Published in final edited form as:

Heart Rhythm. 2015 July ; 12(7): 1683–1690. doi:10.1016/j.hrthm.2015.03.022.

Preclinical Testing and Optimization of a Novel Fetal Micropacemaker

Yaniv Bar-Cohen, MD^a, Gerald E. Loeb, MD^b, Jay D. Pruetz, MD^a, Michael J. Silka, MD^a, Catalina Guerra, DVM^c, Adriana N. Vest^b, Li Zhou^b, and Ramen H. Chmait, MD^d

^aDivision of Cardiology, Children's Hospital Los Angeles, Keck School of Medicine University of Southern California, 4650 Sunset Boulevard, MD #34, Los Angeles, CA 90027

^bDepartment of Biomedical Engineering, University of Southern California, 1042 Downey Way, DRB-B11, Los Angeles, CA 90089

^cC. W. Steers Biological Resources Center, Los Angeles Biomedical Research Institute, Harbor - University of California, Los Angeles, 1124 W. Carson St., E-2 North, Torrance, CA 90502

^dDepartment of Obstetrics and Gynecology, Keck School of Medicine, University of Southern California, 1300 North Vermont Avenue #710, Los Angeles, CA 90027

Keywords

fetus; heart block; pacemaker; heart failure; bradycardia

Introduction

Complete heart block (CHB) in the human fetus may result in progressive bradycardia with the development of hydrops fetalis in a quarter of these pregnancies.^{1,2} Once hydrops fetalis occurs, if the fetus cannot be delivered due to prematurity or other clinical concerns, fetal demise is nearly inevitable. Although the degree of myocardial dysfunction due to antibody-mediated damage in congenital CHB is variable, successful pacing of a fetus with CHB and hydrops fetalis could theoretically allow resolution of hydrops in several weeks and permit an otherwise normal gestation. Historical attempts at pacing human fetuses, however, have invariably failed with no survivors to date.^{3–6} To address this problem, we have designed a single-chamber pacing system (Figure 1) that is self-contained and can be percutaneously implanted in the fetus without exteriorized leads, thereby permitting subsequent fetal movement without risk of electrode dislodgement.

Correspondence: Yaniv Bar-Cohen, MD, Children's Hospital Los Angeles, 4650 Sunset Blvd, MS #34, Los Angeles, CA 90027, Phone: (323) 361-2461, Fax: (323) 361-1513, ybarcohen@chla.usc.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Disclosure: Patent applications filed relating to the micropacemaker device

Previous preclinical animal studies in adult rabbits have demonstrated the viability of our implantation scheme and have allowed us to optimize the electronics of our pacing system.⁷ We now report on implantation outcomes of functional micropacemakers in fetal sheep.

Materials and Methods

Device design and function

In order to meet our rigorous requirements regarding device size, power consumption and development cost, we utilized a simple relaxation oscillator based on a single transistor.⁸ The device functions in a fixed-rate mode (i.e., VOO) with a rate that predictably varies with the battery voltage (generally 100–110 bpm). The output pulse exponentially decays with a total charge that is fixed by the electronic component values and is independent of electrode impedance.⁷ In 6 of 7 experiments, the devices had a 3 μC output pulse (3V peak, 250 μs time constant for fully charged battery); this corresponds approximately to a conventional 2 V over 0.4 ms square pulse. The 3mAh lithium battery cell in the implants sustains pacing for approximately 6 days and can be recharged by inductive coupling of a 6.78 MHz electromagnetic field from a transmitting coil positioned outside the maternal body. With the 6th implant, a higher output device (7 μC ; 3-day recharging interval) was used.

Device implantation

Implantation experiments were performed on pregnant fetal sheep (*ovis aries*, Rambouillet and Columbia mix breed) at 112–128 days of gestation with only singleton pregnancies included. The protocol conformed to the Guide for the Care and Use of Laboratory Animals and was approved by the Institutional Animal Care and Use Committees at the University of Southern California and the Los Angeles Biomedical Research Institute. The ewes were anesthetized with ketamine and atropine, followed by isoflurane. Oxacillin and gentamicin were given preoperatively. Dissection down to the uterine wall was made via a midline abdominal incision, and the uterus was externalized. A purse-string suture was placed and a uterine incision was made. The fetal thorax was exposed and electrodes were placed in triangulated locations surrounding the fetal heart to allow fetal ECG evaluation.

