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## CYANIDE: FIRE SMOKE'S OTHER "TOXIC TWIN"

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On December 2, 2010, the International Association of Fire Chiefs and the International Association of Fire Fighters launched an educational campaign called "The Silent Killer" ([www.thesilentkiller.net](http://www.thesilentkiller.net)) to emphasize the hazards of occupational exposure to carbon monoxide (CO) and to reduce the known risk factors that can kill or injure firefighters. This campaign is highly laudable and can do a great deal toward increasing awareness of the hazards of CO on the fireground and the necessity for proper wearing and use of respiratory protection and air discipline.

What this campaign *does not* emphasize is the significant role of the other "toxic twin" in fire smoke: cyanide (CN<sup>-</sup>). This review was performed to emphasize the role of cyanide in the acute toxicity of smoke inhalation and its probable role in line-of-duty deaths (LODDs) and sudden cardiac illness.

This article is intended to accomplish two objectives. The first is to raise awareness on the issue of the toxicity of fire smoke in general and to more clearly focus on CN<sup>-</sup>'s role in smoke-related illness. This point should not be underestimated; CN<sup>-</sup> is truly as much or more of a silent killer than CO or any other fire gas. The second is to provide you with the scientific research to support the claim that CN<sup>-</sup> is present, to some degree, at virtually every fire to which you respond *and* should be considered when treating firefighters or civilians suffering from smoke inhalation.

Factual information must be used when making claims regarding the composition of fire smoke and its constant presence in a firefighter's line of work. Additionally, solid and factual medical information should be used when training personnel responsible for assessing and treating smoke inhalation victims.

In 1974, George Kimmerle listed eight major factors that can cause death in fires,<sup>1</sup> which follow:

1. Direct consumption by the fire (flame contact).
2. Very high temperatures.
3. Oxygen deficiency.
4. Presence of CO.
5. Presence of other toxic gases.
6. Presence of smoke.
7. Development of fear, shock, and panic.
8. Secondary fire effects because of mechanical reasons (trauma, bone fractures, and so on).

This article focuses on numbers 5 and 6 of Kimmerle's factors: the presence of other toxic gases (i.e., CN-) and the presence of smoke. Fire smoke is a highly complex mixture of liquid and solid aerosols, fumes, gases, and vapors that are produced by thermal decomposition or pyrolysis.<sup>2-5</sup> The effects of the potentially large number of chemical substances in fire smoke and the low-oxygen environment produced by fires can be divided into three categories,<sup>6</sup> which follow:

- Thermal injury—caused by intense heat, heated air, or steam.
- Chemical injury—caused by any number of chemical substances in fire smoke that can cause upper and/or lower airway irritation.
- Hypoxia and asphyxia—because of the decrease in oxygen in the breathing atmosphere and depletion by combustion and systemic asphyxiants, particularly CO and CN-.

Death by smoke inhalation has been known since antiquity. In some ancient conflicts, captured enemy soldiers were executed by placing them in cages over fires fueled with green wood. Although CO poisoning as a cause of serious poisoning or death in smoke inhalation victims has long been recognized, it was only in the 1960s to 1980s when the

potential for a significant CN- poisoning component contributing to or, in some cases, being the major cause of serious poisoning or fatality in smoke inhalation victims began to be recognized.<sup>7-10</sup> Further such cases were reported in the 1990s<sup>11-15</sup> and later.<sup>16-19</sup>

In 1966, a study conducted by J.R. Wetherell showed that 39 of 53 fire victims were found to have both CO and CN- in their bodies; CN- was found in all but 14 of these victims. (7) Some victims were badly burned, while others were hardly burned at all. Wetherell noted, "It would be particularly embarrassing to overlook such a potent cause of death" [CN-]. Despite this admonition, 45 years later, victims of fatal smoke inhalation and firefighters with possible smoke inhalation during LODDs may not have analytical samples obtained for CN- at autopsy. Thus, CN- poisoning may go unrecognized and undiagnosed in smoke inhalation victims because, in fatal cases, blood CN- levels may not be measured during autopsy; this is particularly disturbing in firefighter LODDs.

