Dual-Chamber Pacemaker Implantation via the Cephalic Vein in Healthy Equids

G. van Loon, W. Fonteyne, H. Rottiers, R. Tavernier, L. Jordaens, L. D'Hont, R. Colpaert, T. De Clercq, and P. Deprez

The purpose of the present study was to develop a feasible and safe technique for dual-chamber pacemaker implantation in healthy horses. Implantation was performed in a standing, tranquilized horse and in ponies. Atrial and ventricular leads were transvenously inserted through the cephalic vein, and a subcutaneous pacemaker pocket was created between the lateral pectoral groove and the *manubrium sterni* in 6 equids. Positioning of each lead was guided by echocardiography and by measuring the electrical characteristics of the lead. The implantation procedure lasted about 4 hours in each animal and was well tolerated. In all animals, dual-chamber pacemaker function was obtained, and these results remained good throughout the follow-up period. At the time of implantation, atrial and ventricular sensing were between 2.1 and 7.2 mV and 7.8 and 16.8 mV, respectively, and atrial and ventricular pacing thresholds at 0.5 millisecond varied from 0.5 to 0.7 V and from 0.3 to 1.0 V, respectively. Six months after the implantation, sensing values varied from 2 to 10 mV for the atrial lead and from 2 to 16 mV for the ventricular lead, while pacing thresholds at 0.5 millisecond varied from less than 0.5 to 2.5 V for the right atrium and from less than 0.5 to 5.0 V for the right ventricle. Atrial lead dislodgment occurred in 2 animals, requiring insertion of a new lead. Ventricular lead dislodgment was not observed.

Key words: Cardiac pacing; Echocardiography; Electrical pulse generator; Heart; Horse.

A cardiac pacemaker is an implantable device that delivers battery-supplied electrical stimuli through electrodes in contact with the myocardium in order to produce an artificially triggered depolarization of the atria, the ventricles, or both.^{1,2} The electrodes can be attached to the epicardial surface, which necessitates a thoracotomy, or they can be attached to the endocardium, by means of transvenous lead insertion.³ The pacing system contains 1 or 2 leads to perform single- or dual-chamber pacing. Compared with a single-chamber pacemaker, dual-chamber systems ensure a more physiological cardiac function.^{2,4} This system not only allows a physiological rate responsiveness but also reestablishes atrioventricular (AV) synchrony, which preserves the contribution of the atrial contraction to the ventricular filling, preventing systemic and pulmonary venous congestion, decreasing mitral valve regurgitation, and thus increasing cardiac output.⁵

Transvenous lead placement makes cardiac pacing relatively simple and safe and is widely used in human medicine. In small animal medicine, permanent pacing has gained considerable importance in the management of bradycardias caused by 3rd-degree AV block, high-grade 2nd-degree AV block (Mobitz type II), sick sinus syndrome, or persistent atrial standstill.^{2,4,6–10} Besides the therapeutic use in cases of rhythm disturbances, pacemakers can also be

used for diagnostic or investigational purposes. With a pacemaker, electrophysiological measurements can be made, and arrhythmias can be artificially induced. Many animal studies with sheep,^{11,12} goats,^{13–16} pigs,^{17,18} and especially dogs^{19–24} have been described, but the authors found no published data on equids.

To our knowledge, dual-chamber pacemaker implantation in horses has only been described twice for the treatment of a 3rd-degree AV block, and the applied techniques were quite diverse. In 1986, Reef et al²⁵ implanted a dual-chamber pacemaker in the ventral neck region of a horse by means of transvenous electrodes through the jugular vein. This horse survived for 3 years.²⁶ In 1993, Pibarot et al²⁷ described the implantation of an AV pacemaker in a donkey by means of epicardial electrodes. They created a subcutaneous pacemaker pocket on the lateral chest wall.

The purpose of the present study was to develop a reproducible technique for dual-chamber pacemaker implantation in healthy equids and to investigate the feasibility and safety of the procedure.

Materials and Methods

Animals

This study was approved by the Ethics Committee of the Faculty of Veterinary Medicine, Ghent University.