In the first three animals, the fetal subxyphoid area was exposed via the uterine incision for insertion of the percutaneous implantation trocar and cannula (3.8 mm internal diameter, 4.5 mm external diameter; Figure 1) (Richard Wolf Inc., Vernon Hills, IL). In the latter four animals, the trocar and cannula were advanced percutaneously through an intact area of the uterine wall and into the fetus at the subxyphoid region. Under ultrasound guidance, the trocar and cannula were advanced through the fetal diaphragm and towards the ventricles. Once ultrasound imaging suggested that the cannula and trocar were against the epicardial surface, the trocar was removed and the pacemaker implantation system was advanced through the cannula. After the distal electrode screw reached the epicardium, the device was rotated clockwise into the myocardium. The device was then released (Figure 2) by slowly withdrawing the cannula while keeping the device in place with a pushrod. In cases where successful pacing could not be demonstrated with the initial device, a second and once a third device implantation attempt was performed in the same animal.

After initial experiments demonstrated difficulty in correctly positioning the implantation cannula at the epicardial surface, a pericardial effusion was created in the four latter experiments. This was performed in order to more closely replicate the clinical anatomy in a human fetus with hydrops fetalis. After the uterine wall was exposed and fetal electrodes were placed, a 17 cm, 21-gauge needle was advanced to the epicardial surface (Figure 3A). When the needle appeared to have entered the pericardium, saline was injected until ultrasound demonstrated the presence of a small pericardial effusion (Figure 3B). The cannula and trocar was then inserted as described above with improved visualization of the epicardial surface.

After device deployment, the purse string uterine incision was closed and the uterus was returned to the abdominal cavity. The fetal electrodes were delivered through a small incision in the maternal abdomen for postoperative access. Final ultrasound imaging of the fetus was performed to evaluate the heart rate and rhythm, cardiac function, electrode and device position and overall fetal wellbeing. The sheep was awakened from anesthesia, administered Penicillin G, and returned to the animal housing.

Fetal sheep follow-up and device recharging

Repeat ultrasound evaluations were performed in the first 24–48 hours of follow-up and once to twice per week thereafter. In addition, the externalized fetal skin electrodes were used to obtain a fetal ECG during follow-up. In the last four animals, recharging of the fetal pacemaker was attempted using an external recharging ring that was specifically developed for the micropacemaker.⁸ The design of the pacing circuit was modified to increase the pacing rate modestly (5–10 bpm) in proportion to the received strength of the RF magnetic field. By evaluating the pacing rates when the recharging ring was deactivated (i.e., no external voltage applied), the battery voltage and state of charge could be determined.

Evaluation of pacing

Because the pacing rates in VOO mode (100–110 bpm) were generally slower than the fetal heart rates (130–180 bpm), ventricular capture was detected by the presence of an irregular fetal heart rate. This was identified by a pacing stimuli occurring after the ventricular refractory period and resulting in a premature QRS complex on the fetal ECG (Figure 4). The pacing system did not allow for wireless threshold and impedance measurements after implant, but direct measurements of pacing lead capture threshold and impedances were performed in sheep #5 just prior to euthanasia.

Device explantation

The animals were euthanized (pentobarbital / phenytoin solution) after the follow-up period was completed, and a fetal thoracotomy was performed to determine the device location (micropacemaker, flexible lead and electrode). In addition, injury and inflammation of the surrounding tissues were evaluated, and necropsy specimens were sent for histological evaluation.

Results

Evolution of Acute Implantation Technique

Device implantations were performed in seven pregnant sheep. Table 1 summarizes the procedure results, with ventricular capture after device placement demonstrated in five of the seven animals. Due to intermittent failure of device output with the first implant, the circuit board design was altered and no further electronic device failures were seen. In addition, there was prolonged exteriorization of a large portion of the fetus outside the uterus during implant, associated with spontaneous pleural and pericardial effusions and ascites on post-operative day (POD) #1 which resolved during follow-up. Exteriorization of the fetus from the uterus was minimized in subsequent procedures, and significant effusions were not seen in follow-up after the first experiment.

Blunted screw electrode tips were identified as the cause of failure to penetrate the myocardium with the second procedure and felt to be due to the storage process. The recharging storage system was altered to avoid tip blunting due to electrode tip chafing. In addition, refinements of electrode design and screw bevel were made after a series of experiments on cadaver chicken hearts.

