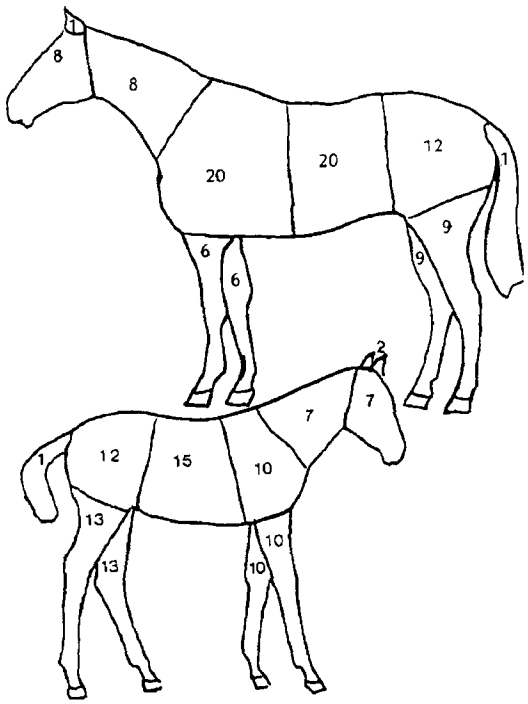

Figure **1.** Percent body surface area.

20 percent body surface area affected causes cellular and humoral immunity compromise and depressed cardiac function (myocardial depressant factor).

Burn depth is classified as partial or full thickness. Partial thickness involves the epidermis and is either superficial (first degree) or deep (second degree). Full thickness involves all the epidermis and dermis (third degree). Determining burn depth is not as readily evident in animals as in people but can be estimated by vision and palpation. Partial thickness burns are characterized by pain, erythema, edema and blisters. Full thickness burns are characterized by eschar formation, no pain and hair that easily epilates.

Mortality is related to extent of full thickness plus 1/4 partial thickness injury.

$$\text{Percent of Body Surface Area Involved} = \frac{\text{Full thickness area} + 1/4\ \text{Partial thickness area}}{\text{Total body surface area of animal}} \times 100$$

The total body surface area of an animal can be taken from the attached conversion table or calculated using the formula:

$$S(m^2) = 0.1X\ w\ (kg)^{2/3}$$

The percentage of body surface area involved can be estimated by allotment of percentages to various body areas. In people, the "Rule of Nines" is used. Percent body surface area allotments have been estimated for an adult horse and a two-week-old foal and are shown in Figure 1.

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SMOKE INHALATION INJURY IN THE HORSE

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Smoke inhalation is a major cause of death in fire victims of all species. The presence of inhalation injury is the major limiting factor in burn mortality. Therefore, early recognition of the clinical signs and understanding the pathophysiology of the injury is essential for timely, appropriate therapeutic intervention. This presentation reviews the mechanism of smoke injury, the pathophysiology, clinical signs, diagnosis and therapy of inhalation injury in the horse.

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Mechanism of Injury

Smoke damages the airways of the horse by two different mechanisms, direct thermal injury and toxicity of the chemicals in smoke. Direct thermal injury is the result of contact of smoke with the face and mucosa of the upper airways. Resultant signs include singeing of the mane and forelock, periorbital burns with singed eyelashes, soot at the nares and muzzle burns. Mucosal burns result in upper airway edema which is most profound at 18-24 hours after the fire. Laryngospasm and an efficient heat exchange mechanism prevent the thermal burn from extending into the lower airways. Severe edema and laryngospasm may cause upper airway obstruction and may necessitate emergency intubation or tracheostomy.

The second mechanism of smoke induced injury is through toxicity of noxious chemicals in smoke. There are three categories of toxic inhalants; asphyxiants, systemic toxins and irritants. Carbon monoxide is an asphyxiant of major concern. Carbon monoxide is responsible for death in greater than 80% of smoke inhalation fatalities. It is colorless, tasteless, odorless and non-irritating, making it difficult to appreciate in the environment. It is also difficult to appreciate clinically in the poisoned victim. Mucous membranes are not cyanotic, thoracic auscultation is normal. Arterial oxygen tension (PaO_2) is also within normal limits.

Carbon monoxide is particularly hazardous due to its high affinity for hemoglobin. It has a 210 times greater affinity for hemoglobin than does oxygen. Due to this, exposure to only small amounts of carbon monoxide will cause severe clinical signs. In addition to binding to hemoglobin, carbon monoxide shifts the oxygen dissociation curve to the left. This increases affinity of hemoglobin for oxygen. This has the effect of decreasing the ability of the tissues to extract oxygen from the blood, worsening the hypoxia.