Five ponies and a Thoroughbred horse were selected. Body weight and height at the withers ranged between 250 and 440 kg and between 1.25 and 1.55 m, respectively (Table 1). All animals were healthy, as indicated by the clinical and biochemical exam. There were no abnormalities on ECG and echocardiographic examination.

Equipment

Dual-chamber pacemakers^a were used for implantation. Table 1 displays the different lead types that were used. Unipolar as well as bipolar transvenous leads were implanted. For the right ventricle, leads with an active (screw-in) or passive (tines) fixation were used, whereas for right atrial pacing, only active fixation leads were applied. Five out of 10 active fixation leads were of the steroid-eluting type (Table 1). The length of the implanted leads varied between 52 and 110 cm. In the lead body, a stylet could be inserted to supply stiffness and to

From the Department of Large Animal Internal Medicine, Faculty of Veterinary Medicine, University of Ghent, Merelbeke, Belgium (van Loon, Rottiers, Deprez); the Department of Cardiology, University Hospital, University of Ghent, Ghent, Belgium (Fonteyne, Tavernier, Jordaens, D'Hont, Colpaert); and the Department of Medical Imaging of Domestic Animals, Faculty of Veterinary Medicine, University of Ghent, Merelbeke, Belgium (De Clercq).

Reprint requests: G. van Loon, DVM, Department of Large Animal Internal Medicine, Faculty of Veterinary Medicine, University of Ghent, Salisburylaan 133, B-9820 Merelbeke, Belgium; e-mail: Gunther.vanLoon@rug.ac.be.

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Table 1. For each animal, the height at the withers and the characteristics of the leads (Medtronic, Minneapolis, MN) are displayed. The last column indicates how long the pacemaker had been implanted at the time of submission of the paper.

Horse	Height (m)	Atrial Lead					Ventricular Lead					Time Im- planted (months)
		Length (cm)	Polarity	Fixation	Steroid- Eluting	Model No.	Length (cm)	Polarity	Fixation	Steroid- Eluting	Model No.	
1	1.25	58	Bipolar	Screw-in	Yes	4068	58	Bipolar	Screw-in	Yes	4068	12
2	1.32	52	Bipolar	Screw-in	No	4058M	58	Unipolar	Tined	No	6971	44
3	1.25	52	Bipolar	Screw-in	No	4058M	52	Unipolar	Tined	No	6971	46
4	1.37	85	Bipolar	Screw-in	Yes	4068	85	Bipolar	Screw-in	Yes	4068	29
5	1.30	52	Bipolar	Screw-in	No	4058M	52	Unipolar	Screw-in	No	4057M	27
6	1.55	58	Bipolar	Screw-in	No	4058M	110	Bipolar	Screw-in	Yes	4068	19

provide shapeability. During implantation, a base apex ECG was continuously recorded.^b Cardiac ultrasonography^c was performed from the right cardiac window. To measure electrical characteristics, including capture threshold at 0.5 millisecond, lead impedance, and sensing voltage of P waves and R waves, the lead was connected to a pacing system analyzer (PSA)^d by sterile connector cables. The cathode was connected with the distal (tip) electrode. The anode was connected to the proximal (ring) electrode for a bipolar lead or with the subcutaneous tissue for a unipolar configuration. After implantation, lateral radiographs of the chest were obtained to verify the lead position. After 1, 2, and 3 days, after 1 and 2 weeks, after 1, 2, and 6 months, and every 12 months, an ECG and echocardiographic examination were performed. Lead position was verified by echocardiography, and special attention was paid to the presence of any irregularity near the lead tip, on the lead body, or on cardiac valves that might indicate inflammatory reaction of a fibrinous layer. Color-flow Doppler of the tricuspid valve was performed to detect valvular regurgitation. At the same time, pacemaker function was analyzed with a telemetric pacemaker programmer^e connected to a surface ECG. Atrial and ventricular sensing threshold was measured during sinus rhythm. Subsequently, atrial and ventricular pacing and sensing were analyzed during atrial, ventricular, and dual-chamber pacing at a pacing rate of 60 beats per minute. For these measurements, the sensed and paced AV intervals were programmed to 350 milliseconds; the atrial and ventricular blanking were 100 and 28 milliseconds, respectively; the postventricular atrial refractory period was 150 milliseconds; and the ventricular re-

