Acute hemorrhagic pulmonary infarction and necrotizing pneumonia in horses: 21 cases (1967–1993)

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Objective—To characterize history, clinical signs, and pathologic findings in horses with histologically confirmed acute hemorrhagic pulmonary infarction and necrotizing pneumonia.

Design—Retrospective study.

Animals—21 horses.

Results—19 of the 21 horses were Thoroughbred racehorses in training. Eighteen horses had had strenuous exercise immediately prior to onset of illness. Fifteen horses had a serosanguineous nasal discharge during hospitalization. Seventeen horses had radiographic evidence of pulmonary consolidation and pleural effusion. Nine of 14 horses had ultrasonographic evidence of large pulmonary parenchymal defects consistent with consolidation. Pleurocentesis yielded a suppurative, serosanguineous effusion in the 14 horses in which it was performed. Bacteria were isolated from all transtracheal aspirates (14) and from 6 of 12 pleural fluid samples. *Actinobacillus suis*-like organisms and *Streptococcus equi* subsp *zooepidemicus* were most commonly isolated. Nineteen horses were hospitalized and treated. Mean duration of treatment was 5 days, and most horses were euthanatized because of secondary complications, continued costs of medical treatment, or poor prognosis for future performance. Pathologic lesions included well-demarcated regions of hemorrhagic pulmonary infarction with necrosis and a serosanguineous pleural effusion. Thrombosis of pulmonary vessels was found in 11 horses.

Clinical Implications—An acute or peracute onset of severe respiratory distress, with serosanguineous nasal discharge, ultrasonographic and radiographic evidence of severe pulmonary consolidation, and serosanguineous suppurative pleural effusion, is strongly suggestive of pulmonary infarction in horses. Horses with pulmonary infarction responded poorly to conventional treatment for pleuropneumonia and had a poor prognosis for recovery. (*J Am Vet Med Assoc* 1997;210:1774–1778)

Bacterial pleuropneumonia is a well-recognized disease in horses. Predisposing causes of bacterial pleuropneumonia include viral infection, stressful events (eg, transportation, poor ventilation, strenuous exercise, or general anesthesia), thoracic trauma, exercise-induced pulmonary hemorrhage (EIPH), neoplasia, and aspiration.[1-3] Common clinical signs during the acute stages of illness include pyrexia, mucopurulent nasal discharge, respiratory distress, cough, pleurodynia, and toxic mucous membranes. Initial retrospective studies have documented survival rates of 38 and 43%, although later studies have reported survival rates up to 90%.[4-6] The most common pathologic findings at necropsy include pneumonia, pulmonary abscesses, and pleuritis.[7] In our practice, we recently recognized a subset of horses with pleuropneumonia that also had evidence of hemorrhagic pulmonary infarction, necrotizing pneumonia, and pulmonary thromboembolism. These horses responded poorly to conventional treatment for pleuropneumonia and had a low survival rate. The purposes of the study reported here were to characterize history, clinical signs, and pathologic findings in this subset of horses.

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Criteria for Selection of Cases

Medical records of all horses admitted to the University of California Veterinary Medical Teaching Hospital between 1967 and 1993 were reviewed. Horses were included in the study if, at necropsy, they had evidence of hemorrhagic pulmonary infarction with or without evidence of pulmonary thromboembolism. Horses were excluded if they had developed pulmonary thromboembolism secondary to some other primary illness such as disseminated intravascular coagulation or jugular thrombosis, if they had a chronic underlying pulmonary disease, or if documented evidence of thoracic or esophageal trauma was found. Information obtained from medical records of horses included in the study consisted of signalment, history, clinical findings at admission, progression of clinical signs and clinicopathologic abnormalities, results of thoracic radiography, and pathologic findings at necropsy.

Results

Twenty-one horses met the criteria for inclusion in the study. Nineteen were Thoroughbreds, and 2 were Standardbreds. Five were sexually intact males, 8 were geldings, and 8 were sexually intact females. Horses ranged from 2 to 19 years of age, but 13 of the 21 were between 2 and 4 years of age.

