Periparturient hemorrhage in mares: 73 cases (1998–2005)

Carolyn E. Arnold, DVM, DACVS; Maria Payne, DVM; James A. Thompson, DVM, DVSc, DACT, DACVPM; Nathan M. Slovis, DVM, DACVIM; Fairfield T. Bain, DVM, DACVIM, DACVP, DACVECC

Objective—To determine signalment, physical examination and clinicopathologic abnormalities, outcome, and subsequent fertility of mares with periparturient hemorrhage (PPH) and identify factors associated with outcome (ie, survival vs death).

Design—Retrospective case series.

Animals—73 mares.

Procedures—Medical records were reviewed for information on age, breed, initial complaint, physical examination and clinicopathologic abnormalities, treatment, outcome, and subsequent fertility.

Results—Median age was 14.0 years (range, 5 to 24 years), and median number of foals produced prior to the diagnosis of PPH was 8 (range, 1 to 16). Ten (14%) mares had prepartum hemorrhage and 63 (86%) had postpartum hemorrhage. Treatment was aimed at restoring cardiovascular volume, enhancing coagulation, controlling pain, and reducing the effects of endotoxemia. Sixty-one (84%) mares survived and 12 (16%) died or were euthanized. Common complications included fever, leukopenia, retained fetal membranes, increased digital pulses, thrombophlebitis, and cardiac arrhythmias. Of the 53 surviving mares for which subsequent breeding information was available, 26 (49%) produced 1 or more foals after recovering from PPH.

Conclusions and Clinical Relevance—Results suggested that PPH can develop in mares of any age and parity. Treatment was associated with a good prognosis for survival and a reasonable prognosis for future fertility. (*J Am Vet Med Assoc* 2008;232:1345–1351)

Periparturient hemorrhage is an important cause of illness and death in broodmares. It has been estimated, for instance, that PPH affects 2% to 3% of broodmares[1] and accounts for 40% of periparturient deaths in mares.[2] Hemorrhage generally occurs within 48 hours after foaling, although prepartum hemorrhage has also been reported.[3–5] Typically, hemorrhage originates from the uterine artery and results in a hematoma if bleeding is contained within the broad ligament or hemoabdomen if it is not.[6,7] Histologic examination of ruptured uterine arteries has revealed degenerative changes of the internal elastic membrane and fibrosis of the intima presumably related to advanced age and parity.[5] Other vessels that have been identified as less common causes of PPH included the ovarian, external iliac, and vaginal arteries.[3–5,8] It has been suggested that multiparous mares and mares with dystocia are at greater risk for PPH.[3,5,6] To our knowledge, however, there have been no studies that have substantiated this clinical impression.

Abbreviation

PPH Periparturient hemorrhage

Clinical signs of PPH are typically nonspecific and may range from lethargy to those associated with abdominal pain (eg, rolling, pawing, sweating, muscle fasciculation, and flank watching) or cardiovascular shock (eg, tachycardia and pale mucous membranes).[5,9–11] For this reason, obtaining an antemortem diagnosis of PPH can be difficult, although rectal palpation, transabdominal or transrectal ultrasonography, and cytologic analysis of peritoneal fluid may assist in the diagnosis of PPH.[5,10,12] However, many clinicians warn against the use of invasive diagnostic procedures, particularly rectal palpation, because of concerns that these procedures could agitate the mare and precipitate an episode of uncontrolled hemorrhage.[5,9,10] Hematologic and biochemical abnormalities may support the diagnosis of PPH and provide an indication of the severity of hemorrhage, but only in the more advanced stages of the disease process, after compensatory mechanisms have taken effect. Other clinicopathologic abnormalities such as azotemia and coagulation deficiencies may also be indicative of PPH,[12,13] but can be nonspecific and time dependent. Obtaining a diagnosis and instituting treatment during the acute stage may be crucial for the survival of mares with PPH.

From the Department of Large Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX 77843 (Arnold, Thompson); Rockwall Equine Center, 9385 County Rd 2432, Terrell, TX 75160 (Payne); the Department of Internal Medicine and Critical Care, McGee Medical Center, Hagyard Equine Medical Institute, 4250 Iron Works Pike, Lexington, KY 40511 (Slovis); and Woodside Equine Clinic, 13011 Blanton Rd, Ashland, VA 23005 (Bain).

Supported by the Link Equine Research Endowment, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, and the Department of Clinical Sciences, College of Veterinary Medicine, Kansas State University.

Address correspondence to Dr. Arnold.

Treatment of mares with PPH is generally supportive because surgical access to the reproductive tract vasculature is limited. Proposed medical treatments include administration of fluids, oxygen-carrying medications, antimicrobials, nonsteroidal anti-inflammatory agents, analgesics, sedatives, and medications to enhance coagulation.[5,6,8–10,14] Although previous reports[14–16] have suggested that the outcome is uniformly unfavorable for mares with PPH, recent studies[17,18] involving limited numbers of mares have reported survival rates ranging from 0% to 88%.