fractory period was 300 milliseconds. During the whole follow-up period, horses were kept in stalls, and every 2 weeks, they were given free access to an arena. The pacemaker was intermittently active to perform cardiac studies, including echocardiographic examinations and blood pressure measurements. Except from horse 1, which developed chronic laminitis, each horse completed treadmill exercise, including performing at walk, trot, canter, and gallop, with heart rates up to 175 beats per minute. Horses 2, 3, 4, 5, and 6 completed 12, 4, 16, 12, and 6 treadmill sessions, respectively. Treadmill exercise and free access to an arena started 2 months after implantation.

Implantation Procedure

The animals were given 15 µg/kg detomidine^f IV and 3 µg/kg butorphanol^g IV and remained in standing position during the implantation. The horses were placed in stocks to allow the surgeons to sit in a safer position in front of the horse. If the horse showed any discomfort during the implantation, 7 µg/kg detomidine was administered IV. The ventral neck and pectoral region was clipped. The region at the level of the lateral pectoral groove was surgically prepared and was blocked with 2% lidocaine.^h An incision of about 8 cm was made along the lateral pectoral groove, and the cephalic vein was exposed by blunt dissection. A sutureⁱ was placed around both ends of the exposed vein, but only the distal part of the vessel was ligated, preventing blood from the leg entering the surgical site. Between the 2 sutures, a partial venotomy was made (Fig 1). The vein lumen was

Table 2. The electrical characteristics of the atrial and ventricular lead at the time of implantation and 6 months after the implantation, including the stimulation threshold at 0.5-millisecond pulse duration, shown in volts (V) and milliamperes (mA), the lead impedance (ohms, Ω), and the sensing amplitude (mV), are shown for each horse.

Horse	Time	Atrial Lead				Ventricular Lead			
		Threshold (0.5 millisecond)		Impedance	Sensing mV	Threshold (0.5 millisecond)		Impedance	Sensing mV
		V	mA			V	mA		
1	Implantation	0.6	1.5	410	2.1	0.5	1.1	440	11.2
	6 months	<0.5	<0.8	587	2.8	<0.5	<0.8	610	8
2	Implantation	0.5	1.0	490	3.5	0.6	1.5	415	8.9
	6 months	1	1.3	741	2.8	5.0	6.7	752	2
3	Implantation	0.7	1.7	420	3.9	0.6	0.7	860	16.8
	6 months	2	3.4	592	2	1.5	1.3	1,116	15.7
4	Implantation	0.7	1.6	440	2.6	1.0	1.6	620	7.8
	6 months	0.5	0.7	741	4.2	0.5	0.6	791	16
5 ^a	Implantation	0.5	0.8	405	7.2	0.3	0.6	520	16.1
	6 months	0.5	1.3	396	5.6	1.6	1.9	855	8.2
6 ^a	Implantation	0.6	0.9	450	6	0.9	1.5	610	13.6
	6 months	2.5	3.9	634	10	<0.5	<0.6	784	8

^aIndicates the values of the re-implanted lead.

Fig 1. The circle, pointed out by the large arrow, indicates the place for the venotomy and the location of the pacemaker pocket. The course of the cephalic vein (black arrowhead) and the jugular vein (transparent arrowhead) are shown.

opened with a vein lifter, and the atrial lead, with a straight stylet inserted, was introduced in cardiac direction. The lead was advanced until it became ultrasonographically visible in the right atrium. Subsequently, the ventricular lead, with a straight stylet inserted, was also advanced in the cephalic vein. The lead became visible in the right atrium, and, under echocardiographic guidance, the lead tip was maneuvered into the right ventricular apex. If the lead was fitted with an active fixation mechanism, the helix was extended into the myocardium by rotation of a connector pin at the external part of the lead.