After the third procedure, a pericardial effusion was created for subsequent implants to improve imaging and enlarge the pericardial space for percutaneous access. There were no major procedural changes in the final four experiments.

Follow-up and recharging

Five of the seven animals were followed after the acute implant, with two euthanized at the initial implant (Table 1). An increase in capture thresholds during follow-up was suggested in three animals (Sheep #5, 6, 7) with either intermittent or no capture seen despite pacemaker stimuli. Recharging was attempted in the last four animals and confirmed in three.

For sheep #4, only a weak recharging field was seen by the device, due to difficulty determining the micropacemaker orientation by ultrasound imaging in addition to malfunction of the recharging system. The external recharging system electronics were augmented, and the pacing circuit was modified to generate a larger increase in the pacing rate in proportion to the received strength of the RF magnetic field. Device recharging was demonstrated in subsequent animals.

In sheep #5, lead characteristics were manually determined for the two implanted devices just prior to euthanasia on POD#15. For the first device, the ventricular capture was 3.75 V @0.5 ms and 5.0 V @0.2 ms; the impedance was 258 Ohms. For the second device, the ventricular capture was 2.5 V @0.5 ms and 5 V @0.1 ms with impedance of 230 Ohms.

Necropsy and Histology

Maternal uterine evaluation suggested normal healing at the purse string incision and only a small scar at sites where the cannula directly penetrated the uterine wall. Similarly, external

evaluation of the fetal chest and abdomen showed a small scar at the site of cannula insertions. The location of the pacing system was also determined (Figure 5).

Necropsy identified the ventricular locations where each electrode had been implanted. In the first two experiments, the disks at the proximal end of the electrode screw were seen to be entirely within the pericardium, suggesting that the sheath had penetrated the pericardium. In the remaining experiments, the disk was seen outside the pericardium. Histology was performed to identify the degree of myocardial inflammation where the electrodes were implanted. When the electrode had penetrated the epicardium, a significant inflammatory response was identified (Figure 6). In the fourth sheep, the necropsy report concluded that a placental infection was the most likely etiology of the fetal demise, although the specific microorganism was not identified. Necropsy did not identify a clear cause of death for sheep #6 and 7.

X-Ray imaging of the micropacemaker system and surrounding tissues were performed after necropsy in sheep #1, 4 and 5 in order to determine the integrity of the flexible lead / electrode prior to removal of the hardware from the tissue. A broken flexible lead was seen with the first device (fracture at the insertion of the lead into the micropacemaker) and was determined to be secondary to a weakening of the wire at that location during the fabrication process. This joint was augmented for future experiments and there were no lead fractures for the subsequent six experiments (Figure 7).

Discussion

CHB in the human fetus with hemodynamic compromise presents a significant therapeutic challenge. While many fetuses with CHB survive to birth, a quarter will develop hydrops fetalis, resulting in nearly universal mortality unless early delivery can be achieved.¹ Cardiac pacing is the standard therapy for heart block, but a method to successfully pace a fetus has been elusive, as no human fetuses have survived attempts at fetal pacing to date. Our fetal micropacemaker addresses the two major challenges of fetal pacing by 1) being a fully implantable pacing system, and 2) allowing for percutaneous implantation without the need for open fetal surgery. The micropacemaker system is designed to be implanted completely inside the fetus, in contrast to previous fetal pacing attempts which have placed electrodes on the fetal myocardium with pacing wires exiting the fetal body into a pacemaker implanted in the maternal abdomen.³⁻⁵ The presence of the extracorporeal pacing wire is problematic in that fetal movement may result in lead dislodgment or impingement of other structures (including the umbilical cord). Furthermore, with extracorporeal procedures, there are maternal as well as fetal risks to be considered (maternal laparotomy and hysterotomy).^{6,8} The high risks associated with open fetal surgery were a primary impetus for developing a percutaneous delivery system and a micropacemaker that was small enough to be implanted completely within the fetus via instruments and approaches in routine use for other fetal interventions.¹⁰

Our initial targets for the fetal micropacemaker will be fetuses with CHB and hydrops fetalis. In the human fetus, we expect to engage the ventricles directly through the chest, as opposed to the subxyphoid approach used in our fetal sheep model. These fetuses are

expected to have pericardial and pleural effusions, which should facilitate access to the fetal heart and micropacemaker implantation. A major challenge of our experimental model was accurately placing the cannula tip within the pericardium (past the parietal pericardium) and directly against the epicardial surface. While alteration of our experimental protocol to include creation of a pericardial effusion resulted in improved visualization, appropriate placement of the needle tip in the pericardial space remained challenging.

