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Nonthermal Inhalation Injury

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Abstract

Inhalation injury is one of the leading factors in the deaths of burn patients and is the leading cause of death at the scene of fires. Research into sepsis and burn care have taken precedence since 1960. Only recently have researchers taken a closer look at smoke inhalation and its effects.

With the increased use of synthetic materials in buildings, furniture and clothes there is a need for emergency personnel to understand the effects of the various byproducts of combustion on the human body. A thorough knowledge of the physiological mechanisms, relevant assessment parameters and treatment modalities related to smoke inhalation enables the emergency CNS to provide expert care. The CNS must then use this knowledge to provide education to emergency and prehopital staff. The emergency CNS may also play a large role in the current research on new treatments.

This paper reviews the physiology, assessment and treatment of inhalation injuries. Suggestions are also made regarding how the emergency CNS can incorporate this knowledge into the roles of the CNS.

NONTHERMAL INHALATION INJURIES

Chapter 1: Introduction

History

With recent innovations in burn treatment technology, mortality rates in fire victims have dropped considerably. Now, the leading cause of mortality in fire victims is complications of smoke inhalation. Most burn patients who die have concomitant smoke inhalation (Blinn, Slater, & Goldfarb, 1988). Therefore, research emphasis is once again focusing on the assessment and treatment of inhalation injuries.

Until the 1940s, little attention was paid to the effects of smoke inhalation. It took a fire of epic proportions at the Cocoanut Grove nightclub in Boston in 1942 to show the importance of smoke inhalation as a cause of death in fires (Haponick & Munster, 1990). Four hundred ninety-one people died in that fire, the majority of deaths occurring without burn injury. The only other significant research in inhalation injuries prior to 1942 was during World War I. This research, however, was limited to chemical inhalants such as chlorine gas, phosgene, and mustard gas. Research on smoke inhalation continued full force until about 1960

when investigative interests switched to sepsis and burn wound care (Mosely, 1988). More recent events such as the Dupont Plaza fire and MGM Grand Hotel fire in Las Vegas have emphasized the lethality of smoke inhalation (Lybarger, 1987). Eighty-six people were killed in the MGM fire with only two deaths resulting from burns (Beretic, 1990). Most of these deaths occurred many floors above the level of the fire. Birkey and Clark (1981), in a study of 530 fire victims at one hospital, found 11% of deaths were related to burns and 89% were related to the inhalation of toxic smoke and hot gases. These statistics were the major impetus that pushed researchers to further explore nonthermal inhalation injuries.

Aside from the increase in mortality that smoke inhalation causes, there is an extension of hospital stay of those patients who do survive. On the average, burns complicated by smoke inhalation increase the hospital stay by two weeks (Sanderson, Buffler, Pery & Blackwell, 1981). The high cost is related to the use of expensive technologies such as ventilatory therapy. Prompt recognition and treatment of inhalation victims can keep mortality and hospital

stay to a minimum.

Anatomy of a Fire

As with any trauma, mechanism of injury is an important factor in assessment. The anatomy of fire related inhalation injury differs slightly from fire to fire and in each person in that fire (Hill, 1989). As materials burn, heat and toxic gases are spewed. The composition of these gases varies according to the fuel being burned, the temperature at which it is being burned and the amount of oxygen present (Haponik et al., 1988).

Respiratory injury can occur through heat, asphyxia, and/or toxic poisoning. Direct thermal injury is usually limited to the upper airways (Haponik et al., 1988). True thermal injury below the vocal cords is rare due to the reflex closing of the glottis and the ability of the airways to effectively cool the inhaled hot air. The exception to this rule is in the case of steam inhalation. Steam has about 400 times the heat carrying capacity of dry air (Cioffi & Rue, 1991; Lybarger, 1987). Since the water vapor retains its heat, inhaled steam carries super heated air down to the lung parenchyma where direct thermal damage can occur. A recent example of steam

inhalation injury was during the Persian Gulf crisis when a steam blast aboard ship caused the death of four seaman. On rare occasions, some heat injury may occur in the lower airways via soot particles that retain heat longer (Haponik & Munster, 1990).

Toxic Inhalants

Carbon monoxide and hydrogen cyanide are the principle asphyxiants in smoke inhalation. Both substances bind with cytochromes and interfere with oxygen delivery. In addition, carbon monoxide displaces oxygen from the hemoglobin molecule causing tissue hypoxia despite adequate ventilation and diffusion. Carbon monoxide may account for as much as 80% of the smoke inhalation fatalities (Haponik et al., 1988). Hydrogen cyanide is the most dangerous byproduct of combustion. Its rapid interference with cytochrome oxidase and consequent effects on oxygen utilization at the tissue level incapacitate inhalation victims more quickly than carbon monoxide and may prevent possible escape (Hill, 1989). Add to this the lack of oxygen already in the environment due to combustion and asphyxiation is more likely (Cioffi & Rue, 1991). Ambient air oxygen concentrations may drop as low as 5% in some fire environments (Beritic,

1990).

With the use of more and more synthetic materials, toxic injury has increased (Markowitz, Gutterman, Schwartz, Link & Gorman, 1989). This is mainly due to the fact that smoke contains both toxic gases and particles that are coated with toxic materials (Vanacker, Boeckx, Van Aken & Gruwez, 1989). Some of the toxic byproducts of incomplete combustion include: aldehydes, ammonia, chlorine, hydrogen chloride, acrolein, sulfur dioxide, nitrogen oxides, and phosgene. Some of these toxic gases are more lethal than others. Their lethality is due in large part to their water solubility as toxins with high water solubility tend to damage only the upper conducting airways. Also, their resultant injury appears clinically much sooner as the toxins are quickly absorbed into the upper airway tissue (Wald & Balmes, 1987). Conversely, toxins with low water solubility tend to pass completely through the air passages and down to the lung parenchyma. Low water solubility toxic injury tends to be more insidious (Haponik et al., 1988; Beritic, 1990).

This paper examines the physiology, assessment and immediate treatment of nonthermal inhalation

injuries. The insidious nature of these injuries requires a thorough knowledge of the proper assessment and prompt treatment to prevent unnecessary death and complication.

Chapter 2

In 1986, there were 4,885 fire-related deaths in the home in the United States (Loke, 1988). In the home, there are many forms of combustible materials including wood furniture, carpets, wall paper, plastic paneling, upholstery, plywood and particle board paneling, cellulose fiber, polyurethane material, papers and clothing (Loke, 1988). As a consequence of incomplete combustion of these materials, multiple toxic byproducts may be liberated in the form of gas and smoke.

Two forms of thermal degradation exist: combustion and pyrolysis. *Combustion* occurs when the thermal degradation process has enough oxygen to support flaming. Pyrolysis, on the other hand, takes place when there is insufficient oxygen to support complete combustion. The resultant chemical degradation creates smoldering and increases emission of toxic gases (Levin, Paabo, Fultz & Bailey, 1985).

