

Clinicopathologic evidence of myocardial injury in horses with acute abdominal disease

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Objective—To determine whether there is evidence of myocardial injury in horses with acute abdominal disease.

Design—Prospective case series.

Animals—18 healthy horses and 69 horses with acute abdominal disease.

Procedures—18 healthy horses had been admitted to the hospital for investigation and were assigned to group 1. Horses examined for acute abdominal disease were assigned to 3 groups: strangulating obstruction, nonstrangulating obstruction, or inflammatory disease (groups 2, 3, and 4, respectively). Heart rate, Hct, and blood lactate and cardiac troponin I (cTnI) concentrations were measured at initial examination. Myocardial function was assessed by echocardiographic measurement of fractional shortening and left ventricular ejection time (LVET). Heart rhythm was evaluated via ECG.

Results—The proportion of horses with high (> 0.03 ng/mL) cTnI concentration was significantly greater among horses with strangulating (9/25 [36%]) or inflammatory (9/19 [47%]) lesions, compared with healthy horses (0/18). The proportion of horses with high cTnI concentration was significantly greater among nonsurvivors (12/24 [50%]) than among survivors (10/45 [22%]). Serum cTnI concentration was positively correlated with Hct, heart rate, and blood lactate concentration and negatively correlated with LVET.

Conclusions and Clinical Relevance—Evidence of myocardial injury was observed in horses with acute abdominal disease, and this injury was associated with severity of illness. Recognition of myocardial injury could improve treatment of acute abdominal disease in horses. (*J Am Vet Med Assoc* 2012;241:1202–1208)

Decreased myocardial function is well recognized in human patients with sepsis.^{1–5} Elevated blood concentration of cTn in humans with sepsis occurs independently of underlying cardiac disease and is a strong indicator of a poor prognosis.^{1–3,6,7} The pathogenesis of elevated cTn concentration can involve direct myocardial effects of inflammatory mediators, hypotension, endothelial dysfunction, septic microembolic disease, or high doses of vasoactive drugs.^{1,2,7} Left ventricular dysfunction in humans with sepsis, determined by means of echocardiography, is correlated with elevated cTn concentration.^{3,5,8} Myocardial dysfunction, evidenced by reduced FS and increased pre-ejection period-to-LVET ratio, occurs in dogs with critical illnesses including sepsis and neoplasia.⁹ Elevated cTnI concentration in dogs with gastric dilatation-volvulus correlates with the severity of abnormalities detected via ECG and is a negative prognostic indicator for survival.^{10,11} Myocardial depression and ventricular dysfunction are suspected

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ABBREVIATIONS

cTn	Cardiac troponin
DIC	Disseminated intravascular coagulation
FS	Fractional shortening
LV	Left ventricle
LVET	Left ventricular ejection time
LVID _d	Left ventricular internal diameter diastole
LVID _s	Left ventricular internal diameter systole

to occur in horses with sepsis but have not been well documented.^{12–14}

Sepsis, hypovolemia, and endotoxemia have been observed to varying degrees in horses with acute abdominal disease, contributing to the release of inflammatory mediators and inducing shock.^{15,16} Cardiovascular status at admission is strongly associated with short- and long-term survival of horses with surgical colic.^{17–22} Elevated cTnI concentration occurs in horses with colic^{13,a,b} and is significantly associated with the occurrence of ventricular arrhythmias^b and death.^{13,b} Elevated cTnI concentration in horses recovering from colic surgery indicates a poor prognosis for survival.¹³ Experimentally induced endotoxemia in horses causes moderately elevated cTnI concentration and precipitates cardiac rhythm disturbances.¹⁴ The association between cTnI concentration and ventricular function in horses with sepsis associated with acute abdominal disease is yet to be determined.

In the study reported here, it was hypothesized that horses with acute abdominal disease would have evidence

of myocardial injury and ventricular dysfunction. Secondary hypotheses were that elevated blood cTnI concentration at admission would be a negative prognostic indicator and would correlate with the severity of illness. The purpose of the study reported here was to evaluate myocardial function in horses with acute abdominal disease through measurement of cTnI concentration, echocardiographic evaluation of LV function, and electrocardiographic detection of cardiac arrhythmias. Furthermore, we sought to determine the relationship between blood cTnI concentration and other indicators of illness, including heart rate, Hct, blood lactate and total solids concentrations, FS, and LVET.

