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Uroperitoneum in 32 Foals: Influence of Intravenous Fluid Therapy, Infection, and Sepsis

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Foals may present to a referral hospital with the primary diagnosis of uroperitoneum (UP), or they may develop UP while hospitalized for other reasons. Historical, physical, laboratory, and diagnostic variables of foals presenting with UP were compared to those developing UP while hospitalized. Emphasis was placed on the presence of electrolyte abnormalities, evidence of sepsis or infection, and development of anesthetic complications during surgical correction of the defect. Foals developing UP while in the hospital frequently had a history of dystocia and presented at a very young age (<48 hours) with primary clinical signs compatible with intrauterine compromise or presumed hypoxic or ischemic insult with or without sepsis. Foals referred with suspected UP often had additional problems unrelated to the urinary system. These foals had hyponatremia and hyperkalemia on presentation, whereas foals receiving intravenous fluid therapy consisting of a balanced electrolyte solution did not develop the classical pattern of electrolyte abnormalities, yet a similar increase in serum creatinine and, frequently, decreasing urine production were noted. Infection was present in 63% of the foals, and 78% of foals revealed signs suggestive of sepsis or infection. Intrauterine compromise, presumed hypoxia or ischemia, and sepsis may predispose foals to development of UP. Anesthetic complications occurred in 16% of the foals undergoing surgical correction of the defect, although hyperkalemia was only present in half of the foals with anesthetic complications.

Key words: Anesthetic complications; Electrolytes; Equine; Neonate; Urachus; Urinary bladder.

Uroperitoneum (UP) in the foal is a long-recognized and well-documented disease that occurs mainly in newborn foals. Early reports indicated a sex predilection for males[1–3] and emphasized the presence of the characteristic electrolyte abnormalities of hyponatremia, hypochloremia, and hyperkalemia in conjunction with azotemia.[4,5] The urinary tract defects were thought to occur during parturition, most commonly along the dorsal bladder wall, secondary to pressure applied to a full urinary bladder.[6] As surgical repair is the treatment of choice with some rare exceptions, emphasis has been placed on preoperative stabilization of the patient as a result of the fear of anesthetic complications related to electrolyte abnormalities, primarily hyperkalemia and metabolic acidosis.[5]

A recent retrospective study demonstrated that the traditionally described presentation for equine neonatal UP does not necessarily apply to the population of a referral hospital. No sex predilection was observed, nor were the classic electrolyte abnormalities present in more than 50% of the documented cases.[7] The authors also found septicemia and critical illness to be major factors affecting outcome, with half of the foals with UP having a positive sepsis score.[7,8] Foals receiving fluid therapy were more likely to be septic and to have normal serum electrolyte concentrations.[7] The authors postulated a potential contribution of sepsis and critical illness to the development of UP in foals. Infection of the urinary tract has previously been suggested as a cause of UP, and similar observations made in other referral hospitals have prompted further evaluation of the subject.[3]

This study was conducted to address 4 objectives. We compared signalment, presenting symptoms, physical parameters, laboratory findings, rupture sites, complications occurring during treatment, and outcome among foals presenting with UP (primary presenting complaint [PPC]) and foals developing UP while hospitalized (secondary complaint [SC]). We attempted to delineate if the 2 groups represented different underlying etiologies leading to the same clinical manifestation or if these foals all had one or more predisposing events—such as hypoxia, infection, or sepsis—in common with more critically ill foals seeking veterinary attention earlier, before either development or diagnosis of UP.

Our second hypothesis was that foals developing UP while hospitalized (SC) and receiving intravenous fluids would not demonstrate the typical electrolyte abnormalities classically associated with UP, whereas foals presenting with a primary complaint of UP (PPC) without previous intravenous fluid therapy would. Third, we further hypothesized that sepsis and infection would be common findings in foals with UP, regardless of whether UP developed before or during hospitalization. Fourth, we hypothesized that anesthetic complications due to electrolyte imbalances were less common than anecdotally feared.