The lead was connected to the PSA for measurement of electrical variables. Electrical variables for the right ventricular lead were acceptable when the capture threshold did not exceed 1 V at 0.5-millisecond pulse width, the lead impedance was between 400 and 1,000 ohms, and the sensed R wave was at least 4.0 mV.^{3,5,28} For the active fixation leads, 5 minutes were allowed to meet the above-mentioned implantation criteria. If electrical parameters did not match these criteria, the lead was repositioned until an acceptable location was reached. The stylet was removed, and the lead was further inserted into the vein for about 4 cm to provide sufficient slack. The ventricular lead was secured to the underlying tissue by an anchoring sleeve to protect the lead insulation.

For atrial lead positioning, the stylet was 1st removed to be curved manually while the lead was left in the right atrium. Re-insertion of this curved stylet provided a J shape to the lead and supplied body. After connection of the PSA and under echocardiographic control, the lead was slowly manipulated to obtain contact with the endocardium. Once in the right position, the helix of the active fixation mechanism was extended into the endocardium, and electrical parameters were checked. A good atrial position was achieved when the capture threshold at 0.5 millisecond did not exceed 1.5 V, lead resistance was between 400 and 1,000 ohms, and P wave sensing was at least 1.5 mV.^{3,5,28} After withdrawal of the stylet, pacing was performed at 5 V and 0.5 millisecond to check if no diaphragmatic stimulation occurred. If the electrical characteristics did not match the implantation criteria or if diaphragmatic stimulation occurred, the lead was repositioned. If the lead was properly positioned, it was advanced slightly into the vein, and the proximal part was secured to the underlying tissue by the anchoring sleeve.¹ Gentle traction was applied to the ventricular and the atrial lead to verify if lead migration occurred, and electrical variables were again examined. Subsequently, the proximal part of the cephalic vein was ligated.¹ Between the lateral pectoral groove and the *manubrium sterni*, a subcutaneous pacemaker pocket was created by blunt dissection (Fig 1). The pacemaker was connected to both leads and inserted in the pocket, taking care that no acute angulations of the electrodes occurred. Redundant leads were looped along the sides of the device or underneath it. The pocket was irrigated with antibiotics,^k and the subcutaneous tissue and skin were closed in a routine manner. Flunixin meglumine^l (0.3 mg/kg IV q8h) and sodium ceftiofur^m (2 mg/kg IV q24h) were given for 5 days. Oral trimethoprim sulphadiazin treatmentⁿ (1 mg/kg trimethoprim and 5 mg/kg sulphadiazin PO q12h) was continued for 10 days. During this recovery period, recumbency of the horse was prevented by attaching the horse to the stable wall.

Results

The whole implantation procedure took about 4 hours and was well tolerated. The cephalic vein could be easily exposed, and its diameter was sufficiently large to bare both the atrial and the ventricular lead. Positioning of the atrial or ventricular lead often initiated ectopy. However, these arrhythmias were always short in duration and self-terminating. The pacemaker leads could clearly be visualized on echocardiography and on chest radiographs. On the ultrasonographic image, the lead body appeared as a smooth, linear, hyperechoic structure, whereas the lead tip was characterized by more irregular hyperechoic reflections (Fig 2). However, even if the lead tip was clearly identifiable on echocardiography, the presence of endocardial contact could not always be ascertained and had to be confirmed by determination of electrical variables. During ventricular lead positioning, echocardiographic control was useful to follow the lead entering into the right ventricular apex and to avoid its migration toward the pulmonary artery. With a straight stylet inserted, the lead easily entered the right ven-

Fig 2. Right parasternal long-axis echocardiogram optimized to view the right ventricle (RV). The lead body (arrowheads) is visible as a smooth hyperechoic structure underneath the tricuspid valve. The lead tip (arrows) is localized in the right ventricular apex and shows more irregular reflections (RVFW = right ventricular free wall; RA = right atrium; RV = right ventricle; IVS = interventricular septum; displayed depth = 12 cm).