Possible complications that can occur in mares with PPH that survive include sepsis of the broad ligament, uterine hematoma, and subsequent infertility.[3,8] However, rates of these complications and rate of recurrence have not been reported.[5]

Much, therefore, is unknown about PPH in horses, and additional information is needed on factors that can be used to identify horses with PPH and outcome of affected horses following treatment. The purpose of the study reported here was to determine signalment, physical examination and clinicopathologic abnormalities, outcome, and subsequent fertility of mares with PPH. In addition, we wanted to determine whether any physical examination or clinicopathologic abnormalities identified at the time of initial examination were significantly associated with outcome (ie, survival vs death).

Materials and Methods

Case selection criteria—Medical records of broodmares treated at the McGee Medicine Unit at Hagyard Equine Medical Institute between January 1, 1998, and April 30, 2005, were searched to identify mares in which a diagnosis of uterine artery hemorrhage or rupture, broad ligament hematoma, hemoperitoneum, hemoabdomen, PPH, vaginal hemorrhage, uterine wall hematoma, or hemorrhage from the uterine lumen had been made. Mares were included in the study only if the episode of hemorrhage had occurred within 60 days before or after parturition and there was ultrasonographic or necropsy evidence of PPH or results of abdominocentesis were consistent with hemoperitoneum.

Medical records review—Information obtained from medical records of mares included in the study consisted of age, breed, date of admission to the hospital, whether there was any history of complications associated with this or previous pregnancies, initial complaint, whether the mare was recumbent at the time of admission, physical examination findings at the time of admission, and results of clinicopathologic testing. In mares in which transabdominal or transrectal ultrasonography was performed, information was obtained on whether fluid was seen within the uterus or an excessive amount of free peritoneal fluid or a hematoma of the reproductive tract was seen. If an excessive amount of free peritoneal fluid was seen, sonographic characteristics of the fluid were recorded. If a hematoma of the reproductive tract was seen, its location and appearance were recorded. In mares in which abdominocentesis was performed, information was obtained on results of fluid analysis, including WBC count, neutrophil and mononuclear cell fractions, RBC count, Hct, total protein concentration, and whether bacteria were seen, and results of bacterial culture. Details of medical and surgical treatments were also recorded. Finally, information on outcome; duration of hospitalization; complications that developed during hospitalization; and, for mares that died or were euthanized, results of necropsy were recorded. Information on parity at the time PPH was diagnosed and whether mares that survived subsequently produced a foal was obtained from Jockey Club Information Systems[a] and the American Trotting Horse Association.[b]

Statistical analysis—χ^2 Analysis was used to test for associations between categoric clinical findings and outcome (survived vs died or euthanized). Categoric clinical findings that were analyzed included parity (primiparous vs multiparous), timing of hemorrhage (prepartum vs postpartum), occurrence of dystocia (yes vs no), recumbency at admission (yes vs no), whether examination per rectum was performed (yes vs no), and location of hemorrhage (broad ligament hematoma, hemoperitoneum, bleeding into the lumen of the uterus, uterine wall hematoma, or vaginal laceration). χ^2 Analysis was also used to test for an association between outcome and age, by dichotomizing mares as younger or older than the median age, and for associations between outcome and hypothermia at the time of initial examination (yes vs no), hypothermia during hospitalization (yes vs no; determined on the basis of lowest rectal temperature recorded during hospitalization), hyperthermia at the time of initial examination (yes vs no), hyperthermia during hospitalization (yes vs no; determined on the basis of highest rectal temperature recorded during hospitalization), tachycardia at the time of initial examination (yes vs no), tachycardia during hospitalization (yes vs no; determined on the basis of highest heart rate recorded during hospitalization), tachypnea at the time of initial examination (yes vs no), tachypnea during hospitalization (yes vs no; determined on the basis of highest respiratory rate recorded during hospitalization), anemia at the time of initial examination (yes vs no), anemia during hospitalization (yes vs no; determined on the basis of lowest PCV recorded during hospitalization), hypoproteinemia at the time of initial examination (yes vs no), and hypoproteinemia during hospitalization (yes vs no; determined on the basis of lowest total protein concentration recorded during hospitalization). For these analyses, hypothermia was defined as a rectal temperature < 37.2°C (99°F), hyperthermia was defined as a rectal temperature > 38.6°C (101.5°F), tachycardia was defined as a heart rate > 100 beats/min, tachypnea was defined as a respiratory rate > 40 breaths/min, anemia was defined as a PCV < 20%, and hypoproteinemia was defined as a total protein concentration < 4.0 g/dL. For contingency tables with a value of 0 in one of the cells, a value of 0.5 was used to compute confidence intervals. All analyses were performed with standard software.[c] Values of $P \leq 0.05$ were considered significant.