Material and Methods

Case Selection

Medical records of foals with a diagnosis of UP between 1987 and 2004 were reviewed. Diagnosis of UP was determined by findings during exploratory celiotomy, postmortem examination, or, in medically treated foals, ultrasonographic findings suggestive of UP and a peritoneal creatinine : serum creatinine concentration ratio of >2. Foals in which the diagnosis of UP was established within 24 hours of admission were assigned to the PPC group, whereas foals diagnosed at >24 hours after admission were assigned to the SC group. Sepsis was defined by a positive blood culture or documented infection from a local site of suspected infection.[9] Documented infection was defined

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Table 1. Comparison of data obtained from all foals (overall) on admission, foals presenting with uroperitoneum (primary presenting complaint, PPC) on admission, and foals developing uroperitoneum while hospitalized on admission (secondary complaint: SCa) and on the day of diagnosis of uroperitoneum (secondary complaint, SCb).

	Overall	PPC	SCa	SCb
Age (days)	6.5 ± 10.1	9.5 ± 10.9	2.6 ± 7.9*	6.6 ± 7.5
WBC ($10^3/\mu L$)	9.4 ± 8.0	12.7 ± 8.2	5.5 ± 5.8*	7.9 ± 5.4
Neutrophils ($10^3/\mu L$)	8.7 ± 8.8	12.3 ± 9.3	4.0 ± 5.7*	ND
Fibrinogen (mg/dL)	399 ± 272	515 ± 208	256 ± 283*	ND
Na^+ (mmol/L)	131 ± 12	127 ± 14	135 ± 6*	136 ± 8*
Cl^- (mmol/L)	94 ± 13	91 ± 16	98 ± 8*	94 ± 6
K^+ (mmol/L)	5.0 ± 1.0	5.5 ± 1.1	4.4 ± 0.5*	4.5 ± 1.4
Creatinine (mg/dL)	5.6 ± 4.3	5.1 ± 2.1	6.1 ± 5.9	3.6 ± 1.7
IgG (mg/dL)	624 ± 279	770 ± 111	467 ± 324*	ND
Glucose (mg/dL)	123 ± 75	155 ± 78	88 ± 57*	ND

WBC, white blood cell count; Na, serum sodium concentration; Cl, serum chloride concentration; K, serum postassium concentration; IgG, immunoglobulin G; ND, not determined.

* Statistically different from PPC ($P < .05$).

as a positive culture result or microscopic evidence of intra- and extracellular bacteria in physiologically sterile samples. In an attempt to identify all foals with infection or sepsis, cases involving negative culture results were reviewed and classified as suspected sepsis or infection if the presenting complaint and clinical course were consistent with an infectious process and if the foal had marked leukopenia, a significant left shift, or clinical and laboratory signs consistent with sepsis or SIRS (2 or more of the abnormalities: fever [temperature >102.5°F], hypothermia [temperature <97.5°F], tachycardia [>120], tachypnea [>40], leukocytosis [normal 5,500–12,500 cells/μL], leucopenia, or greater than 10% immature granulocytes).[10] Anesthetic complications were defined as any unexpected hemodynamic or respiratory events occurring during induction or maintenance of general anesthesia. Anesthesia records were reviewed by one of the authors (KO).

Information obtained from the medical records included signalment; presenting complaint; clinical and clinicopathologic data on admission, and, in foals assigned to the SC group, the day-before, the day-of, and the day-after diagnosis of UP; type of intravenous fluids administered (sodium-containing versus sodium-free fluids); exploratory celiotomy and postmortem findings; and complications. Recorded laboratory values included routine CBC, serum chemistry, venous and arterial blood gases, peritoneal fluid analysis (including peritoneal creatinine concentration), and any culture results. Outcome was recorded based on whether the foal was discharged, was euthanized, or died.

Statistical Analysis

Tests of association of categorical variables were performed by Fisher's exact test, and tests of differences of interval variates among outcome classes were performed by the Kruskal-Wallis test and the Student's *t*-test, depending on sample size and data normality. Additionally, tabulation facilities and box plots were used to create tables and graphics for this article. The software, Stata 8.2,[a] was used for all analyses, Minitab 12[b] was used to generate the graphs, and a *P* value of .05 was routinely used to separate differences due to chance from differences due to test variables.

Results

Thirty-two foals met the criteria for inclusion into the study: 18 (56%) foals were assigned to the PPC group and 14 foals comprised the SC group. The age at presentation ranged from 0 (<24 hours old) to 42 days; the mean age was 6.2 days. The average age of foals in the PPC group was 9.5 ± 10.8 days (range 2–42 days). Foals in the SC group were significantly younger ($P < .001$), with a mean age of 0.46 ± 0.52 days on admission. All foals in the SC group, with the exception of a 30-day-old foal, were <48 hours old; 7 were <24 hours old. UP was diagnosed in the SC group between 1 and 11 days after admission, with an average diagnosis time of 4 ± 2.6 days postadmission at an average age of 6.64 ± 7.48 days.

