Smoke Inhalation
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INTRODUCTION
Animals caught around fire are not only at risk for burn injury but also for smoke inhalation. Damage to the animal results from heat damage as well as inhalation of toxicants. The nature of the fire will dictate which toxins are the most abundant. Mortality rates reported for serious cases are as high as 73%.1 Caring for those animals implies a good understanding of the physiopathologic consequences of smoke inhalation.

PHYSIOPATHOLOGY
Animals exposed to fires can be exposed to extreme heat and inhalation of toxins. Direct thermal damage is most frequent in the upper airways and can lead to edema, which might create increased resistance to inspiration or even complete obstruction.2 The rest of the respiratory tree can also suffer from damages induced by the heat or heated-particles, which can lead to cell death, edema, and loss of tissue.3

Smokes are made up of various compounds, which will vary with the nature of the substrate on fire. Solid particles may aggregate and occlude small airway via both mechanical obstruction and reflex bronchoconstriction. Mucociliary clearance is also diminished. Pulmonary inflammation and acute respiratory distress syndrome may ensue.

Various toxic gases such as chlorine or ammonia can be present in smokes but the most common ones are carbon monoxide (CO) and cyanide.

CO is formed by combustion of carbon-containing substances and is often involved in the clinical progression of animals exposed to smoke. CO’s effects on the body are the results of its interaction with hemoglobin. CO will bind hemoglobin with a much higher affinity than oxygen, thereby decreasing the amount of oxygen carried by hemoglobin. Also, CO will shift hemoglobin’s equilibrium curve to the left, which decreases the amount of oxygen delivered to the tissues. CO also promotes cellular damage via indirect effects, especially in the brain. It can lead to inflammation, oxidative stress, and also act as a neurotransmitter increasing the brain’s metabolic demand. Neurological signs due to CO intoxication can be acute or delayed (up to 6 days after the initial injury, in one report in dogs).

Hydrogen cyanide is another toxin frequently encountered in smokes. It can be produced by combustion of rubber, plastics, wool, etc. It exerts its effects by inhibiting the electron transport chain in the mitochondria, rendering cells unable to produce energy.

PHYSICAL EXAMINATION
Upon presentation, patients should undergo a triage examination to ensure airway patency and cardio-respiratory stability. A thorough physical examination should be performed in order to understand the extent of the damage for the individual patient. Animals suffering from smoke inhalation might present with superficial burns. Special attention should be paid to examination of the respiratory tree, as respiratory distress has been reported in animals exposed to fire.1

Clinical signs of respiratory distress

<table>
<thead>
<tr>
<th>Clinical signs</th>
<th>Description</th>
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<tr>
<td>Increased rate or effort</td>
<td>Crickles</td>
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<tr>
<td>Lack of sleep, appetite, water intake</td>
<td>Wheezes</td>
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<tr>
<td>Orthopneic posture</td>
<td>Abnormal chest wall excursions</td>
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<td>Extended neck</td>
<td>Paradoxical movements</td>
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<tr>
<td>Lip commissures retraction</td>
<td>Chest wall injury</td>
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<tr>
<td>Staring at wall or cage</td>
<td>Open mouth breathing</td>
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<tr>
<td>Stridor</td>
<td>Sternal recumbency</td>
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<tr>
<td>Stertor</td>
<td>Nares flaring</td>
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Severe cough  
Cyanosis  
Decreased lung sounds  
Borborygmus* in the chest

Neurologic signs such as obtundation, stupor (responsive only to noxious stimuli), coma (absence of response to noxious stimuli), or seizures might be present, especially in cases of CO intoxication.¹

A careful ocular examination should also be performed, as corneal damage may also be present.

