Use of biphasic electrical cardioversion for treatment of idiopathic atrial fibrillation in two horses

Melinda A. Frye, DVM, MS, DACVIM; Corine G. Selders, DVM; Khursheed R. Mama, DVM, DACVA; Ann E. Wagner, DVM, MS, DACVP, DACVA; Janice M. Bright, DVM, MS, DACVIM

- For horses with idiopathic atrial fibrillation, the standard method of conversion is administration of quinidine, but adverse effects are common.
- Biphasic electrical cardioversion may be efficacious in horses with atrial fibrillation of short duration.

A 15.5-year-old Quarter Horse gelding weighing 550 kg (1,210 lb) was referred to the **Colorado State University Veterinary Teaching Hospital (CSU-VTH)** for evaluation and treatment of an irregular heart rhythm of approximately 2 years' duration. The rhythm was initially detected during a routine examination; the horse did not have exercise intolerance at that time. However, the owner believed that the horse had developed reduced stamina during the 12 months preceding admission. A cough possibly caused by allergy to dust had been successfully managed with daily administration of prednisone within the year preceding admission.

On admission the horse was bright and alert, with pink mucous membranes and capillary refill time of approximately 2 seconds. The horse was afebrile and had a respiratory rate within reference range. An irregular heart rhythm was auscultated, and mean heart rate was 40 beats/min. No murmur, dyspnea, nasal discharge, cough, or abnormalities of the jugular vein were detected. Results of the remainder of the physical examination were unremarkable.

Results of an ECG confirmed that the irregular rhythm was atrial fibrillation, with normal QRS complexes and a slow ventricular response. Doppler echocardiographic examination revealed normal cardiac structure and function. There was a small amount of mitral regurgitation that was believed to be secondary to the arrhythmia. The owner was given 2 treatment options, including administration of quinidine sulfate in an attempt to pharmacologically convert the heart to normal sinus rhythm, and biphasic electrical cardioversion. Potential adverse effects of quinidine administration were explained, and the owner was informed that the prognosis for successful and permanent conversion was guarded, given the duration of the arrhythmia. It was suggested, on the basis of the prolonged duration of the atrial fibrillation and data from human patients, that electrical cardioversion might be more effective than pharmacologic conversion.[1-4] The owner was informed that electrical cardioversion was not known to have been attempted in horses, and the risks of general anesthesia were explained. Possible risks associated with electrical cardioversion were described, including ventricular tachyarrhythmias, cardiac arrest (asystole or ventricular fibrillation), and myocardial damage. The owner chose to take the horse home and consider the information.

Three months after the initial visit, the owner returned the horse to the CSU-VTH for electrical cardioversion. Results of auscultation and electrocardiography confirmed that the horse remained in atrial fibrillation. Results of a CBC revealed a mild leukopenia (5.9×10^3 cells/µl; reference range, 6.5 to 12.5×10^3 cells/µl) with neutrophil and lymphocyte values within reference ranges. A serum biochemical profile revealed low serum magnesium concentration (1.2 mg/dl; reference range, 2 to 2.8 mg/dl) and high serum globulin concentration (4.0 g/dl; reference range, 2.4 to 3.5 g/dl). Fractional excretions of sodium and potassium were within reference ranges. Serum concentration of **cardiac troponin I (cTn I)**, measured by use of a commercially available ELISA kit,[a] was 0.097 ng/ml, which is within the reference range reported for horses.[5]

In preparation for electrical cardioversion, the horse was sedated by IV administration of incremental doses of xylazine hydrochloride (0.25 mg/kg [0.11 mg/lb] and 0.3 mg/kg [0.14 mg/lb]) prior to anesthetic induction with guaifenesin (50 mg/kg [22.73 mg/lb], IV), diazepam (0.02 mg/kg [0.01 mg/lb], IV), and ketamine hydrochloride (2 mg/kg [0.91 mg/lb], IV). An orotracheal tube (26 mm in diameter) was placed, and anesthesia was maintained with sevoflurane (2.5 to 5.5%) in oxygen (6 to 10 L/min) delivered via a standard large-animal anesthetic breathing circuit. Ventilation was controlled at a mean rate of 4 breaths/min by use of a ventilator.[b] A balanced electrolyte solution[c] and dobutamine (1 to 2 µg/kg per minute [0.45 to 0.91 µg/lb per minute]) were administered IV during the procedure in an effort to maintain a mean arterial pressure of 60 mm Hg.

After induction of anesthesia and positioning of the horse in dorsal recumbency with suspension of all 4 limbs, the lateral aspect of the thorax was clipped on each side from the axilla caudally and ventrally for approximately 25 cm. Self-adhesive cardioversion-defibrillation pads[d] with pre-applied gel were applied to each side of the thorax over the clipped areas. Electrocardiographic electrodes were also attached to the skin in a base-apex configuration and connected to the ECG leads of a rectilinear biphasic cardioverter-defibrillator.[e] The synchronous mode (cardioversion

From the Cardiovascular Pulmonary Research Laboratory, Department of Medicine, University of Colorado Health Sciences Center, University of Colorado, Denver, CO 80220 (Frye); and the Department of Clinical Sciences, College of Veterinary Medicine, Colorado State University, Fort Collins, CO 80523 (Selders, Mama, Wagner, Bright).
Address correspondence to Dr. Frye.