Clinical Stages of Smoke Injury in the Horse

There are three clinical stages of inhalation injury described in the horse, acute pulmonary insufficiency, pulmonary edema and bronchopneumonia. Acute pulmonary insufficiency occurs during the first 36 hours after exposure to the fire. This phase involves both mechanisms of injury, direct thermal burns and asphyxiant smoke toxicity. In addition there is loss of surfactant activity which results shortly after exposure to smoke. Surfactant functions to decrease surface tension in the alveoli thereby increasing compliance of the lungs. Surfactant helps exclude interstitial fluid from the alveoli and functions to prevent lower airway atelectesis. As surfactant activity decreases after smoke exposure, there is a decreased compliance and an increased ventilation-perfusion mismatch. Ciliary function also decreases during the stage of acute pulmonary insufficiency. It is therefore difficult for the patient to clear particulate matter which becomes introduced to the airways during the fire or as a result of resuscitation efforts.

The second stage of smoke induced injury is pulmonary edema. This stage is generally most profound 24 to 48 hours after the initial insult. Pulmonary edema results from increased permeability of the microvasculature as a result of two mechanisms of injury. First there is direct injury of the epithelium of the alveoli by the irritant chemicals of smoke. Second, there is an influx of neutrophils which release cytotoxic mediators such as proteases, arachidonic acid metabolites and oxygen free radicals. The interstitial edema is made more profound by a redistribution of blood flow following inhalation injury which results in a 10-fold increase of bronchial blood flow.

During the second stage of inhalation injury there is also a necrosis and sloughing of the mucosa of tracheal and bronchial airways as well as the type I alveolar cells. This may result in a pseudomembrane which, if dislodged, can cause small airway obstruction, and resultant ventilation-perfusion mismatch. Obstruction of small airways may also lead to emphysema and pneumothorax if severe.

The third phase, bronchopneumonia, occurs on an average 7 days after the fire. The pneumonia is likely a result of both decreased bacterial clearance and increased susceptibility. Bacterial clearance is impaired by the damage to the lining of the respiratory tree and loss of ciliary clearance mechanisms. Also there is a decrease in function of the alveolar macrophages after smoke exposure. Susceptibility of the patient is increased as immunosuppression and sepsis result from burn trauma. Additionally, resuscitation efforts often result in inoculation of microorganisms in the lower airways.

Diagnosis of Smoke Toxicity

A complete assessment of the patient as well as characterization of the fire will help in predicting the probability of smoke inhalation injury. Enclosed fires have a greater risk of inducing asphyxiant toxicity and hypox-

emia. Fires involving plastics are likely to cause hydrogen cyanide toxicity. Steam is likely to cause direct thermal injury to the lower as well as upper airways. The presence of facial burns and a soot-like nasal discharge indicate likely inhalation injury.

A complete physical assessment of the patient is essential in formulating therapeutic plans and establishing a prognosis. In addition to assessing the respiratory system, particular attention should be paid to the cardiovascular and central nervous systems as these are most susceptible to the effects of hypoxemia. A complete ophthamalogical examination including fluorescein staining of the corneas is indicated. Evaluation of the degree of cutaneous burn should be included.

A minimal clinicopathological data base including a complete blood count, serum biochemistry, arterial blood gas and acid-base evaluation should be obtained. Often these values are normal at the onset of smoke induced injury. This information may be useful in assessing progression of disease and response to therapy. Due to the likelihood of edema and fluid shifts in smoke injured patients, packed cell volume, serum total solids and albumin should be frequently monitored. Carbon monoxide exposure can be diagnosed by measuring carboxyhemoglobin levels in venous blood. This test is generally available at human hospitals. Thoracic radiographs may also prove useful in assessing disease progression, but the changes seen on radiographs usually lag behind the clinical pathology. Fiber-optic bronchoscopy is useful in visualizing the degree of direct thermal injury to the mucosa. This will assist in deciding when to intervene with a tracheostomy or intubation. Transtracheal lavage or bronchoalveolar lavage is indicated if bronchopneumonia is suspected.

Therapy of Smoke Inhalation in the Horse

There are six major goals of therapy.

Maintain a patent airway. Correct hypoxemia. Remove mucus and debris from the respiratory tract. Decrease inflammation and minimize edema formation. Prevent secondary complications. Provide analgesia.

As previously discussed, maintaining an airway may involve emergency intubation or tracheostomy. It is important to remember the upper airway edema continues to develop for 18–24 hours after the fire. Therefore, it is important to continue to closely monitor the patient during this time period for signs of upper airway obstruction.