Materials and Methods

Horses—Four groups of horses were used in this study. Healthy Thoroughbred racehorses ($n = 18$; group 1) were identified from local racehorse trainers and underwent cardiac evaluation. This included clinical examination, resting echocardiography, and ECG. Horses were retained in the study if they did not have any clinical signs of cardiac dysfunction, and blood sampling for cTnI analysis was performed. For group 1 horses, Hct and blood lactate and total solids concentrations were not measured. Group 1 horses were evaluated from February 2007 until December 2008.

From September 2007 until March 2009, horses were included in the study if they were > 12 months of age and were evaluated at the Equine Centre, Werribee, Australia, for abdominal disease including colic or typhlocolitis. Horses examined because of colic were excluded from the study if they responded to medical treatment alone and were not determined to have typhlocolitis, septic peritonitis, or duodenitis-proximal jejunitis. Horses that had intestinal obstructions identified at surgery or necropsy were divided into strangulating (group 2; $n = 25$) or nonstrangulating (group 3; 25) obstruction. Group 4 were horses ($n = 19$) determined to have on the basis of clinical examination or necropsy one of the following inflammatory intestinal diseases: typhlocolitis, septic peritonitis, or duodenitis-proximal jejunitis.

Clinical laboratory tests—Blood was collected at the time of admission via the jugular or other peripheral vein into a plain evacuated glass vial (10 mL) and an evacuated vial containing lithium heparin anticoagulant (10 mL). Serum was harvested through centrifugation and frozen at -20°C within 45 minutes after collection. The samples were transferred within 1 week to a second freezer, on-site at the Equine Centre, and kept at -80°C until analysis for cTnI concentration. At the conclusion of the study, all samples were transported on dry ice to an off-site laboratory for measurement of cTnI via immunoassay, with lower and upper limits of sensitivity of 0.006 to 50 ng/mL.^c Cardiac troponin I concentration ≤ 0.01 ng/mL was reported as 0.01 ng/mL. Blood collected with lithium heparin anticoagulant was used to measure blood lactate concentration by use of 1 of 2 devices.^{d,e} Blood collected with lithium heparin anticoagulant was also aspirated into a microcapillary tube for measurement of Hct (by centrifugation) and plasma total solids concentration (with a refractometer).

Timing of echocardiography and ECG—In the healthy horses (group 1), echocardiography and ECG were performed on the day of admission. In horses that underwent surgery (groups 2 and 3), ECG was performed throughout the duration of anesthesia, and echocardiography and ECG were performed within 24 hours after recovery from anesthesia. Echocardiography and ECG were performed within 24 hours after hospital admission in horses with inflammatory disease (group 4).

Procedures—Standard echocardiographic views²³ were obtained with an ultrasound unit^f with a 2- to 4-MHz phased array transducer.^g Fractional shortening was calculated from the equation $\text{FS} = \text{LVID}_d - \text{LVID}_s / \text{LVID}_d$, and LVET was calculated from an M-mode short-axis view of the aorta. A reference interval for FS of 28.44% to 50.32% was adopted.²⁴ Left ventricular ejection time > 338 milliseconds was considered normal.²⁵ Telemetric ECG^h was performed stall side and observed for a period of 1 hour.

Statistical analysis—The Fisher exact test was used to compare proportions. Residuals of cTnI concentration and FS were tested for normality via the Shapiro-Wilk test and both were nonnormal; hence, nonparametric analysis was performed. The Kruskal-Wallis test and the Mann-Whitney U test were used to compare values of cTnI concentration and FS between groups. Correlations among cTnI concentration, blood lactate concentration, LVET, and heart rate were assessed by use of the Spearman rank correlation coefficient (r_s) from data of horses with abdominal disease. The variance of FS in group 1 was compared with that combined in groups 2, 3, and 4 by use of the robust modified Levene test.²⁶ This test was robust to nonnormality. Survival was defined as the horse surviving or not surviving to discharge. Horses that were euthanized because of financial constraints were allocated to the survival or nonsurvival groups on the basis of the outcome most likely to occur (ie, survival or nonsurvival) if finances had not been limited. This decision was made by a panel of 3 equine internists on the basis of clinicopathologic and postmortem data. The likely outcome was decided by a simple majority vote. Exact logistic regression estimated the OR of nonsurvival as a function of the cTnI concentration.²⁷ A statistical software programⁱ was used to perform the data analyses. Values of $P \leq 0.05$ were considered significant for all analyses. Mean \pm SD are reported.