Unauthenticated | Downloaded 12/05/23 11:04 PM UTC

mode) of the cardioverter-defibrillator was selected to prevent current delivery during the ventricular repolarization phase of the cardiac cycle. Atracurium (0.09 mg/kg [0.04 mg/lb], IV) was administered prior to starting the procedure. The first shock was 120 J with a measured impedance of 38 Ω and a delivered current of 16 A. This shock had no apparent effect on the cardiac rhythm. Additional ketamine (0.36 mg/kg [0.16 mg/lb]) was administered IV after the initial shock because of an increase in blood pressure and heart rate. Subsequent delivery of 1 shock of 150 J and 4 shocks of 200 J failed to elicit a sustained conversion to sinus rhythm. Transthoracic impedance during these attempts ranged from 36 to 41 Ω, and the current delivered ranged from 18 to 23 A. Shocks were delivered 3 to 4 minutes apart. After delivery of the first 3 shocks, an attempt was made to administer magnesium chloride (Mg^{2+}) IV (0.2 mEq/kg [0.09 mEq/lb]). However, it rapidly became apparent that administration of the magnesium chloride was inducing an appreciable decrease in arterial blood pressure, and the infusion was abruptly terminated.

After the delivery of the third and fourth shocks, transient alterations in the electrocardiographic tracing that lasted 5 to 7 cardiac cycles were detected (**Fig 1**). Because these tracings revealed a transient regularity in R-R interval, there may have been brief conversion to sinus rhythm. However, because P waves were not distinct in the single-lead rhythm strips, the rhythm could not be determined with certainty.

Time from induction to standing recovery was 60 minutes. The horse was given flunixin meglumine (1.1 mg/kg [0.50 mg/lb]) IV during recovery to aid in relieving muscle discomfort caused by the cardioversion, and phenylbutazone (1.82 mg/kg (0.83 mg/lb]) was administered orally twice daily for 2 additional days. Heart rate and rhythm were monitored hourly for 20 hours after the procedure, and an irregular rhythm with a rate of 32 to 40 beats/min was consistently recorded. Serum **creatine kinase (CK)** activity measured 20 hours after the cardioversion attempt was 9,006 U/L (reference range, 130 to 470 U/L). However, serum concentration of cTn I at this time was 0.047 ng/ml. Complications included mild erythema and edema of the thoracic skin underlying the cardioversion pads. The skin remained intact, and the reaction resolved without intervention. The horse also had shortened forward phase of the stride bilaterally and reluctance to ambulate, without sensitivity to use of hoof testers, for 48 to 72 hours after the procedure.

A 393-kg (865-lb) 6-year-old Arabian gelding was referred to the CSU-VTH for evaluation of atrial fibrillation. Three weeks prior to referral, the horse had been disqualified from an endurance race at the 15-mile veterinary check because of irregular cardiac rhythm. Atrial fibrillation was confirmed electrocardiographically. Results of physical examination and cardiac auscultation prior to the endurance race had been unremarkable. The horse had no history of reduced exercise tolerance and no clinical signs of congestive heart failure.

At the time of admission to the VTH, the horse had an irregular cardiac rhythm with heart sounds of varying intensity and variable arterial pulse quality. No other physical abnormalities were detected. Electrocardiography revealed sustained atrial fibrillation with a mean heart rate of 42 beats/min. Doppler echocardiography revealed no abnormalities except slightly reduced internal dimensions of the left ventricle during diastole (9.9 cm; reference range, 10.5 to 13.3 cm), perhaps because of mild dehydration.

Because the horse had no evidence of cardiac disease, the owner was given 3 treatment options: no treatment, pharmacologic cardioversion to sinus rhythm with quinidine, or biphasic electrical cardioversion. The owner chose biphasic cardioversion.

A CBC and a serum biochemistry panel were performed prior to the cardioversion procedure. Results of these blood tests were within reference ranges except for high serum glucose concentration (149 mg/dl; reference range, 65 to 100 mg/dl) and slightly low serum CK (124 U/L; reference range, 130 to 470 U/L) and aspartate aminotransferase (AST; 154 U/L; reference range, 185 to 375 U/L) activities. Serum electrolyte concentrations were within reference ranges. On the morning of the procedure, serum electrolyte concentrations were reevaluated, and although still within reference range, the potassium concentration had decreased from 3.5 to 3.1 mEq/L, and the magnesium concentration had decreased from 2.0 to 1.6 mg/dl.