Results

Seventy-three mares met the criteria for inclusion in the study. This included 68 Thoroughbreds, 2 Stan-

dardbreds, 1 Rocky Mountain Horse, 1 Paso Fino, and 1 Warmblood. Median age was 14.0 years (range, 5 to 24 years; **Figure 1**). Median number of foals produced prior to diagnosis of PPH was 8 (range, 1 to 16; **Figure 2**). Ten mares (14%) had prepartum hemorrhage (ie, between the 10th month of gestation and the day prior to the expected foaling date), and 63 (86%) had postpartum hemorrhage.

Only 4 of the 73 (5%) mares had reportedly had a previous episode of PPH. Fourteen (19%) mares had a history of dystocia during the current pregnancy. Of these, 3 underwent assisted vaginal delivery while anesthetized, 1 underwent fetotomy, and 4 underwent cesarean section. Additional information was not available for the remaining 6 mares that had had dystocia.

Other reported complications associated with the current pregnancy included placentitis (n = 1), abortion (1), laminitis (1), colitis (1), and uterine prolapse (1). Median time interval between foaling and admission to the hospital for the 63 mares with postpartum hemorrhage was 12 hours (range, 0 to 120 hours).

Twenty of the 73 (27%) mares were examined because of PPH diagnosed prior to referral. Initial complaints for the remaining mares varied, with 46 (63%) examined because of signs of colic; 8 (11%) examined because of vaginal hemorrhage; 5 (7%) examined because of clinical signs consistent with cardiovascular shock; 4 (5%) examined because of ataxia; and 1 (1%) each examined because of uterine prolapse, diarrhea, retained fetal membranes, syncope, signs of depression, weight loss, and fever. Sixteen (22%) mares had multiple complaints at the time of admission, with the most common combination being signs of colic and PPH.

Eight of the 73 (10.9%) mares were recumbent at the time of admission. Data related to vital signs were summarized (**Table 1**). All mares reportedly had abnormal mucous membrane color, ranging from pale pink to white, at some time while hospitalized. Median capillary refill time was 3 seconds (range, 1 to 5 seconds).

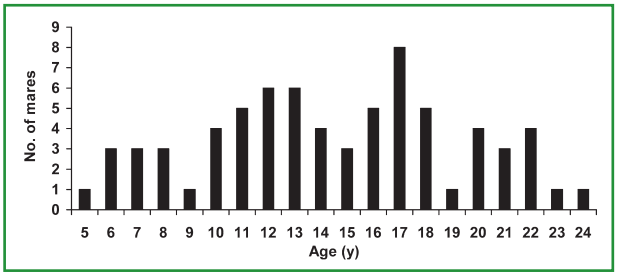

Figure 1—Distribution of age for 71 mares with PPH.

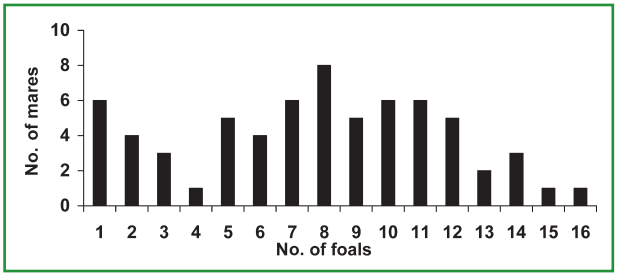

Figure 2—Distribution of parity for 66 mares with PPH.

Rectal palpation was performed by a hospital clinician in 22 of the 73 (30%) mares.

In 71 of the 73 mares, the site of hemorrhage was determined by means of transrectal or transabdominal ultrasonography (**Table 2**). Although size of the hematoma within the broad ligament was not consistently recorded, reported sizes ranged from 3×3 cm^2 to a hematoma that extended from the broad ligament along the ventral aspect of the abdomen to the diaphragm. The depth of the abdominal fluid was measured ultrasonographically in 23 of the 29 mares with hemoabdomen; median depth was 5 cm (range, 1 to 23 cm). Abdominal fluid had a homogenous echogenic appearance characteristic of hemoabdomen in all 29 mares. In addition, in 20 mares, the fluid also had a swirling pattern indicative of movement within the abdomen secondary to respiration or active hemorrhage.

Abdominocentesis was performed in 19 of the 73 (26%) mares. Gross evaluation of the peritoneal fluid was used to confirm hemoabdomen in 10 mares; in the remaining 9 mares, the fluid was submitted for cytologic evaluation. For these 9 mares, median WBC count

Table 1—Vital signs at the time of initial examination and while hospitalized in 73 mares with PPH.