Results

Horses—Eighty-seven horses were included in the study. This included 18 horses in group 1, 25 horses in group 2, 25 horses in group 3, and 19 horses in group 4. All horses in group 1 were Thoroughbreds that ranged in age from 3 to 8 years old, with a mean and median age of 4.1 and 4 years, respectively. Horses in groups 2, 3, and 4 consisted of 38 Thoroughbreds, 9 warmbloods, 4 Standardbreds, 4 Arabians, 2 Quarter Horses, 2 Friesians, 1 Appaloosa, 1 Clydesdale, 6 ponies, and 2 Miniature Horses. Horses in groups 2, 3, and 4 ranged in age from 1 to 29 years old, with a mean age of 10.0 years. In group 2, there were 15 horses with small intestinal lesions, 8 horses with large intestinal lesions, and 2 horses with diaphragmatic herniation of both

the small and large intestines. In group 3, there were 7 horses with small intestinal lesions, 16 horses with large intestinal lesions, 1 horse with peritonitis and 1 horse with a nonstrangulated diaphragmatic hernia that both had laparotomies. In group 4, 18 horses had large intestinal disease and 1 horse was determined to have peritonitis. Twelve of 25 (48%) horses in group 2, 19 of 25 (76%) horses in group 3, and 14 of 19 (74%) horses in group 4 survived to discharge ($P = 0.097$). There were 7 horses euthanized because of financial constraints. Of these, 5 were allocated as likely survivors (1 horse in group 2, 3 horses in group 3, and 1 horse in group 4) and 2 were allocated as likely nonsurvivors (1 horse in group 2 and 1 horse in group 4).

Case management—Horses were treated according to the directions of the senior clinician. Horses received medical treatment consisting of crystalloid fluids, antimicrobials, NSAIDs, lidocaine constant rate infusion, hyperimmune plasma, synthetic colloids, partial parenteral nutrition, unfractionated heparin, and pedal supports as deemed necessary. Horses that did not survive underwent necropsy for the purposes of lesion identification.

cTnI—All horses in group 1 ($n = 18$) had a cTnI concentration ≤ 0.03 ng/mL (Figure 1). The proportion of horses with elevated cTnI concentration (> 0.03 ng/mL) was significantly higher in group 2 (9/25 [36%]; $P = 0.006$) and group 4 (9/19; $P = 0.001$), compared with group 1 (Table 1).

The Kruskal-Wallis test indicated that there were significant differences between groups in cTnI concentration ($P = 0.007$). Serum cTnI concentrations were significantly associated with Hct ($r_s = 0.38$; $n = 65$; $P = 0.002$), blood lactate concentration ($r_s = 0.43$; $n = 63$; $P = 0.000$), heart rate ($r_s = 0.47$; $n = 69$; $P < 0.000$), and LVET ($r_s = -0.41$; $n = 25$; $P = 0.041$). Serum cTnI concentration was not associated with concentration of total solids ($r_s = 0.19$; $n = 64$; $P = 0.13$) or FS ($r_s = -0.015$; $n = 31$; $P = 0.93$). Serum cTnI concentration was elevated in 10 of 45 (22%) survivors and 12 of 24 (50%) nonsurvivors ($P = 0.029$). The OR of nonsurvival for a 1 ng/mL increase in cTnI concentration was 1.82 (95% confidence interval, 1.07 to 3.73; $P = 0.022$).

Echocardiography—Fractional shortening data were acquired from 49 horses. There was less variability in the FS for horses in group 1 (mean \pm SD, 36.6 ± 4.8 [median 36.5; range, 26 to 48]), compared with groups 2, 3, and 4 (mean, 37.6 ± 8.6 [median, 37; range, 17 to 54]; $P = 0.030$).

