251–1258)*

implantable cardioverter-defibrillator, implantable intravascular cardioverter-defibrillator, percutaneous implantation, chronic canine model

Introduction

A percutaneously placed implantable cardioverter defibrillator (PICD) has been developed which has shown superior defibrillation thresholds (DFTs) when compared to conventional devices in animal models.[1] The device utilizes a unique defibrillation vector that includes the inferior-posterior segments of the right ventricle, shocking from a right ventricular coil (anode) to titanium cans located in the superior vena cava (SVC) and inferior vena cava (IVC), respectively (Figs. 1A and B).[2] The PICD is rapidly implanted (average of 14 minutes) from the femoral vein and designed to be completely removed when explant or replacement is required.[1] The device has a 4.5-year battery life and removal is by an entirely percutaneous technique, which has recently been described.[3]

Despite multiple studies as well as guidelines recommending ICD placement for primary prevention, it is estimated that up to 60% of patients

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Conflicts of interest: Dr. Sanders was a consultant for Synecor. Dr. Reddy was a consultant for InnerPulse.

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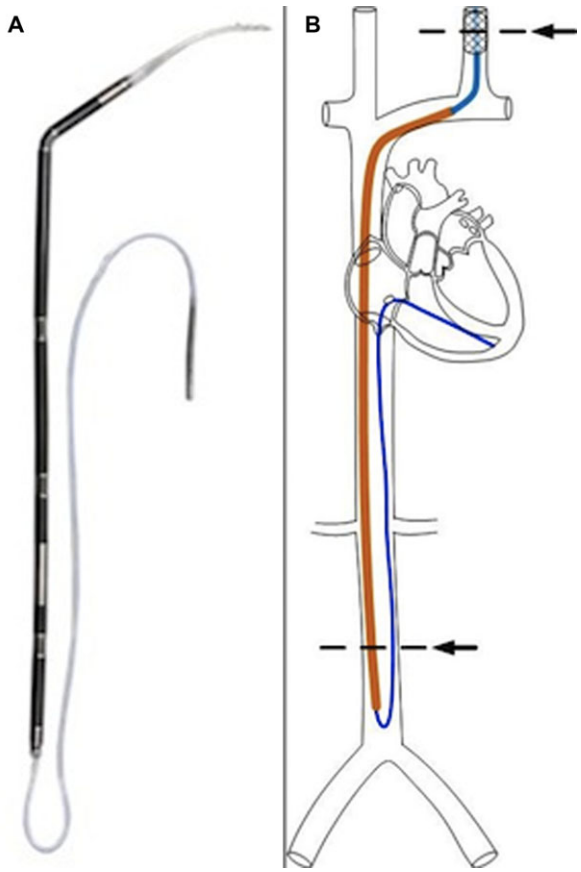

Figure 1. *(A) Percutaneous intravascular defibrillator (PICD) with right ventricular lead system. (B) Typical position of PICD in the canine venous vasculature. Arrows mark the anchoring region and caudal aspect of the device in the left external jugular vein and inferior vena cava, respectively.*

eligible for this potentially life-saving therapy go untreated.[4–7] The reasons for underutilization of the ICD in this population remains uncertain and a topic of vigorous debate.[8] The requirements of a surgical procedure and implantation by highly trained subspecialist may play a role.[4]

A percutaneously placed, intravascular device could provide certain technical implantation advantages and reduce specific surgical complications, but little is known regarding venous device compatibility. This study evaluated the implant techniques, device stability, vessel patency, and anchor histology of the PICD over 9 months in a canine model.

Methods

This study was approved by the Synecor Institutional Animal Use and Care Committee and The Arrhythmia Research Foundation of Semmelweis University prior to its initiation.

Study Design

Three groups of canines were studied based on the timing and total number of venograms that were obtained in each animal. The first group of the canines (Group 1, n = 13) had serial venograms performed at 7 days, 14 days, and 28 days. In the second group (Group 2, n = 6) and the third group (Group 3, n = 5), venograms were also performed at 90 days and 270 days, respectively. A total of 24 canines were implanted and underwent evaluation. Six canines (Group 2) were sacrificed and anchor histologic examination done at 90 days.