Variable	No. of mares	Median	Range
Rectal temperature (°C)			
At initial examination	66	37.7	34.9–39.4
Maximum while hospitalized	28	38.6	38.7–40.6
Minimum while hospitalized	19	36.4	35.0–37.2
Heart rate (beats/min)			
At initial examination	72	60	36–128
Maximum while hospitalized	73	80	44–152
Respiratory rate (breaths/min)			
At initial examination	66	20	8–80
Maximum while hospitalized	73	24	12–120
PCV (%)			
At initial examination	69	38.4	11.4–70.0
Minimum while hospitalized	69	21.2	10.0–48.7
Total protein (g/dL)			
At initial examination	69	5.0	3.2–9.2
Minimum while hospitalized	69	4.8	2.2–8.0

Table 2—Location of hemorrhage in 71 mares with PPH.

Location	No. (%) of mares that survived	No. (%) of mares that died
Abdomen	23 (79)	6 (21)
Left broad ligament	7 (100)	0 (0)
Right broad ligament	5 (100)	0 (0)
Uterine lumen	5 (100)	0 (0)
Uterine wall	3 (100)	0 (0)
Vagina	0 (0)	1 (100)
Multiple locations	18* (86)	3† (14)
Total	61 (86)	10 (14)

*Abdomen and broad ligament (n = 8); abdomen and uterine lumen (3); broad ligament and uterine lumen (2); abdomen and uterine wall (2); and right and left broad ligaments, broad ligament and uterine wall, and uterine lumen and wall (1 each). †Abdomen and broad ligament, broad ligament and uterine wall, and uterine wall and vagina (n = 1 each).

of the peritoneal fluid was 5,100 cells/µL (range, 1,280 to 50,000 cells/µL), and median RBC count was 6.48 × 10^6 RBC/µL (range, 1 × 10^3 to 9.2 × 10^6 RBC/µL). Median Hct was 39% (range, 19.5% to 44%), and median total protein concentration was 4.4 g/dL (range, 0.4 to 5.8 g/dL). Bacteria were evident in peritoneal fluid from 6 mares. Peritoneal fluid samples from 10 mares were submitted for bacterial culture, and 2 samples yielded bacterial growth. *Escherichia coli, Klebsiella pneumoniae*, group B *Streptococcus equi* subsp *zooepidemicus*, and *Staphylococcus aureus* were isolated from one mare; *E coli* was isolated from the other.

A nasogastric tube was inserted to check for reflux in 50 of the 73 mares. Reflux was obtained from 1 mare that had concurrent small intestinal volvulus.

Results of a CBC performed at the time of initial examination were available for 50 mares. Median WBC count was 9,105 cells/µL (range, 1,200 to 25,000 cells/µL). Median neutrophil count was 7,880 neutrophils/µL (range, 1,080 to 23,250 neutrophils/µL), and median band neutrophil count was 194 band neutrophils/µL (range, 0 to 1,534 band neutrophils/µL). Results of CBCs performed during hospitalization were available for 56 mares. Twenty-one of the 56 (38%) mares were leukopenic, with a median WBC count of 2,850 cells/µL (range, 640 to 4,910 cells/µL). In all but 2 mares, the leukopenia resolved within 3 days. The 2 mares in which leukopenia was slower to resolve developed complications including colitis and uterine incision dehiscence following cesarean section.

Treatment of mares with PPH was intended to restore cardiovascular volume, aid in hemostasis, provide antimicrobial prophylaxis, reduce the effects of endotoxemia, and provide analgesia and sedation. Hemostatic treatments (**Table 3**) included administration of aminocaproic acid, blood transfusions, or plasma transfusions. Sixty-seven of the 73 (92%) mares were given aminocaproic acid. The standard dosing regimen consisted of IV administration of a loading dose of 20 g of aminocaproic acid mixed in 1 L of isotonic fluids followed by IV administration of 10 g mixed in 10 L of isotonic fluids every 6 hours. Treatment was continued until there was a clinical impression that hemostasis had been achieved. Blood transfusions were administered to 33 (45%) mares. The decision to administer a blood transfusion was made when the mare was estimated to have lost ≥ 25% of total blood volume or had rapid, uncontrolled hemorrhage. Prior to transfusion, mares were cross-matched, and donor blood was collected in glass bottles containing acid-citrate-dextrose. If tolerated by the patient, the transfusion was administered at a flow rate of 8 L over 40 minutes or approximately 20 to 30 mL/kg/h (9.1 to 13.6 mL/lb/h). If this high flow rate was not tolerated by the patient (ie, hives, tachycardia, or sweating was seen), the rate was reduced. Plasma was administered to 11 (15%) mares. Other hemostatic treatments administered to small numbers of mares included administration of 10% formalin (30 mL of 10% formalin in 1 L of isotonic fluids, IV, q 12 to 24 h; 1 mare), conjugated estrogens (0.05 to 0.1 mg/kg [0.022 to 0.045 mg/lb], IV, q 12 to 24 h; 2 mares), and yunan paiyao (8 mg/kg [3.7 mg/lb], PO, q 6 h; 2 mares).