There was no significant difference in FS ($P = 0.61$) between groups. There was 1 of 18 horses in group 1, 2 of 10 horses in group 2, 3 of 15 horses in group 3, and 1 of 6 horses in group 4 with FS values outside the reference range (28.44% to 50.32%; Figure 2).²⁴

Data for LVET were acquired from 28 horses. There were 0 of 3 horses in group 1, 5 of 8 horses in group 2, 5 of 14 horses in group 3, and 3 of 3 horses in group 4 with an LVET < 338 milliseconds.²⁵ For the 25 horses with acute abdominal disease for which serum cTnI concentration, LVET, and heart rate were recorded, the Spearman partial correlation between cTnI concentration and LVET was -0.39 ($P = 0.057$).

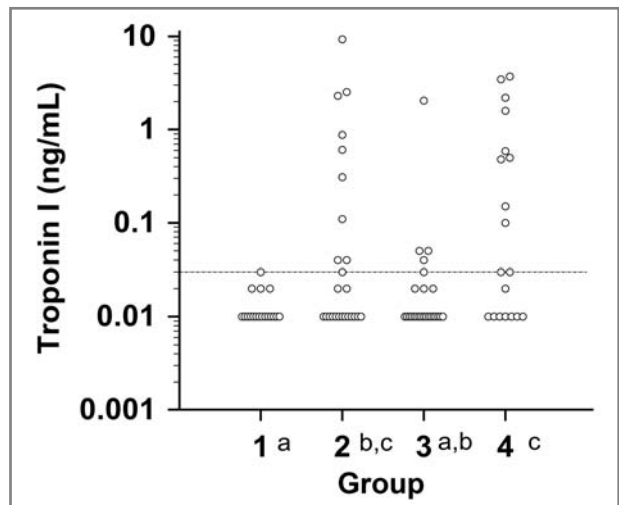


Figure 1—Dot plot of cTnI concentration (ng/mL) for healthy ($n = 18$) horses versus concentrations for horses examined because of acute abdominal disease (69). Dotted line indicates cTnI concentration of 0.03 ng/mL. Groups with different letters are significantly ($P < 0.05$) different as determined by the Mann-Whitney U test and Fisher exact test. Group 1 = healthy horses; group 2 = horses with strangulating intestinal lesions ($n = 25$); group 3 = horses with nonstrangulating intestinal lesions (25); and group 4 = horses with inflammatory abdominal disease (19).

Table 1—Median (range) and proportion of horses with cTnI values > 0.03 ng/mL for healthy horses ($n = 18$) and horses examined because of acute abdominal disease with strangulating or nonstrangulating intestinal obstructions or inflammatory lesions (69).

Group	Median (range)	Proportion of horses with cTnI values > 0.03 ng/mL
Healthy ($n = 18$)	0.01 (0.01–0.03) ^a	0/18 (0%) ^a
Strangulating obstruction ($n = 25$)	0.01 (0.01–9.31) ^{b,c}	9/25 (36%) ^{b,c}
Nonstrangulating obstruction ($n = 25$)	0.01 (0.01–2.05) ^{a,b}	4/25 (16%) ^{a,b}
Inflammatory lesion ($n = 19$)	0.03 (0.01–3.71) ^c	9/19 (47%) ^c

^{a-c}Values within a column with differing superscript letters are significantly ($P < 0.05$) different.