Some examples of the toxic byproducts of combustion or pyrolysis are: hydrogen chloride and carbon monoxide from polyvinylchloride in wall and floor coverings; isocyanates and hydrogen cyanide from the polyurethane in upholstery; acrolein from cotton

and wood products; hydrogen cyanide, ammonia, and acrolein from carpets and carbon monoxide from polystyrene in styrofoam insulation products (Loke, 1988, Lybarger, 1987, Beretic, 1990, Haponik & Munster, 1990). It is most important to understand that few materials are pure. The result is a combination of these products being liberated in most fires. Therefore, the physiology which occurs as a result of smoke inhalation provides for an extremely complex picture.

Asphyxiants

The products of smoke can be divided into asphyxiants and irritants. The most dangerous of these products are the asphyxiants. These may cause incapacitation which can hamper escape efforts thereby prolonging exposure and causing an early death. Two of the chief asphyxiants are carbon monoxide and hydrogen cyanide.

Released during the incomplete combustion of most materials, carbon monoxide is the single greatest cause of death at the scene of a fire (Lowry, Juarez, Petty & Roberts, 1985). Carbon monoxide a colorless, odorless, and tasteless gas, has a 200 fold greater affinity for hemoglobin than does oxygen (Lybarger,

1987; Haponik, 1990). Furthermore, it shifts the oxyhemoglobin disassociation curve to the left thus impairing the ability of hemoglobin to release oxygen at the tissue level (Cioffi & Rue, 1991). In addition, because it binds to cytochrome oxidase, it is a putative inhibitor of oxidative phosphorylation. (Haponik, 1990).

Nonirritating to mucus membranes, carbon monoxide is not thought to directly cause lung damage (Mosely, 1988; Shimazu et al., 1990). Although oxygen saturation (SaO2) is decreased as carbon monoxide takes the place of oxygen on the hemoglobin molecule, arterial oxygen (PaO2), which measures the oxygen in the plasma, may be normal despite the decreased delivery to the tissues. Therefore, there may be no increase in ventilation until lactic acidosis occurs (Mosely, 1988).

Because the brain and heart are most susceptible to hypoxia, they are the organs at highest risk of injury due to carbon monoxide poisoning (Haponik, 1990; Shimazu, 1990; Sugi, Theissen, Traber, Herndon & Traber, 1990). Elevated heart rate and mean arterial pressures are seen in the early stages of carbon monoxide poisoning in sheep (Sugi et al., 1990). In

humans, it is likely that the same elevations would occur as the body attempts to correct the tissue hypoxia. Central nervous system findings correlate in large part with the level of exposure. Symptoms can range from headache to convulsions and death with the level of hypoxemia dependent on the length of exposure and the oxygen demand (Loke, 1988). Charts that create ranges of toxicity for certain symptoms presented are at best guesses. Current research findings have shown that incapacitating carbon monoxide levels may be much lower than previously thought (Hill, 1989). As a result, a victim has less potential escape time.

The second asphyxiant gas associated with fires is hydrogen cyanide. This colorless gas has a characteristic smell of burnt almonds. However, realistically this smell would be difficult to detect in the presence of other gases in a fire. Nitrogen containing materials, particularly plastics and polyurethane, will produce hydrogen cyanide during their combustion (Davies, 1986). Aircraft fires, where a good portion of the inside of the aircraft is plastic and seat cushions are polyurethane, have a high incidence of hydrogen cyanide production (Hill,

1989). In seconds a victim may begin to feel toxic effects such as headache, nausea, vertigo, and weakness. In acute exposures there may be severe nausea and vomiting, convulsions, and coma with death occurring in minutes (Loke, 1988). Physiologically, hydrogen cyanide interferes with the utilization of oxygen at the cellular level and inhibits cytochrome oxidase in the mitochondria. Thus, transported oxygen is unable to be utilized by the tissues and anaerobic metabolism ensues with a resultant lactic acidosis (Hall & Rumack, 1986). The associated respiratory stimulation further increases injury as hyperventilation occurs and toxicity increases.

Carbon monoxide and hydrogen cyanide function synergistically due to their additive effects. Carbon monoxide blocks oxygen uptake and release at the tissue level and hydrogen cyanide blocks oxygen utilization at the cellular level (Hill, 1989).

High Water Soluble Irritant Gases

As previously stated, the seriousness of damage from toxic gases depends largely on their solubility in water. Highly water soluble gases include: acrolein, ammonia, chlorine, hydrogen chloride, and sulfur dioxide (Haponik & Munster, 1990). Compounds

with low water solubility are the nitrogen oxides and phosgene (Haponik & Munster, 1990).

Onset of symptoms from the highly water soluble group occurs rapidly, but, reflex laryngeal spasm usually protects against lower airway damage during brief exposures. Consequently, the upper airway is usually the most severely damaged (Wald & Balmes, 1987). In conjunction with airway injury, these irritants also cause rhinitis and conjunctivitis. Lower airway injury is possible, however, when respirations are increased as with physical exertion, anxiety or metabolic acidosis (Haponik & Munster, 1990). A loss of consciousness increases the likelihood of long term exposure and subsequent lower airway injury. Irritant chemicals may also adhere to soot particles that may find their way to the lower airways causing direct injury to the lung parenchyma (Wald & Balmes, 1987).

One of the most highly researched chemicals is the aldehyde acrolein (Clark, Nieman & Hakim, 1990; Salahadin et al., 1990; Hales et al., 1989; Clark, Grossman, Ritter-Hrncirik & Warner, 1988; Niehas et al., 1990; Kimura et al., 1988; Abdi et al., 1990). Found in the combustion of wood, cotton, paper, and

petroleum products, acrolein exposure can occur in almost all modern day fires. Research findings have demonstrated concentrations of acrolein to be much higher in wood combustion than kerosine which was thought to be the most potent source of acrolein (Zikria, Ferrer & Floch, 1972). This fact suggests that acrolein exposure should be suspected in most smoke inhalation victims. A powerful irritant, acrolein in high concentrations may cause death in as soon as ten minutes of exposure (Terrill, Montgomery & Reinhardt, 1978). Acrolein, along with other aldehydes, causes denaturation of proteins in the airways and lungs which leads to pulmonary edema and death (Salahadin et al, 1990; Zikria et al., 1972).

Ammonia, commonly found in household cleaning products, is a colorless and highly pungent gas. Like acrolein it is highly irritating to the airway epithelium. Inhalation injury can occur while working with ammonia in a closed environment, such as a bathroom. Other sources of ammonia include: explosives, fertilizers, pharmaceuticals, and other chemicals. In fires, it may be produced by the combustion of wool, silk, nylon, and some plastics (Wald & Balmes, 1987). When ammonia contacts the

water in the upper airways ammonium hydroxide, a strong alkali, is formed. The result is liquefaction necrosis with edema and sloughing of the airway mucosa (Schwartz, 1987). Resultant injuries may include pulmonary and airway edema, airway obstruction, pneumonitis, bronchiectasis and bronchitis (Haponik & Munster, 1990). Ammonia may also cause acute blindness which may hinder the victims escape thereby increasing the length of exposure (Terrell et al., 1978).