Surgical Preparations and Monitoring

All animals were sedated with a 5.5-mg/kg bolus of ketamine and 0.275 mg/kg of diazepam. Canines were intubated and ventilated (FG- Hallowell EMC model 2000 ventilator, Hallowell EMC, Pittsfield, MA, USA). Anesthesia was maintained using inhaled 1–5% isoflurane supplemented with 100% oxygen. Jaw relaxation was monitored to ensure the appropriate level of anesthesia. Femoral arterial pressure and surface electrocardiogram lead II were monitored continuously.

Device Positioning and Electrode Configuration

A 27Fr custom sheath (InnerPulse model 13427–01-A, InnerPulse Inc., Research Triangle Park, NC, USA), which allowed rapid exchange of multiple catheters through a single port, was placed in the right femoral vein using the Seldinger technique. Placement of the InnerPulse sheath was entirely percutaneous and required no cut down of the vessel. In a similar fashion, a 6Fr sheath (model no. 406112, St. Jude Medical, Minnetonka, MN, USA) was positioned in the left femoral artery and arterial pressure monitoring was performed via this access. After sheath placement, a bolus of 2,000 units of heparin was administered. Two custom guidewires (260 cm, 0.035″, Lake Region Medical, Inc., Chaska, MN, USA) were positioned in the left external jugular vein (LEJV) using a 9Fr standard guide catheter. The PICD (InnerPulse models #12529–34-A or #12529–38-A, InnerPulse Inc.) was advanced over one wire from the right femoral vein such that the titanium electrodes (cathodes) were located in the SVC and IVC (Figs. 1 and 2). The SVC electrode was positioned close to the brachiocephalic-SVC junction in the cephalic portion of the SVC. The IVC electrode was located just below the right atrium at the level of the diaphragm. The right ventricular (RV) lead (model #12529–38-A, InnerPulse Inc.) was introduced with a delivery catheter (model #13554–1, InnerPulse Inc.) into

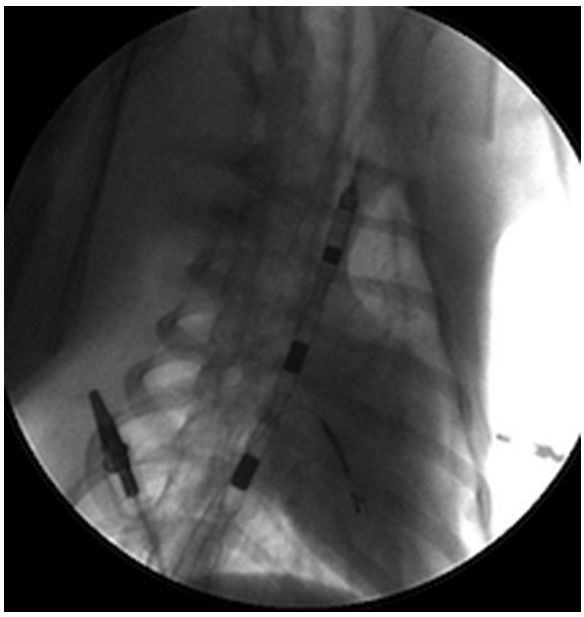

Figure 2. *Fluoroscopy of the percutaneously placed implantable cardioverter defibrillator body and right ventricular lead in the canine heart.*

Figure 3. *Interaction of the silicone rubber cephalic anchoring area of the percutaneously placed implantable cardioverter defibrillator with the nitinol anchor.*

the femoral vein, advanced to the heart through the usual course, positioned in the apex of the right ventricle, and screwed into place. Over the second wire, a self-expanding nitinol anchor (InnerPulse model #13521 or #13617, 13–18 mm, InnerPulse Inc.) was advanced to the LEJV and deployed over the PICD cephalic tip, securing the device to the vessel wall (Fig. 3). Anchor size was chosen to achieve between 15% and 45% oversizing of the vessel from the baseline measurement of LEJV diameter. All catheters, wires, and sheaths were removed. Hemostasis was achieved and all canines fully recovered from the procedure.

Stability and Migration Evaluation

Anchor stability was evaluated using relative changes in the anchor location as demonstrated with fluoroscopic/cineographic imaging. Care was taken to ensure that each animal was reproducibly positioned in an anterior-posterior position. The animal was positioned on the procedure table in a dorsally recumbent position with lateral external rotation of the forelimbs. Initial implant location was documented in reference to boney structures with the animal appropriately positioned. The line of reference was a perpendicular line to the target vessel between the shoulder girdle and the adjacent vertebrae. Relative anchor migration >2 cm by fluoroscopic examination was considered significant.