Prior to anesthesia, the horse was sedated by administration of xylazine (0.25 mg/kg, IV). Anesthesia was induced by IV administration of keta-

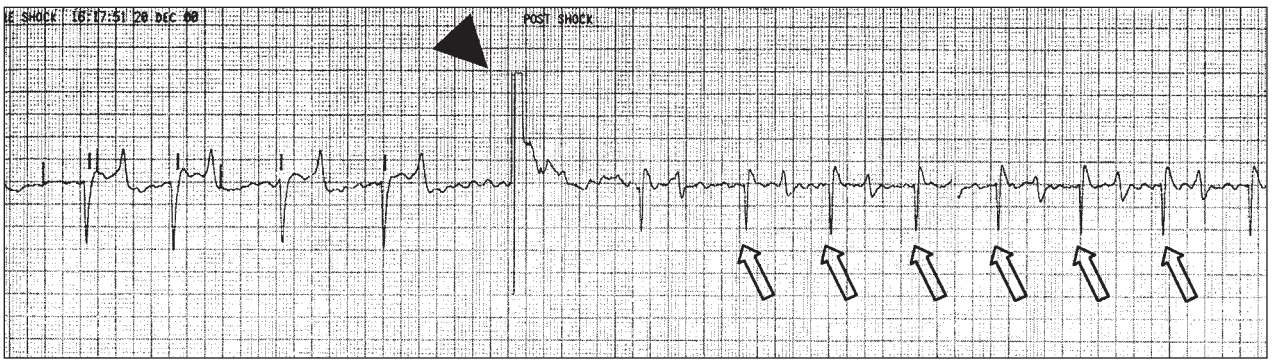

Figure 1—Electrocardiographic rhythm strip obtained during delivery of a third cardioversion shock in a horse. Notice atrial fibrillation with irregular R-R intervals. After delivery of the shock (arrowhead), there is an initial QRS complex that is followed by 6 cardiac cycles (open arrows) with a constant R-R interval of 0.96 seconds, which may represent transient conversion to sinus rhythm with isoelectric P waves. Base-apex lead; paper speed, 25 mm/s.

Unauthenticated | Downloaded 12/05/23 11:04 PM UTC

mine (2.2 mg/kg [1.0 mg/lb]) and guaifenesin (64 mg/kg [29 mg/lb]), and anesthesia was maintained with sevoflurane. During anesthesia, a balanced electrolyte solution[c] with an additional 20 mEq of potassium chloride/L was infused at a rate of 5 ml/kg (2.27 ml/lb) per hour. After induction of anesthesia, the horse was positioned in dorsal recumbency with hind limbs and forelimbs supported by ties to a hoist. An area (approx 25 × 25 cm) was clipped and shaved on each side of the thorax from the level of the shoulder joint ventrally and from the axillary region caudally. Cardioversion-defibrillation pads[d] were attached to the skin in the shaved areas. Self-adhesive electrocardiographic electrodes were also attached to the skin in a base-apex lead configuration and connected to the ECG leads of the defibrillator-cardioverter.[e]

The synchronized (cardioversion) mode of the defibrillator-cardioverter was used to administer an initial series of 4 shocks with progressively increasing energy (120, 150, 200, and 200 J), with no apparent effect on cardiac rhythm. Transthoracic impedance ranged from 30 to 31 Ω during these shocks. The horse was hypotensive (mean arterial pressure, 52 to 55 mm Hg) during anesthesia and received dobutamine (2 µg/kg [0.91 µg/lb] per minute, IV). In an attempt to facilitate conversion to sinus rhythm, slow IV administration of magnesium chloride (0.2 mEq/kg) was begun but was quickly terminated because of worsening hypotension. The defibrillation pads were exchanged for paddles, and 3 additional shocks were administered (200 J; impedance, 27 and 28 Ω). The procedure was terminated, and the horse recovered from anesthesia without complications.

After 2 days, a second attempt at electrical conversion to sinus rhythm was made at the owner's request. Several variations to the previous cardioversion protocol were planned, including IV administration of magnesium and potassium prior to induction of anesthesia and the use of 2-dimensional echocardiography after positioning the horse in dorsal recumbency to determine optimal placement of the cardioversion paddles over the atria. If cardioversion was unsuccessful despite these measures, administration of a low dose of quinidine gluconate IV during the procedure was planned, to aid in cardioversion or maintenance of sinus rhythm after cardioversion.

Food was withheld from the horse for 12 hours, and serum electrolytes were measured prior to anesthesia on the morning of the procedure. Serum potassium concentration was 3.3 mEq/L, and serum magnesium concentration was 1.6 mg/dl. During 3 hours, the horse received a balanced electrolyte solution[c] (25 ml/kg [11.4 mg/lb], IV) that contained potassium chloride (45 mEq/L) and magnesium chloride (1.5 g/L). Serum electrolyte concentrations were measured hourly. At the time of the procedure, serum potassium concentration was 3.9 mEq/L, and the serum magnesium concentration was 2.3 mg/dl.

The horse was anesthetized in a manner similar to the first cardioversion attempt. Because the horse was hypotensive while under anesthesia (mean arterial pressure, 52 mm Hg), dobutamine was infused IV (2 to 4 µg/kg [0.91 to 1.82 µg/lb] per minute). After verifying the position of the atria echocardiographically, the shoulder joints were extended and the cardioversion paddles were placed more cranially on the thorax than during the first cardioversion attempt, in the axillary region over the atria. Three shocks were delivered, using 200 J. Each of these shocks briefly converted the cardiac rhythm from atrial fibrillation to atrial flutter **(Fig 2)**. Transthoracic impedance ranged from 19 to 23 Ω during these cardioversion attempts, and the current delivered was 23 A. A low dose of quinidine gluconate (0.25 mg/kg) was slowly administered IV during a 2- to 3-minute period. Mean blood pressure and heart rate increased slightly after quinidine administration, and a second bolus of quinidine (0.25 mg/kg) was administered IV slowly. A fourth shock was delivered at 200 J, which resulted in conversion to sinus rhythm **(Fig 3)**. The transthoracic impedance recorded during the final shock was 19 Ω, and the current delivered was 24 A. An additional bolus of quinidine (0.25 mg/kg) was administered IV immediately after conversion to sinus rhythm to prevent relapse to atrial fibrillation. A venous blood sample was obtained, and the serum was submitted to determine quinidine concentration.