Widely used in industry, chlorine is extensively used as a bleaching agent. Like ammonia, chlorine gas also has a pungent odor. This yellow-green gas is highly irritating to the airway epithelium as well. When combined with water vapor it produces hydrochloric acid and hypochlorous acid. Hypochlorous acid is about 20 times more toxic than hydrochloric acid (Wald & Balmes, 1987). Although highly soluble, it is the least soluble in this category. During the combustion of products that liberate chlorine gas, oxygen free radicals are generated (Schwartz, 1987). These radicals further damage the airway lining by reacting with polyunsaturated fatty acids in cell membranes causing loss of membrane fluidity and loss

of secretory function (Baue, 1990). Like many of the toxic gases, chlorine also irritates the eyes and can cause laryngospasm, bronchospasm, and pulmonary edema (Haponik & Munster, 1990).

Hydrogen chloride a colorless, corrosive gas with a pungent odor is generated through the pyrolysis and combustion of polyvinylchloride, acrylics and fabrics with fire retardant materials. Dyes, fertilizers and rubbers also contain hydrogen chloride. The clinical picture of a victim with hydrogen chloride toxicity may include acute bronchitis, airway edema, laryngospasm and pulmonary edema (Schwartz, 1987). In combination with water vapor, hydrogen chloride forms hydrochloric acid. This corrosive chemical damages the mucosal membranes and hinders mucociliary function (Loke, 1988).

Chemical, paper, and smelting industries like steel mills widely use sulfur dioxide. It is the major air pollutant produced by the combustion of sulfur containing fossil fuels (Leaderer, 1982). Twice as dense as air, sulfur dioxide has a strong suffocating odor. In low exposures it may cause eye irritation and bronchoconstriction. Danger of fatality increases if sulfur dioxide attached to

particulate matter is carried to the lower airways. Resultant vasoconstriction can increase hydrostatic pressure and increase the severity of pulmonary edema (Schwartz, 1987).

Low Water Soluble Irritant Gases

Low water soluble gases are more likely to cause lower airway damage and to have a delayed onset of detectable injury. Little eye or nasal irritation occurs which may cause the victim to stay in the environment longer and increase his/her length of exposure.

Nitrogen oxides are found in explosives, chemical welding, and cleaning industries. They are produced by the combustion of fabrics and celluloid products. There are few if any immediate symptoms and virtually no upper airway warning symptoms. Acute injury may not be evident for 5 to 72 hours (Gosselin et al., 1976). It is the acids formed when these nitrogen oxides combine with water that are thought to create the most damage (Haponik & Munster, 1990).

Phosgene has been examined extensively in the military field as it has been used as a chemical weapon against troops. Phosgene is used in the synthesis of organic compounds and in the separation

of metals. In fires, it is produced through the combustion of polyvinylchlorides. Quantities in most fires are very small and contribute little to the toxic effects of smoke (Parkes, 1982). Phosgene is considerably more toxic than chlorine. In high concentrations, phosgene may cause a cough but usually has little or no initial symptoms. The delayed primary sites of injury are the small airways and the alveoli (Wald & Balmes, 1987). Phosgene's carbonyl group reacts with a variety of amines and hydroxyl groups thus affecting cell wall stability particularly in the capillaries, thereby, affecting permeability (Loke, 1988). *Pulmonary edema* and pneumonia can occur as the result of necrosis of the epithelial layer (Loke, 1988).

While understanding what each of the individual toxic gases may contribute to injury is helpful in assessing the smoke inhalation patient, rarely do health providers know exactly how much or to what specific chemicals patients have been exposed. It is, therefore, important to realize that combinations of these toxic gases are what occur in smoke inhalation. A thorough assessment and history will narrow the list of possible toxic exposures so that treatment,

specific to the likely substances, may begin as soon as possible.

Overview of Smoke Inhalation Physiology

The physiologic mechanisms of injury from smoke inhalation are multiple and complex. Even with the exclusion of toxic gases the victim may be hypoxic due to a lack of oxygen in the ambient air as the process of combustion utilizes the oxygen in the environment. This lack of oxygen is more likely in a closed space (Large, Owens & Hoffman, 1990). Concurrently, asphyxiation is also possible then through either carbon monoxide or hydrogen cyanide exposure. Asphyxiation leads to cerebral hypoxia which can render a victim unconscious, reduce his chances for escape and prolong his exposure causing greater inhalation of asphyxiants and other toxic gases. This vicious cycle is the most common mechanism of death at the scene (Hill, 1989).

Toxic gas exposure precipitates a long chain of damaging events. Consequences of this exposure can include: impairment of mucociliary function, mucus hypersecretion, inflammatory responses such as bronchitis and bronchiolitis, alteration in the biochemical factors in the lung, increased vascular

permeability which can lead to pulmonary edema, and bronchoconstriction and mucus plugging which can lead to increased shunting (Loke, 1988; Barrow, Morris, Linares & Herndon, 1991).

More specifically smoke inhalation can cause direct damage to the epithelial layer. Partial denudation of the tracheal epithelium may occur as early as 30 minutes after inhalation (Salahadin et al., 1990; Barrow et al., 1991). The inflammatory process is initiated by direct injury to the epithelial cells from chemicals in the smoke (Cioffi & Rue, 1991). Cells are sloughed off as they lose their attachments to the basement membrane (Salahadin et al., 1990). This sloughing can lead to airway blockage since mucociliary function can not adequately remove the debris (Cioffi & Rue, 1991).

In smoke inhalation, alveolar macrophages, the lung's main defensive mechanism, are activated (Riyami et al., 1991; Clark et al., 1988). Being the principle phagocytic cell in the lung, macrophages engulf smoke particles and debris that enter the lung, but not without further consequence. The activation of these cells give rise to a series of mediator cascades. Chemotactic substances are released and

neutrophil margination occurs. As neutrophils phagocytise the debris, oxygen free radicals as well as proteases are released (Ghio, Kennedy, Hatch & Tepper, 1991). Further cell death and sloughing of the epithelial layer occurs. Airway casts created by mucin, leukocytic components of the blood, fibrin and the trapping of epithelial cellular debris in the smaller airways may also develop in 12 to 36 hours thus creating a greater ventilation perfusion mismatch as poorly ventilated areas of the lung are perfused (Haponik et al., 1988).

Direct damage to type II cells in the lung leads to decreased surfactant. The resultant loss of surface tension and loss of the fluid barrier leads to atelectasis and pulmonary edema (Wang, Li & Yang, 1990).