History prior to onset of illness was available for 20 horses. Nineteen were racehorses in training. Of these, 17 had raced or had had a strenuous workout within 7 days prior to onset of illness, 1 had been transported immediately prior to onset of clinical signs and 1 had raced and was transported within 7 days

prior to onset of illness. The remaining horse for which history was available was a pleasure horse without any history of strenuous exercise or transportation prior to onset of clinical signs.

Initial clinical findings reported by referring veterinarians were available for 19 horses and included a febrile episode within 24 hours after transportation or exercise (19/19) and a bloody nasal discharge within the first 24 hours after onset of clinical signs (10/19). Three additional horses developed a bloody nasal discharge within 3 days after strenuous exercise or transportation. Mean duration of illness prior to admission to the veterinary medical teaching hospital was 5 days (range, 4 hours to 3 weeks). Reasons for referral, when reported, included poor response to conventional treatment for pleuropneumonia and deterioration of the horse's condition.

At initial examination, 15 of the 21 horses had a serosanguineous nasal discharge. This discharge often recurred while horses were hospitalized. Only 2 horses, both of which had been ill for 2 to 3 weeks, had a mucopurulent nasal discharge at the time of admission. Other common clinical findings at admission included tachypnea (respiratory rate > 20 breaths/min; 16/21), tachycardia (heart rate > 44 beats/min; 15/21), and toxic appearing mucous membranes (15/21). Thirteen horses were febrile (rectal temperature > 38.3 C [101 F]) at the time of admission, despite recent treatment with nonsteroidal anti-inflammatory drugs in 6. Eleven horses had coughs, and 4 had petechial hemorrhages on the nasal mucous membranes. Twelve horses had complete or partial anorexia at admission or at some time during hospitalization. Abnormal lung sounds were detected on auscultation of the thorax in all horses. Seventeen horses had ventral dullness, consistent with pleural effusion or consolidation.

Eleven horses were subjectively judged to be severely ill (ie, evidence of severe endotoxemia, dehydration, pleurodynia, and continuous serosanguineous nasal discharge) at the time of admission, and 8 of these 11 were examined at the teaching hospital within the first 4 days of illness. Seven horses were judged to be less severely ill and in stable condition at the time of admission, and 6 of 7 had been ill for ≥ 1 week. The remaining 3 horses could not be subjectively classified into these 2 main categories and had been ill for various times prior to admission.

Thoracic radiography was performed on 18 horses at the time of admission. Seventeen had radiographic evidence of ventral consolidation or pleural effusion. Eleven had well-circumscribed soft-tissue opacities in the lung fields, consistent with a mass, abscess, or infarction of the pulmonary parenchyma (Fig 1). The most common sites for these well-circumscribed lesions were the mid-caudal (8), caudodorsal (4), caudoventral (1), and cranioventral (1) lung fields. Although absence of typical radiographic findings cannot be used to rule out the possibility that horses had EIPH, only 3 horses had the classic radiographic changes associated with EIPH.[8]

Thoracic ultrasonography was performed on 14 horses. Typical findings were pleural effusion (14), pleuritis with fibrin tags visible on the parietal pleural surface (10), and pulmonary parenchymal defects consistent with consolidation or pneumonia (9). Five of 6 horses had ultrasonagraphic evidence of large parenchymal lesions that appeared to have been hepatized. These lesions were subsequently found at necropsy to correspond to areas of hemorrhagic pulmonary infarction.