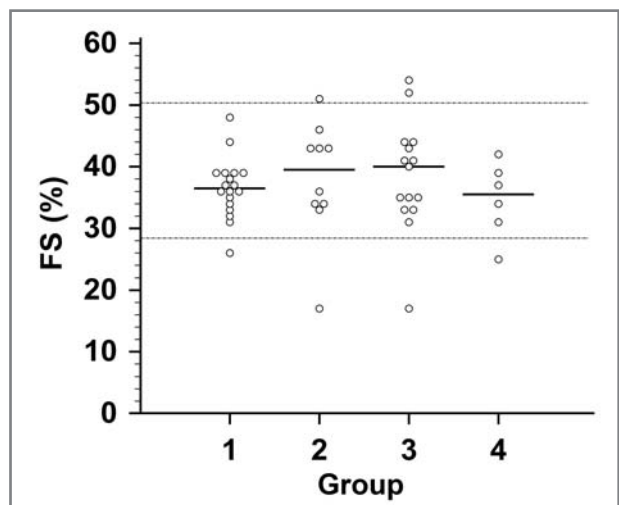


Figure 2—Dot plot of FS (%) for horses in groups 1, 2, 3, and 4. Dotted lines represent the reference interval (28.44% to 50.32%)²⁴; solid lines are the median values. See Figure 1 for remainder of key.

ECG—No clinically important arrhythmias were detected in any of the horses at any time during the study.

Discussion

The present study demonstrated that myocardial injury occurs in horses with acute abdominal disease. Horses with strangulating intestinal or inflammatory abdominal lesions had elevated blood concentration of cTnI, and in the horses evaluated in the present study, elevated cTnI concentration was associated with death and increased severity of illness, as assessed by heart rate, Hct, and blood lactate concentration.

Cardiac troponin is released into plasma from cardiac myocytes in response to injury. It is a sensitive and specific biomarker of acute myocardial injury in humans.^{28,29} Cardiac troponin I and cTnT have high tissue specificity, and assays designed for humans are suitable for measuring cTnI and cTnT concentrations in horses.^{30,31} Reference ranges for cTn concentration reported for healthy horses were < 0.15 ng/mL³² and < 0.35 ng/mL.³³ However, when assays with greater sensitivity are used, the reference range is from 0.00 to 0.03 ng/mL.^{34,35} On the basis of these more recent studies and the values obtained using our assay for group 1 horses, we chose a cutoff of 0.03 ng/mL for cTnI concentration in healthy horses.

The echocardiographic measurements obtained in the present study provided an estimate of systolic LV function. A limitation of our study was that both indices of LV function that were used are dependent on heart rate and mechanical loading. Fractional shortening is an approximation of the ejection fraction and is the most commonly used index of LV systolic function in horses.³⁶ However, FS did not detect myocardial dysfunction in a consistent manner in our study. A limitation of the use of FS as an indicator of myocardial dysfunction is that it is a dynamic measurement with a wide reference interval. Fractional shortening can be affected by a variety of factors including inotropic, lusitropic, and chronotropic effects of sympathetic tone; influence of circulating volume and vascular tone on venous return and afterload; and systolic and diastolic myocardial function.^{37–39} Evidence of sympathetic activation in horses with colic has been demonstrated through measurement of epinephrine and cortisol concentrations in blood and is positively associated with illness severity.²² Gastrointestinal fluid loss and pooling as well as vascular changes induced by endotoxemia can cause reduced venous return, lowered blood pressure, and reduced vascular resistance in horses with acute abdominal disease.^{12,40,41} Rapid IV administration of fluids enhances circulating volume, venous return, and stroke volume in horses with colic.^{41,42} Therefore, wide variation within groups in FS is not an unexpected finding in the horses of our study.

Left ventricular ejection time provided a second indicator of LV function, which is independent of ventricular shape and geometry and could be a more accurate assessment of LV function than FS.³⁶ In the present study, the negative association between cTnI concentration and LVET was not significant when the effect of heart rate

was taken into account. A second limitation of our study was that echocardiographic measurements of FS and LVET were not obtained in every patient, thus making statistical comparisons less powerful. More advanced evaluation of cardiac function is required to determine whether myocardial dysfunction, in addition to vascular changes and hypovolemia, contributes to poor cardiac performance in horses with acute abdominal disease.

