Treatment of smoke inhalation in five horses

Tina Kemper, DVM; Sharon Spier, DVM, PhD; Simon M. Barratt-Boyes, BVSc; Rick Hoffman, DVM, PhD

Five Quarter Horses, 2 to 10 years old, were admitted to the teaching hospital 5 hours after a barn fire in which they sustained inhalation injuries. The barn, which was built from cinder blocks, had been completely closed when the tack room caught fire, exposing the horses to thick smoke without access to fresh air. Four horses were found dead from smoke inhalation. The duration of exposure was not known, but was < 6 hours. None of the horses suffered surface burns, but all horses admitted had singed eyelashes, mane, and tail from the intense heat. All horses received furosemide, flunixin meglumine, dexamethasone, and penicillin at the scene of the fire.

A 3-year-old gelding (horse 1) and a 10-year-old mare (horse 2) had mildly high heart rates (54 and 44 beats/min, respectively) on admission; respiratory rates were 36 and 24 breaths/min, respectively, and rectal temperatures were 38.3 and 38.4°C, respectively. Both horses were bright, alert, and responsive, and coughed intermittently. Thoracic auscultation revealed diffuse wheezes throughout the lungs; in horse 2, wheezes were loudest caudodorsally. Arterial blood gas analysis revealed hypocapnia and low bicarbonate values in horse 1; results were within normal limits in horse 2 (Table 1). The horses were treated for pulmonary edema with furosemide (1 mg/kg of body weight, IV, q 12 h) and dexamethasone sodium phosphate (0.05 mg/kg, IV, q 12 h) for 24 hours. Aminophylline (7 mg/kg, PO, q 12 h) and terbutaline sulfate (0.05 mg/kg, PO, q 12 h) were used for bronchodilation. Trimethoprim/sulfamethoxazole was given prophylactically (5 mg of the trimethoprim fraction/kg PO, q 8 h) for 5 days. Thoracic radiography on the fifth day of hospitalization revealed a diffuse, mild bronchointerstitial pattern in both horses, with pulmonary consolidation ventrally in horse 2. Both horses recovered without complications and were discharged 6 to 7 days after admission.

On reexamination of horse 2, 2 months after admission, physical abnormalities were not evident and results of thoracic auscultation were normal. Results of a CBC were within normal limits. Thoracic radiography revealed a continuing diffuse, mild bronchointerstitial pattern, but no consolidation was found. The owners were advised to start the horse back to work.

A 2-year-old filly (horse 3) had a heart rate of 48 beats/min, respiratory rate of 24 breaths/min, and rectal temperature of 37.2°C at admission. Loud wheezes and crackles could be auscultated throughout the lungs. This horse had paroxysmal coughing episodes, during which it would strike out with its forelimbs and appear to be distressed. Arterial blood gas analysis revealed hypoxemia, mild hypocapnia, and low bicarbonate concentration (Table 1). This horse was treated by use of a protocol similar to that used for horses 1 and 2, but in addition, received meperidine HCl (1 mg/kg, IM) to suppress coughing, and later was given butorphanol (0.025 to 0.05 mg/kg, IV) for cough suppression when anxious behavior was observed.

Thoracic radiography 5 days after admission revealed a diffuse, moderate bronchointerstitial pattern. The horse recovered and was discharged 7 days after admission.

Horse 3 had normal results on physical examination 2 months after hospitalization. Normal bronchovesicular sounds were heard throughout the lungs. Thoracic radiography revealed a continuing mild bronchointerstitial pattern. Results of a CBC were within normal limits. The owners were advised to resume training of this horse.

A 2-year-old colt (horse 4) was admitted in

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Table 1—Results of arterial blood gas analysis at admission in five horses with smoke inhalation

<table>
<thead>
<tr>
<th>Horse No.</th>
<th>$P_{O_2}$ (mm Hg)</th>
<th>pH</th>
<th>$P_{CO_2}$ (mm Hg)</th>
<th>HCO$_3^-$ (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>89.3</td>
<td>7.466</td>
<td>23.8</td>
<td>16.3</td>
</tr>
<tr>
<td>2</td>
<td>94.1</td>
<td>7.440</td>
<td>33.7</td>
<td>24.0</td>
</tr>
<tr>
<td>3</td>
<td>77.0</td>
<td>7.485</td>
<td>21.5</td>
<td>17.0</td>
</tr>
<tr>
<td>4</td>
<td>50.4</td>
<td>7.439</td>
<td>44.3</td>
<td>29.0</td>
</tr>
<tr>
<td>5</td>
<td>57.1</td>
<td>7.383</td>
<td>39.8</td>
<td>22.8</td>
</tr>
</tbody>
</table>

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From the Veterinary Medical Teaching Hospital, School of Veterinary Medicine, University of California, Davis, CA 95616. Dr. Kemper's present address is Chino Valley Equine Hospital, 13385 Yorba Ave, Chino, CA 91710.