The U.S. Fire Administration's Firefighter Autopsy Protocol (U.S. Fire Administration, 2008) specifically lists hydrocyanic acid [Hydrogen Cyanide (HCN)] under Blood Analysis in its Toxicological Examination section. However, one of the author's conversations during attendance and presentations at forensic toxicology meetings, together with the paucity of recent publications describing postmortem blood CN- levels in smoke inhalation victims and firefighter LODDs, suggests that this portion of the Autopsy Protocol is not being routinely followed. This is important to consider because CN- may be less evident as a cause of death when it is not specifically tested for. Consequently, the level of concern may be lower than it should be because of the lack of data when it comes to whole blood CN- testing.

Today, with the proliferation of plastics and synthetic polymer building materials, the risk of a significant CN- poisoning component in victims of enclosed-space fire smoke inhalation has increased.<sup>20,21</sup> There are approximately 4,000 smoke inhalation deaths each year in the United States; 50 to 80 percent of them may be because of the CN- poisoning component. Firefighters are especially at risk as well as children, the elderly, and those with preexisting cardiac or respiratory disease. An ideal CN- antidote for prehospital administration on the fireground would have the following characteristics<sup>22</sup>:

- A rapid onset of action.
- Neutralize CN- without interfering with oxygen transport.

- Safety and tolerability profile would be conducive for use in prehospital settings, the emergency department, or the intensive care unit (ICU).
- Safe for use in smoke inhalation victims.
- Not harmful if administered to nonpoisoned patients.
- Easy to administer.

Hydroxocobalamin, of all the CN- antidotes in clinical use worldwide, best meets these criteria. Hydroxocobalamin (as Cyanokit® Hydroxocobalamin 5 Grams) is the only Federal Drug Administration-approved antidote in the United States that meets all these criteria for known or suspected CN- poisoning, including smoke inhalation victims.

The Paris (France) Fire Brigade's years of experience have shown that early and efficacious prehospital administration of hydroxocobalamin can be safely done using the following criteria (18):

- Extricated from an enclosed-space fire scene with smoke.
- Soot in the nose, mouth, or throat or sooty expectorations.
- Any alteration of consciousness.
- Hypotension. Although hypotension need not be present for the decision to administer hydroxocobalamin, hydroxocobalamin is most efficacious when administered as soon as possible following extrication and thus should be available on the fireground for prehospital administration.

#### **WHY IS CN- POISONING IN SMOKE INHALATION NOT RECOGNIZED?**

Despite recent attempts to further the recognition of CN- as a significant part of the toxicity of fire smoke inhalation, the diagnosis is often missed because it is not considered; a significant point about recognition of the illness lies in this statement. Remember, you can't treat it if you don't recognize it.

It has been known since the 1960s that significant CN- poisoning can occur in smoke inhalation (7) and was further recognized in the '70s and '80s.<sup>23-28</sup> However, the lack of a readily available analytical method for emergent measurement of blood CN- concentrations and the nonspecific and nonpathognomonic nature of CN- poisoning signs and symptoms continue to hamper making the diagnosis.<sup>29</sup>

A 2007 NIOSH publication, "Preventing Fire Fighter Fatalities Due to Heart Attacks and Other Sudden Cardiovascular Events," noted that HCN is formed by incomplete combustion of any substance that contains carbon and nitrogen (both naturally occurring and synthetic) and that airborne concentrations exceeding those of established occupational exposure limits occur in structural fires.<sup>30-32</sup> It also acknowledges that CN<sup>-</sup> impairs cellular use of oxygen, which can result in cellular hypoxia and a variety of cardiac manifestations.<sup>33</sup> A study in dogs exposed to both CO by inhalation and CN<sup>-</sup> by intravenous infusion to mimic inhalation exposure showed that CO had little or no effect on cardiovascular parameters, whereas CN<sup>-</sup> severely depressed such functions.<sup>34</sup>

One study of air monitoring at more than 200 fires in Boston, Massachusetts, did not find significant amounts of HCN present,<sup>35</sup> while an earlier study found CN<sup>-</sup> present in about 50 percent of fires.<sup>(32)</sup> The difference may have been because of the types of materials that were burning, the instrumentation used, or the air-monitoring protocols in place at the time of these studies.

Based on the Paris, France, protocol now generally adopted in the United States, CN<sup>-</sup> poisoning should be considered in any patient who has been extricated from an enclosed-space fire scene; has soot in the nose, mouth, or throat or has carbonaceous sputum; or has any alteration in the levels of consciousness. Hypotension is a particularly ominous sign in such patients. Administration of a CN<sup>-</sup> antidote, preferably hydroxocobalamin (Cyanokit® 5 grams), is indicated for known or suspected CN<sup>-</sup> poisoning.