tricular apex (Fig 2), and an acceptable position was readily accomplished. Atrial lead positioning was more difficult. The atrial lead had to curve in the right atrium toward the atrial wall in order to allow the helix to be extended into the myocardium. On echocardiography, the most dorsal parts of the right atrium could not completely be imaged, and the visualization of both the atrial and the ventricular lead in the right atrium complicated ultrasonographic guidance. Therefore, echocardiography served mainly to prevent atrial lead fixation too close to the tricuspid valve, and lead positioning was performed relying mainly on the measurements of electrical variables. Although satisfactory electrical characteristics were often readily achieved after extension of the helix, withdrawal of the stylet sometimes resulted in an immediate loss of capture or sensing, indicating that active fixation of the lead had not been accomplished. Several attempts were sometimes necessary to achieve active fixation of the atrial lead. Furthermore, diaphragmatic stimulation occasionally occurred at 5 V and 0.5 millisecond, which also necessitated lead repositioning. However, lead implantation criteria could always be met (Table 2).

On postoperative radiographs, the ventricular lead tip was visible in the right ventricular apex in all animals. The atrial lead tip was identifiable in the right atrium and was located in the cranial region of the right atrium in pony 3 and in the horse, in the mid-portion in pony 2, and in the caudal part in ponies 1, 4, and 5.

In all animals, the dual-chamber pacemaker was successfully implanted, and atrial and ventricular sensing and pacing were all achieved, allowing an effective atrial, ventricular, and dual-chamber pacing function (Fig 3). In the

Thoroughbred horse (animal 6), at a heart rate of 30–35 beats per minute, the normal intrinsic AV interval was about 400 milliseconds, which is within normal limits. Due to a maximal programmable AV interval of the pacemaker of 350 milliseconds, however, in the dual-chamber pacing mode, ventricular pacing occurred at slow heart rates, although AV conduction in that horse was normal.

All animals had a normal wound healing with formation of a small seroma during the 1st days postimplantation. Ligation of the cephalic vein caused no circulatory problems, and the position of the pacemaker did not impede equids in lying down or performing treadmill exercise. In none of the horses was treadmill exercise associated with lead dislodgment. Atrial lead dislodgment occurred 2 days after implantation in pony 5, causing a loss of atrial capture and atrial sensing. On echocardiography, the lead tip was identified in the right ventricle near the tricuspid valve, and radiographs were taken to confirm the lead displacement. The pacemaker pocket was reopened, and, after retraction of the helix, the lead was removed and replaced by a new one. In the horse, the atrial lead dislodgment occurred 1 month after implantation. At that time, treadmill exercise had not been performed. Atrial capture could no longer be obtained, even at 1.5 milliseconds and 7.5 V. Atrial sensing dropped from 8 mV on the previous day to 1.4 mV on the day of dislodgment. On cardiac ultrasound, the dislodged lead tip was identifiable in the right atrium near the tricuspid valve. The lead, which was of the steroid-eluting type, was withdrawn, and a new nonsteroid-eluting lead was inserted in the contra-lateral cephalic vein. After positioning and fixation of this new lead, the proximal part was tunneled subcutaneously and connected to the pacemaker, re-

Fig 3. Print from the pacemaker programmer showing a surface ECG (1), calibrated at 0.1 mV/mm, a simultaneous intra-atrial and intraventricular ECG (2), calibrated at 1 mV/mm, and a marker channel (3) indicating atrial pacing (AP) and ventricular pacing (VP). The pacemaker is programmed at dual-chamber pacing and sensing (dual-chamber mode) at 60 beats per minute with an atrioventricular interval of 350 milliseconds. Each atrial and ventricular stimulus is followed by a P wave and QRS complex on the surface ECG, respectively, indicating successful dual-chamber pacing.

storing normal pacemaker function. Ventricular lead dislodgment was never observed.

Long-term follow-up revealed no abnormalities on echocardiographic examinations. The appearance and position of each lead remained identical. The lead body surface remained smooth, and no irregularities were detected near the lead tip. The tricuspid valve showed no valvular lesions or valvular regurgitation. In all animals, atrial and ventricular sensing remained possible, and both chambers could be successfully stimulated with acceptable threshold values, indicating a good pacemaker function (Table 2). Arrhythmias, other than occasional 2nd-degree AV block, were not seen. In the horse, diaphragmatic stimulation occasionally occurred during atrial pacing. Pony 1 had to be euthanized 1 year after implantation because of a chronic laminitis. Postmortem examination revealed that both the atrial and the ventricular lead tip were closely attached to the myocardium and were surrounded by a small amount of fibrous tissue. There were no lesions on the tricuspid valve. A small fibrous layer was present on the proximal intravascular part of the lead. A firm fibrous capsule surrounded the pacemaker box.