Sixty-nine of the 73 (95%) mares received either lactated Ringer's solution or another polyionic solution; polyionic solutions were administered IV via a 14-gauge catheter in 10- to 20-L boluses every 6 hours. Hypertonic saline (7.5% NaCl) solution (2 to 4 mL/kg [0.9 to 1.8 mL/lb], IV bolus) was used as resuscitative fluid treatment in 24 (33%) mares. Five (7%) mares were given a hemoglobin-based oxygen-carrying solution[d] (3 mL/kg [1.36 mL/lb], IV), and 4 (5%) were given hetastarch (10 mg/kg [4.5 mg/lb], IV).

Sixty-seven (92%) mares received antimicrobials while hospitalized, including 53 mares treated with potassium penicillin (22,000 U/kg [10,000 U/lb], IV, q 6 h) and gentamicin (6.6 mg/kg [3 mg/lb], IV, q 24 h), penicillin (22,000 U/kg, IV, q 6 h) and enrofloxacin (7.5 mg/kg [3.4 mg/lb], PO, q 24 h), ceftiofur (2.2 mg/kg [1 mg/lb], IV, q 12 h) and gentamicin (6.6 mg/kg, IV, q 24 h), ceftiofur alone (2.2 mg/kg, IV, q 12 h), or trimethoprim-sulfonamide (30 mg/kg [13.6 mg/lb], PO, q 12 h). Fourteen mares were also treated with metronidazole (15 mg/kg [6.8 mg/lb], PO, q 8 h).

Seventy-two of the 73 (99%) mares received flunixin meglumine (1.1 mg/kg [0.5 mg/lb], IV, q 12 h) for analgesia. In addition, 22 (30%) mares received morphine (0.22 mg/kg [0.1 mg/lb], IV, q 24 h), and 7 (10%) received xylazine (0.17 mg/kg [0.077 mg/lb], q 24 h), 2% mepivacaine hydrochloride (5 to 7 mL), or morphine (0.1 mg/kg [0.045 mg/lb], q 6 to 24 h) by means of caudal epidural administration.

Sedation was used to control mares with clinical signs of pain or agitation related to separation from the

Table 3—Treatments administered to 73 horses with PPH.

Treatment	No. of mares that survived	No. of mares that died	Median	Range
Hemostatic treatment				
Aminocaproic acid (No. of doses)	57	10	6.0	1–17
Blood (L)	25	8	8.0	2–12
Plasma (L)	8	3	3.0	1–5
Crystalloid or colloidal fluids				
Polyionic fluids (L)	59	10	60	5–455
Hypertonic saline solution (L)	16	9	1.5	1–3
Hetastarch (L)	3	1	4.5	4–5
Oxygen-carrying solution (mL)	4	1	NA	NA
Other				
Naloxone	18	6	1	1
Oxytocin	13	1	NA	NA

NA = Not applicable.

foal. Twenty-two (30%) mares did not require any sedation. The remaining 51 (70%) mares were given 1 to 12 doses of a sedative while hospitalized. Acepromazine (0.02 to 0.04 mg/kg [0.009 to 0.018 mg/lb], IV, q 6 to 12 h) was used in 13 mares. Butorphanol (0.02 to 0.1 mg/kg [0.009 to 0.045 mg/lb], IV or IM, q 6 to 12 h) was administered in 6 mares. Xylazine (0.5 to 1.1 mg/kg [0.23 to 0.5 mg/lb], IV, q 6 to 12 h) was used in 1 mare. Detomidine (0.01 to 0.02 mg/kg [0.004 to 0.009 mg/lb], IV or IM, q 6 to 12 h) was used in 3 mares. Combinations of these medications were given to 27 mares.

Other treatments were administered to reduce the effects of endotoxemia (pentoxyphylline and polymyxin B), attenuate the effects of cardiovascular shock (naloxone), or aid in the expulsion of retained fetal membranes (oxytocin). Thirty-two (44%) mares received pentoxyphylline (7.5 mg/kg [3.4 mg/lb], PO, q 12 h), and 4 (6%) received polymyxin B (6,000 U/kg [2,700 U/lb], IV, q 8 h). Twenty-four (33%) mares received naloxone (0.03 to 0.08 mg/kg [0.014 to 0.036 mg/lb], IV, once). Fourteen (19%) mares received oxytocin (20 units, IM, q 6 h until membranes were passed).