In the present study, elevated serum cTnI concentration was also associated with elevated heart rate and Hct. Increased heart rate and Hct are consistent indicators of disease severity and are negative prognostic indicators in horses with acute abdominal disease.^{17,43–46} Consistent with other studies, elevated serum concentrations of cTnI in this study were also associated with elevated blood lactate concentration.^{a,b} Increased blood lactate concentration in horses with colic reflects severity of illness and is associated with nonsurvival.^{13,22,47,48} The association between elevated blood lactate concentration and cTnI concentration in horses in our study is likely because elevated blood lactate concentration reflects severity of disease in these cases.²² In horses undergoing colic surgery, median blood cTnI concentration was higher in nonsurvivors than survivors at all time points, although this was only significant at time points after surgery.¹³ In the same study, median blood lactate concentration was higher in nonsurvivors than survivors at admission and at several time points after surgery.¹³ This correlates well with our study, in which elevated cTnI concentration at admission occurred with greater frequency in nonsurvivors than survivors. Reduced cardiac contractility can in itself cause hyperlactatemia through reduced cardiac output and impaired tissue perfusion.⁴⁹ In addition, elevated blood lactate concentration could reflect systemic hypoperfusion, microvascular thrombosis, or both with the release of cTnI due to ischemic myocardial cell necrosis.³

The observed associations between simple clinical tests such as heart rate, Hct, and blood lactate and cTnI concentrations in the present study demonstrate why these clinical tests are so important in assessment of horses with colic. This observation provides further evidence for the types of pathophysiologic processes occurring in horses with acute abdominal disease, which are reflected by heart rate, Hct, and blood lactate concentration.

Endotoxemia is a well-recognized component of acute abdominal disease,^{15,16,50,51} and it has been speculated to be an important cause of secondary cardiac problems in critically ill adult horses^{14,34,52} but without direct empirical evidence. Infusion of endotoxin causes myocardial depression in humans.⁵³ Inflammatory mediators released in association with endotoxemia in horses include tumor necrosis factor- α , interleukin-6, interleukin-8, and thromboxanes.^{16,54} Although experimental infusion of endotoxin causes elevated cTnI concentration and induces rhythm disturbances in horses, elevated cTnI concentration in horses with colic is not associated with blood concentration of endotoxin.^a The influence of endotoxemia on ventricular function and cTnI concentration in acute abdominal disease is difficult to elucidate because it is recognized that endotoxin is not readily detected in blood. Inflammatory cytokine concentrations or neutrophil chemiluminescence activity might be more useful indicators of endotoxemia in hors-

es.^{16,51} Our investigation detected evidence of myocardial injury in horses, which were likely subject to the effects of endotoxemia; however, further research is needed to determine the extent to which endotoxemia causes myocardial injury in horses.

Increased cTnT concentration occurs in foals with sepsis and is thought to reflect myocardial injury.⁵⁵ Inflammatory mediators, released in association with sepsis, cause changes to vascular tone and vascular permeability, inducing vasodilation and shock.¹² Vascular endothelial injury is associated with cardiac dysfunction, hypoperfusion of the splanchnic and other tissues, and DIC.^{56,57} Human patients with septic shock have increased coronary blood flow.⁵⁸ However, perturbations in regional coronary blood flow and microvascular failure could contribute to myocardial ischemia in humans with sepsis.⁵⁹ Humans with sepsis have interstitial myocarditis, interstitial edema, and muscle fiber necrosis.⁶⁰ Septic foals with cardiac lesions identified at necropsy have cTnI concentrations above the median values for the general septic foal population.⁵⁵ Excessive deposition of fibrin occurs in capillaries of the kidneys, lungs, liver, spleen, heart, and brain of humans with DIC, and these lesions also occur in the kidneys, lungs, and liver of horses with gastrointestinal disease.⁵⁷ Subclinical DIC occurs in 32% of horses with acute colitis and is significantly associated with a poor prognosis.⁵⁶ These results suggest that horses with intestinal disease are subject to microvascular thrombosis and hypoperfusion, potentially contributing to elevations in cTnI.⁵⁷ Examination of cardiac tissue of horses dying from acute abdominal disease could determine the extent to which microvascular thrombosis and fibrin deposition contribute to myocardial injury in horses with acute abdominal disease. Additionally, elevations in cTn concentration have been observed after racing, strenuous treadmill exercise, and endurance events.^{34,61,62} The influence of prolonged tachycardia and myocardial exhaustion on cTnI concentration in horses with acute abdominal disease is not clear.