The drug of choice for correcting hypoxemia is humidified oxygen. This is most commonly provided to the awake, adult horse by nasal insufflation. Nasal insufflation is technically easy to accomplish and well tolerated by the horse. Inspired oxygen levels of 30–40% may be achieved with oxygen flow rates of 15L/minute if the nasal tube is properly placed, such that it extends into the pharynx. A lower value will result from a tube which ends in the nasal passage. In foals higher arterial oxygen tensions were achieved using percutaneous transtracheal catheter systems for oxygen delivery. To my knowledge, a similar study has not been reported in adult horses. The use of masks for oxygen delivery in the adult horse is limited by patient tolerance. In addition, high flow rates are necessary to accommodate the large tidal volume of the mature horse.

Pharmacological bronchodilation may also help correct hypoxemia by relieving the reflex bronchoconstriction that results from smoke exposure. Phosphodiesterase inhibitors, such as aminophylline (3–5 mg/kg PO BID–TID) have been used in horses following inhalation injury. Beta-2 agonists, such a terbutaline sulfate (0.05 mg/kg PO BID), have also been employed. Often the two types of bronchodilators are combined.

Removal of mucus and debris from the airways is facilitated by preventing these secretions from becoming tenacious. Maintaining adequate systemic hydration will help in achieving this goal. This may be a difficult order due to the tendency of these animals to develop edema. The addition of local airway hydration methods such as nebulization may assist in achieving the desired effect. Nebulization may be done with sterile saline alone or in combination with other drugs. Investigators have advocated the addition of anti-inflammatory drugs such as acetylcysteine or dimethylsulfoxide and bronchodilators such as glycopyrrolate. In some species the use of airway lavage and suction has proven effective in aiding in the clearance of debris. If large casts form in the small airways it may be an advantage to perform a tracheostomy to allow direct removal of these plugs. A tracheostomy will eliminate the animal's ability to cough, however, thereby reducing his clearance abilities.

Reduction of edema and inflammation is an important pharmacological goal. Upper airway and interstitial edema can be controlled through the use of a diuretic such a furosemide (1 mg/kg IV ql2 hours.) Once the patient has been rehydrated and renal function has been established to be normal the use of non-steroidal anti-inflammatory drugs may be beneficial. Corticosteriods are considered controversial by some clinicians due to their immunosuppressive effects and the risk of secondary infections in burn victims. However, due to their marked anti-inflammatory activity, many clinicians advocate the use of corticosteroids in the

animal that has incurred smoke toxicity with minimal cutaneous burns.

Avoiding secondary complications may be facilitated by prophylactic use of antibiotics, strict hygiene, and excellent nursing care. In humans, the use of antibiotics to prevent pneumonia is contraindicated due to the emergence of resistant strains of bacteria. This phenomenon has not been documented in the horse. The use of a broad spectrum antibiotic is recommended. Maintaining a clean tracheostomy site and protecting the site with some means of filter to avoid inhalation of particulate matter is beneficial. Providing excellent nursing care and analgesia will also improve the prognosis for recovery and shorten the duration of rehabilitation time.

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BARN FIRE PREVENTION

William and Jori Miller

Fire Prevention

Fire prevention is a simple combination of three key elements: awareness, concern, and knowledge; issues which basically point to one common factor, "common sense." Personal commitment to your facility and animals will prevent the unthinkable disaster of a barn fire.

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A formula for fire prevention includes:

a. facility maintenance/cleanliness
b. pre-planning
c. constant vigilance/awareness
d. education of other facility users
e. the elimination of the sense that "it will never happen to me."

How To Get Started

Review your situation - step back and look at your facility and picture how and where a potential problem might exist. Request a fire inspection from your local fire department, or County Fire Marshal's Office. Inspection by these departments either is free or carries a minimal fee. Fire Marshals in most states will provide this service as a courtesy. There are very few code requirements that would necessitate mandated changes.

This Fire Department meeting will also serve as a familiarization vehicle for the local fire service representatives. They are always interested in better serving you, and will be glad to hear your comments, concerns, and questions about their protection of your facility.

Barn Fire Prevention - Electrical Issues

Faulty electrical wiring and connections are the leading cause of barn fires. This is obviously frustrating for the fire service, because this cause is certainly easily eliminated and preventable.

To eliminate fires of electrical origin wiring should absolutely meet local building code requirements. Wiring in areas with animal access should be in conduit. Curious horses and rats will easily chew through wiring sheath insulation.

Install an adequate size breaker box to eliminate circuit overloads. This also gives the fire department ready access to an electrical shut off.

Extension cords, even those that are permanently mounted, and strip or multiple plug-in devices should never be used. All electrical appliances should be unplugged after their use. Coffee pots, radios, fans, tack room portable heaters, lights.

Barn Fire Prevention - Maintenance Issues

One of the best ways to prevent fires is good housekeeping. Cobwebs, dust, and debris are fuels waiting for something to ignite them. It takes very little time to keep these items from accumulating.

Feed, hay, and bedding areas should be maintained as "no public access" areas. By keeping casual visitors out of these areas you can eliminate the fire potential from people