Insert Figure 1 about here

Activation of phospholipase initiates the arachadonic acid cascade with all its constituents. These constituents include: leukotrienes, prostaglandins and thromboxane. The coagulation and compliment cascades are also initiated causing

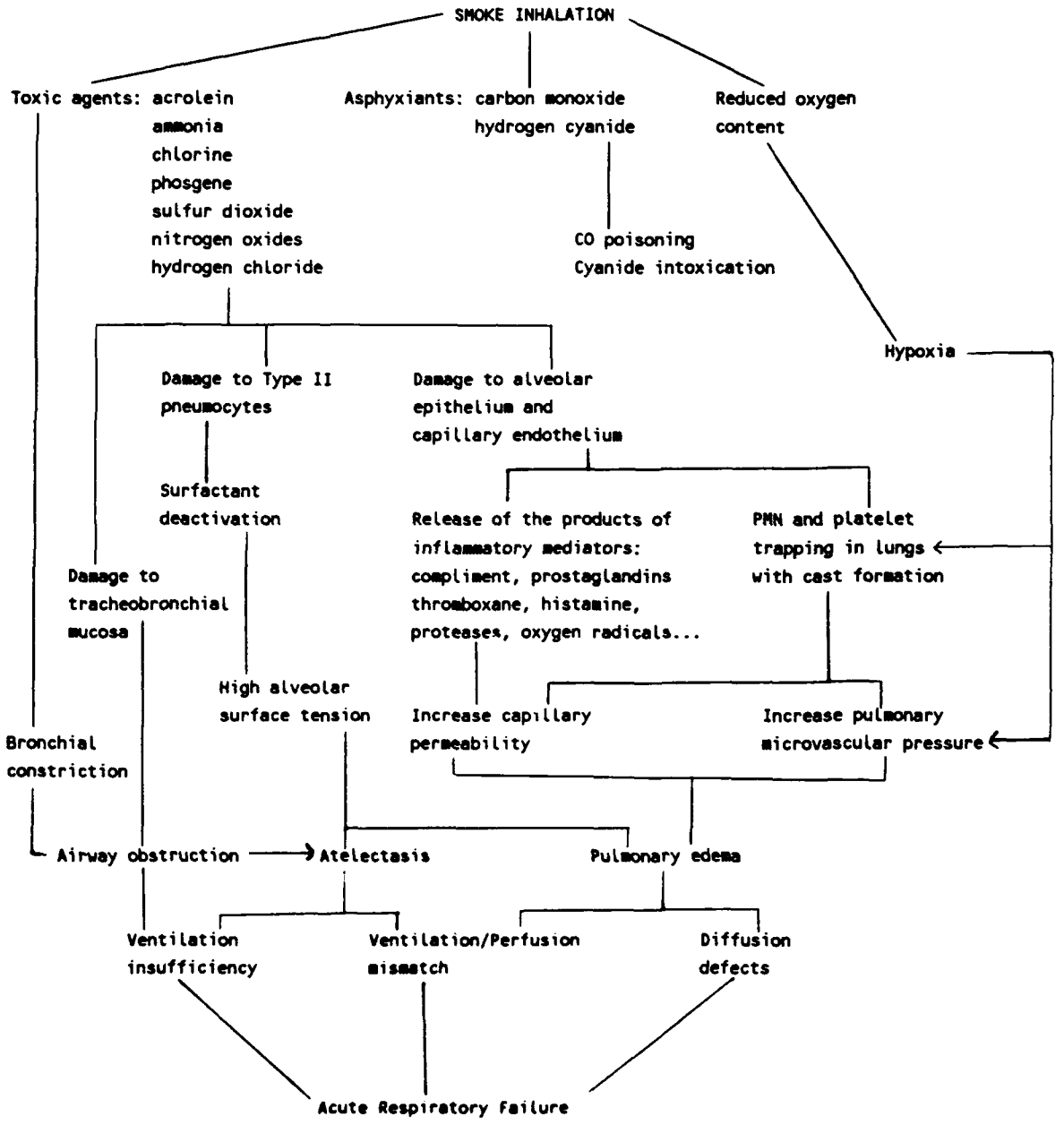

Figure 1. Cascade of events in smoke inhalation.

(Adapted from Wang et al., 1990)

platelet and neutrophil aggregation further blocking airways and releasing oxygen free radicals (Haponik & Munster, 1990). Figure 1 is an overview of the complex cascade of events.

Chapter 3

Assessment is the key to early recognition of potential inhalation injury victims. Due to its incipient nature, accurate and thorough assessment may be the difference between life and death to these patients. Although there are many assessment factors likely to rule in inhalation injury, their absence does not rule inhalation injury out (Haponik & Munster, 1990). Herein lies the main difficulty in recognition of potential inhalation injury victims.

History of the mechanism of injury is very important. It is vital to gain as much information on the patient scene as possible. Color and density of the smoke can be an indicator of the type and concentration of the toxic chemicals. Level of consciousness at the scene may be an indicator of the level of carbon monoxide or cyanide exposure. This type of information is best obtained from Emergency Medical Service (EMS) personnel at the fire. Aside from the physical care given at the scene, prehospital personnel have an important role in future treatment by noting the environment and obtaining information on the toxins involved. A notation of the time oxygen was supplied should be made so that hospital personnel

can approximate the peak level of carbon monoxide. Additionally, bystanders may be helpful in determining the approximate length of exposure.

Whenever possible, the fuel burned should be identified. Knowing what fuel was burned will aid in identifying the possible toxins inhaled. If the fire occurred at an industrial site, plant management or other employees are likely to know exactly what has burned. In a house fire it must be assumed that the smoke was a combination of most of the toxins discussed earlier. Figure 2 includes a list of important questions to ask any patient with a potential inhalation injury.

Insert Figure 2 about here

When firefighters are patients, it is important to obtain the following information: what job was held in fighting the fire, what protection was worn, duration of their work, and whether or not they worked with smoldering material. In addition to knowing if they wore protective equipment, it is important to know whether or not they had their mask on continuous or intermittent and whether the mask was set to low

QUESTIONS FOR THE SMOKE INHALATION VICTIM

1. How long was exposure?
2. What color and denstiy was the smoke?
3. Do you know what was burning?
4. Were you above or below the flames?
5. Did you lose consciousness?
6. Was it in an open area or an enclosed space?
7. What symptoms did you have while exposed?
8. Do you have any past history of respiratory or cardiac illness?
9. Have you had alcohol or any drugs?
10. Did you have any chemicals stored near the area of the fire?
11. Did you notice any hoarseness or difficulty in swallowing?
12. Was there more fire or smoldering?

Figure 2. Pertinent questions in the smoke inhalation victim.

flow or demand. A continuous system at low flow will pressurize the mask and continually flush the air around the sides of the mask (Crapo & Ellis, 1980). This is the best method of protection, but it is also the quickest way to empty a tank of air. Studies have indicated, however, that the use of an intermittent mask is as effective as no mask at all and offers virtually no protection against carbon monoxide poisoning (Levine, 1979).

Findings in the physical assessment can range from a patient with burns of the face, soot in the airway, altered level of consciousness, altered blood gases and airway obstruction; to a patient who merely has the subjective feeling of shortness of breath without other obvious signs. Inhalation injury may occur without cutaneous burn injury (Cioffi & Rue, 1991).