Six (8%) mares underwent general anesthesia for abdominal exploration. One mare had a concurrent small intestinal volvulus, and another mare developed cecal necrosis 3 days after PPH was diagnosed. In the remaining 4 mares (3 with postpartum hemorrhage and 1 with prepartum hemorrhage), abdominal exploration ruled out the presence of a gastrointestinal tract lesion and confirmed the diagnosis of PPH. Two of the mares with postpartum hemorrhage died during recovery from anesthesia; the mare with prepartum hemorrhage aborted during recovery.

Median hospitalization time for the 73 mares was 5 days (range, 1 to 30 days). Sixty-one (84%) survived and 12 (16%) died or were euthanatized. Thirteen of the 61 (21%) mares that survived did not have any complications; complications identified in the remaining surviving horses included fever of > 24 hours' duration (n = 27 [44%]), increased digital pulses (13 [21%]), retained fetal membranes (7 [11%]), cardiac arrhythmia (6 [10%]), thrombophlebitis (6 [10%]), signs of colic that persisted for > 24 hours (5 [8%]), stranguria alleviated by means of urinary catheterization (5 [8%]), radiographically evident laminitis (5 [8%]), lower limb edema (5 [8%]), vaginal discharge (4 [7%]), colitis (3 [5%]), peritonitis (3 [5%]), transfusion reaction (2 [3%]), ataxia (1 [2%]), seizures (1 [2%]), blindness (1 [2%]), coagulopathy (1 [2%]), and dehiscence of the uterine incision (1 [2%]). Fifty-seven of the 61 mares that survived and were discharged were being treated with trimethoprim-sulfonamide (30 mg/kg, PO, q 12 h) at the time of discharge.

Information on subsequent fertility was available for 53 of the 61 mares that survived. Twenty-seven of the 53 (51%) mares had not produced another foal (live or dead) by the end of the follow-up period, whereas 26 (49%) did, including 10 mares that produced a single foal, 6 mares that each produced 2 foals, 3 mares that each produced 3 foals, 2 mares that each produced 4 foals, 2 mares that each produced 5 foals, 2 mares that each produced 6 foals, and 1 mare that produced 7 foals. Six of the 26 mares that eventually produced foals were reportedly barren for 1 to 3 years after the episode of PPH despite known breeding attempts. It was not known whether any of the surviving mares had a recurrence of PPH.

Table 4—Results of bivariate analyses of associations between various factors and outcome (survived vs died or euthanized) in 73 horses with PPH.

Factor	Odds ratio	95% CI	*P* value
Older than median age	1.23	0.33–4.61	0.763
Multiparous	2.23	0.36–13.73	0.382
Prepartum hemorrhage	0.25	0.06–1.08	0.054
Dystocia	1.34	0.31–5.68	0.696
Recumbency	0.61	0.11–3.44	0.576
Rectal palpation	1.61	0.45–5.81	0.470
Location of hemorrhage			
Abdomen	1.14	0.33–3.91	0.833
Left broad ligament	1.20	0.29–5.05	0.805
Right broad ligament	0.30	0.036–2.54	0.249
Uterine lumen	0.00	0.01–2.88	0.096
Uterine wall	1.38	0.25–7.54	0.714
Vagina	26.30	1.18–584.57	0.022
Hypothermia			
At initial examination	1.85	0.47–7.32	0.382
While hospitalized	1.71	0.12–17.63	0.650
Hyperthermia			
At initial examination	0.65	0.03–13.51	0.431
While hospitalized	3.00	0.55–16.26	0.191
Tachycardia			
At initial examination	25.43	1.14–565.44	0.003
While hospitalized	8.83	2.15–36.25	0.001
Tachypnea			
At initial examination	1.02	0.19–5.47	0.980
While hospitalized	1.68	0.28–9.95	0.568
Anemia			
At initial examination	1.35	0.14–13.37	0.798
While hospitalized	0.31	0.03–2.82	0.280
Hypoproteinemia			
At initial examination	1.56	0.28–8.72	0.616
While hospitalized	3.91	0.69–22.09	0.107

For each factor, the odds ratio represents the odds that a mare with the stated factor would die or be euthanized, relative to the odds for a mare without the stated factor.
CI = Confidence interval.

A necropsy was performed on 11 of the 12 mares that died or were euthanatized because of PPH. Eight mares were determined to have had a rupture of the uterine artery (right uterine artery in 4 mares and left uterine artery in 2; side was not specified in the other 2). The other 3 mares died of rupture of the adrenal, external iliac, and vaginal arteries.

Of the variables studied, only tachycardia at the time of admission, tachycardia during hospitalization, and hemorrhage localized to the vagina were significantly associated with outcome (**Table 4**).

Discussion

Results of the present study indicated that PPH can develop in mares of any age or parity. The prognosis with medical treatment was good, with 61 of 73 (84%) mares surviving and 26 of 53 (49%) mares that survived and for which follow-up information was available subsequently producing a foal.

