How to Cope With Barn Fires

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1. Introduction

Barn fires can cause horrific injuries to horses, with high morbidity and mortality rates. Equine practitioners can be consulted for disaster planning advice and can be called to the scene of a barn fire to provide urgent care. In these situations, emotions run high, and making critical decisions can be difficult. The latent nature of many of the manifestations of thermal damage and smoke inhalation compound this difficulty. Effective communication, rapid and efficient triage, fluid resuscitation, and referral for specialized therapy can offset some of the devastating effects of barn fire disasters.

Practical Measures for Fire Prevention

In terms of advice for prevention, you may be asked to comment on the design of a barn or find yourself in barns that pose obvious fire hazards. Equipping yourself with a basic knowledge of fire hazard safety and who to contact for an expert consultation will enable you to help your clients, as well as save them heartache and financial losses. Many insurance companies will reduce premiums by 5% for demonstrable fire prevention measures. This figure can be as high as 10% for the installation of a sprinkler system. Expert assistance with reducing fire risk can be obtained by consulting the local fire chief and insurance agent.

Briefly, the following is a list of simple measures that may be employed to reduce fire risk, but it is by no means an exhaustive summary:

- Barn design. Stall door hardware should operate smoothly to ease evacuation in case of an emergency. Consider front and back stall doors; ensure sufficient electrical outlets to service the number of stalls; employ a design that incorporates insulation and ventilation to reduce the need for summer fans or winter heating. Reduce the use of wood when possible, especially wood treated with highly flammable preservative agents (such as railroad ties). Place the manure pile away from the barn (this will reduce flies also).
- Spring clean the barn. Remove debris from any electrical equipment such as fans; remove organic debris from the barn; remove cobwebs and accumulations of dust; remove all accumulations of flammable materials such as feedbags, hay waste, and baling string. This includes pruning adjacent shrubbery, storing the least amount of hay and straw adjacent to the horses, and removing chemicals that are not essential for daily horse care (such as herbicides and paint).
- Install warning systems such as heat or smoke sensors. Consult the fire chief or a licensed
install lightening rods.
- Install a sprinkler system. This can save the lives of horses and salvage the barn.
- Install and learn how to use an all-purpose fire extinguisher. In case of an extinguished fire, be sure to still call the fire brigade, as 5% of barn fires are due to rekindling.
- Inspect electrical fittings. Faulty wiring, overloaded sockets, and multiple electrical appliances increase risk considerably. Inspection by an electrician is money well spent.
- Avoid storing hay/straw near the horses. This is especially relevant for storing freshly cut hay that may retain enough moisture to heat up.
- Motorized equipment and vehicles. Fuel leaks and hot exhaust fumes are fire hazards, both individually and combined. Hot exhausts have been known to ignite stored hay. Fuel in vehicles and stored fuel cans are potent fire hazards and accelerants and should be stored away from livestock when possible.

Pathophysiology

The pathophysiology of the responses to thermal damage and smoke inhalation are complex. Most of our knowledge is extrapolated from human data, with the addition of small case series written up in the veterinary literature. This can make interpretation of human literature somewhat challenging because our patients do not react identically and are not housed in sterile burn care facilities. The nature of the fire, location within it, degree of combustion, and materials burned are all variables to consider. Although treatment will commence without this exact knowledge being available, having thought about it beforehand will equip you with an ability to adapt therapy as clinical manifestations present. For example, an open barn structure may reduce smoke inhalation, partial combustion may result in a greater degree of carbon monoxide toxicity, and some modern construction materials can generate hydrogen cyanide on combustion.

The initial thermal insults cause microvascular damage and direct tissue coagulation. This rapidly leads to systemic shock with the triggering of the inflammatory and coagulation cascades and decreased cardiac output. Microvascular damage, both direct and via inflammatory cytokines, leads to an increased capillary pressure, with leaky capillaries followed by the formation of edema. This occurs both locally and at distant sites, especially with extensive thermal damage. The activation of neutrophils compounds endothelial damage as they generate free radical species and marginate. Ischemia-reperfusion injury results from direct vascular damage and the effects of the systemic inflammatory response. This causes further tissue destruction, particularly in the form of oxidant damage and membrane lipid peroxidation.