Discussion

The described standardized technique for dual-chamber pacemaker implantation in horses proved to be efficient and safe. The whole procedure could be performed in the standing horse and was well tolerated. Successful sensing and pacing of the atrium as well as the ventricle were obtained on long-term follow-up.

Transvenous implantation of a permanent pacemaker is the preferred method in both human and small animal medicine.^{2,10} In contrast to epicardial lead placement, it is much less invasive, cheaper, and more rapidly accomplished. A transvenous approach avoids major surgery and can be performed without general anesthesia,^{4,6} which is an advantage in animals with compromised cardiovascular systems in which general anesthesia is associated with considerable risk of adverse events.^{29,30} The occurrence of a lethal ventricular fibrillation during general anesthesia has been described in a horse during an attempt to implant a pacemaker.³¹ In equids requiring pacemaker implantation because of a symptomatic bradycardia, administration of detomidine could worsen cardiac function because of its negative chro-

notropic effect. In these animals, a temporary pacing catheter should therefore be inserted into the right ventricular apex before sedation, to allow pacing during the implantation procedure.

The only way to perform transvenous pacemaker implantation in equids is to use a superficial vein that is located close to the heart and that is large enough to accommodate 2 leads. The vein will be ligated. Furthermore, the pacemaker pocket has to be located close to the vein and close to the heart to avoid the use of extremely long leads and be implanted at a safe location in order to allow recumbency and exercise. For these reasons, the cephalic vein at the level of the lateral pectoral groove was chosen for lead insertion. The left as well as the right cephalic vein were used without a noticeable difference in the procedure. Although the jugular vein, which was used by Reef et al,²⁵ is also close to the heart, the authors preferred the smaller cephalic vein to be ligated permanently. Consequently, both jugular veins were preserved, and venous access was facilitated during subsequent experimental studies. Furthermore, in large breed dogs with lead insertion in the jugular vein, excessive neck motion is suspected to be a contributing factor in the higher incidence of lead dislodgment in these animals.^{7,10} The placement of the lead and pacemaker pocket near the *manubrium sterni* was thought to be more stable than localization in the neck. The lateral thoracic vein is large enough to bare 2 leads, but its subcutaneous part is too remote from the heart. Moreover, creating a pacemaker pocket near this vein could impede the use of a girth or recumbency of the horse.

Although complications of the pacemaker pocket were not encountered, the subcutaneous position of the pocket implies a possible risk for injury by biting, rubbing, or trauma.

The requisite length of the ventricular lead was about 50 cm for the ponies and about 55 cm for the Thoroughbred horse. Because of the availability of a 110-cm lead, this lead was implanted in the right ventricle of the Thoroughbred horse, and a redundant lead was looped along the sides of the pacemaker. The atrial lead had to be at least 45 cm for the ponies and 50 cm for the Thoroughbred horse. Passive as well as active fixation leads were used. Passive fixation leads possess tines or fins to enhance entanglement within the trabeculae of the myocardium and were applicable for implantation in the right ventricle. Active fixation leads penetrate the myocardium by grasping screws, and they were used for implantation in the right ventricle and the right atrium. Passive fixation leads were never used in the right atrium because we feared that lead dislodgment would occur more easily.

