Inhalation Injury and Toxic Industrial Chemicals (TICs)

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Several TICs act on the respiratory tract. This information sheet will review the top 4 most common and toxic TICs. More data are available e.g. from the CDC at http://www.bt.cdc.gov/agent/agentlistchem.asp, and in the *Textbook of Military Medicine*, http://www.bordeninstitute.army.mil/cwbw/default.htm.

In general, the treatment of acute lung injury secondary to TICs is similar to that for smoke inhalation injury: i.e., it is largely supportive with a focus on (1) airway management, (2) lung-protective ventilation, (3) pulmonary toilet, and (4) avoidance of volume overload or excessively rapid fluid infusion that might worsen pulmonary edema. All patients who require mechanical ventilation secondary to TIC inhalation are likely at high risk of ventilator-associated pneumonia and should be monitored as such. The drug treatments mentioned below are largely based on animal experiments, so that evidence in support of clinical use is often weak. A specific drug regimen is likely to work best immediately after injury. Finally, these patients should be transferred to a burn center with expertise in inhalation injury, if feasible.

Chlorine (Cl_2). Used abundantly in industry. Common cause of industrial and transportation accidents. Dissolves in water to form HCl and HOCl acids; all 3 species participate in pathogenicity. Causes both small airway and alveolar injuries. Proposed treatments include inhaled (nebulized) corticosteroids (e.g. budesonide), nebulized sodium bicarbonate in water (e.g. 3.75 to 4.2%), nebulized beta agonists (e.g. terbutaline), and prone ventilation.[1]

Phosgene ($COCl_2$). New-mown hay smell. Used in production of plastics, drugs, and polyurethane. Most lethal chemical agent during WWI. Classic case is that of delayed-onset pulmonary edema (casualty may be seen and discharged, only to come back in a few hours with lethal edema, triggered by exertion). Pathophysiology includes oxidative stress and influx of neutrophils into lung. Proposed treatments focus on these mechanisms, and include N-acetylcysteine, ibuprofen, aminophylline, isoproterenol, and colchicine.[2]

Hydrogen sulfide (H_2S). Rotten eggs smell. Seen in petroleum, waste management ("dung lung"), and natural gas industries. Enters the bloodstream via the lungs, binds to cytochrome c oxidase (similar to cyanide); prevents oxygen utilization by the cells. "Knockdown": sudden loss of consciousness due to effect on brainstem mitochondria. Other effects are seizures, myocardial ischemia. Direct toxic effect on lungs causes pulmonary edema. Direct effect on cornea causes keratoconjunctivitis ("gas eye"). Treatment may include i.v. nitrites, as for cyanide poisoning--with caution to avoid excessive levels of methemoglobinemia.[3]

Anhydrous ammonia (NH_3). Fertilizer, refrigeration, petroleum, explosives industries. Transported in liquid form, at sub-zero temperatures. Reacts quickly upon release to form NH_4, ammonium hydroxide, a strong base. Causes alkali skin burns and frostbite. May cause rapid, severe upper airway obstruction if inhaled, followed by pulmonary edema. Inhaled corticosteroids were not effective in animal models, suggesting the directly destructive mechanism of this chemical. Patients with skin and ocular involvement must be copiously irrigated as for any alkali injury.

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1. Wang J et al. Administration of aerosolized terbutaline and budesonide reduces chlorine gas-induced acute lung injury. *Journal of Trauma.* 56(4):850-62, 2004 Apr.

2. Sciuto AM, Hurt HH. Therapeutic treatments of phosgene-induced lung injury. *Inhalation Toxicology*. 16(8):565-80, 2004 Jul.

3. van Aalst JA et al. Hydrogen sulfide inhalation injury. *Journal of Burn Care & Rehabilitation*. 21(3):248-53, 2000 May-Jun.

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