The provider may find the patient with an inhalation injury on presentation to have normal breath sounds, normal chest radiographs and normal blood gases (Blinn et al., 1988). This does not rule out inhalation injury. These tests, however, must be taken for baseline analysis and comparison of future values. Those patients who enter the emergency room

with positive findings such as altered blood gases, abnormal chest radiographs or noisy breath sounds can have a rapidly fatal progression.

Carbon monoxide toxicity, upper airway obstruction and tracheobronchial obstruction are early events and usually present within the first hours after exposure (Haponik & Munster, 1990). Therefore, these areas should be evaluated first. Carbon monoxide affects the amount of oxygen attached to hemoglobin and cyanide affects the utilization of oxygen at the tissue level. Neither, affects the plasma level of oxygen. Blood gas determination, therefore, should include carboxyhemoglobin levels and direct oxygen saturation levels, since PaO2, which only measures plasma oxygen, may be normal in the presence of carbon monoxide or hydrogen cyanide poisoning. Direct visualization of the airway should be performed if the patient has numerous risk factors and signs such as: facial or neck burns, altered level of consciousness, alcohol or drug intoxication, singed nasal hairs, expectoration of carbonaceous sputum, physical impairment that may have hampered escape, history of an exposure in a enclosed space or known involvement with respiratory corrosive chemicals

(Cioffi & Rue, 1990). As with any patient, past medical history and age are also important assessment factors.

Insert Table 1 about here

Signs and symptoms that may occur are listed in Table 1. Any number of these may be present in an inhalation victim. Dyspnea, cough, and hoarseness are very common in smoke inhalation victims, but are often transient and self-limiting findings which may clear shortly after removal from the smoke filled environment (Haponik & Munster, 1990). Chest tightness and pain may also occur. Dyspnea may suggest early parenchymal injury, wheezing may indicate early bronchitis from chemical irritation, and hoarseness may be an early sign of oropharyngeal injury and swelling (Haponik & Munster, 1990). Diagnosis is very difficult in the early stages. Time and serial assessment are the only truly useful tools in these instances.

Altered breathing patterns are vital clues to underlying pathology. Tachypnea, rapid breathing, may be a sign of inhalation injury or anxiety. Hyperpnea,

Aphyxia	Altered sensorium, coma, nausea, head ache, seizure
Upper airway obstruction	Stridor, sore throat, dysphagia, hoarseness, pharyngeal edema, history of flash exposure, respiratory orifice burns or burns of the neck, use of accessory muscles to breathe
Lower airway obstruction	Dyspnea, tachypnea, wheezing, rhonchi, carbonaceous sputum
Parenchymal injury	Dyspnea, tachypnea, rales

Table 1. Signs and symptoms at different levels of injury.

abnormally deep breathing, may indicate a metabolic acidosis as the body attempts to blow off excess carbon dioxide (Haponik & Munster, 1990). Greater force is required to move air through narrowed airways, therefore, use of accessory muscles may be an important sign of respiratory distress and possible obstruction. An unstable pattern of breathing or apnea are ominous signs and require prompt airway support.

Although chest auscultation may not rule out inhalation injury, it continues to be an important assessment tool. Auscultation should take place over all lung fields as well as the neck. Stridor is the high pitched sound created as air attempts to pass through a narrowed upper airway, and is indicative of upper airway obstruction. This narrowing demands immediate attention. An altered voice and difficulty swallowing also fall into this category. Almost half of the victims of smoke inhalation experience wheezing at some point during their course secondary to bronchospasm and excess secretions (Haponik & Munster, 1990). Smoke exposure can exacerbate bronchospasm in those with a prior history of asthma or chronic obstructive pulmonary disease. The lower pitched

rhonchi are commonly heard in the initial care and are related to bronchorrhea and are indicative of chemical injury. Early lower airway rales indicate bronchitis, whereas late rales may be more indicative of pulmonary edema (Clark, 1990). The silent chest is the most ominous sign of all. A patient who clears wheezing, but has diminished breath sounds is likely to be in worse shape than during the wheezing. The reason for this is that air flow at this point is so low there is not enough air flow to create a wheeze.

Spirometry may assist in identifying those with delayed symptoms of inhalation injury. With inhalation injury there is usually a decrease in the forced expiratory volume in one second (FEV1) after 4-6 hours. FEV1 less than one liter should be regarded as a severe defect (Haponik & Munster, 1990).

Neurologic alterations vary depending on the type of exposure. Signs and symptoms such as headache, dizziness, hallucinations, and seizures may be important indicators of exposure to asphyxiants. Nausea, vomiting, and abdominal pain are more prevalent in cases of chemical toxin exposure.

Cyanide blood levels are necessary when there is persistent acidemia and obtundation that is

inappropriate for the carboxyhemoglobin level (Haponik et al. 1988). In one study, 90% of 43 patients who were dead on arrival had abnormal cyanide levels, with two thirds having lethal levels (Symington & Anderson, 1978).

Radionucleotide scanning with Xenon is another evaluative tool, but it is expensive (Peitzman, Shires, Teixdor & Curreri, 1989). While somewhat specific, it too can create false positives, especially with smokers (Clark et al., 1988). A new substance is needed that will more accurately reflect airway and parenchymal damage. Currently, studies on dogs are in progress which examine the use of aerosolized Tc-Diethylenetriaminepentacetate clearance for diagnosing inhalation injury. Animal tests have thus far shown it to be more reliable than chest radiographs or Xenon scans and may prove valuable in the years ahead (Clark et al., 1988).

If the patient is being assisted with ventilations, compliance and resistance values are helpful in diagnosing and locating injury. With inhalation injuries, compliance is decreased due to parenchymal destruction and surfactant loss and resistance is increased as bronchoconstriction and

swelling narrow the airways. Static compliance should be used to better differentiate true elastic changes versus airway resistance changes (Jones, Barrie, Madden, Finkelstein & Goodwin, 1988). Dynamic compliance uses peak inspiratory pressure as one of its components, therefore, it does not separate out airway resistance factors.

Assessment of the smoke exposed patient is very complicated. Prediction of clinical course and extent of damage is very difficult. Assessment over time is necessary to truly rule out an inhalation injury. Often it may take as long as 4-6 hours for a patient to show any signs or symptoms. Table 1 is a synopsis of the clinical findings present at the different levels of injury. There is much overlap, but, it does provide general guidelines.

Chapter 4

As stated previously, there have been fewer advances in the care of inhalation injuries than there have been in burn care (Langford & Armastrong, 1989). Parenchymal injury has especially poor patient outcomes. Currently, researchers are studying numerous agents that may aid in the prevention of developing parenchymal injury.

One of the few treatments researchers unanimously support is the use of oxygen as an initial treatment. In fact, the mere history of smoke inhalation warrants oxygen administration. Initial care must be aimed at securing the airway as well as providing adequate oxygenation. This is accomplished in a variety of ways. The most obvious treatment is the administration of 100% oxygen via face mask starting at the scene. Early oxygen administration decreases the half-life of carbon monoxide and prevents hypoxia. Carbon monoxide is the leading cause of death at the scene of house fires, accounting for 66% of all house fire deaths in 1980 (Baker, 1984). Therefore, immediate on-scene oxygen administration may lower mortality associated with smoke inhalation.