To avoid lead tip distortion, a straight stylet was fully inserted into the lead during introduction and while advancing the lead, to supply a degree of stiffness and shapeability to the lead. Echocardiography facilitated atrial and ventricular lead positioning, but measurements of electrical parameters were crucial for final adjustments during lead placement, because they allow selection of the optimal electrode stimulation and sensing site.² For the electrical measurements, the distal (tip) electrode of the lead was always connected with the cathode, because cathodal stimulation is less likely to induce arrhythmias.³² The same acceptance values for the electrical parameters of the atrial and ventricular lead were

taken as in human medicine. Because the short- and long-term success of the pacing system is related to the initial lead position, effort was expended to obtain the best possible initial location in terms of both stability and electrical performance.³ For the active fixation leads, 5 minutes were sometimes allowed for the lead to meet the implantation criteria, because it is common for capture thresholds to decrease markedly shortly after active fixation.^{3,33}

The ventricular lead was positioned 1st, because it may supply back-up pacing in animals with severe bradycardia and because it is usually considered the most important of the leads.³ Ventricular lead placement was performed with a straight stylet inserted in the lead body to guide the lead tip straight toward the right ventricular apex. Except from an occasional insertion of the tip between the right ventricular wall and a papillary muscle or a migration toward the pulmonary artery, the lead tip readily moved into the right ventricular apex to remain in a stable position. The active as well as the passive fixation lead remained well in place, and lead dislodgment was never encountered.

Atrial lead positioning was much more difficult because (1) the atrial lead had to bend with its tip toward the atrial endocardium by means of a curved stylet, and (2) a firm endocardial contact had to be obtained with the active fixation mechanism. Even when acceptable electrical parameters were obtained after extension of the helix, atrial capture was sometimes lost when the stylet was withdrawn. Possibly, the helix only partially entered the myocardium, or the lead tip was located parallel to the endocardium instead of perpendicular, preventing the helix from entering the myocardium sufficiently. Furthermore, the presence of large atrial trabeculae might have interfered with the lead fixation. In contrast with the ventricular lead, the exact anatomical position of the atrial lead in the right atrium could not always be determined. However, any atrial position that allows adequate pacing and sensing thresholds can be used as long as no extracardiac stimulation occurs with pacing.⁵

Occasionally, diaphragmatic stimulation was observed during atrial pacing at 5 V and 0.5 millisecond. Stimulation of the lateral, dorsal part of the right atrium, at the level of the right diaphragmatic nerve, was likely to be the cause.³⁴ When diaphragmatic stimulation occurred, the lead was always repositioned.

Fluoroscopy is used during pacemaker implantation in humans and small animals and could have been helpful during the implantation in horses. Especially during atrial lead placement, fluoroscopy might have been useful to identify the typical to-and-fro motion of the lead tip with atrial activity. However, ultrasonography proved to be sufficient and is also used in human medicine when fluoroscopy is contraindicated, as in pregnant women.^{3,35–37} Postoperative echocardiographic examinations are useful, not only to check lead position but also to look for strands or vegetative lesions, because the latter can often be found in cases of lead infection.^{38–40}

Once satisfactory lead position was obtained, the lead was advanced slightly into the vein to impart a small bend and thereby allow for movements of the neck and foreleg that might otherwise exert traction on the lead.^{1,2,19} However, care was taken not to enter the lead too far in the vein. Too large a loop may predispose to ectopy, lead displace-

ment, or myocardial perforation at the tip.^{2,3} When securing the outer part of the lead to the underlying tissue and when closing the pocket, special care was taken not to damage the lead insulation.

After lead implantation, the tissue around the lead tip becomes inflamed due to the presence of foreign material in the body and due to the pressure of the lead-electrode system in contact with the myocardium.^{2,41} With time, the inflammatory reaction subsides, leaving a thin capsule of fibrous tissue between the electrode and the active myocardium supplying an extra fixation of the lead tip to the myocardium.⁴¹ However, the inflammatory reaction can be excessive, causing the capture threshold to rise, sometimes above the output of the pacemaker, causing exit block. In this context, the use of a steroid-eluting electrode may offer benefits in terms of subacute and chronic thresholds.³ Such an electrode elutes small amounts of dexamethasone from its tip, which attenuates the local inflammatory reaction and reduces the thickness of the fibrous capsule that surrounds the electrode, lowering the chronic threshold for stimulation.^{3,33} This study suggests that lower chronic threshold values are observed with the steroid-eluting leads (Table 2).