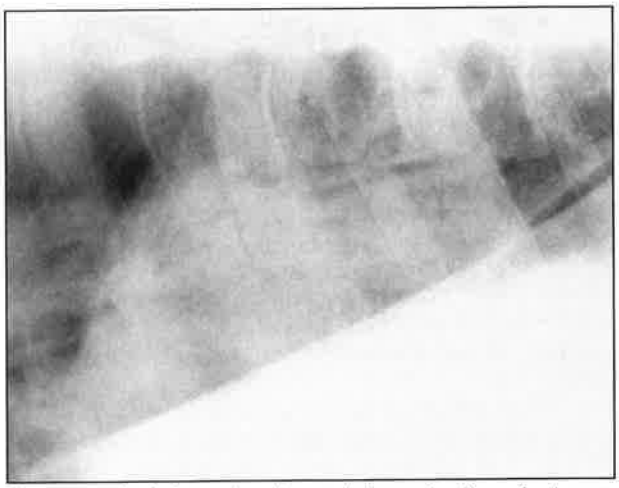

Figure 1—Left lateral radiographic projection of a horse with clinical signs of acute hemorrhagic pulmonary infarction and necrotizing pneumonia. A large, well-circumscribed opacity was seen in the ventral lung field. An area of pulmonary infarction was found in this region at necropsy.

A CBC was performed on 20 horses. A high serum fibrinogen concentration (> 400 mg/dl) was found in 18 horses, and toxic neutrophils with a degenerative left shift were found in 9. Four horses had neutropenia, and 11, 6 of which had been sick ≥ 4 days prior to examination, had neutrophilia. Mean serum total protein concentration was 6.6 g/dl (range, 5.5 to 8.3 g/dl). Platelet counts were normal (100,000 to 350,000/ml) in all horses except 1, which had a high platelet count. Abnormalities were detected in 5 of 6 horses on which coagulation panels were performed and included prolonged prothrombin time (3) and partial thromboplastin time (5) and a high concentration of fibrin degradation products (4).

Pleurocentesis was performed on 13 horses and yielded a serosanguineous suppurative effusion in all 13. Pleural fluid from 12 horses was examined cytologically; WBC count ranged from 3,900 to 66,000/ml, and RBC count ranged from 0.0028 to 1.4 × 10^6/ml. Most of the nucleated cells were neutrophils (range, 63 to 97%). Pleural fluid total protein concentration ranged from 3.9 to 5.7 g/dl, and bacteria were seen in pleural fluid from 3 of 12 horses.

Bacterial culture of pleural fluid was performed in 12 cases. *Actinobacillus suis*-like organisms were isolated from 6 horses, *Streptococcus equi* subsp *zooepidemicus* from 1, and *Staphylococcus* sp from 1. Bacterial growth was not obtained from samples collected from 6 horses, 3 of which were known to be receiving antimicrobials at the time of sample collection.

Transtracheal wash was performed on 14 horses. Fluid from 13 horses was evaluated cytologically and

was indicative of a septic suppurative process in 12 of the 13. Fluid from all 14 horses was submitted for bacterial culture, and bacteria were isolated from all samples. Most commonly isolated were *A suis*-like organisms (9/14) and *S equi* subsp *zooepidemicus* (8/14). Other organisms isolated from transtracheal wash samples included *Klebsiella pneumoniae* (2), *S viridans* (2), *Escherichia coli* (2), *Pasteurella* sp (2), *Bacteroides* sp (2), *Clostridium* sp (1), and *Micrococcus* sp (1). Multiple organisms were cultured from 9 horses, and 2 of the 9 had a mixed anaerobic/aerobic infection. Of the 14 horses on which a transtracheal wash was performed, 10 had previously been treated with antibiotics.

When results of bacterial cultures of pleural fluid and transtracheal wash samples were considered together, 2 of 16 horses were infected with anaerobic bacteria (both were also infected with aerobic bacteria), 10 were infected with *A suis*-like organisms (7/10 had polymicrobic infections), and 8 were infected with *S equi* subsp *zooepidemicus* (7/8 had polymicrobic infections).