Angiographic Luminal Patency

Angiography (venography) was employed to evaluate luminal patency of all vessels examined at each time point. At implant, venograms were obtained initially with the injection catheter positioned in the LEJV just proximal to the deployed anchor and, subsequently, proximal to the bottom of the PICD located in the IVC (Figs. 4A and 5A). At appropriate time intervals for each group of canines, peripheral upper extremity venograms and catheter injection venograms were obtained to determine continued patency of the LEJV anchor region and the IVC, respectively (Figs. 4A and B and 5A and B). Any obstruction >70% was deemed significant and all measurements were compared to baseline.

Anchor Site Tissue Evaluation

In the group of six canines sacrificed at 90 days, histopathology of the anchoring region was performed to establish safety based on appropriate vessel healing without evidence of significant hemorrhage or vein perforation. Tissues surrounding the point of anchor placement were examined to determine the degree, if any, of collateral injury to adjacent organs or structures. In addition, the target vessel patency was histopathologically determined.

Histopathology Preparation

All gross necropsies were performed under the supervision of a board-certified veterinary pathologist. The LEJV anchoring region was exposed and an adequate vascular segment, which incorporated the anchor and the silicone superior tip of the PICD device, was removed. The vascular segment included >1 inch of marginal vascular tissue both distal and proximal to the implanted anchor. All tissues for histology assessment were stored in 10% buffered formalin and sent to the histology laboratory where the tissue trimming was performed.

Histology Evaluation, Light Microscopy, and Staining

Tissues were stained with hematoxylin and eosin prior to evaluation by light microscopy. Light microscopy was also performed on tissue sections taken adjacent, subjacent, or distal to the implant site to determine and characterize cellular changes.