None of the horses in the present study developed cardiac arrhythmia during electrocardiographic observation. Other studies^{63,64} have shown frequent arrhythmias in horses with acute abdominal disease and electrolyte disturbances or induced endotoxemia.¹⁴ Thirty-eight of 110 horses examined at an equine referral center for colic had abnormal cTnI concentrations at admission, and elevated cTnI concentrations were significantly associated with the occurrence of ventricular arrhythmias.^b The absence of arrhythmias in our study is an interesting finding, given the evidence for concurrent myocardial injury in many of the horses. It is possible that monitoring during anesthesia, in addition to 1 hour of telemetric recording, was insufficient to capture occasional rhythm disturbances in these patients. Electrocardiography was not performed in any of the horses prior to the commencement of treatment, which might also have precluded the detection of occasional rhythm disturbances. Constant rate infusion of lidocaine was administered after surgery in horses thought to be at risk for ischemia-reperfusion injury of the intestine.⁶⁵ Lidocaine is a potent type I antiarrhythmic agent used in the treatment of ventricular dysrhythmias. Tight

control of electrolyte abnormalities and the administration of lidocaine to many of the horses after surgery could have contributed to the absence of arrhythmias in horses in the present study.

This investigation found that elevated cTnI concentration occurred frequently in horses with acute abdominal disease and was associated with myocardial dysfunction and nonsurvival. It is not intended for cTnI analysis to be introduced as a commonly used prognostic indicator in horses with acute abdominal disease. Cardiac troponin analysis and echocardiography might be useful for select cases in which myocardial dysfunction is suspected and for patients that have been nonresponsive to routine treatments. Indices such as heart rate, Hct, and blood lactate concentration are fast, inexpensive, and consistent indicators of prognosis.^{13,19,21,22,43-46} These simple clinical tests have been shown to accurately reflect pathophysiologic processes, including myocardial injury, which contribute to a poor outcome in horses with abdominal disease. Importantly, myocardial injury appears to be common in horses with severe, acute abdominal disease, and case management should incorporate this consideration.

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From this month's AJVR

Plasma 25-hydroxyvitamin D₃ concentrations in Hermann's tortoises (*Testudo hermanni*) exposed to natural sunlight or one of two artificial ultraviolet radiation sources

Paolo Selleri and Nicola Di Girolamo

Objective—To determine the effect of various UVB radiation sources on plasma 25-hydroxyvitamin D₃ concentrations in Hermann's tortoises (*Testudo hermanni*).

Animals—18 healthy Hermann's tortoises.

Procedures—Tortoises were exposed to sunlight in an outdoor enclosure located in the natural geographic range of Hermann's tortoises (n = 6 tortoises) or a self-ballasted mercury-vapor lamp (6) or fluorescent UVB-emitting lamp (6) in an indoor enclosure for 35 days. Plasma samples were obtained from each tortoise on the first (day 0) and last (day 35) days of the study, and concentrations of 25-hydroxyvitamin D₃ were determined. Amount of UVB radiation in enclosures was measured.

Results—Mean ± SD plasma 25-hydroxyvitamin D₃ concentrations for tortoises exposed to the mercury-vapor and fluorescent lamps were significantly lower on day 35 (155.69 ± 80.71 nmol/L and 134.42 ± 51.42 nmol/L, respectively) than they were on day 0 (368.02 ± 119.34 nmol/L and 313.69 ± 109.54 nmol/L, respectively). Mean ± SD plasma 25-hydroxyvitamin D₃ concentration for tortoises exposed to sunlight did not differ significantly between days 0 (387.74 ± 114.56 nmol/L) and 35 (411.51 ± 189.75 nmol/L). Mean day 35 plasma 25-hydroxyvitamin D₃ concentration was significantly higher for tortoises exposed to sunlight versus those exposed to mercury-vapor or fluorescent lamps. Sunlight provided significantly more UVB radiation than did the mercury-vapor or fluorescent lamps.

Conclusions and Clinical Relevance—Plasma 25-hydroxyvitamin D₃ concentrations differed between tortoises exposed to sunlight and those exposed to artificial UVB sources. Exposure to sunlight at a latitude similar to that of the natural geographic range is recommended for healthy and calcium-deficient tortoises. (*Am J Vet Res* 2012;73:1781–1786)



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