As shock progresses, tissue perfusion is further compromised, vascular permeability increases, and protein leaks into interstitial spaces, furthering circulatory collapse and edema formation. The massive release of catecholamines drives the metabolism to a hypermetabolic state leading to insulin resistance, increased cardiac output, increased oxygen consumption, and ultimately fat and protein wasting. The redistribution of circulation leaves the renal and splanchnic circulation at risk of hypoperfusion, which can result in renal damage and translocation of gastrointestinal bacteria, respectively.

Intravascular hemolysis has been noted in both human and equine burn victims. This is currently thought to be the result of direct thermal injury and massive oxidant challenge from free radicals. Pigment nephropathy is a significant factor in these cases, no doubt compounded by inadequate renal perfusion.

Inhalation injury is a combination of smoke and thermal damage. In humans, damage to the lower respiratory tract is a serious source of morbidity and mortality. This seems not to be so pronounced in the horse—most likely due to a comparatively longer upper respiratory tract. The insult varies according to the heat generated, the type of fuel burned, and how completely it was burned. The respiratory tract therefore suffers direct thermal damage, the effects of toxic gases, and hypoxia. Thermal damage, as mentioned above, is mostly directed against the upper respiratory tract but causes massive sloughing of the respiratory membrane. The physical presence of this necrotic debris may necessitate a tracheostomy to aid in its removal. Incomplete combustion of fuels generates carbon monoxide. Carbon monoxide has a 250 times greater affinity for hemoglobin, and together they form carboxyhemoglobin, whose oxygen dissociation curve is shifted massively to the left, resulting in tissue hypoxia. Oxyhemoglobin and carboxyhemoglobin are indistinguishable on pulse oximetry, and a different spectrometer is required for assessment.

Tissue hypoxia also occurs during the fire event itself as the horse inspires a reduced oxygen environment along with the smoke. This sets up free radical production in all hypoxic tissues and causes derangements of blood perfusion. Ultimately, the systemic inflammatory response, thermal necrosis, and progressive edema generate bronchoalveolar irritation and bronchoconstriction. Initially, pulmonary edema may develop, which can be fatal and difficult to reverse. In the medium to longer term, the lower respiratory tract can suffer alveolar damage when pseudomembranous cast formation blocks the small airways. This sets the stage for the development of bronchopneumonia approximately 7 to 14 days after the insult.
Clinical Signs

A variety of symptoms may develop over the first few hours to days after rescue from a barn fire. Thermal damage is typically seen on the dorsum and head. The eyes may be swollen shut or exhibiting blepharospasm, making the evaluation of corneal damage difficult. The degree of thermal skin damage is measured in both surface area and depth. More superficial burns tend to be painful because damaged nerves are exposed. These horses are much more likely to be pruritic and may self-traumatize. Third- and fourth-degree burns (third degree is through all layers of the skin, and fourth degree includes damage to underlying muscle and bone) are less painful because they have destroyed the cutaneous nerve endings, but they result in more scarring and disfigurement. The skin becomes hard and leathery when its full depth is damaged.

The rapid onset of shock and widespread edema causes tachypnea, tachycardia, and injected mucous membranes. Carbon monoxide toxicosis and severe hypoxemia will manifest as depression, disorientation, and/or ataxia. Neither a pulse oximeter or blood gas analysis can diagnose carboxyhemoglobin. On a pulse oximeter, it is indistinguishable from oxyhemoglobin, and blood gas analyses measure oxygen dissolved in plasma. Unfortunately, prior to pink froth being seen at the nostrils, signs of pulmonary edema are indistinguishable from hypoxemia.