The population in the present study consisted of mares examined at a single referral practice, and results

may not be applicable to mares from other populations. In particular, the severity of the disease process in this group of horses may not be reflective of the true spectrum of disease associated with PPH. In particular, mares with mild forms of PPH that were treated on the farm and not referred would have been excluded from the study, as would have been mares with severe forms of PPH that were found dead or did not survive transport to the hospital. Thus, mares with the most and least severe forms of PPH may have been excluded from the study, and this fact should be taken into account when interpreting the results and conclusions.

Our findings demonstrated that broodmares of any age and parity were susceptible to PPH. Median age of affected mares in the present study was 14 years, which was similar to age reported in another study.[18] Sixty of 66 (91%) mares in the present study were multiparous, and 52 of 66 (79%) had previously delivered > 4 foals. Although age and parity were not significantly associated with outcome, no attempts were made in the present study to identify factors associated with development of PPH.

Many mares in the present study were initially examined because of signs of colic or cardiovascular shock, and development of similar clinical signs during the periparturient period should prompt consideration of PPH as a possible cause. However, there were a number of mares in the present study in which the initial complaint seemed unrelated to the reproductive tract. Subjectively, these appeared to be mares in which PPH developed > 24 hours after foaling, and most of these mares eventually developed signs relating to hypovolemia and hypoxia. Mares were included in the present study only if PPH developed within 60 days before or after foaling, but while reviewing medical records for the hospital, we identified a subset of mares that had signs of PPH as early as the fifth month of gestation or as late as 4 months after parturition. Our impression was that mares in which PPH was diagnosed months after parturition typically had signs of sepsis, such as fever and weight loss, consistent with an intra-abdominal abscess or peritonitis. Thus, we recommend that PPH should still be considered as a possible cause when signs of hemorrhage are observed outside of the periparturient period.

For most of the mares in the present study, results of clinicopathologic testing performed at the time of initial examination were normal. Owing to the time-dependent nature of the effects of hemorrhage on clinicopathologic values, this was not unexpected, as most mares included in the present study lived within approximately 30 miles of the hospital and were examined shortly after signs developed. In general, it was our impression that abnormalities such as tachycardia, hypothermia, hyperthermia, leukopenia, and anemia worsened over time. Of the variables studied, only tachycardia at the time of admission, tachycardia during hospitalization, and hemorrhage localized to the vagina were significantly associated with outcome, whereas other physical examination and clinicopathologic findings were not. Importantly, findings may not apply to other populations or areas where management of broodmares does not allow for early detection of PPH and rapid referral for treatment.

In our experience, mares with PPH rarely have evidence of external blood loss. Instead, hemorrhage typically occurs internally and can be associated with several locations. Hematomas within the broad ligaments and hemorrhagic peritoneal effusions are presumed to originate from the uterine artery. Other locations of hemorrhage include the placental attachment to the uterus, with intra-uterine accumulation of blood, and intramural hematomas of the uterus and vagina. In 8 of 11 horses in the present study that underwent necropsy, the source of hemorrhage was confirmed to be the uterine artery. However, the remaining 3 mares had a rupture of the adrenal, external iliac, or vaginal artery. In the present study, having hemorrhage localized to the vagina was significantly associated with outcome; however, the low number of mares (n = 2) with hemorrhage in this location casts uncertainty over the interpretation of this result.

Establishing a diagnosis of PPH can be challenging because of the frequency of nonspecific clinical signs, the difficulties associated with diagnostic imaging of the abdomen in periparturient mares, and the time delay required until clinicopathologic abnormalities characteristic of hemorrhage develop. Although we did not specifically examine whether time to treatment was associated with outcome in the present study, it is our impression that delays in treatment can result in a fatal outcome. In our review of the medical records, it appeared that attending clinicians typically depended on the historical time frame in relationship to foaling, clinical signs, ultrasonographic evidence of hemorrhage, and clinicopathologic abnormalities to arrive at a diagnosis of PPH. Although the possibility of potentiating hemorrhage has been cited as a reason to avoid use of invasive diagnostic procedures, such as abdominocentesis and transrectal ultrasonography, such procedures were commonly performed on mares in the present study, and importantly, whether rectal palpation was performed was not significantly associated with outcome. For 2 mares in the present study, the diagnosis of PPH was complicated by the presence of concurrent gastrointestinal tract disease (small intestinal volvulus and cecal necrosis).

Medical management of mares in the present study was relatively uniform, with therapeutic efforts aimed at enhancing cardiovascular volume and perfusion (eg, administration of polyionic fluids), obtaining hemostasis (eg, administration of aminocaproic acid and blood transfusions), controlling signs of pain and agitation, and preventing endotoxemia. The use of hetastarch in 4 mares was inappropriate because of hetastarch's potential negative effects on coagulation; however, these mares were initially believed to have primary gastrointestinal tract abnormalities. Because treatment was not randomly applied, it is inappropriate to draw conclusions about the efficacy of individual treatment modalities, and further study is needed to identify the most efficacious treatments in mares with PPH.