The potential for or presence of upper airway

obstruction, as evidenced by stridor, hoarseness, pharyngeal edema or unconsciousness, necessitates endotracheal intubation. At a minimum, these patients should have an oral or nasal airway inserted and a 100% oxygen administered.

Once in the emergency room, aerosolized racemic epinephrine in combination with elevation of the head of the bed may be all that is necessary to treat mild to moderate upper airway edema (Cioffi & Rue, 1991). It must be kept in mind that upper airway edema can increase and these patients should be monitored closely in an intensive care environment. Airway management equipment should be readily available at the patient bedside. Endotracheal intubation as well as cricothyrotomy equipment should be close at hand in the event that an alternate airway is needed emergently.

In cases of severe upper airway edema, endotracheal intubation is necessary. Nasotracheal intubation is preferred as it is better tolerated by the patient and can be more firmly secured. In nonthermal injuries, upper airway injury due to smoke usually resolves in three to five days so that oral intubation would not be disadvantageous and may be

more easily inserted by some hands (Haponik & Munster, 1990).

Mild tracheobronchial injury may be treated with the use of frequent pulmonary toilet such as coughing, deep breathing and incentive spirometry since mucociliary function may be decreased. In the intubated patient, suctioning should be considered if a patient shows signs of hypoxia such as aggitation, restlessness, cyanosis, decrease level of consciousness, and rapid heart rate or if arterial blood gases show hypoxia or low oxygen saturation. Suctioning for its own sake should be avoided as it too lowers oxygen blood levels temporarily (Cioffi & Rue, 1991). If suctioning is necessary, hyperventilation and preoxygenation with 100% oxygen should be used to avoid additional hypoxia.

Bronchospasm may be treated with bronchodilators such as albuterol, metaproterenol and terbutaline. Their prophylactic use however, has not been shown to be successful (Haponik & Munster, 1990). Aminophylline is the intravenous drug of choice (Cioffi & Rue, 1991). Parasympatholytic agents such as atropine may be useful if given aerosolized and in low doses that do not create cardiac side effects

(Haponik & Munster, 1990). More studies with the use of Atropine in this manner are needed to prove its efficacy.

The use of steroids is controversial. Steroids, when used in patients with no thermal injuries, are useful in decreasing airway inflammation in the acute stages during the first twenty four hours. In inhalation injuries with concomitant thermal burns researchers have shown that steroids have no effect and may even increase the mortality rate up to four times those not treated with steroids (Haponik et al., 1988).

The effect of post-end expiratory pressure (PEEP) has also been examined in sheep with smoke inhalation (Abdi et al., 1990). With PEEP levels of <=15mm cm H2O, bronchial blood flow was shown to decrease with no effect on hemodynamics. In theory, damage due to neutrophil margination may be slowed if the bronchial flow is decreased. The less blood to the site the less neutrophils to the site. One such study revealed a decrease in bronchial flow without change in hemodynamics but failed to show a significant change in mortality of the sheep (Abdi et al., 1990). With the known value of PEEP in preventing atelectasis and

the potential value of decreasing mediator injury it would seem prudent to use low levels of PEEP as long as there was no hemodynamic compromise. Further research is needed in this area on animals and eventually on humans before the use of PEEP is conclusive.

For patients with suspected or proven carbon monoxide poisoning, hyperbaric oxygenation is an option. Hyperbaric oxygenation can reduce the half-life of carbon monoxide from 5-6 hours to 30 minutes by increasing the partial pressure of oxygen. This greater availability of oxygen aids in more quickly displacing carbon monoxide from the hemoglobin molecule (Meyer, Hart & Strauss, 1991). It is not, however, a panacea. Oxygen alone can reduce the half-life of carbon monoxide to as low as one hour (Mellins & Park, 1975). Hyperbaric oxygenation may produce little if any effect in some cases and has potential complications. This therapy is most appropriately used with a comatose patient who on admission has a carboxyhemoglobin of greater than 30% or when there are neurological deficits present and a chamber is nearby (Haponik & Munster, 1990). Patients with severe burns or other trauma must be evaluated

carefully. Hyperbaric treatment should begin only after the risk to life, engendered by the delay in other treatments that hyperbaric therapy may cause, has been weighed against possible benefits. While quite successful in lowering carbon monoxide blood levels, hyperbaric oxygenation has yet to prove effective in changing final patient outcome in inhalation injury (Haponik et al., 1988).

Cyanide intoxication is one of the most underassessed and underestimated problems. In fires where there have been a large amount of plastics or furniture burned, cyanide poisoning should be considered. Symptoms are similar to those of carbon monoxide poisoning, which should not be surprising given their asphyxiant effects. Cyanide inhibits the final step in oxidative phosphorylation. Aerobic metabolism is halted, resulting in lactic acidosis despite normal blood oxygen values. Cyanide poisoning should be suspected and treated in the presence of lactic acidosis with nonresolving obtundation (Haponik et al., 1988). Serum levels may take as long as four to six hours to run. Cyanide levels >.2mg/L should be treated (Haponik & Munster, 1990). The cyanide antidote regimen of inhaled amyl nitrite, sodium

nitrite, and sodium thiosulfate must be started promptly. Amyl nitrite and sodium nitrite are used to produce methemoglobin that competitively binds with cyanide thus displacing it from cytochrome oxidase. Sodium thiosulfate irreversibly binds with cyanide to form thiocyanate thus preventing redevelopment of symptoms (Haponik & Munster, 1990). The synergistic effects of carbon monoxide and cyanide can and do prove rapidly fatal.

Fluid administration should be based on whether the inhalation injury occurs alone or combined with surface burns and according to what other injuries or diseases may be present. Although the reason is not yet known, greater fluid is required when burn injuries have concomitant inhalation injuries (Navar, Saffle & Warden, 1985). With or without inhalation injury the patient should be carefully monitored for signs of pulmonary overload such as increased dyspnea, orthopnea and wet lung sounds. A delicate balance needs to be maintained. Not enough fluid will disrupt hemodynamic balance. Urinary output, hemodynamic status, and respiratory status should be used to guide the amount of fluid administered. Strict adherence to any one formula without careful monitoring could prove

fatal since adherence to these formulas does not guarantee against fluid overload and further respiratory compromise.

The administration of prophylactic antibiotics has not been shown to prevent subsequent pneumonia. They may, in fact, create a more resistant strain of bacteria if and when pneumonia does develop. Therefore, their use in the initial treatment of inhalation injuries is not recommended (Haponik et al., 1988).