Organ dysfunction, even in the successfully fluid-resuscitated horse, can occur in the first several days after the insult. However, repeated clinical examinations, serial plasma lactate concentration, hematology and serum biochemistry analyses, central venous pressure when possible, and urine output and analysis are the mainstays of monitoring. When possible, colloid oncotic pressure and serum albumin level are useful to monitor protein loss as a surrogate for endothelial damage. Particular attention should be paid to hemolysis in the plasma. This was reported in a cohort of horses from a barn fire in Texas, which resulted in significant pigment nephropathy contributing to the morbidity and mortality of these horses. Of this group, some were not found until several hours after the fire broke out, probably compounding shock and catecholamine-related renal hypoperfusion.

Initial Therapy

This paper will be confined to the initial treatment period for horses rescued from barn fires. More extensive discussions of burn wound management and respiratory therapy are listed in the bibliography. When called to a barn fire, take instruction from the fire department on-site leader. Treat the horses that have already been removed from the fire and do not enter the barn fire area. The use of ice to cool horses off is not recommended by virtue that it elicits peripheral vasoconstriction in the skin, poten-
very short course of prophylactic antimicrobial therapy. Avoiding drugs such as aminoglycosides, which carry a risk of nephrotoxicity, is prudent. Drugs such as doxycycline (10 mg/kg PO q 12 hours) or enrofloxacin (5 mg/kg IV q 24 hours) are likely to be adequate. Topical antimicrobial therapy is universally accepted to prevent severe wound contamination. Silver sulfadiazine was developed with burn patients in mind, and it is still a mainstay of treatment. It is worth noting that skin barrier function is severely compromised. Because of this immunocompromise, it is prudent to observe strict hygiene and wear gloves, and even disposable gowns when possible, when treating these patients.

Therapy directed against free radicals can be helpful. There is modest support in the literature for the use of intravenous dimethyl sulfoxide (DMSO 100 mg to 1 g/kg; 50 to 500 mL/500-kg horse diluted to <10% solution IV). In humans, the xanthine oxidase inhibitor allopurinol may be used. Pharmacokinetic studies have shown it to be safe in horses at 30 mg/kg PO or 5 mg/kg IV and to reduce free radical formation at exercise. Allopurinol is available in tablet form in human pharmacies because it is used in gout sufferers due to its role in uric acid metabolism. The use of N-acetylcysteine is also showing promise as an antioxidant therapy in human burn patients but has not been evaluated in horses. The use of “anti-oxidant” drugs seems as logical as other agents that we freely reach for such as pentoxifylline (8.5 mg/kg PO q 12 hours) or DMSO.

The placement of a tracheostomy is rarely needed in the immediate post-rescue phase but is a consideration in an acute case of inspiratory distress, when the glottis may be reduced due to laryngeal swelling. Should the tracheostomy not relieve the respiratory distress, then it may be concluded that pulmonary edema, bronchoconstriction, and/or carbon monoxide poisoning are the cause of the hypoxia. Flow by intranasal oxygen may help, but marginally so, and hyperbaric oxygen therapy is the best treatment for these conditions, particularly the reduction of carboxyhemoglobin. The half-life of carboxyhemoglobin is reduced when breathing oxygen and dramatically reduced when breathing hyperbaric oxygen (average t½ at 1 ATM, 15 L/min = 71 minutes versus 26 minutes at 1.58 ATM).

In a recent review by Dr. Nathan Slovis, the additional benefits of hyperbaric oxygen therapy (HBOT) are outlined. The significant reduction in edema and increased plasma oxygen concentrations, in addition to the reduction of carboxyhemoglobin levels, make HBOT the single most attractive treatment modality for burn patients after IV fluid resuscitation. Referral to a facility for HBOT treatment is recommended if geography and finances allow. A list of public equine treatment chambers is listed in Table 1. Should this therapy be an option, the horse should be taken to the facility as soon as he is stable to travel, so that HBOT can commence immediately after evaluation. Obviously, skilled veterinary critical care must be available at whatever site is chosen.