Two horses were euthanatized shortly after initial examination. Treatment for the 19 horses that were hospitalized included administration of broad-spectrum antibiotics (19), nonsteroidal anti-inflammatory drugs (17), acepromazine maleate (2), hyperimmune plasma (2), and sodium iodide (1). One horse died within 4 hours after initial examination; the other 18 were euthanatized (mean duration of hospitalization, 5 days). Reasons reported for euthanasia included secondary complications such as laminitis (7), colitis (1), and myonecrosis (1), as well as cost for continued medical treatment (6) and a poor prognosis for future performance (8).

On gross examination at necropsy, the pulmonary parenchyma appeared to be diffusely congested in all horses. Serosanguineous pleural effusion with fibrin tags and adhesions in various stages of maturation and severity were also detected. There was a sharp line of demarcation between grossly normal and grossly abnormal lung, which was palpably firm and dark red to black on the cut surface (Fig 2). Thrombosis of large vessels supplying these lesions was found during gross examination in 9 horses. Chronic lesions were usually tan to brown and had a friable necrotic center surrounded by a fibrous capsule. Three horses had well-circumscribed abscesses within the pulmonary parenchyma. In 1 horse that had been ill for several months, a piece of necrotic lung was found free within the pleural cavity.

Common histologic lesions were parenchymal hemorrhage, necrosis, demarcation or encapsulation (depending on chronicity), inflammation, sepsis, and pleuritis. Thrombosis of major pulmonary vessels was detected histologically in 2 horses, in addition to the 9 in which pulmonary thrombosis was evident during gross examination of the lung. Failure to find thrombotic lesions in the remaining horses may have been related to inadequate dissection of the circulation to all these lesions. Thrombosis involved arteries in 10 horses and veins in 1. Pulmonary infarction more frequently involved the right (20/21 horses) than the left (10/21) lung. In the right lung, gross lesions were distributed in the cranial (14/28) and caudal (14/28) lobes. In the left lung, gross lesions were distributed more frequently in the cranial (7/12) than caudal (5/12) lobes.

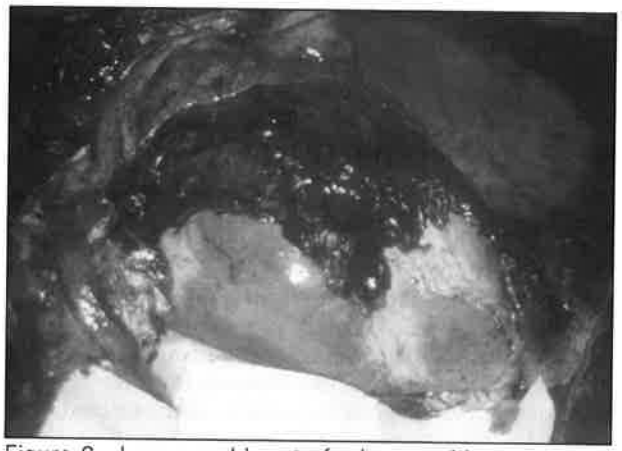

Figure 2—Lungs and heart of a horse with acute hemorrhagic pulmonary infarction and necrotizing pneumonia. In the right cranial lung lobe, there was a line of demarcation between grossly normal and grossly abnormal lung tissue. Abnormal lung tissue was firm and dark red on the cut surface.

Discussion

To our knowledge, hemorrhagic pulmonary infarction has not previously been reported in horses. In human beings, pulmonary embolism is a common pathologic finding with pulmonary infarction being a relatively rare sequela.[9] Criteria for antemortem diagnosis of pulmonary embolism and infarction in human patients include radiographic evidence of pulmonary infiltrates that persist for several days to weeks, angiographic evidence of pulmonary vascular occlusion, pleuritic pain, hemoptysis, dyspnea, cough, and pleural friction rubs. Although pulmonary angiography is not routinely done in horses, most of the horses in this study met ≥ 2 of these criteria. In human beings, a predisposition to pulmonary infarction is seen with septic emboli, pulmonary hypertension, and polycythemia.[10-12] Three horses in this study had a walled-off pulmonary abscess, which could have initiated septic embolization. Bacteria were isolated from all horses on which a transtracheal wash was performed. Whether bacterial infection was the cause of hemorrhagic pulmonary infarction and necrotizing pneumonia in these horses or merely a reflection of secondary bacterial colonization of severely compromised, infarcted lung remains unresolved. The most commonly isolated bacteria were *S equi* subsp *zooepidemicus* and *A suis*-like organisms. These were also the most commonly isolated organisms from horses with bacterial pleuropneumonia without pulmonary infarction in our hospital.[13]