*Lasix, Hoechst-Roussel, Somerville, NJ.
*Azium, Schering Corp USA, Kenilworth, NJ.
*Aminophylline, Westward Pharmaceutical Corp, Eatontown, NJ.
*Brethine, Ciba-Geigy Corp, Ardsley, NY.
*Trimethoprim/sulfamethoxazole (DS), Vitarine Pharmaceuticals Inc, Springfield Gardens, NY.

*Demerol, Winthrop Pharmaceuticals, New York, NY.
*Torbugesic, Fort Dodge Laboratories Inc, Fort Dodge, Iowa.
respiratory distress. This horse was reported to be severely affected and had been given emergency oxygen treatment by the referring veterinarian at the scene of the fire.

The heart rate was 80 beats/min, respiratory rate was 28 breaths/min, and rectal temperature was 37.8°C. Respiration were labored, with marked inspiratory and expiratory effort. On auscultation, severe inspiratory and expiratory wheezes and crackles were heard bilaterally, with little evidence of bronchovesicular sounds. Oral mucous membranes were cyanotic. The horse immediately was treated with nasal oxygen insufflation at 15 L/min. Analysis of a sample of arterial blood obtained after the onset of oxygen treatment revealed marked hypoxemia, with an arterial partial pressure of oxygen (PaO₂) of 50.4 mm Hg (Table 1). The hematocrit was 45%, and total plasma protein concentration was 8.3 g/dl.

Horse 4 was treated by use of a protocol similar to that for horse 3, but received dexamethasone sodium phosphate at a higher dosage (0.1 mg/kg, iv, q 12 h) and flumixin meglumine1 (1 mg/kg, iv, q 12 h). This horse also was given lactated Ringer's solution, iv, at 1.5 L/h. Humidified oxygen was administered via nasal insufflation (15 L/min) for the first 10 hours of hospitalization. The horse's condition continued to deteriorate, with an increased heart rate of 100 beats/min and a decreased PaO₂ of 44.7 mm Hg while breathing oxygen. It intermittently expectorated large pseudomembranous tracheobronchial casts, which periodically obstructed the airway. The horse became severely dyspneic and partially collapsed several times in the restraining stocks. A tracheostomy was performed to facilitate removal of the large fibrin casts. Humidified oxygen treatment was changed from nasal to tracheal insufflation, and tracheal suction was performed every 2 hours to remove secretions. By 72 hours after admission, the horse's condition had stabilized, as evidenced by decreased respiratory rate and effort and improved attitude. Oxygen treatment was discontinued, although the PaO₂ remained at 50.3 mm Hg.

Tracheal aspirates obtained 3 and 5 days after admission contained a large number of degenerate neutrophils, but pathogens were not found on bacterial culturing of these samples. Thoracic radiography 3 days after admission revealed a diffuse, moderate bronchointerstitial pattern, with mild pleural effusion and pneumomediastinum. Analysis of a sample of arterial blood obtained on day 13 of hospitalization revealed a mild persistent hypoxemia (PaO₂, 77.5 mm Hg), although the respiratory rate was 24 breaths/min.

Horse 4 was discharged the next day, with instructions to the owners to continue terbutaline sulfate (0.05 mg/kg, po, q 12 h) and to keep the horse in a dust-free environment. At examination 2 months later, the horse was bright, alert, and responsive. The only abnormalities found on physical examination were bilateral wheezes on thoracic auscultation. A single dose of 5 mg of atropine1 was administered iv for bronchodilation, and resulted in a marked reduction of auscultatory wheezes, indicating a persistent bronchoconstrictive component. Thoracic radiography revealed marked improvement; pneumomediastinum had resolved and pleural effusion was not evident. A minimal interstitial pattern still was detected.