The main complication of the transvenous implantation technique is a lead displacement during the early postoperative period^{4,6,7,10} as was seen in pony 5. Therefore, exercise should be restricted during the 1st weeks after implantation because, at that time, a fibrous connection of the lead tip is not yet achieved.^{4,6} In the horse, lead dislodgment occurred 1 month after implantation. Such a late dislodgment was probably related to the animal's movement and was caused by inadequate fixation of the anchoring sleeve to the lead or to the surrounding tissue, insufficient slack, or a poor fixation of the lead tip to the endocardium.⁴¹ The latter could have been influenced by the use of the steroid-eluting electrode in this horse, because the elution of dexamethasone could have reduced the thickness of the fibrous capsule surrounding the lead tip.

The maximal programmable AV interval for the pacemaker was limited to 350 milliseconds. In a dual-chamber pacing mode, this means that, if the pacemaker has not sensed a ventricular depolarization within 350 milliseconds after an atrial event, a ventricular stimulus will be delivered. At slow heart rates, horses with a normal AV conduction can present an interval between P wave and R wave of the QRS complex of more than 350 milliseconds. This implies that, even in the presence of a normal AV conduction, dual-chamber pacing can give rise to unnecessary ventricular pacing because of the restricted programmable AV interval. When the heart rate increases slightly, however, the PR interval reduces to below 350 milliseconds, and inappropriate ventricular pacing ceases.

We can conclude that the implantation of a dual-chamber pacemaker with endocardial leads via the cephalic vein is feasible and safe in healthy equids and can be performed on the standing animal. The advantage of the described method is that the cephalic venous access and the subcutaneous implantation of the pacemaker near the *manubrium sterni* require only minor surgery and can be performed under local anesthesia, thus avoiding general anesthesia. The cephalic venous approach preserves both jugular veins, facilitating experimental studies after pacemaker implanta-

tion. The subcutaneous position of the pacemaker permits postoperative reprogramming. No major complications were encountered in this study, and the implanted pacemakers did not impede the ability of the horse and ponies to lie down or to perform treadmill exercise. Limitations of the technique included the lack of fluoroscopy and the limited echocardiographic visualization of the high right atrium, which hampered atrial lead positioning. Implantations were performed in healthy animals, and further studies are required to investigate the applicability of the technique in clinical patients who would need temporary pacing before sedation. The use of transvenous endocardial leads can imply an increased risk for lead dislodgment. Therefore, regular examination of pacemaker function should be performed in a horse with symptomatic bradycardia, especially if the animal is intended to perform exercise. Although no problems were encountered with pacemaker pockets in this study, the subcutaneously positioned pocket could be injured by external trauma. Besides the implantation of a pacemaker in animals with extreme bradycardia, this relatively simple technique also provides an excellent tool to collect information about cardiac arrhythmias and electrophysiological characteristics of the equine heart. In combination with various drugs, the influence of these drugs on electrophysiological properties of the heart could be studied. Furthermore, arrhythmias can be artificially induced, which makes research about their effect on heart function and about the application of a feasible therapy possible.

Footnotes

- ^a Thera D(R), Medtronic, Minneapolis, MN
 - ^b Cardiolife TEC-7511K, Nihon Kohden, Tokyo, Japan
 - ^c Vingmed CFM 800 SV, GE Vingmed, Horten, Norway
 - ^d PSA Model 5309, Medtronic, Minneapolis, MN
 - ^e Programmer 9790®, Medtronic, Minneapolis, MN
 - ^f Domosedan®, Pfizer Animal Health, Nosssegem, Belgium
 - ^g Temgesic®, Schering-Plough, Brussels, Belgium
 - ^h Xylocaine 2%®, Astra Pharmaceuticals, Brussels, Belgium
 - ⁱ Vicryl 4/1, Johnson & Johnson, Dilbeek, Belgium
 - ^j Mersutures 4/1, Johnson & Johnson, Dilbeek, Belgium
 - ^k Kanacillin Trifortis Vet®, Continental Pharma, Brussels, Belgium
 - ^l Finadyne®, Schering-Plough, Brussels, Belgium
 - ^m Excenel®, Upjohn, Puurs, Belgium
 - ⁿ Tribriksen Oral Paste, Mallinckrodt Veterinary, Brussels, Belgium
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