Sixty-nine percent of the foals were colts and 31% were fillies. Although no statistical difference in males versus females ($P = .22$) could be documented, 78% of the PPC group foals were colts, compared to 57% males in the SC group. Breeds included 69% Thoroughbreds, 6% Standardbreds, 9% Warmbloods, and 16% other breeds consisting of 1 each of Miniature Horse, Quarter Horse, Appaloosa, and Paint. Eighteen foals were delivered unassisted without complications; no information regarding birth was available for 3 foals. Dystocia was significantly more common in SC group foals ($P = .014$), with 6 of 12 foals experiencing dystocia, 2 resulting in Caesarean section, compared to 2 of 17 in the PPC group. Birth was induced in 2 foals in the SC group and in 1 foal in the PPC group. Most foals (25/32) presented with more than one complaint, including not nursing/depression (20/32), signs related to the gastrointestinal tract (11/32), abdominal distension (15/32), straining to urinate (7/32), suspected respiratory disease (6/32), prematurity (6/32) and suspected sepsis (6/32) (with sepsis and prematurity being more common in SC group foals), fever (2/32), neurologic signs (3/32); 1 foal each displayed herniation through the umbilical remnant, renal failure, urethral obstruction, and an unspecified umbilical problem. Findings on physical examination were nonspecific; abdominal distension was noted, with one exception, almost exclusively in PPC foals ($P < .001$). Only 9 foals presented with complaints directly related to the urogenital system.

Hematologic abnormalities included leukopenia (Reference range: 5,500–12,500 10^6 cells/μL) (14/31) and leukocytosis (8/31). White blood cell ($P = .004$) and neutrophil count ($P < .002$) were significantly higher in PPC foals. There was no statistical difference in packed cell volume, lymphocyte count, the presence of nonsegmented neutrophils, or any arterial blood gas variables (Table 1). Mean plasma fibrinogen concentration was 399 ± 273 mg/dL and

was significantly ($P = .001$) higher in foals of the PPC group. Plasma immunoglobulin G (IgG) ($P = .05$) and blood glucose ($P = .024$) concentrations were significantly lower in SC foals. The most common recorded rupture site (75%) was the bladder. The dorsal surface was affected 8 times and the ventral aspect 7 times, 2 ruptures occurred on the bladder apex, and in 4 foals the exact location of the bladder defect could not be identified from the record. Defects in the urachus were present in 7 foals (6 PPC foals versus 1 SC foal). One foal had a ureteral lesion; in 3 foals the rupture site was not recorded. Although by inspection, rupture of the urachus appeared to be more common in PPC foals, there was no statistical difference in rupture site when we compared both groups. There was no statistical association between rupture site and peritoneal creatinine concentration or between the peritoneal creatinine concentrations in both groups. Forty-six percent of the rupture sites (7 in PPC foals and 6 in SC foals) revealed gross or histopathologic evidence of necrosis or infection; 9 of 15 bacteriologic cultures of tissue obtained from rupture sites were positive, comprising 3 urachal and 6 bladder specimens. Two of the positive rupture sites within the bladder were adjacent to an umbilical infection. Infection of the bladder was more frequent in PPC foals ($P = .026$).

Surgical repair was performed in 25 foals; 1 foal was treated medically and recovered. A second surgery resulting from failure of the repair site was necessary in 2 foals, and 1 additional foal developed a second tear perpendicular to the original rupture site. All foals requiring a second surgery belonged to the PPC group and all survived to discharge from the hospital. In none of these foals was compromise of the bladder wall, beyond the primary defect, recorded during the first surgery. Nineteen foals (59%) survived to discharge from the hospital; 72% in the PPC group versus 43% in the SC group. There was no statistical difference in survival between the 2 groups ($P = .09$), although there was a trend toward greater survival in PPC foals. Eleven foals were euthanized, 10 (3 in the PPC group versus 7 in the SC group) due to the overall poor prognosis resulting from multiple organ dysfunction, 6 of these (1 in the PPC group versus 5 in the SC group) upon discovery of the UP without attempted surgical repair. One foal was euthanized during surgery due to severe peritonitis. Two foals died.