In addition to nonsteroidal anti-inflammatory drugs for the control of pain, opioids and opiates may be used with caution. In the immediate post-rescue period, you may be confined to using butorphanol, but the use of morphine, when combined with a background of α-2 agonist drug, can be safe, effective, and relatively inconsequential for cardiovascular parameters. Both drugs can be used intravenously or intramuscularly, provided that α-2 agonist drugs are on board. The author prefers morphine to butorphanol for pain control with sedation (morphine sulfate 0.3 to 1 mg/kg, slow IV or IM—the author finds that 150 mg IM is typically satisfactory in a 500-kg horse with sufficiently rapid onset). The use of a “ketamine stun” will give a practitioner 5 to 10 minutes of adequate chemical restraint time in a horse manic with pain. This small window of time should be sufficient to insert an IV catheter and for the subsequent safe delivery of other drugs. At subanesthetic doses, ketamine is a potent analgesic. The administration of 100 mg/450 kg IV (0.22 mg/kg) on a background of α-2 agonist sedation can be very effective in intractably painful horses, in which safety is a concern. The use of continuous-rate infusions of drugs such as lidocaine, ketamine, morphine, or combinations of these agents can be very helpful in managing painful and self-traumatizing patients once they are in a hospital setting.

Summary
The immediate treatment of horses rescued from fires poses many challenges, not least of all keeping yourself out of harm’s way. The most urgent needs are to gain venous access and institute immediate and aggressive fluid resuscitation to offset shock. Additional therapy should be directed at limiting oxidative damage and hypoxia, controlling pain, and triaging the cases to a care facility. Hyperbaric oxygen is an excellent therapeutic modality and should be provided if at all possible. An initial assessment of prognosis is often difficult; it can be hard to appreciate the extent of cutaneous, ocular, and systemic organ damage immediately to extrapolate the cost and length of care of these cases. However, in cases of severely affected patients, euthanasia may be the most reasonable and ultimately humane option.

References and Footnotes


**Table 1. List of Equine Hyperbaric Oxygen Facilities**

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<tr>
<th>Facility Name</th>
<th>City, State</th>
<th>Contact Person</th>
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<tr>
<td>Alamo Pintado Equine Medical Center</td>
<td>Los Olivos, CA</td>
<td>Dr. Doug Gerthel</td>
</tr>
<tr>
<td>Vladimir Cerin Training Center</td>
<td>Santa Anita, CA</td>
<td>Mr. Vladimir Cerin</td>
</tr>
<tr>
<td>Reid &amp; Associates</td>
<td>Wellington, FL</td>
<td>Drs. Byron Reid and Meg Miller</td>
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<tr>
<td>KESMARC Florida, Ocala, FL</td>
<td>Operation suspended at the time of print February, 2012</td>
<td></td>
</tr>
<tr>
<td>Hagyard Equine Medical Institute</td>
<td>Lexington, KY</td>
<td>Dr. Nathan Slovis</td>
</tr>
<tr>
<td>KESMARC, Versailles, KY</td>
<td></td>
<td>Ms. Kirsten Johnson</td>
</tr>
<tr>
<td>Delta Equine</td>
<td>Vinton, LA</td>
<td>Dr. Larry Findley</td>
</tr>
<tr>
<td>New Jersey Equine, Millstone Twp, NJ</td>
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<td>Dr. Scott Palmer</td>
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<tr>
<td>University of Tennessee, Knoxville, TN</td>
<td></td>
<td>Dr. Dennis Geiser</td>
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<tr>
<td>Equine Sports Medicine &amp; Surgery</td>
<td>Weatherford, TX</td>
<td>Dr. Reese Hand</td>
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<td>Pegasus Thoroughbred Training Center</td>
<td>Redmond, WA</td>
<td>Dr. Mark Dedominico</td>
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Provided by Dr. Fairfield Bain.

(This list is not exhaustive, is written alphabetically by state, and is not intended as an advertisement)