The horse recovered routinely from anesthesia, with a total anesthesia time of 50 minutes. Phenylbutazone (5 mg/kg [2.27 mg/lb]) was administered IV to prevent skeletal muscle soreness that might result from the electrical shocks. During the next 24 hours, the horse received quinidine gluconate (1.0 mg/kg [0.45 mg/lb], IV) every 6 hours to promote maintenance of sinus rhythm. A venous blood sample was obtained 36 hours after cardioversion for measurement of serum cTn I concentration. The horse remained in sinus rhythm and was discharged 48 hours after cardioversion.

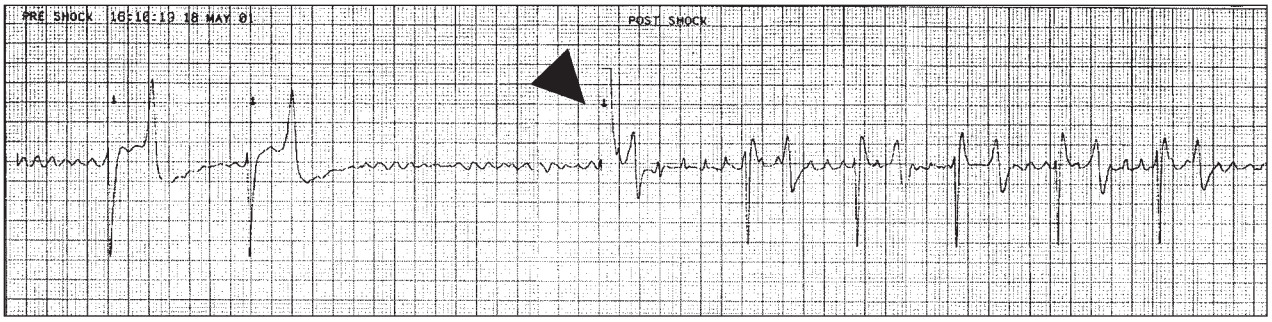

Figure 2—Electrocardiographic rhythm strip obtained during the first shock (arrowhead) used in a second cardioversion procedure in a horse. Notice that the shock briefly converts the cardiac rhythm from atrial fibrillation to atrial flutter. Base-apex lead; paper speed, 25 mm/s.

Unauthenticated | Downloaded 12/05/23 11:04 PM UTC

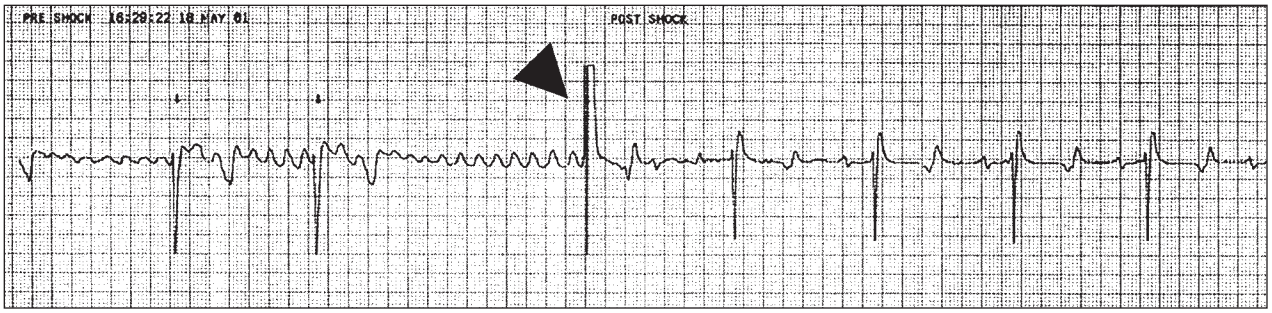

Figure 3—Electrocardiographic rhythm strip obtained during successful cardioversion in a horse. Delivery of the shock is indicated by the arrowhead. Base-apex lead; paper speed, 25 mm/s.

Serum quinidine concentration determined immediately after cardioversion was 0.3 µg/ml; 2 to 4 µg/ml is the therapeutic range in humans and horses.[6] Serum cTnI concentration 36 hours after cardioversion was within reference range (< 0.3 ng/ml; reference limit, < 0.4 ng/ml) despite delivery of 11 biphasic electrical shocks.

The horse was rested for 6 weeks after cardioversion and returned to endurance activities. At 6-month follow-up, the horse remained in sinus rhythm.