Hyponatremia (reference range: 132–141 mM/L) was the most common electrolyte abnormality, followed by hyperkalemia (reference range: 2.7–4.9 mM/L) and hypochloremia (reference range: 94–102 mM/L) (Table 1). Three foals demonstrated hypernatremia (2 in the PPC group versus 1 in the SC group) and 5 demonstrated hyperchloremia (2 in the PPC group versus 3 in the SC group). Hypokalemia was not detected in any patient. Sodium ($P = .001$) and chloride ($P = .001$) were significantly lower and potassium ($P = .008$) significantly higher in PPC foals on the day of admission (Table 1; Fig 1). Comparing electrolyte concentrations on the day of diagnosis of UP between the 2 groups, only sodium was significantly ($P = .03$) lower in PPC foals (Fig 1); differences in chloride ($P = .16$) and potassium ($P = .06$) concentrations did not reach statistical significance, although there was a trend toward higher potassium in PPC foals. There was no statistical difference in creatinine concentration or blood urea nitrogen on the day of admission (PPC: mean 5.12 ± 2.13 mg/dL; SC: mean 6.13 ± 5.89 mg/dL) or on the day of diagnosis of UP (SC: mean 3.6 ± 1.7 mg/dL) between the 2 groups. The creatinine concentration in SC significantly ($P = .03$) decreased from the day of admission to a mean of 2.7 ± 1.7 mg/dL on the day before diagnosis of UP followed by a nearly significant ($P = .058$) increase to 3.6 ± 1.7 mg/dL, with concurrent decrease in micturition, on the day of diagnosis of UP. Data regarding intravenous fluid therapy received before and during admission were available in 10 foals; most received sodium-free and sodium-containing fluids concurrently. Three foals in the SC group received only sodium-free fluids; 2 of these were hyponatremic on the day of diagnosis of UP.

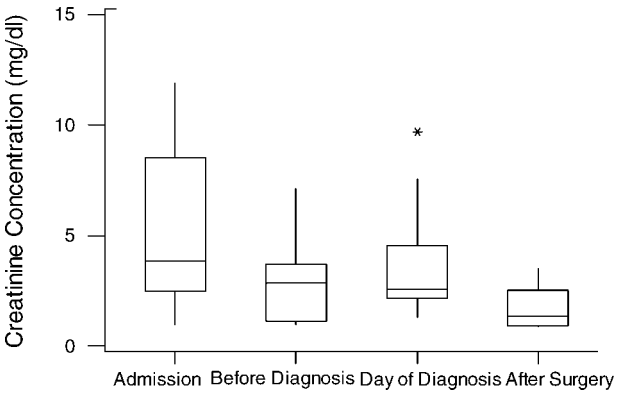

Fig 1. Box-and-whiskers plot of plasma creatinine concentration of secondary complaint (SC) foals (foals developing uroperitoneum [UP] while hospitalized for other primary complaints) at admission, the day before diagnosis of UP, the day of diagnosis of UP, and the day post-surgery. This graph displays the median as a horizontal line within the box, the top and bottom horizontal lines represent the 25th and 75th percentiles, and * represents outliers. Creatinine concentration was significantly decreased between admission and the day before diagnosis.

Documented infection at any body site was identified in 20 of 32 (63%) foals (11/18 PPC versus 9/14 SC foals); there was no statistical difference between the 2 groups. Seven foals had documented infection at more than 1 body site. A positive blood culture was obtained from 7 of 25 (28%) foals; (5/12 PPC versus 2/13 SC foals). Bacterial growth from the urinary tract (rupture site specimens collected during surgery or urine) was detected more commonly in PPC foals, as stated above: 8 of 18 PPC foals versus 1 of 13 SC foals. Including 5 additional foals classified as 'suspected infection,' the total number of documented infection and ''suspected infection'' in foals was 25 of 32 (78%). There was no statistical difference in the presence of any infection or sepsis between the 2 groups. In comparing foals with and without documented infection, no statistical difference was detected in any clinical, hematologic, or biochemical value.