While there are various methods to create fetal hydrops,^{11,12} they are limited by significant hemodynamic compromise and fetal morbidity. In addition, although fetal sheep models of CHB have been proposed,¹³ creating iatrogenic fetal heart block was not necessary, as we were able to clearly identify ventricular capture (as well as ascertain the pacing rate) despite a faster intrinsic fetal heart rate.

Because our pacing system does not allow for alterations in pacing outputs after implantation, we chose an output based on the limited literature on pacing human fetuses, as well as data from our rabbit experiments.⁷ A single case report provides a pulse-duration curve for a human fetal pacing⁴ and is consistent with epicardial pacing parameters in neonates, children and adults.¹⁴ However, as the literature suggests that fetuses exhibit less fibrosis than children and adults,¹⁵ we did not anticipate the observed degree of myocardial inflammation and subsequent capture threshold elevations after electrode placement. After observing the inflammatory reaction that was present histologically at the electrodermyocardial interface, it is clear that a significant inflammatory response does occur in the fetal myocardium. As a result, we are considering including a steroid-eluting component in our fetal micropacemaker electrodes in addition to ultimately using a higher output device.

Our micropacemaker has a diameter less than 3.5 mm diameter and is 5 to 7 times smaller than the newly developed leadless pacemakers currently under investigation for transvenous implantation [Micra™ by Medtronic, Inc. (Minneapolis, MN) and Nanostim™ by St. Jude Medical, Inc. (St. Paul, MN)]. The very small size (0.15 cc volume) and cylindrical shape of our pacemaker necessitates a small battery with limited charge capacity. As a result, we designed and incorporated a recharging system to allow the pacemaker device to function for the duration of the pregnancy (anticipated 3–5 months). While the technical details of this recharging system are reported separately,⁷ it is worth noting that the electronic circuit was redesigned during these experiments such that small fluctuations in the recharging field seen by the micropacemaker would result in relatively large changes in the pacing rate. This modification proved very useful for optimizing the recharging time since changes in fetal and maternal positioning could be quickly recognized (by a decrease in the pacer rate) and the recharging coil re-positioned accordingly.

During the follow-up period, three fetuses expired and were prematurely delivered. Although the necropsy suggested that infection was the cause of death for sheep #4, a cause of death was not determined for sheep #6 and #7. Good fetal health during the follow-up periods had been suggested by ultrasound, suggesting that the animals had tolerated the initial implantation procedures. The deaths were therefore unexpected in both cases. The micropacemaker implantation procedures in the fetal sheep model were significantly more

invasive compared to the planned percutaneous insertion in a human fetus. This was due to the need to surgically open the uterus for placement of fetal ECG leads for confirmation of ventricular capture in the setting of rapid intrinsic fetal heart rates. These more invasive procedures performed in the fetal sheep model may have significantly contributed to the fetal demise.

The results of our acute and chronic fetal sheep testing are encouraging for the viability of this system for human fetal use. Although the lack of a significant pericardial effusion in the fetal sheep model resulted in a more complex implantation procedure than we expect in a human fetus with hydrops fetalis, we were able to successfully implant the electrode into the fetal myocardium and confirm ventricular capture. In addition, we were able to recharge the device wirelessly through the maternal abdomen. Our device has been granted a Humanitarian Use Device designation by the Food and Drug Association, and we ultimately plan to progress to a clinical trial for device approval under a Humanitarian Device Exemption.

Limitations

The development of the fetal micropacemaker has been an iterative process, which has inevitably required re-design and alterations of our initial protocol, particularly in the first three experiments. In addition, the ability to introduce the cannula and directly engage the fetal myocardium, even in the presence of a pericardial effusion, has remained a technical challenge. For clinical use, however, we expect to only target human fetuses with larger pericardial effusions due to hydrops fetalis, thereby facilitating the implantation procedure. Lastly, while fixed rate VOO pacing may not be the optimal pacing modality, the current capabilities of our device are limited by finite size considerations. Since most fetuses with CHB and hydrops fetalis will have intrinsic rates <60 bpm, we do not expect the intrinsic rates to compete with a paced VOO rate of >100 bpm.