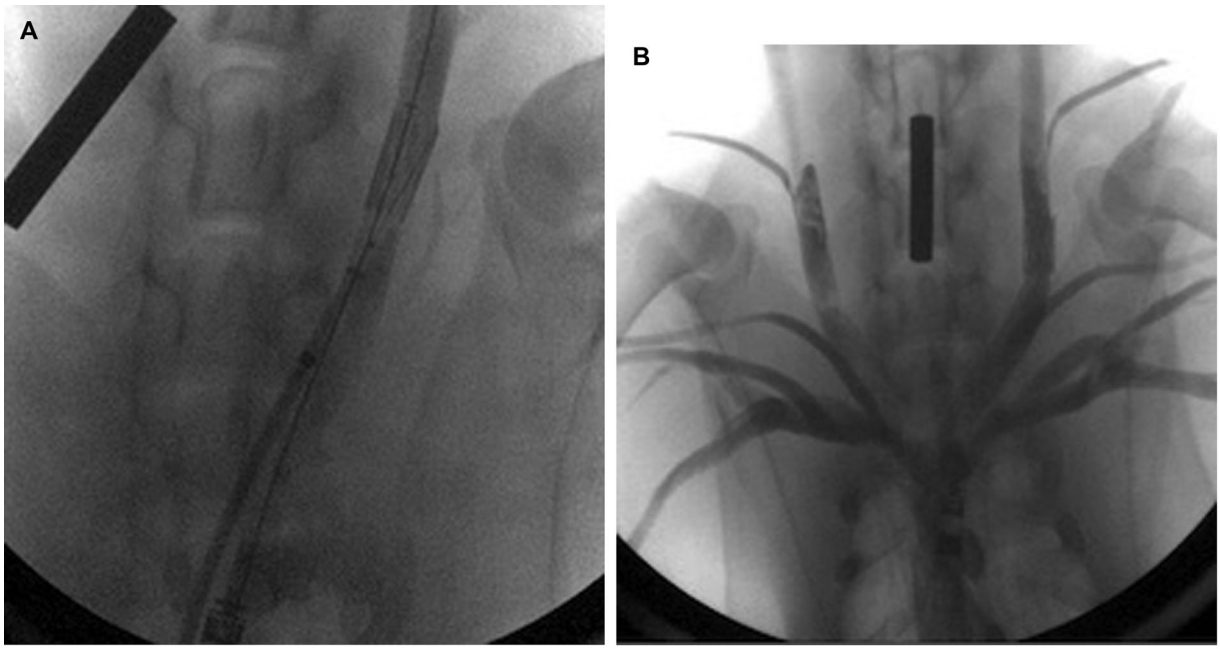

Figure 4. *Anchor and cephalic portion of the percutaneously placed implantable cardioverter defibrillator with venograms at implant and 9 months. (A) Venogram at the time of implant. (B) Upper extremity venogram showing patent anchor and normal venous vasculature at 9 months.*

Anchor: Tissue Processing

The explanted vascular segment containing the anchor was submitted intact for histopathologic assessment. The vascular segment was trimmed with sufficient margins to maintain the integrity of the areas of interest. Each vascular segment was embedded in plastic, cross-sectioned, and stained. Inclusion of anchor and tissues in polymethymethacrylate permitted thin slides and preserved the capacity to perform the appropriate staining.[9] The vascular segment was cross-sectioned in a minimum of four segments. The segments were evaluated for inflammation, capsule fibrosis, hemorrhage, endothelialization, necrosis, and luminal patency.

Results

Implantation and Positioning

The PICD was successfully implanted on the first attempt in all animals. Appropriate electrode positioning was achieved in all cases with anchor deployment in the LEJV. RV leads were screwed into locations in the apex or septum. There were no acute dislodgements of the anchor or lead. The average implantation time for this device is 14.3 $\pm$ 4.9 minutes (range 7–24 minutes). The time for implantation does not include that which is required for assessment of DFT or pacing parameters.

General Health

Canines were formally examined by a veterinarian weekly and were observed daily to detect any change in behavior. All canines were deemed in excellent health during the duration of the trial.

Baseline and Follow-Up Venograms

Venography was performed at baseline following completion of the implant procedure. Measurements of vessel diameter at the site of the anchor and the IVC at the level of the base of the PICD were obtained. All subsequent diameter evaluations at specified time points were compared to baseline. Venograms demonstrated widely patent LEJVs and IVCs in all animals at all times point tested (Figs. 4A and B and 5A and B). When compared to implant position, no significant movement of any anchor was observed and all PICDs remained in the same position. In Group 2 (n = 6) of canines, which were sacrificed at 90 days, formal vessel measurement comparisons were calculated and are shown in Tables I and II.

Anchor Histopathology

A veterinary pathologist directed all tissue preparation and performed all histopatholgic evaluations. In the six canines sacrificed at 90 days, necropsy and gross evaluation showed no

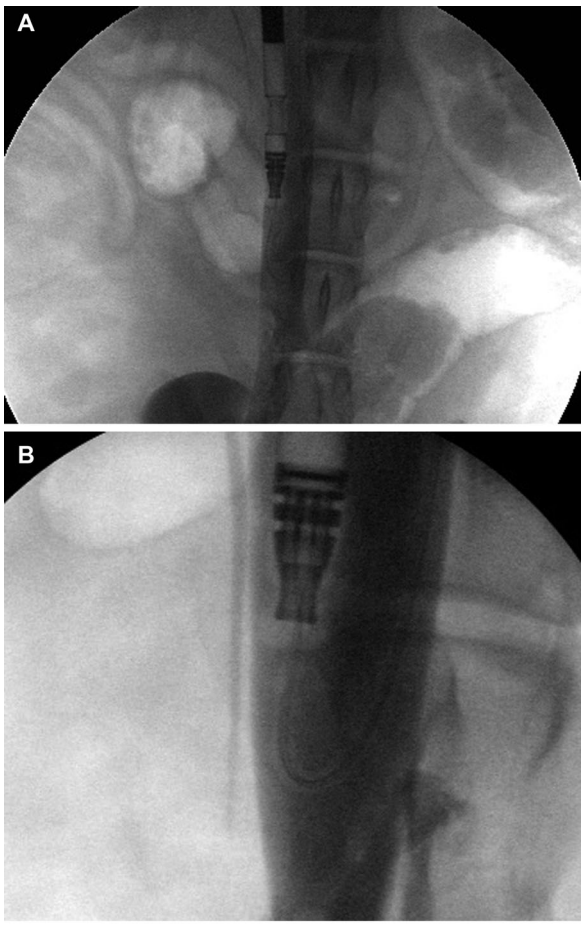

Figure 5. *Venograms of inferior vena cava with percutaneously placed implantable cardioverter defibrillator (PICD) in appropriate position at implant and 9 months. (A) Implant. (B) Nine months postimplant. Distal venous system is normal. Persistent slight dilatation of the IVC, which contains the PICD. No evidence of thrombosis or collateral formation.*