**DIAGNOSTICS AND MONITORING**

Oxygenation function should be evaluated in patients exposed to smoke. Oxygenation can be assessed either via pulse oximetry or arterial blood gas measurement. Pulse oximetry measurement is an easy diagnostic tool. It relies on light transmission and assesses hemoglobin saturation with oxygen. Nonetheless, it carries some limitations: the readings are not always accurate and the instrument might be sensitive to patient motion, mucosal pigmentation, and vasomotor tone. Importantly, animals suffering from CO intoxication might have a normal pulse oximetry reading while being severely hypoxemic. Ideally, definite assessment of oxygen saturation function should be based on arterial blood gases analysis. This remains frequently not feasible because of cost, technicality, or availability. This is also a more reliable tool but obtaining a sample is often a challenge, especially in patients with respiratory distress.

CO binds hemoglobin to form carboxyhemoglobin (COHb). COHb blood levels can be measured by co-oximetry.

Thoracic imaging should be considered in animals with overt respiratory distress. Radiographs are the first line for many animals. It is important to ensure patients have been stabilized as much as possible prior to taking radiographs. Stress should be minimized. An exam of the larynx can be performed under sedation if the animal presents signs of upper airway obstruction (stridor, stertor, inspiratory respiratory distress). Bronchoscopic exam might provide more information on the extent of the damage.

Other diagnostic tools such as complete blood counts or chemistry panel should be considered as clinically indicated at the clinician’s discretion.

**TREATMENTS**

**General Approach**

Patient should be assessed for stability during triage examination. Should the upper airway be obstructed, intubation or tracheotomy should be considered. If respiratory distress is recognized, oxygen therapy via either face mask or flow-by should be immediately initiated. If clinical signs of shock are present (increased heart rate, poor pulse quality, prolonged or shortened capillary refill time, pale or hyperemic mucous membranes, cold extremities, altered mental status), intravenous fluid resuscitation should be initiated.

Analgesia is an important part of the management of this patient as significant pain might arise from the injuries induced by the fire.

**Carbon Monoxide Intoxication**

The mainstay of therapy for animals intoxicated with CO is oxygen administration. As discussed above, oxygen and CO compete for their binding sites on hemoglobin. Enriching inhaled air with oxygen allows for a reduction in CO half-life from 4 to 6 hours on room air down to 40 to 80 minutes on 100% oxygen.

**Cyanide Intoxication**

Oxygen therapy is also the first line treatment for cyanide intoxication; oxygen increases dissociation of cyanide from mitochondrial cytochromes. Nitrite administration is recommended for the treatment of cyanide intoxication as it converts hemoglobin to methemoglobin and cyanide has a high affinity for methemoglobin. Its use is however NOT recommended in patients with smoke inhalation as increasing the concentration of methemoglobin would worsen the oxygen carrying capacity already altered by CO intoxication. Animals can be administered 25% sodium thiosulfate (150–500 mg/kg IV bolus or CRI), which promotes the conversion of cyanide to thiocyanide, which is then eliminated in the urine.
Hydroxycobalamin (vitamin B12, 75–150 mg/kg IV) can be administered as it binds free cyanide to form cyanocobalamin, which is then excreted in the urine.

**Respiratory Failure**

Some animals might suffer from such upper airway swelling that emergency tracheotomy might be needed to maintain upper airway patency. In one report, 8% of dogs with smoke inhalation required a tracheotomy.¹

The extent of the parenchymal injury might be such that mechanical ventilation might be required. This should be considered in animals with severe hypoxemia (partial pressure of oxygen in arterial blood < 60 mmHg or pulse oximetry reading < 90%, on oxygen supplementation), severe hypoventilation (partial pressure of carbon dioxide in arterial blood > 60 mmHg), or impending respiratory arrest. A recent report⁴ described the management of such patient that underwent mechanical ventilation successfully.

**CONCLUSION**

Smoke inhalation is a serious problem and is often accompanied by other problems such as burns. It is important to carefully evaluate the patient with special attention to the respiratory and neurological system. Intensive care and continuous monitoring are important for serious cases and contribute to improved outcome.

**REFERENCES**