The most severely affected horse admitted was a 2-year-old filly (horse 5) that had respiratory distress. Heart rate was 100 beats/min, respiratory rate was 40 breaths/min, and rectal temperature was 38.9°C. Mucous membranes were cyanotic, and sclerae were markedly injected. Pulmonary edema was so severe in this horse that normal bronchovesicular sounds were not detected, despite marked inspiratory and expiratory efforts. Foam exuded from both nostrils and only minimal airflow could be detected. Arterial blood gas analysis while the horse was being treated with nasal oxygen insufflation (15 L/min) revealed marked hypoxemia (PaO₂, 57.1 mm Hg; Table 1); hematocrit was 52%, and total protein concentration was 7.0 g/dl.

Treatment was similar to that of horse 4. Like that horse, horse 5 expectorated large proteinaceous tracheobronchial casts that, on microscopic examination, contained carbon particles. Horse 5 also had multiple episodes of partial collapse. Tracheostomy was performed 10 hours after admission, and humidified oxygen (15 L/min) was administered through the tracheostomy. The horse's condition continued to deteriorate, with severe hypoxemia (PaO₂, 43.1 mm Hg) on the third day of hospitalization. Oxygen treatment was changed to a high-frequency jet ventilator delivering humidified oxygen at a rate of 120 pulses/min. The horse also was given 1 ml of 10% acetylcysteine1 via a humidification system every 2 hours in an attempt to partially dissolve or loosen the proteinaceous casts. The trachea was suctioned every 2 hours.

On the fourth day of hospitalization, horse 5 developed severe hemorrhagic colitis with metabolic acidosis and collapsed in the restraining stocks. The horse was euthanatized at that time because of the poor prognosis. Gross necropsy revealed diffuse congestion of the larynx and proximal portion of the trachea. Six centimeters distal to the arytenoid cartilages, the tracheal mucosa was diffusely rough and was covered with 1 to 2 mm of loosely adhered fibrin. The fibrin plaques extended the remaining length of the trachea and obstructed small bronchioles. The lungs had areas of bullous emphysema cranioventrally, and areas of congestion and atelectasis. Histologic examination revealed extensive pulmonary parenchymal collapse, edema, and congestion. Bronchi and bronchioles

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1Atropine sulfate, Western Medical Supply Inc, Arcadia, Calif.
1Mucomyst, Bristol Laboratories, Evansville, Ind.

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were obstructed with proteinaceous exudate, neutrophils, sloughed epithelial cells, and large aggregates of carbon particles (Fig 1). Marked mucosal squamous metaplasia of the bronchioles was found, with focal areas of necrosis often containing neutrophils. Alveoli were flooded with edema containing neutrophils and mononuclear cells. In other areas, alveolar destruction and emphysema were observed.

Gross and microscopic examination of the alimentary tract did not reveal notable lesions in the esophagus or small intestine. The cecal mucosa was diffusely hemorrhagic but not ulcerated. The large colon contained focal reddened regions; the small colon was unremarkable. Salmonella sp was not found on bacterial culture of fecal samples.

Most of the pulmonary damage acquired by human beings or animals during exposure to a fire is caused by the decreased oxygen content of inspired air, as well as by exposure to various noxious gases. The character of such noxious gases, which may include carbon monoxide and cyanide, depends on the type of material that is burning. In experiments, superheated air has been shown to rarely reach the lower airways because of the efficient heat-exchange mechanism of the lungs and the upper airways. A fire hot enough to induce lower-airway thermal damage causes sufficient damage to the upper airway to result in severe laryngeal edema and suffocation.

Histopathologic changes in the lungs caused by smoke inhalation have been documented by studying sheep exposed to burning cotton toweling. Within 8 hours of smoke inhalation, diffuse tracheobronchial mucosal sloughing developed. Progressive separation of the epithelium created pseudomembranous casts that caused partial or complete airway obstruction. The pseudomembranous casts were composed of mucus, cellular debris, neutrophils, bacteria, and fibrin. Pulmonary edema developed concurrently as the mucosal lining was destroyed. In clinical studies of human beings and dogs, as well as in experiments with goats and rabbits, histopathologic findings similar to those in the 1 nonsurviving horse of this report were observed.