Atrial fibrillation is a common pathologic arrhythmia and a frequent cause of poor performance in horses.[7,8] Horses with idiopathic atrial fibrillation (lone atrial fibrillation) are candidates for conversion to sinus rhythm. The standard method of conversion is to administer quinidine PO or IV.[6,9] However, serious adverse effects often develop while attempting pharmacologic cardioversion.[2,6,9] For cardioversion, the required plasma quinidine concentration ranges from 2 to 4 µg/ml and toxicosis has been reported at 5 µg/ml.[6] Adverse effects of quinidine in horses include nasal mucosal edema with possible upper airway obstruction, diarrhea, seizures, urticaria, colic, ataxia, and laminitis.[2,6,8,10] In addition, adverse cardiovascular effects are common and include hypotension, decreased cardiac contractility, congestive heart failure, prolongation of the QRS complex, rapid supraventricular tachycardia, ventricular tachycardia, and sudden death.[1,2,8,9] Administration of quinidine for cardioversion of atrial fibrillation requires continuous or frequent electrocardiographic and hemodynamic monitoring[9] and can be time-consuming and expensive. Successful conversion with quinidine is reportedly achieved in 75 to 90% of horses that do not have substantial underlying heart disease.[1,10] However, chronic atrial fibrillation is associated with a lower success rate, more frequent recurrence, and greater incidence of adverse effects.[1]

In humans with chronic atrial fibrillation, electrical cardioversion is preferred over pharmacologic cardioversion.[3,4] During monophasic electrical cardioversion, in use since the 1960s, a single phase (single direction) of current is generated, and current delivery to the heart is highly dependent on transthoracic impedence.[11] However, external direct-current shock, using conventional monophasic current, is less effective in horses than in humans because of the large thoracic volume that diffuses the current, thereby reducing current density in target tissues (atrial or ventricular myocardium).

Recently, a new generation of cardiac cardioverters-defibrillators has become available, and these newer devices deliver a biphasic energy waveform rather than monophasic current. Biphasic cardioverters-defibrillators deliver current in a positive direction for a predetermined length of time, and the direction of current flow is then reversed for the last few milliseconds of discharge. The biphasic technology adjusts for transthoracic impedance, providing delivery of a constant current through the heart at substantially lower energy levels.[11,12] Biphasic technology incorporates several variables, including waveform (eg, rectilinear, damped sin or Gurvich, and truncated exponential),[11,13] tilt (slope) of waveform discharge,[13] and phase duration, which is the percentage of the total waveform duration occupied by the first phase.[13] Manipulation of these variables may affect defibrillation threshold in a beneficial or adverse manner; thus, observations associated with 1 type of biphasic cardioverter-defibrillator cannot be applied to all types of units. New-generation biphasic cardioversion devices that incorporate these waveforms have greater efficacy and safety (eg, fewer ECG abnormalities, less post-shock myocardial dysfunction) in humans and in animal models.[11-16] In humans and swine, first-shock efficacy with a low-energy biphasic defibrillator is greater than with high-energy monophasic shocks for both ventricular defibrillation and atrial cardioversion.[11-14] Furthermore, because low-energy doses can be used, there is less myocardial dysfunction,[14-18] fewer ECG abnormalities,[16] and less burning of the skin.

Successful conversion from atrial fibrillation to sustained sinus rhythm by use of rectilinear biphasic cardioversion was attained in 1 of 2 horses reported here. There are several factors that may explain the difference in outcome between horses. Duration of atrial fibrillation adversely affects the likelihood of conversion to sinus rhythm in horses and humans.[1,6,19,20] Chronic atrial fibrillation causes electrophysiologic and structural remodeling of atrial myocytes (fibrosis, decreased duration of action potentials, and decreased effective refractory period), which perpetuates the arrhythmia.[4,20] Thus, atrial fibrillation that is initially responsive to pharmacologic or electrical cardioversion tends to become resistant and cannot then be converted to sinus rhythm.[20] It is not surprising, therefore, that cardioversion did not provide lasting conversion to sinus rhythm in the first horse, in which fibrillation had been present for > 24 months. In contrast, the

Unauthenticated | Downloaded 12/05/23 11:04 PM UTC

horse in which cardioversion was successful had been in atrial fibrillation for only approximately 1 month.

The fact that the initial cardioversion attempt failed in horse 2 indicates that there are factors other than chronicity of the arrhythmia that may also affect outcome, such as placement of the cardioversion-debrillation pads or paddles. In horse 1 and in the first cardioversion attempt in horse 2, the location of the atria was not confirmed ultrasonographically. After the initial failure at cardioconversion in horse 2, echocardiography was used to verify positioning of the paddles over the atria. This necessitated moving the forelimbs farther cranially than during initial attempts. Placement of the pads (or paddles) over the atria is important for achieving peak current flow through the atrial myocardium and maximizing the opportunity for successful cardioversion. It may be helpful, therefore, to include echocardiographic location of the atria after placement of the patient in dorsal recumbency as part of routine preparation for cardioversion.