Most foals were induced with a mask and inhaled isoflurane; 5 foals received xylazine (0.18–0.4 mg/kg); and 2 foals received diazepam (0.08 and 0.18 mg/kg) as premedication. All foals were maintained on inhaled isoflurane. Anesthetic complications were noted in 4 foals (16%) during induction and general anesthesia; all 4 belonged to the

PPC group and none of them had received anesthetic premedications other than isoflurane. Two foals developed atrioventricular (AV) blocks during induction and anesthesia, respectively, and in both foals, hyperkalemia was present at the time ([K^+] 6.9 mM/L and 6.0 mM/L, respectively. In 2 additional foals, conduction blocks, which resolved with dobutamine and epinephrine infusions, respectively, were observed. Both foals were mildly acidemic at the time the arrhythmias were observed (pH 7.32 and pH 7.28, respectively). Serum electrolytes were within normal limits with the exception of hyponatremia in 1 foal [Na^+]120 mM/L).

Discussion

Our study agrees in many aspects with the previously reported findings of foals with UP in a referral hospital population.[7] Despite attempts to delineate distinguishing features between foals in PPC and SC population, little statistical difference was found between the 2 populations, and most of the difference present could be explained by the presenting complaint and age at presentation. Nevertheless, comparison of historical, clinical, and clinicopathologic data of the 2 groups raised several points. We were unable to document a statistical difference in the sex distribution between the 2 groups; gross inspection of the data indicates that there is a trend toward more colts than fillies in the PPC group, while the sex distribution is more equal in the SC group. A larger study population is needed to further evaluate this part of the hypothesis. Foals in the SC group presented at a younger age and were significantly more exposed to dystocia, likely contributing to the early presentation of these foals to a referral hospital with primary complaints compatible with hypoxic/ischemic injury, sepsis, or both. Independent of whether the foals had been hospitalized or not, foals were diagnosed with UP at a similar age. Physical parameters on presentation were similar; there were no significant differences in any of the analyzed clinical or laboratory parameters between the groups, with the exception of IgG, blood glucose, and serum electrolyte concentrations. Lower IgG and blood glucose concentrations were most likely attributed to the young age and suspected intrauterine compromise, while electrolyte abnormalities were masked by the intravenous administration of balanced electrolyte solutions to foals in the SC group. The lack of statistical difference in the plasma creatinine concentration on admission was not surprising, as the SC group was almost entirely composed of severely compromised neonates, with azotemia most likely being reflective of either intrauterine abnormalities or delayed transition from fetal to neonatal renal function rather than postrenal failure. We suspect that in many of the SC foals, the urinary tract was intact at the time of admission, supported by the fact that creatinine values initially decreased, followed by a sudden increase (in combination with decreased urine production) several days later, leading to the detection of UP. Comparing the plasma creatinine concentration of the 2 groups on the day of diagnosis of UP, our results are in agreement with findings from the previous study[7] in that fluid therapy affects serum electrolytes but not plasma creatinine concentration.

The exact date of rupture of the urinary bladder was difficult to determine because few foals underwent abdominal ultrasonographic evaluation if UP was not suspected. Time for occurrence of significant abdominal fluid accumulation depends on the site and size of the defect, presence of complete or partial postrenal failure, as well as fluid intake. A 3-fold increase in serum creatinine concentration was observed within 20 hours of experimentally induced UP in adult horses and serum: peritoneal creatinine concentration became greater than 2 within 2 hours.[11] Although we are confident that the main defect in the majority of SC foals developed during hospitalization, we cannot exclude the existence of a small leak present since birth.

The similarity in many analyzed parameters between PPC and SC foals could indicate a fairly homogeneous population and could signify that the division in the 2 groups is arbitrary. Foals developing UP may have similar predisposing factors, such as infection, sepsis, and hypoxic ischemic injury. All tissue samples obtained from the urinary tract submitted for histopathology and culture in one report and a large proportion of samples submitted from our study population yielded evidence of ischemia, necrosis, and infection.[7] It is tempting to speculate that infection, sepsis, and the associated local malperfusion may predispose the foal to focal lesions within the lower urinary tract, with subsequent development of urinary leakage, either during birth or at a later point in time. If the predisposing compromise is mild, the foal may be first presented to a veterinarian once intra-abdominal urine accumulation becomes extensive or once dysuria is noted (commonly at 4–5 days of age). This is supported by the fact that few foals present with a single complaint, and the urinary system is rarely the only body system affected. More severely affected foals, in which compromise is evident at birth, are already hospitalized at this age and receive fluid therapy, masking the typical electrolyte abnormalities. Although there was no statistical difference between groups regarding outcome, there was a trend toward greater survival in PPC foals, and there were more foals euthanized due to multiple organ dysfunction in the SC group, lending further evidence to the assumption that foals in the SC group were more critically ill patients.