Lesions in these horses were strikingly similar to those reported in association with acute *A pleuropneumoniae* infection in swine.[14] *Actinobacillus pleuropneumoniae* has several known virulence factors, including lipopolysaccharide, capsular antigen, hemolysins, and cytolysins.[15] Lung lesions similar to the natu-

rally occurring lesions in pigs infected with *A pleuropneumoniae* can be experimentally produced by administering culture supernatant containing exotoxins of this organism. *Actinobacillus suis*-like organisms are closely related to *A pleuropneumoniae*, and highly virulent strains of this organism may play an important role in this syndrome. Although *A suis*-like organisms were isolated from transtracheal wash samples from only 9 of the 14 horses, 10 of the 14 were being treated with antibiotics before samples were obtained. Concurrent antimicrobial administration may have affected our ability to isolate this and other organisms. Surprisingly, despite appropriate broad-spectrum antimicrobial treatment in 10 horses, organisms considered to be primary respiratory pathogens were isolated from transtracheal wash samples of all 10.

Eighteen horses in this study had a history of strenuous exercise immediately prior to the onset of illness. A reduction in pulmonary macrophage phagocytic ability and respiratory burst activity has been documented in horses undergoing strenuous exercise, and this may have contributed to the development of pulmonary infection.[16,17] Shipping fever (pneumonic pasteurellosis) in cattle is recognized to result from the synergistic effects of stress (most commonly, transportation), viral infection, and immunosuppression, followed by secondary invasion by bacteria. A similar scenario may be occurring in this population of horses, with the initiating stressful event being strenuous exercise. Viral isolation and serologic tests were not performed, making it difficult to determine the role that viral infection may have played. Arguments against the theory that stress and viral infection played a role include the rapid onset of illness and the fact that cases were sporadic. In addition, *S equi* subsp *zooepidemicus* and *A suis*-like organisms have been reported to be primary pathogens in horses.[18-22]

Transient polycythemia, with Hct in excess of 60%, has been detected in horses exercising at high intensity and is attributed to mobilization of splenic erythrocyte reserves.[23] An increase in blood viscosity associated with this elevation in Hct could result in slowing of blood flow and increased turbulence, thus predisposing to clot formation.[23] Furosemide, commonly used prior to racing to prevent EIPH, is also known to cause an increase in blood viscosity in exercising horses, perhaps further compounding the potential for infarction.[24]

Marked pulmonary hypertension has been documented in horses galloping on a treadmill, with pressures of 120 and 70 mm of Hg recorded in the pulmonary artery and left atrium, respectively.[25] The high pulmonary capillary pressures could, in turn, result in stress failure of the capillary walls.[26] The resulting endothelial damage could trigger vascular thrombosis and hemorrhagic infarction. The cause of EIPH is still under debate, but it is possible that factors important in the development of EIPH may also be important in the development of acute hemorrhagic pulmonary infarction.

In this study, 5 of 6 horses had evidence of a coagulopathy. Thromboembolism in these horses could have been a result of coagulopathy; however, coagulopathies have also been reported to be a sequela of gram-negative endotoxemia,[27] and more than half of the horses in this series had clinical signs consistent with endotoxemia. Thus, it is difficult to determine whether coagulopathies in these horses were a result of overwhelming endotoxemia or a primary factor in the initiation of the disease process.

The most common site of well-circumscribed, soft-tissue opacities identified radiographically (mid-caudal lung fields) was not the same as the most common site of pulmonary infarctions found during post mortem examination (cranial lung lobes). This discrepancy was thought to be a result of shadowing of cranioventral lesions by the cardiac silhouette or pleural fluid during radiography.