The first horse was slightly hypomagnesemic prior to attempted cardioversion. Although an attempt was made to administer magnesium IV to this horse during anesthesia, magnesium administration was discontinued because of worsening hypotension. Similarly, administration of magnesium chloride to horse 2 while under anesthesia was terminated because of hypotension during the first cardioversion attempt. In contrast, during the second and ultimately successful cardioversion attempt in horse 2, electrolytes (magnesium and potassium) were administered prior to anesthesia, and serum electrolyte concentrations were within reference ranges immediately prior to anesthetic induction. Increasing serum magnesium and potassium concentrations to the middle of the reference range may have had a favorable electrophysiologic effect, because the shocks resulted in brief conversion of the rhythm from atrial fibrillation to atrial flutter.

In humans, magnesium administration is useful in preventing and treating some supraventricular and ventricular arrhythmias, including atrial fibrillation,[21,22] even in patients with serum magnesium concentrations with the reference range.[23,24] Recently, decreased protein content of several potassium channels, as a result of atrial fibrillation, has been observed in humans.[25] Consequently, administration of potassium ion along with magnesium ion may be required for optimal antiarrhythmic effect.[24] Nonetheless, it remains to be proven whether augmenting magnesium and potassium concentrations in patients with concentrations of these electrolytes that are within reference ranges is helpful in prolonging the atrial action potential and effective refractory period[26,27] and in optimizing the resting membrane potential.[28]

Both horses received an IV infusion of dobutamine during general anesthesia to maintain mean arterial pressure > 60 mm Hg. Doses ranged from 2 to 4 μg/kg per minute; the higher dose was used during successful cardioversion in horse 2. The effects of β-adrenergic stimulation on defibrillation and cardioversion remain poorly elucidated, and most efforts have been directed at defining the effects on ventricular, rather than atrial, defibrillation thresholds. In a study that examined the effects of α- and β-adrenergic stimulation on atrial defibrillation threshold in human patients, the authors reported no change in atrial defibrillation threshold despite profound atrial electrophysiologic alterations.[29] Epinephrine and dopamine, but not dobutamine, augment the rate of successful defibrillation in dogs with ventricular arrhythmias.[30] It is unlikely that the use of dobutamine had a substantial effect in the outcome of cardioversion attempts in the horses reported here, considering the low doses that were administered and the equivocal benefits of dobutamine on cardioversion.

The electrical shock delivered immediately after administration of a modest dose of quinidine gluconate resulted in successful conversion to sinus rhythm in horse 2. Serum quinidine concentration immediately after cardioversion was 0.3 μg/ml, far less than the concentration needed for pharmacologic cardioversion (2 to 4 μg/ml).[6] These findings suggest that little quinidine is needed to sufficiently enhance the efficacy of electrical cardioversion. Such a low dose of quinidine, although unlikely to cause adverse effects, can apparently facilitate electrical cardioversion, presumably by inhibiting vagal tone and prolonging refractoriness. Although results of some experimental studies[31,32] indicate that quinidine increases the energy needed for cardioversion, these findings could not be reproduced in other studies.[33] It is recommended that human patients receive quinidine 24 to 48 hours prior to cardioversion to prevent early relapse of atrial fibrillation.[33] In humans, the effect of administration of quinidine before electrical cardioversion remains controversial but has been associated with decreasing the number and energy of shocks required for successful conversion as well as reduction of postcardioversion arrhythmias and recurrence of atrial fibrillation.[3]

Measurement of serum CK activity is neither a sensitive nor specific indicator of myocardial damage in horses.[34] In contrast, cTn I and cTn T are specific for cardiac muscle.[28,35] When myocardial damage occurs, measurement of cTn I and cTn T in serum will reveal an early increase that is attributable to release from the cytoplasm, and sustained increases of serum cTn I and cTn T are detected for several days because of myofilament degeneration.[5] Although the first horse in this report had an increase in serum CK activity after the cardioversion attempt, the cTn I concentration remained within reference range. Together, the CK and cTn I findings indicate that the increase in serum CK activity was attributable to skeletal muscle damage rather than myocardial damage. After 11 biphasic electrical shocks delivered during a 48-hour period, serum cTnI concentration in the second horse was within reference range (< 0.3 ng/ml) 36 hours after cardioversion, which indicates that there was no appreciable damage to the myocardium. These data are similar to data obtained from human patients, in whom cTn I concentrations are within reference range or only mildly increased after transthoracic cardioversion.[36] The commercially available ELISA used to measure cTn I in these 2 horses has been validated for use in horses.[5,35]

Complications from anesthesia and biphasic electrical cardioversion were limited to mild erythema and

Unauthenticated | Downloaded 12/05/23 11:04 PM UTC

edema of the skin underlying the cardioversion pads and shortened forward phase of the stride bilaterally with reluctance to ambulate in horse 1. This horse did not have sensitivity to use of hoof testers; therefore, the signs were attributed to generalized muscle soreness secondary to the electrical shocks or recumbency. The weight of horse 1 may have contributed to muscle damage as a result of recumbency, although duration of anesthesia was not excessive (60 minutes).

It is possible that the greater body weight of horse 1 (550 kg vs 393 kg for horse 2) accounted for the increased impedance in this horse (36 to 41 Ω vs 19 to 31 Ω for horse 2). Increased body weight is associated with increased transthoracic impedance in humans.[11] Interestingly, impedance in both horses was less than the mean impedance value of 70 Ω reported for human patients.[37] This would suggest that impedance was not a critical factor in determining the success of cardioversion in these 2 horses.