Overall, 61 of the 73 (84%) mares in the present study survived, including 6 of the 10 mares with prepartum hemorrhage and 55 of the 63 (87%) mares with postpartum hemorrhage. In the present study, timing of hemorrhage (prepartum vs postpartum) was not signif-

icantly associated with outcome. In contrast, in a previous study,[17] none of the horses with prepartum PPH survived. We attribute the successful treatment of mares with prepartum hemorrhage in the present study to early recognition of hemorrhage, close proximity of the referral center, and aggressive medical management.

Our findings illustrate that mares that survive an episode of PPH may still be fertile, in that 26 of 53 (49%) mares for which follow-up information was available were found to have produced a foal after recovering from PPH. Records for the 27 surviving mares that did not produce a foal by the end of the follow-up period indicated that at least 6 of these mares had been bred but failed to conceive or produce a live foal. However, we were unable to determine whether the remaining 21 mares had ever been bred again or, for mares that had not been bred, whether this was because of presumed infertility, the potential for recurrence, or some other reason. The lack of information regarding historical difficulties with pregnancy and parturition and our inability to obtain breeding information following recovery prevented us from determining the risk of recurrence of PPH and its effect on future fertility. Nevertheless, our findings do suggest that treatment of mares with PPH can be associated with a good prognosis for survival and a reasonable prognosis for future fertility.

a. Jockey Club Information Systems, Lexington, Ky.
b. American Trotting Horse Association, Columbus, Ohio.
c. SAS, version 9.1, SAS Institute Inc, Cary, NC.
d. Oxyglobin, Biopure Corp, Cambridge, Mass.

References

1. McCarthy PF, Hooper RN, Carter GK, et al. Postparturient hemorrhage in the mare: managing ruptured arteries of the broad ligament. *Vet Med* 1994;89:147–152.
2. Dwyer R. Post partum deaths of mares. *Equine Dis Q* 1993;2:5.
3. Pascoe RR. Rupture of the utero-ovarian or middle uterine artery in the mare at or near parturition. *Vet Rec* 1979;104:77.
4. Rossdale PD. Differential diagnosis of post parturient haemorrhage in the mare. *Equine Vet Educ* 1994;6:135–136.
5. Rooney JR. Internal hemorrhage related to gestation in the mare. *Cornell Vet* 1964;54:11–17.
6. Zent WW. Postpartum complications. In: Robinson WB, ed. *Current therapy in equine medicine*. 2nd ed. Philadelphia: WB Saunders, 1987;544–547.
7. Rooney JR, Robertson JL. Female reproductive system. In: Rooney JR, Robertson JL, eds. *Equine pathology*. Ames, Iowa: Iowa State University Press, 1996;237.
8. Loftstedt RM. Haemorrhage associated with pregnancy and parturition. *Equine Vet Educ* 1994;6:138–141.
9. Britt BL. Postpartum hemorrhage. In: Robinson WB, ed. *Current therapy in equine medicine*. 5th ed. Philadelphia: WB Saunders, 2003;327–330.
10. Frazer GS. Post partum complications in the mare. Part 1: conditions affecting the uterus. *Equine Vet Educ* 2003;15:45–54.
11. Pusterla N, Fecteau ME, Madigan JE, et al. Acute hemoperitoneum in horses: a review of 19 cases (1992–2003). *J Vet Intern Med* 2005;19:344–347.
12. Saxon WD. The acute abdomen. *Vet Clin North Am Small Anim Pract* 1994;24:1207–1224.
13. Frazer G, Burba D, Paccamonti D, et al. The effects of parturition and peripartum complications on the peritoneal fluid composition of mares. *Theriogenology* 1997;48:919–931.
14. Lofstedt RM. Miscellaneous diseases of pregnancy and parturition. In: McKinnon AO, Voss JL, eds. *Equine reproduction*. Philadelphia: Lea & Febiger, 1993;596–603.
15. LeBlanc MM. Diseases of the reproductive tract: the mare. In: Colahan PT, ed. *Equine medicine and surgery*. Goleta, Calif: American Veterinary Publications, 1991;596–603.
16. Asbury AC. The reproductive system. In: Mannsman RA, McAllister ES, eds. *Equine medicine and surgery*. 3rd ed. Philadelphia: Lea & Febiger 1982;1305–1367.
17. Dechant JE, Nieto JE, LeJeune SS. Hemoperitoneum in horses: 67 cases (1989–2004). *J Am Vet Med Assoc* 2006;229:253–259.
18. Dolente BA, Sullivan EK, Boston R, et al. Mares admitted to a referral hospital for postpartum emergencies: 163 cases (1992–2002). *J Vet Emerg Crit Care* 2005;15:193–200.