Although the definitive diagnosis of pulmonary infarction in these horses was made at necropsy, the history, clinical signs, and progression of signs were considered to be sufficiently consistent to allow a tentative clinical diagnosis. An acute or peracute onset of severe respiratory distress and a serosanguineous nasal discharge were typical findings. Ultrasonographic and radiographic findings of severe pulmonary consolidation and pleural effusion along with serosanguineous, suppurative pleural fluid were also strongly suggestive of pulmonary infarction. Although horses with these signs have been treated successfully at the teaching hospital, it is our experience that horses with hemorrhagic pulmonary infarction and necrotizing pneumonia responded poorly to conventional treatment and had a poor prognosis for recovery.

References

1. Sweeney CR. Pleuropneumonia. In: Smith BP, ed. *Large animal internal medicine*. St Louis: CV Mosby Co, 1996;577–580.
2. Raidal SL. Equine pleuropneumonia. *Br Vet J* 1995;151:233–262.
3. Chaffin MK, Carter GK. Equine bacterial pleuropneumonia. Part I. Epidemiology, pathophysiology, and bacterial isolates. *Compend Contin Educ Pract Vet* 1993;15:1642–1650.
4. Raphel CF, Beech J. Pleuritis secondary to pneumonia or lung abscessation in 90 horses. *J Am Vet Med Assoc* 1982;181:808–810.
5. Sweeney CR, Divers TJ, Benson CE. Diseases of the lung: diagnostic approach and management of horses with anaerobic pleuropneumonia, in *Proceedings*. Am Assoc Equine Pract Annu Meet 1984;30:262–273.
6. Seltzer KL, Byars TD. Prognosis for return to racing after recovery from infectious pleuropneumonia in Thoroughbred racehorses: 70 cases (1984–1989). *J Am Vet Med Assoc* 1996;208:1300–1301.
7. Chaffin MK, Carter GK, Byars TD. Equine bacterial pleuropneumonia. Part III. Treatment, sequela, and prognosis. *Compend Contin Educ Pract Vet* 1994;16:1585–1595.
8. Pascoe JR, O'Brien TR, Wheat JD, et al. Radiographic aspects of exercise-induced pulmonary hemorrhage in racing horses. *Vet Radiol* 1983;24:85–92.
9. Smith GT, Dexter L, Dammin GJ. Post mortem quantitative studies in pulmonary embolism. In: Sassahara AA, Stein M, eds. *Pulmonary embolic disease*. New York: Grune and Stratton, 1965;120–130.
10. Dalen JE. Clinical diagnosis of acute pulmonary embolism. In: Murpurgo M, ed. *Pulmonary embolism*. New York: Marcel Dekker Inc, 1994;55–66.
11. Mathes ME, Holman E, Reichert FL. A study of the bronchial, pulmonary and lymphatic circulations of the lung under various pathologic conditions experimentally produced. *J Thorac Surg* 1932;1:339–362.
12. Dalen JE, Haffajee CI, Alpert JS, et al. Pulmonary embo-

lism, pulmonary hemorrhage, and pulmonary infarction. *N Engl J Med* 1977;296:1431–1435.

13. Hirsh DC, Jang SS. Antimicrobic susceptibility of bacterial pathogens from horses. *Vet Clin North Am Equine Pract* 1987;3:181–190.

14. Didier PJ, Perino L, Urbance J. Porcine *Haemophilus* pleuropneumonia: microbiologic and pathologic findings. *J Am Vet Med Assoc* 1984;184:716–719.

15. Fedorka-Cray PJ, Cray WC, Gray JT, et al. *Actinobacillus (Haemophilus) pleuropneumonia*. Part II. Virulence factors, immunity, and vaccines. *Compend Contin Educ Pract Vet* 1994;16:117–125.