Data from human patients indicate that there is an optimal range of current for achieving cardioversion.[37] Extremely low or extremely high current is not only less effective but possibly deleterious. In humans with atrial fibrillation, successful cardioversion was achieved in 79% of patients by use of energy settings that delivered 30 to 33 A.[37] Maximum current delivered to the horses in this study was 23 A (horse 1) and 24 A (horse 2). Higher current may have resulted in successful cardioversion of horse 1 and more immediate cardioversion of horse 2. However, the maximum energy output of the biphasic cardioverter-defibrillator used in this study was 200 J. Methods of achieving higher current in target tissues include delivery of shocks during expiration as well as administration of multiple shocks in rapid succession, because transthoracic impedance diminishes with successive shocks. Other measures to minimize impedance, thereby enhancing current delivery to the myocardium, include use of maximal electrode-thorax contact pressure, placement of electrodes such that a direct pathway for current delivery is created while maximizing spacing between electrodes, application of electrodes with a large surface area, and adequate application of coupling gel.[19] Cardioversion pads were used in the unsuccessful attempt at cardioversion in horse 1. A decline in impedance was noticed in horse 2 after changing from adhesive pads to hand-held paddles.

The experiences with rectilinear biphasic electrical cardioversion in these 2 horses indicate that this method of treatment may offer a safe and effective alternative to pharmacologic cardioversion in horses with idiopathic atrial fibrillation of relatively short duration. Additional experience is needed for perfecting technique, determining overall efficacy, defining the optimal biphasic waveform, and establishing arrhythmia duration criteria for successful outcome. As experience is gained, additional data regarding the relative risk, expense, and efficacy of this treatment modality, compared with pharmacologic cardioversion, can be acquired. Considering the successful cardioversion of 1 horse, the lack of cTn I increase in either horse, the lack of substantial deleterious effects in either horse, and the potentially life-threatening adverse effects of cardioversion with quinidine, additional clinical investigation of biphasic electrical cardioversion in horses is warranted.

[a]AXSYM Troponin I, Abbott Laboratories, Abbott Park, Ill.
[b]Mark 7 respirator, Bird Corp, Palm Springs, Calif.
[c]Normosol, Abbott Laboratories, North Chicago, Ill.
[d]ProPadz, ZOLL Medical Corp, Burlington, Mass.
[e]ZOLL M series, courtesy of Robert Finan, ZOLL Medical Corp, Burlington, Mass.

References

1. Reef VB, Levitan CW, Spencer PA. Factors affecting prognosis and conversion in equine atrial fibrillation. *J Vet Intern Med* 1988;2:1–6.
2. Morris DD, Fregin GF. Atrial fibrillation in horses: factors associated with response to quinidine sulfate in 77 clinical cases. *Cornell Vet* 1982;72:339–349.
3. Sarubbi B, Ducceschi V, D'Andrea A, et al. Atrial fibrillation: what are the effects of drug therapy on the effectiveness and complications of electrical cardioversion? *Can J Cardiol* 1998;14:1267–1273.
4. Naccarelli GV, Dell'Orfano JT, Wolbrette DL, et al. Cost-effective management of acute atrial fibrillation: role of rate control, spontaneous conversion, medical and direct current cardioversion, transesophageal echocardiography, and antiembolic therapy. *Am J Cardiol* 2000;85:36D–45D.
5. Cornelisse CJ, Schott HC, Olivier NB, et al. Concentration of cardiac troponin I in a horse with a ruptured aortic regurgitation jet lesion and ventricular tachycardia. *J Am Vet Med Assoc* 2000;217:231–235
6. Collatos C. Treating atrial fibrillation in horses. *Compend Contin Educ Pract Vet* 1995;17:243–245.
7. Patteson M. Problems in equine cardiology. In: Patteson M, ed. *Equine cardiology*. Oxford, England: Blackwell Science Ltd, 1996;206–231.
8. Deem DA, Fregin GF. Atrial fibrillation in horses: a review of 106 clinical cases, with consideration of prevalence, clinical signs, and prognosis. *J Am Vet Med Assoc* 1982;180:261–265.
9. Reef VB. Arrhythmias. In: Marr CM, ed. *Cardiology of the horse*. Philadelphia: WB Saunders Co, 1999;179–209.
10. Muir WW, Reed SM, McGuirk SM. Treatment of atrial fibrillation in horses by intravenous administration of quinidine. *J Am Vet Med Assoc* 1990;197:1607–1610.
11. Mittal S, Ayati S, Stein KM, et al. Transthoracic cardioversion of atrial fibrillation: comparison of rectilinear biphasic versus damped sine wave monophasic shocks. *Circulation* 2000;101:1282–1287.
12. Greene HL, DiMarco JP, Kudenchuk PJ, et al. Comparison of monophasic and biphasic defibrillating pulse waveforms for transthoracic cardioversion. *Am J Cardiol* 1995;75:1135–1139.
13. Gliner BE, Lyster TE, Dillion SM, et al. Transthoracic defibrillation of swine with monophasic and biphasic waveforms. *Circulation* 1995;92:1634–1643.
14. Tang W, Weil MH, Sun S, et al. The effects of biphasic and conventional monophasic defibrillation on postresuscitation myocardial function. *J Am Coll Cardiol* 1999;34:815–822.
15. White RD, Hankins DG, Atkinson EJ. Patient outcomes following defibrillation with a low energy biphasic truncated exponential waveform in out-of-hospital cardiac arrest. *Resuscitation* 2001;49:9–14.
16. Bardy GH, Marchlinski FE, Sharma AD, et al. Multicenter comparison of truncated biphasic shocks and standard damped sine wave monophasic shocks for transthoracic ventricular defibrillation. *Circulation* 1996;94:2507–2514.
17. Tokano T, Bach D, Chang J, et al. Effect of ventricular shock strength on cardiac hemodynamics. *J Cardiovasc Electrophysiol* 1998;9:791–797.
18. Sun S, Klouche K, Tang W, et al. The effects of biphasic and conventional monophasic defibrillation on postresuscitation myocardial function. *J Am Coll Cardiol* 2001;37:1753–1754.
19. Kerber RE. Transthoracic cardioversion of atrial fibrillation