All of the above methods of treatment are currently used and recognized. They, however, do little to change the outcome of patients with lower pulmonary parenchymal damage. Recent studies are aimed at reducing the destruction of the epithelium in the lower airways. A 1988 study showed nebulized dimethysulfoxide, an oxygen radical scavenger, to be successful in attenuating lung lesions following smoke inhalation (Kimura et al., 1988). Another study in 1990 demonstrated that the intravenous use of the synthetic antiprotease gabexate mesilate in sheep significantly attenuated the transvascular fluid and protein flux in the lung, thereby decreasing diffusion abnormalities (Niehaus et al., 1990). However, to the

contrary, a study in 1990 failed to show any effect of pretreatment with allopurinol, a xanthine oxidase inhibitor, on the lung damage found in sheep with smoke inhalation (Ahn et al., 1990). It remains to be seen if free radicals or proteases are the main culprit in lung parenchymal damage. The success of the first two treatments warrants further research in this area.

Another treatment that requires more investigation is the use of high-frequency percussive ventilation (HFPV) in patients with inhalation injury. HFPV has a ventilatory frequency of greater than 60 breaths per minute, tidal volumes less than dead space and lower peak airway pressures. While revealing their effect in creating better blood gas values and in increasing bronchial clearance, research has yet to prove HFPV's ability to decrease mortality as a final outcome (Cioffi, Graves, McManus & Pruitt, 1989).

Advances in treating lower airway damage will require more study. There seems to be some consensus on neutrophil margination as the culprit (Riyami et al., 1990; Basadre et al., 1988; Barrow, Morris, Basadre & Herndon, 1990). Clinical trials with realistic ways to combat this margination after smoke

inhalation are only just beginning. Further research in lowering the destructive effects on healthy lung tissue from the toxins released by neutrophil phagocytosis of smoke debris is necessary if there is to be a treatment that significantly effects mortality in the smoke inhalation patient.

Chapter 5

The responsibilities of the clinical nurse specialist (CNS) in the emergency setting lie in five roles: expert practitioner, educator, researcher, consultant and administrator. All of these areas are vital to assure quality care for the smoke inhalation patient.

Expert Practitioner

As an expert practitioner, the Emergency CNS models excellence in emergency nursing practice utilizing advanced concepts in all areas of practice (Emergency Nurses Association, 1991). Direct care allows the emergency CNS to attain competency with new equipment, medications and procedures while maintaining competency in previously learned areas. In the smoke inhalation patient, the emergency CNS demonstrates through thorough assessment the ability to adequately triage those cases who have the potential for the development of life-threatening respiratory complications from their smoke inhalation. The CNS also quickly formulates accurate actual and potential nursing diagnosis and plans for the immediate life-saving interventions necessary. Lab values are also interpreted by the CNS and abnormal

results are communicated quickly to the physician for prompt intervention. Care options are discussed with the physician to assure the best possible care for the patient. The emergency CNS also assures that all of the appropriate equipment is in place and functioning while allowing for the most cost effective use of the equipment. Interventions are performed skillfully and timely. Care provided is evaluated constantly to note alteration in patient status or to foresee possible complications of smoke inhalation and intervene in order to prevent their development. Even in the predominantly asymptomatic smoke exposed patient the CNS recognizes the potential threat and provides for the monitoring of such a patient until such time when it is safe to discharge him. Collaboration with emergency physicians on protocols regarding smoke inhalation patients will aid in preventing premature discharge. In advance, the CNS develops algorithms and standards of care for smoke inhalation patients for all staff members to utilize. Figure 3 is one such algorithm.

Insert Figure 3 about here

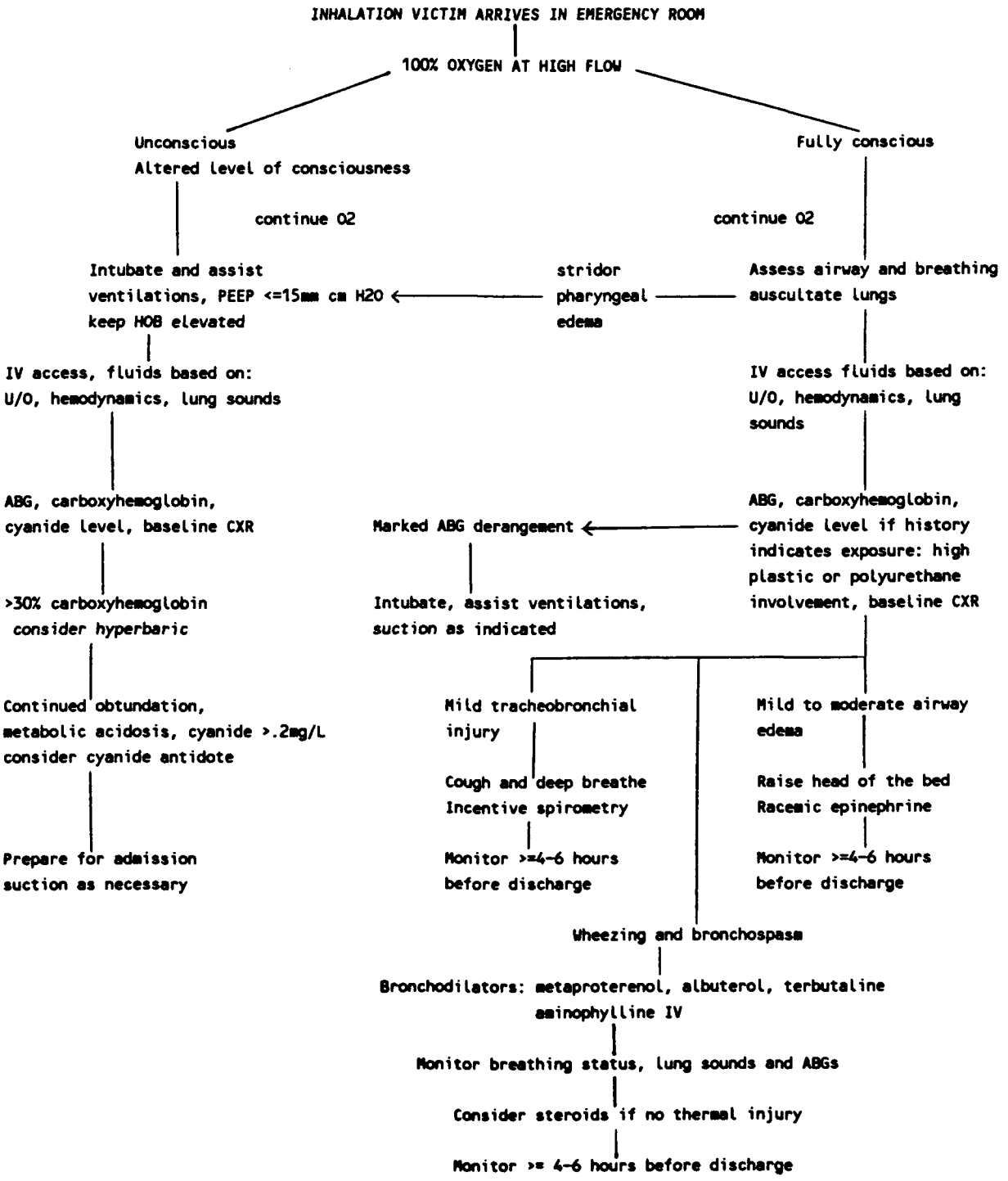

Figure 3. Algorithm for the inhalation injured patient.