evidence of anchor perforation, blood extravasation, or damage to surrounding tissues. The anchors and device tip-anchoring region were encased in a fibrosis capsule. Vein lumen was patent in all instances with no microscopic evidence of perforation or blood in vessel wall or surrounding tissues (Fig. 6). This finding was confirmed in both gross and histopathology evaluations. The histopathologic examination demonstrated that greater than 98% of all anchor wires were endothelialized and were covered with a mature neointima composed of collagenous fibrovascular tissue generally between 0.1-mm and 1-mm thick. No significant inflammation was observed (Fig. 6).

Discussion

Implantable defibrillators are proven to reduce mortality when employed as primary prevention in heart failure patients; however, this lifesaving therapy remains underutilized in the target population. The development of a percutaneously placed device, requiring no surgical technique, which can be achieved more rapidly than conventional ICD insertion, could potentially impact acceptance of this therapeutic approach. The PICD has a battery life of 4.5 years and, because of its unique totally intravascular location, implantation and removal techniques were developed simultaneously.[3] R-wave determination, pacing thresholds, and ventricular fibrillation induction are performed via a hand-held programmer, which has a wireless telemetry range of 6–8 feet. The time required to evaluate typical pacing parameters and DFT is 3–5 minutes.[1,10] At the end of battery life, the device can be removed and a second PICD placed in a similar location. The PICD consists of a series of isodiametric titanium cans connected at manufacturing to an RV lead (Fig. 1A). It represents, to our knowledge, the first defibrillator and lead system specifically designed to be removed.

Venous access was attained utilizing a custom 27Fr sheath in this animal study. In order to ensure adequate hemostasis in these active canines, the vessels and wounds were sutured after sheath removal. No significant groin complications were observed in any animal. A human trial of 10 patients using the same sheath has demonstrated excellent venous hemostasis with 20 minutes of manual compression without a closure device or other intervention.[10] One minor hematoma occurred, which resolved by day 7 postprocedure and required no therapy.[10]

The isodiametric form factor allows the device, when snared from below, to torque in a 1:1 fashion unlike conventional leads. The RV lead has a detachment region just proximal to the screw, which permits lead removal by traction from the IVC without the use of laser, radiofrequency, or extraction sheaths. All pacemaker and defibrillator leads are to varying degrees encapsulated in fibrosis tissue and subsequently either totally or partially endothelialized.[11–13] At points of wall contact, neointimal proliferation and calcification can occur.[14] The PICD has been observed to freely float in the venous blood pool, touching only at the anchoring site and the RV apical screw. The body of the device and lead do experience typical thin fibrosis tissue encapsulation. The isodiameteric form factor and the ability to torque allow sliding if neointimal proliferation or adhesion does occur.

For removal, the IVC segment of the PICD is snared utilizing a custom catheter. The RV lead is separated from the body of the PICD by cutting the lead inside a protective sheath. A novel catheter with a surgical cutting wire is then advanced over

Table I.

LEJV-Original Diameter, Degree of Oversizing with Anchor, Patency, and Diameter at 45 Days and 90 Days Postimplantation

Animal #	OD IID Device Anchor Location	OD IID Target Vessel (mm)	IID Device Anchor Size (mm)	% OS	Baseline Functional Lumen Diameter (mm)	45 Days Follow-Up Functional Lumen Diameter (mm)	45 Days Luminal Patency (%)	90 Days Follow-Up Functional Lumen Diameter (mm)	90 Days Luminal Patency (%)
D-481	LEJV	8.2	11	134.1	8.9	10.7	119.6	10.5	117.3
D-482	LEJV	9.8	13	132.7	9.2	12.2	132.6	13.0	141.9
D-483	LEJV	7.7	11	142.9	8.2	11.1	136.3	10.7	130.5
D-479	LEJV	8.7	11	126.4	8.7	11.3	129.4	10.6	121.4
D-506	LEJV	9.4	13	138.3	10.3	12.1	116.7	12.3	119.4
D-509	LEJV	11.0	15	136.4	10.3	12.7	123.3	14.0	135.7
Mean		9.1		135.1	9.3	11.7	126.3	11.8	127.7
SD		1.2		5.5	0.9	0.8	7.7	1.5	9.9

LEJV = left external jugular vein; SD = standard deviation.