Unauthenticated | Downloaded 12/05/23 11:04 PM UTC

and flutter: standard techniques and new advances. *Am J Cardiol* 1996;78:22–26.

20. Allessie MA, Boyden PA, Camm AJ, et al. Pathophysiology and prevention of atrial fibrillation. *Circulation* 2001;103:769–777.

21. England MR, Gordon G, Salem M, et al. Magnesium administration and dysrhythmias after cardiac surgery: a placebo-controlled, double-blind, randomized trial. *JAMA* 1992;268:2395–2402.

22. Katholi RE, Taylor GJ, Woods WT Jr, et al. Magnesium chloride replacement after bypass surgery to prevent atrial fibrillation: a double blind, randomized trial. *Circulation* 1990;82(suppl 3):III-58.

23. DeCarli C, Sprouse G, LaRosa JC. Serum magnesium levels in symptomatic atrial fibrillation and their relation to rhythm control by intravenous digoxin. *Am J Cardiol* 1986;57:956–959.

24. Iseri LT, Fairshter RD, Hardemann JL, et al. Magnesium and potassium therapy in multifocal atrial tachycardia. *Am Heart J* 1985;110:789–794.

25. Brundel BJJM, Van Gelder IC, Henning RH, et al. Ion channel remodeling is related to intraoperative atrial effective refractory periods in patients with paroxysmal and persistent atrial fibrillation. *Circulation* 2001;103:684–690.

26. Ingemansson MP, Smideberg B, Olsson SB. Intravenous MgSO4 alone and in combination with glucose, insulin and potassium prolong the atrial cycle length in chronic atrial fibrillation. *Europace* 2000;2:106–114.

27. Castellanos A, Kessler KM, Myerburg RJ. The resting electrocardiogram. In: Alexander RW, Schlant RC, Fuster V, et al, eds. *The heart*. New York: McGraw-Hill Book Co, 1998;351–385.

28. Ganong, WF. Excitable tissue: muscle. In: Ganong WF, ed. *Review of medical physiology*. Stamford, Conn: Appleton & Lange, 1999;60–79.

29. Barold HS, Shander G, Tomassoni G, et al. Effect of increased parasympathetic and sympathetic tone on internal atrial defibrillation thresholds in humans. *Pacing Clin Electrophysiol* 1999;22:238–242

30. Otto CW, Yakaitis RW, Redding JS, et al. Comparison of dopamine, dobutamine, and epinephrine in CPR. *Crit Care Med* 1981;9:640–643

31. Babbs CF, Yim GKW, Whistler SJ, et al. Elevation of ventricular defibrillation threshold in dogs by antiarrhythmic drugs. *Am Heart J* 1979;98:345–350.

32. Tacker WA, Niebauer MJ, Babbs CF, et al. The effect of newer antiarrhythmic drugs on defibrillation threshold. *Crit Care Med* 1980;8:177–180.

33. Fuchs T. Cardioversion and defibrillation. In: Podrid PJ, Kowey PR, eds. *Cardiac arrhythmia mechanisms, diagnosis, and management*. Baltimore: The Williams & Wilkins Co, 1995;726–737

34. Argiroudis SA, Kent JE, Blackmore DJ. Observations on isoenzymes of creatine kinase in equine serum and tissues. *Equine Vet J* 1982;14:317–321.

35. O'Brien PJ, Dameron GW, Beck ML, et al. Differential reactivity of cardiac and skeletal muscle from various species in two generations of cardiac troponin-T immunoassays. *Res Vet Sci* 1998;65:135–137.

36. Allan JJ, Feld RD, Russell AA, et al. Cardiac troponin I levels are normal or minimally elevated after transthoracic cardioversion. *J Am Coll Cardiol* 1997;30:1052–1056.

37. Kerber RE, Martins JB, Kienzle MG, et al. Energy, current, and success in defibrillation and cardioversion: clinical studies using an automated impedance-based method of energy adjustment. *Circulation* 1988;77:1038–1046.

Unauthenticated | Downloaded 12/05/23 11:04 PM UTC