This direct care must also include interacting with the families of smoke inhalation patients. Providing information on patient condition and treatments, for example hyperbaric therapy, can help to allay some of the anxiety in the family during the long waiting process. Families also need to be informed about an impending transport, the reason for the transport and the location and capabilities of the receiving facility.

Educator

The CNS is a resource for all staff members in the emergency room. In nursing, the educator roles include patient educator, staff educator, and student educator (Hamric & Spross, 1989).

With the aid of a needs assessement tool, the CNS assesses the level of knowledge currently held by the emergency staff regarding inhalation injuries. Caring for patients with smoke inhalation injuries may rarely be experienced by some nurses. The CNS develops programs that include: the nature and severity of these complex injuries, the toxicity of various chemicals involved in most fires, updates on the latest treatments, toxic substances present in large quantities in the community that may serve as fuel in

a large industrial fire and case scenarios that actively incorporate what they have learned.

In addition, disaster preparedness exercises could include symptomatic and asymptomatic inhalation injury victims as well as those with concomitant burns and those with no burns. Realistic patients would have injuries ranging from severe respiratory distress to those with few or no symptoms that develop respiratory difficulty over time. Hazardous materials exercises should also include inhalation injuries. Although beyond the scope of this paper, information from the knowledge of smoke inhalation can be applied to those who have been exposed to some of the toxins discussed without combustion.

Student education would include the education of new orientees to the care of inhalation injury patients as part of an orientation curriculum. The CNS may also act as an expert lecturer at university schools of nursing, at both the undergraduate and graduate level, in the area of pathophysiology and care of the inhalation injured patient.

As a patient educator, the CNS has the responsbility for preparing the smoke exposed patient for discharge. Instructions would include:

recognition of signs and symptoms of respiratory problems that would indicate a need for further evaluation and treatment, proper dosage and administration of medications, possible side-effects of the medications, medical follow-up plans, and preventative education as warranted. For those patients who are more seriously injured, information on diagnosis, equipment, treatments and procedures will help to alleviate some of the anxiety. Also education on coughing and deep breathing exercises as well as the effective use of incentive spirometry are necessary with increasingly longer stays in the emergency room.

Researcher

The involvement of the emergency CNS in research is vital to the advancement of the profession and the maintenance of quality patient care. With the recent surge in research on inhalation injuries, the CNS has the tough job of keeping up with the latest in treatments. This requires careful analyzing of the latest research literature on smoke inhalation for appropriate research methods and conclusions. The new information can then be disseminated to the entire emergency staff.

If the hospital has the capability and patient population to aid in this research, the CNS collaborates with the medical and research staffs to insure patient safety and research reliability. The CNS also assists in obtaining the grants necessary to perform the research.

Research on nursing interventions related to smoke inhalation is also necessary to provide for optimal patient outcomes. Topics could include the effects of suctioning, percussion, incentive spirometry, positioning and the value of expiratory force measurements as an assessment tool in the emergency room. Fellow staff members need to be recruited to assist with research to allow for more patient sampling and to give the staff the opportunity to experience research first hand. Staff can also assist the CNS in turning clinical problems into research questions suitable for investigation.

Once research is completed on smoke inhalation the CNS writes scholarly publications and disseminates these findings through research presentations. The CNS can also act as proof reader for others who are writing papers in the area of smoke inhalation thus enabling the CNS to receive the latest research

results even sooner.

Consultant

According to the ENA (1991), the emergency CNS has as part of the role of consultant the responsibility for coordinating emergency patient care with appropriate resource referral and utilization of other health care team members. Coordination with other hospitals assures that patients with smoke inhalation are referred to the appropriate facility to meet their needs. Locating the nearest hyperbaric facility and having the predetermined time frame for transit will assist in making triage decisions.

The CNS is a consultant to the health care consumer as well. Education in the emergency setting should also include education in the area of prevention. Emergency rooms should work with EMS personnel, firefighters, local schools and community organizations to prevent inhalation injuries, particularly in the area of fire prevention and fire early detection systems for home and industry. Activities could include school talks, fire prevention pamphlets and courses for occupational health nurses specific to the particular hazards in their setting. New information should be shared with firefighters,

EMS personnel, and occupational health workers who are most likely to become involved with inhalation injury patients. The goal is to decrease the mortality and morbidity of inhalation injury patients and this is best accomplished by educating the people who will first see and treat these victims.

The emergency CNS also uses expert knowledge to persuade legistlators to enact laws that may prevent needless inhalation injury. These could include stricter fire and safety codes in single family dwellings, apartments, hotels and office buildings.

Administrator and Leader

As a manager of an emergency room, the CNS can develop policies and procedures that relate to smoke inhalation. The manager can assure that the information and resources needed to deal with smoke inhalation victims is readily available. This may include phone numbers for poison control or hazardous materials experts, a computer system that can access information on the byproducts of the combustion of various materials, numbers and information on life flight in the area and the nearest hyperbaric chamber as well as protocols for patient referal.

Assessment guidelines and algorithms for the

treatment of inhalation injuries should be readily available in treatment rooms where resuscitation of these victims would take place. Also in this area a cyanide antedote kit and alternate airway equipment such as endotracheal tubes, oral airways, bag valve devices or ventilator, and cricothyrotomy equipment should be near the bedside. Collaboration with the emergency physicians is needed to establish a clear protocol for transfer and use of hyperbaric treatments to prevent unnecessary delays in treatment or unwarranted use of hyperbarics.

In order to maintain the best possible care for patients, the manager develops quality assurance tools to indicate patient outcomes and level of care of the inhalation injured. Problem areas are identified and plans of action are developed to resolve the problem or develop research to answer research questions.

Summary

Inhalation injury is a very complex process. Definitive damage may not be recognized for several hours after injury. Because of this insideous progression it is necessary for the CNS to be well versed on the potential physiologic alterations that may occur as a result of exposure to these toxic

byproducts of combustion. The assessment of the inhalation injured patients then includes all of the items in Figure 2 to narrow the treatment more specifically to the toxins involved.

As an educator, the CNS shares this knowledge with the entire staff to provide continuity of care. As researcher, the CNS researches new drugs currently being studied that may assist in preventing the mortality associated with lung parenchymal damage. These studies are reviewed and when there is sufficient proof of their success the CNS works with medical and research staff to develop studies for their use with smoke inhalation patients.

As a leader, the CNS provides the best chance for survival for smoke inhalation patients by: collaborating with physicians to establish protocols, assuring the adequate supply and quality of equipment and supplies for resuscitation, assuring appropriate referal and transportation to other institutions as warranted, monitoring the implementation of new therapies to assure safety and effectiveness, and by promptly giving direct expert care.

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