16. Huston LJ, Bayly WM, Liggitt HD, et al. Alveolar macrophage function in Thoroughbreds after strenuous exercise, in *Proceedings*. 2nd Int Conf Equine Exerc Physiol 1986;2:243–252.

17. Wong CW, Thompson HL, Thong YH, et al. Effect of strenuous exercise stress on chemiluminescence response of equine alveolar macrophages. *Equine Vet J* 1990;22:33–35.

18. Oikawa M, Kamada M, Yoshikawa Y, et al. Pathology of equine pneumonia associated with transport and isolation of *Streptococcus equi* subsp *zooepidemicus*. *J Comp Pathol* 1994;111:205–212.

19. Jang SS, Biberstein EL, Hirsch DC. *Actinobacillus suis*-like organisms in horses. *Am J Vet Res* 1987;48:1036–1038.

20. Samitz EM, Biberstein EL. *Actinobacillus suis*-like organisms and evidence of hemolytic strains of *Actinobacillus lignieresii* in horses. *Am J Vet Res* 1991;52:1245–1251.

21. Hayakawa Y, Komae H, Ide H, et al. An occurrence of equine transport pneumonia caused by mixed infection with *Pasteurella caballi*, *Streptococcus suis* and *Streptococcus zooepidemicus*. *J Vet Med Sci* 1993;55:455–456.

22. Wood JLN, Burrell MH, Roberts CA, et al. *Streptococci* and *Pasteurella* spp associated with disease of equine lower respiratory tract. *Equine Vet J* 1993;25:314–318.

23. Evans DL. The cardiovascular system: anatomy, physiology and adaptations to exercise and training. In: Hodgson DR, Rose RJ, eds. *The athletic horse*. Philadelphia: WB Saunders Co, 1994;129–144.

24. Geor RJ, Weiss DJ, Burris SM, et al. Effects of furosemide and pentoxifylline on blood flow properties in horses. *Am J Vet Res* 1992;53:2043–2049.

25. Jones JH, Smith BL, Birks EK, et al. Left atrial and pulmonary artery pressures in exercising horses. *FASEB J* 1992;5:A2020.

26. West JB, Mathieu-Costello O, Hones JH, et al. Stress failure of pulmonary capillaries in racehorses with exercise-induced pulmonary hemorrhage. *J Appl Physiol* 1993;75:1097–1109.

27. Hardie EM, Kruse-Elliot K. Endotoxic shock. Part I. A review of causes. *J Vet Intern Med* 1990;4:258–266.

Multi-element assay of mammary secretions and sera from periparturient mares by inductively coupled argon plasma emission spectroscopy—J. S. Rook, W. E. Braselton, R. F. Nachreiner, et al

Objective—To document and determine changes in the mineral profiles of sera and mammary secretions from a population of periparturient mares.

Animals—18 clinically normal periparturient Arabian broodmares.

Procedure—Inductively coupled argon emission spectroscopy was used to measure Ca, Cu, Fe, K, Mg, Mn, Na, P, and Zn concentrations in sera and mammary secretions of periparturient mares. In addition, S was measured in mammary secretions.

Results—Serum concentrations of Ca, Cu, Fe, K, Mg, Na, P, and Zn remained constant throughout late pregnancy and the first 7 days of lactation. Compared with values on day 11 before foaling , mammary fluid concentrations Ca, Cu, K, Mg, P, S, and Zn increased prior to parturition and all element concentrations, except Ca, decreased with the onset of lactation. In contrast, Na concentrations in mammary secretions decreased precipitously as parturition approached. Iron concentrations in mammary secretions remained relatively constant up to the time of parturition, decreased at parturition, and remained constant during lactation.

Conclusions and Clinical Relevance—Prior to foaling, increasing concentrations of Ca, Cu, K, Mg, P, S, or Zn in mammary secretions in concert with precipitous decreases in Na concentrations may provide a predictive index of impending parturition in the mare and a means of assessing fetal readiness for birth. (*Am J Vet Res* 1997;58:376–378)