Alternatively, UP may represent a syndrome with multiple etiologies, such as birth defects, traumatic incidents, or ischemic/infectious injury, as previously suggested in the literature.[3,6,7,12,13] Review of the literature indicates that birth defects are extremely rare and most likely do not account for the majority of UP foals observed. Bain[1] reported on a foal developing UP secondary to a congenital defect manifesting with absence of the ventral bladder wall, while Pascoe[12] described leakage from a small defect at the urachal-bladder junction that, in the author's opinion, resembled rather a defect than tearing. Wellington[13] reported 2 dorsal bladder defects with rounded commissures and minimal hemorrhage in 2 closely related colts, leading to the suspicion of a congenital malformation. A search for other species suffering from similar congenital or developmental bladder defects was unrewarding, with the majority of cases of ''urinary ascites,'' as it is termed in human infants, occurring secondary to iatrogenic trauma or bladder rupture secondary to posterior urethral valves in males.[14] The pos-

sibility that the initiation of healing of the rupture site creates smooth margins, especially in foals in which diagnosis is delayed for several days, offers an attractive alternative explanation.

Trauma to the umbilical remnant during birth, particularly during dystocia, could predispose foals to partial postrenal failure with gradual accumulation of urine in the abdomen. The thin structure of the urachus and exposure to tearing forces during rupture of the umbilical cord make the urachus a more logical candidate for traumatic rupture than a healthy, muscular, elastic urinary bladder wall. Experimentally induced rupture of the neonatal bladder caused exclusively longitudinal rupture along the dorsal surface at the cranial to mid third with minimal bleeding.[15] Again, looking to other species, rupture of the bladder secondary to large-volume urine accumulation does occur in male human infants secondary to posterior urethral valves.[14] The human fetus differs from the equine fetus in that the urachus does not serve as a potential ''pop-off'' valve in the human near term. Other authors suggest a dichotomy with regard to noninfectious (traumatic) and infectious etiology, the latter occurring in critically ill recumbent neonates hospitalized for other reasons.[16] We were unable to confirm these suggestions, because in our study population, infection of the bladder occurred significantly less frequently in foals developing UP while hospitalized. In our opinion, infection and sepsis play significant roles in the development of the majority of UP cases in foals, although occasional traumatic incidents and very rare, as yet-undocumented congenital defects may also be observed.

We did confirm our second hypothesis, documenting that classic electrolyte abnormalities were not present in foals receiving intravenous fluid therapy, which was naturally more common in SC foals. Only 2 foals treated with intravenous fluids developed hyponatremia on the day of UP diagnosis, both foals received almost exclusively 5% dextrose as maintenance fluids. Although there was no statistical difference among the type of fluid used (sodium-containing versus sodium-free fluids), the available data were incomplete, and most foals received both fluid types, albeit at different rates and volumes. As sodium-free fluids have become more commonly used as maintenance fluids in recent years, a difference may become detectable, with electrolyte abnormalities being more pronounced in foals receiving sodium-free fluids.[17]

As presented in our third hypothesis, evidence of infection and sepsis was present in almost all foals on presentation and was not more common in either group. We did not observe any statistical differences in any of the analyzed parameters among foals with documented infection and those without, and we suspect that more foals were septic or suffered from microbial infection than we were able to definitively document. In contrast to other studies, documented sepsis did not influence the outcome in our study population, but appeared to be an important predisposing factor for the development of UP.

Anesthetic complications occurred less frequently than previously reported; all consisted of third-degree AV block. In 2 foals, minimal or no electrolyte and acid-base abnormalities were present at the time of anesthesia and probably did not account for the dysrhythmias.[5] In an early report, 9 out of 18 foals developed life-threatening arrhythmias under halothane anesthesia, most commonly third-degree AV block or cardiac arrest.[5] The lower prevalence of complications may be reflective of less severe electrolyte abnormalities resulting from earlier detection, better preoperative stabilization, or the routine use of isoflurane, rather than halothane, during general anesthesia.

Footnotes

[a] Stata 8.2, Stata Corporation, College Station, TX
[b] Minitab 12, Minitab Inc, State College, PA

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