Conclusions

A percutaneously-implantable fetal micropacemaker system provides a new approach to pacing fetuses with CHB and hydrops fetalis. The device is completely implantable within the fetus and thus minimizes the risks of lead dislodgement due to fetal movement. In addition, the minimally invasive percutaneous implantation approach avoids the risks of open fetal surgery. While the fetal sheep model presented some unique challenges for implantation compared to a hydropic human fetus, our data provide support for and demonstrate the necessity of a clinical trial to confirm that the fetal micropacemaker is a viable option for treatment of this potentially fatal condition.

Acknowledgements

We thank Dr. Joshua Kramer and Charter Preclinical Services for histologic interpretations and analyses of the necropsy specimens. This research was funded by an NIH R01 grant (1R01HD075135), the Southern California Clinical and Translational Science Institute, and the Coulter Foundation.

References

1. Lopes LM, Tavares GM, Damiano AP, Lopes MA, Aiello VD, Schultz R, Zugaib M. Perinatal outcome of fetal atrioventricular block: one-hundred-sixteen cases from a single institution. *Circulation*. 2008; 118:1268–1275. [PubMed: 18765396]
2. Eliasson H, Sonesson SE, Sharland G, et al. for the Fetal Working Group of the European Association of Pediatric Cardiology. Isolated atrioventricular block in the fetus: a retrospective, multinational, multicenter study of 175 patients. *Circulation*. 2011; 124:1919–1926. [PubMed: 21986286]
3. Walkinshaw SA, Wlech CR, McCormack J, Walsh K. In utero pacing for fetal congenital heart block. *Fetal Diagn Ther*. 1994; 9:183–185. [PubMed: 8060513]
4. Assad RS, Zielinsky P, Kalil R, Lima G, Aramayo A, Santos A, Costa R, Marcial MB, Oliveira SA. New lead for in utero pacing for fetal congenital heart block. *J Thorac Cardiovasc Surg*. 2003; 126:300–302. [PubMed: 12878975]
5. Carpenter RJ Jr, Strasburger JF, Garson A Jr, Smith RT, Deter RL, Engelhardt HT Jr. Fetal ventricular pacing for hydrops secondary to complete atrioventricular block. *J Am Coll Cardiol*. 1986; 8:1434–1436. [PubMed: 3782646]
6. Egtesady P, Michelfelder EC, Knilans TK, Witte DP, Manning PB, Crombleholme TM. Fetal surgical management of congenital heart block in a hydropic fetus: lessons learned from a clinical experience. *J Thorac Cardiovasc Surg*. 2011; 141:835–837. [PubMed: 20674938]
7. Loeb GE, Zhou L, Zheng K, Nicholson A, Peck RA, Krishnan A, Silka M, Pruetz J, Chmait R, Bar-Cohen Y. Design and testing of a percutaneously implantable fetal pacemaker. *Ann Biomed Eng*. 2013; 41:17–27. [PubMed: 22855119]
8. Zhou L, Vest AN, Chmait RH, Bar-Cohen Y, Pruetz J, Silka M, Zheng K, Peck R, Loeb GE. A Percutaneously Implantable Fetal Pacemaker. *Conf Proc IEEE Eng Med Biol Soc*. 2014; 2014:4459–4463. [PubMed: 25570982]
9. Golombeck K, Ball RH, Lee H, Farrell JA, Farmer DL, Jacobs VR, Rosen MA, Filly RA, Harrison MR. Maternal morbidity after maternal-fetal surgery. *Am J Obstet Gynecol*. 2006; 194:834–839. [PubMed: 16522421]
10. Chmait R, Kontopoulos E, Quintero R. Fetoscopic management of complicated monochorionic twins. *Clin Obstet Gynecol*. 2009; 52:647–653. [PubMed: 20393415]
11. Andres RL, Brace RA. The development of hydrops fetalis in the ovine fetus after lymphatic ligation or lymphatic excision. *Am J Obstet Gynecol*. 1990; 162:1331–1334. [PubMed: 2339736]
12. Rice HE, Estes JM, Hedrick MH, Bealer JF, Harrison MR, Adzick NS. Congenital cystic adenomatoid malformation: a sheep model of fetal hydrops. *J Pediatr Surg*. 1994; 29:692–696. [PubMed: 8035286]
13. Shiraishi H, Kikuchi Y, Hoshina M, Ohki T, Ayustawati, Momoi MY. Hemodynamic effect of the ventricular pacing site in fetal lambs with complete atrioventricular block. *Pacing Clin Electrophysiol*. 2002; 25:1731–1736. [PubMed: 12520674]
14. Fortescue EB, Berul CI, Cecchin F, Walsh EP, Triedman JK, Alexander ME. Comparison of modern steroid-eluting epicardial and thin transvenous pacemaker leads in pediatric and congenital heart disease patients. *J Interv Card Electrophysiol*. 2005; 14:27–36. [PubMed: 16311936]
15. Dang C, Ting K, Soo C, Longaker MT, Lorenz HP. Fetal wound healing current perspectives. *Clin Plast Surg*. 2003; 30:13–23. [PubMed: 12636212]