Table II.

IVC Patency and Diameter at 45 Days and 90 Days Postimplantation of PICD

Animal #	Baseline IVC Diameter (mm)	45 Days IVC Diameter (mm)	45 Days Luminal Patency (%)	90 Days IVC Diameter (mm)	90 Days Luminal Patency (%)
D-481	15.9	15.0	94.6	17.8	112.5
D-482	13.5	15.9	118.1	21.2	157.6
D-483	12.8	13.6	106.2	18.9	147.0
D-479	12.8	17.0	132.7	18.9	147.9
D-506	16.3	15.4	94.5	20.4	125.0
D-509	17.7	21.0	118.3	19.5	109.9
Mean	14.8	16.3	110.7	19.5	133.3
SD	2.1	2.5	15.1	1.2	20.2

IVC = interior vena cava; PICD = percutaneously placed implantable cardioverter defibrillator; SD = standard deviation.

the device. The PICD acts as a rail to advance the cutting wire to the anchoring region. The silicone segment of the PICD is detached from the nitinol anchor by the cutting catheter and the entire device is removed via the femoral vein. Subsequently, the RV lead is snared. A cutting wire is positioned at a specific detachment region at the tip and the lead is cut and removed through the femoral vein. After device removal, a new PICD can be placed and this procedure has been safely performed in the canine model.

This removal technique employs separation of the lead and anchoring segments by cutting with either a surgical wire or custom intravascular scissors. In order to perform this operation safely, all cutting takes place either inside a sheath or with the device serving as a rail to guide the tool. Although not observed in this trial, vein entrapment or adhesion in the area of cutting could result in vessel damage and possible hemorrhage or thrombosis. Careful fluoroscopic examination of the site prior to tool engagement is important to ensure proper location and that the PICD is freely floating in the vascular space. This removal procedure requires less than 20 minutes and has been performed in canines (N = 10) by independent operators with no complications.[3] Minor variability was observed in time required for removal of the PICD between operators. The difference appeared dependent on a learning curve for the procedure as well as the RV lead position.

Benefits of such a percutaneous system might include the elimination of many of the factors that have traditionally contributed to lead and