When dealing with smoke inhalation, the main goals of treatment are to decrease pulmonary edema, to alleviate bronchoconstriction, to maintain PaO2 within normal limits, and to manage resulting secondary organ failure and bronchopneumonia. Many treatment options are available for human patients with inhalation injury, although some are controversial. Treatment with oxygen at the scene of the fire can be extremely beneficial. The half-life of carbon monoxide in human blood is 240 minutes if the patient is inhaling room air, but decreases to 40 to 50 minutes if the patient is breathing 100% oxygen. The early administration of oxygen to horse 4 at the scene of the fire was considered to be an important factor in its survival.

Humidified oxygen can be delivered to less severely affected horses by nasal insufflation. In severely affected horses that have large, obstructive, tracheobronchial pseudomembranous casts, nasal insufflation may not be adequate to increase the inspired oxygen concentration to compensate for diffusion impairment. The casts, if large enough, can cause respiratory tract obstruction, which contributes to cyanosis. If a tracheostomy is necessary to facilitate removal of large casts, humidified oxygen can be insufflated directly through the tracheostomy tube. One of the disadvantages of performing a tracheostomy, however, is that the horse can no longer elicit an effective cough. This is of little importance if the casts are so large that the horse cannot dislodge them even with an intact cough mechanism.

Ventilatory support is difficult at best in conscious standing horses. High-frequency jet ventilation has been used successfully to ventilate anesthetized horses. The high-frequency jet ventilation system that delivered a large volume of oxygen to horse 5 was well-tolerated by the horse, but because of the severe pulmonary injury, this treatment was not adequate in substantially increasing PaO2 to values compatible with survival. The mortality for human beings with smoke inhalation approaches 50% if ventilatory support is required.

Diuretics such as furosemide are important to facilitate removal of pulmonary interstitial fluid that may result from inhalation injury. Bronchodilators such as terbutaline, used alone or in combination with aminophylline, are also important early in the process to relieve bronchoconstriction caused by smoke irritation. Care must be taken in determining the dose required because toxic con-
Corticosteroids have been used successfully to decrease inflammation in an earlier report of smoke inhalation in horses. A decrease in mortality in rats has been shown if a glucocorticoid was used, whereas mortality increased to 131.6% of expected when a steroid having mineralocorticoid effects was used. The increased mortality was thought to be attributable to retention of sodium, and subsequently fluid, thereby exacerbating pulmonary edema. In studies of human beings with smoke inhalation and surface burns, however, those given corticosteroids were shown to have 4 times the mortality of the placebo group, and developed major infections 3 times as often as did those not given corticosteroids. In human beings with burns, the detrimental immunosuppressive effects of corticosteroids may exceed any benefits; therefore, use of corticosteroids should be limited to those patients with smoke inhalation alone. This logic may be applicable to equine patients as well.

Prophylactic administration of antibiotics has been shown to provide little or no benefit in human patients with smoke inhalation, because of potential development of resistant strains of bacteria. Human patients commonly develop bacterial bronchopneumonia secondary to an inhalation injury, and these patients then are treated with the appropriate antimicrobials. The propensity for developing bacterial pneumonia secondary to smoke inhalation was not observed in rabbits exposed to pine-wood smoke, in 20 previously reported cases of smoke inhalation in horses, or in the equine patients of this report. The prophylactic use of antibiotics may have contributed to the colitis that developed in horse 5.

Many human beings with smoke inhalation develop decreased pulmonary function shortly after injury, but in most cases, function returns to normal within 5 months of inhalation injury. Isolated cases of late bronchial hyperresponsiveness, as well as bronchiectasis and progressive respiratory failure, have been reported, but are rare.

In the 4 surviving horses, the 3 less severely affected resumed training 2 months after inhalation injury, whereas the more severely affected (horse 4) resumed training 5 months after injury. In the 4 years since the fire, all 4 horses have achieved or exceeded the performance that they were capable of prior to the fire. Horse 4, the most severely affected survivor, has been reported to cough when first exercised, but is a successful reining horse and has won many competitions and championships. The inhalation injuries that these horses sustained did not seem to have affected them deleteriously on a long-term basis. Prognosis for horses returning to work therefore is not necessarily poor, even with severe inhalation injuries, if the horse can survive the initial stages of pulmonary damage and secondary organ involvement.