Figure 1. Photographs of the implantation equipment. A: Implantation cannula with trocar inside. B: Sharp tip of trocar protruding from end of cannula. C: Micropacemaker inside its implantation sheath protruding through cannula. D: Micropacemaker device with distal electrode screw connected to the micropacemaker (3.475 mm diameter, 18 mm long) via a coiled flexible lead.

Figure 2.
Micropacemaker deployment: Ultrasound image showing implanted electrode (arrow) and
implantation cannula (arrow) just after deployment of the micropacemaker.

Figure 3.

Creation of pericardial effusion: A long needle (arrow) is inserted into the pericardial space under ultrasound guidance (A). Saline is injected in order to create a transient pericardial effusion (B).

Figure 4.

Demonstration of ventricular capture: Fetal electrocardiogram demonstrating ventricular capture. Stimulus artifacts from the pacemaker are seen to march out at the pacing rate of 118 BPM while the intrinsic fetal heart rate is 170 BPM. Advancement of the QRS (arrow) is seen immediately after a stimulus artifact when ventricular capture occurs.

Figure 5.
Necropsy: Necropsy demonstrating the micropacemaker device (large arrow) lying through the diaphragm. The electrode is seen penetrating the left ventricular epicardium (small arrow) (sheep #4)

Figure 6. Histology at implantation site: Histology from sheep #4 demonstrating the path of electrode (seen as round voids) as it tunneled into the myocardium. There is moderate myocardial degeneration and necrosis with a mixed inflammatory reaction including macrophages, lymphocytes and neutrophils.

Figure 7.
Radiograph of micropacemaker: Radiograph of a fetal sheep heart specimen fixed in formaldehyde (sheep #4). The electrode is connected to the micropacemaker via an intact flexible lead.

Table 1

Implantation and Follow-up

Animal number	Gestational age at implant (days)	Device follow-up time (days)	Total procedure time	Open uterus time	Number of devices	Successful Capture?	Implantation comments	Follow-up
1	112	19	2:15	1:49	1	Yes	Failure of electronics causing intermittent pacemaker output.	POD1: Effusions (pericardial and pleural) seen on. POD1 2: Complete resolution of effusions. POD19: Euthanasia.
2	127	N/A	2:50	2:24	3	No	Electrode screws did not penetrate epicardial surface.	
3	128	N/A	1:40	1:23	2	No	First device did not contact epicardial surface. Second device placement resulted in myocardial perforation and cardiac tamponade	
4	127	5	2:25	0:45	1	Yes	Pericardial effusion created (40 cc)	POD1 and POD3: Capture and good fetal health. POD5: Animal found delivered and deceased.
5	128	15	2:30	0:23	2	Yes	Pericardial effusion created (40 cc). Capture seen on first device after implant and capture of second device	POD1: Good fetal health, 1 device captures POD7: Good fetal health, no capture POD12: 2 devices recharged, no capture. POD15: Elective termination
6	120	5	0:50	0:20	1	Yes	Pericardial effusion created (40 cc)	POD2: Intermittent capture, good health POD5: Fetal death, sheep in labor
7	125	6	1:30	0:17	1	Yes	Pericardial effusion created (50 cc)	POD2: No capture, good fetal health POD6: Fetal death, sheep in labor

POD: Post-operative day