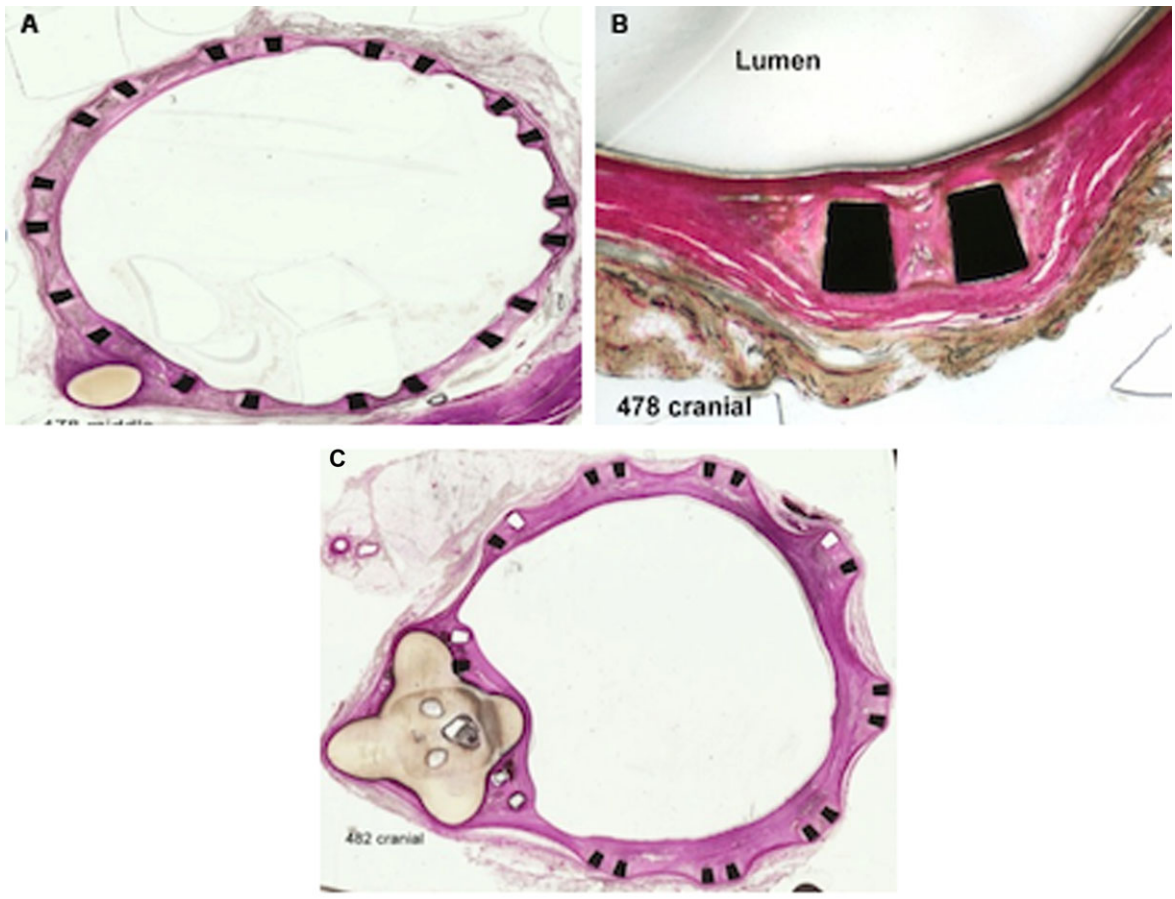

Figure 6. *Histologic sections at 3 months postimplant through anchor in two canines showing silicone rubber percutaneously placed implantable cardioverter defibrillator (PICD) tip in stable position and secured. Vein is widely patent in anchoring region. (A) Middle portion of anchor. Black struts of anchor are evident and confined within the vein wall. No evidence of perforation. Yellow PICD tip pressed against wall in appropriate position. (B) High magnification showing endothelialization of anchor wires. Black struts of anchor are evident and confined within the vein wall. No evidence of perforation. (C) Cranial aspect of the anchor and PICD tip. No evidence of perforation. Yellow PICD tip pressed against wall in appropriate position with tines visible.*

device complications. The PICD lead is not subject to pocket insulation erosion, loose setscrews, subclavian crush, or the forces that result in lead perforation due to its unique attachment to the device and lead location in the vasculature. Device pocket infection or erosion are not possible and there is an obvious cosmetic and comfort advantage for the patient.

Stents in the large veins have been employed to treat a variety of disorders, including Budd-Chiari syndrome and obstructive thrombosis.[15–17] In the IVC, stents have been effectively positioned for the treatment of thrombosis in a vena caval filter.[18] More recently, caval stents have anchored valves for therapy of severe tricuspid regurgitation.[19] However, little is known concerning the long-term vascular response of stenting normal venous structures and even less is understood about employing veins to anchor or secure a medical device. This trial evaluates the anchor vascular effects and histopathologic vessel response to the placement of a percutaneous defibrillator, which is held in place by a nitinol anchor.

This trial showed minimal venous vascular response to the anchoring stent in the LEJV. The anchor uniformly maintained the PICD in the implanted position and all venous structures remained widely patent. Histopathologic examination of anchor region showed incorporation of the anchor into the venous wall and near-complete endothelialization of the wire struts. No damage to surrounding tissues was seen. The LEJV at the level of the anchor at 3 months maintained the oversizing range that was targeted at implant (Table I). The IVC shows modest

dilatation throughout the course of the PICD (Table II).

Conclusions

The PICD can be securely anchored in the canine venous vasculature and has no deleterious effects on the vessel or surrounding tissue. Nitinol anchors similar to stents can be placed in veins and rapidly incorporated into the vessel wall and their lumen surfaces endothelialize within 3 months. Vein patency is maintained with an implanted PICD in a canine model.

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