Ambient air monitoring for CO and CN<sup>-</sup> can now be done on the fireground in real time, which can alert firefighters to the potential for harm. This is an emerging and important fire service topic and is beyond the scope of this article. A significant CN<sup>-</sup> poisoning component should be suspected in smoke inhalation victims with otherwise unexplained respiratory failure or a persistent anion-gap metabolic acidosis.<sup>36</sup>

A study by D.J. Barrillo<sup>37</sup> is often quoted by those who question the frequency and importance of a significant CN<sup>-</sup> poisoning component in smoke inhalation victims. These authors state in their study, which examined the New Jersey State Medical Examiner's Office records from 1985-87 and which focused on fatalities only (not only of victims who were still alive when extricated from a fire scene and who, therefore, might have benefited from specific CN<sup>-</sup> antidote treatment): "Cyanide poisoning is infrequent in fire fatalities ..."

and "Specific assay and treatment for cyanide poisoning is rarely necessary in the treatment of smoke and fire."

Victims had deaths attributed to fires, burns, and smoke inhalation. Thus, unlike Baud's study from Paris,<sup>38</sup> where only living victims with less than 20 percent total body surface area (TBSA) burns were examined, burns were present in 82 percent of cases. The average TBSA for burns was 71 percent, and 61 victims were charred or incinerated (had nonsurvivable burn/thermal injuries). Also, the blood CN- samples were obtained at autopsy, not shortly after extrication from the fire scene, as was done in Baud's study. Only 56 of 433 fire fatalities received prehospital, emergency department, or in-patient medical treatment, and most died within one to two hours. (37) Whole blood CN- levels were obtained in 364 cases. Of these, 85 (23 percent) had no detectable blood CN-, and 31 (nine percent) had blood CN- levels greater than 3 mg/L (milligrams per liter), which is generally considered to be lethal. These authors do not report whether the victims with the potentially lethal blood CN- levels received prehospital or hospital treatment.

Although only nine percent of the fatalities in this study had potentially fatal blood CN- concentrations, patients administered hydroxocobalamin in the prehospital setting with such blood CN- levels have survived with similar blood CN- levels. (18,19) This is clearly NOT a reason to withhold specific antidote treatment from smoke inhalation victims who are extricated from an enclosed-space fire with smoke present; have soot in the nose, mouth, or throat or carbonaceous sputum; have any alteration of the level of consciousness; and especially those who are hypotensive [systolic blood pressure less than or equal to 90 mmHg (millimeters of mercury) in adults].

#### **FIREFIGHTERS/PARAMEDICS CAN MAKE A DIFFERENCE**

In a prospective series of 69 smoke inhalation patients from Paris, France, treated with between five and 15 grams of hydroxocobalamin in the prehospital setting or in the ICU, 50 (72 percent) survived. (18) Of the 67 percent of patients confirmed to have CN- poisoning [whole blood CN- level greater than or equal to 39  $\mu\text{mol/L}$  (1 mg/L)], 67 percent administered hydroxocobalamin survived. No serious adverse events were attributed to administration of the antidote. (18)

The issue is sometimes raised regarding substituting sodium thiosulfate alone for hydroxocobalamin because of the lesser cost. This was reviewed by A.H. Hall, et al.<sup>39</sup> There are much fewer published data on the use of sodium thiosulfate alone than there

are for hydroxocobalamin for the antidotal treatment of CN- poisoning. There are contradictory conclusions regarding the efficacy of sodium thiosulfate in animal studies, and the onset of sodium thiosulfate antidotal action may be too slow for it to be recommended as the only CN- antidote for emergent administration. (39) Because a significant CN- poisoning component in smoke inhalation victims (firefighters and civilians) continues to be underdiagnosed and untreated, departments should consider any action that could increase awareness of the possibility of this potentially fatal and treatable condition.

For those in positions of influence in the fire service, this could include opening a dialog with the local medical examiner's office and strongly urging that all firefighter LODDs and civilian smoke inhalation fatalities that come to autopsy have blood CN- levels determined on a routine basis. The results of such assays should be published and discussed with prehospital care providers, EMS medical directors, and emergency physicians locally at mortality and morbidity conferences and medical grand rounds. Medical examiners and forensic toxicologists should be encouraged to report their findings at regional and national medical meetings.

#### **WHAT MAY BE COMING**

**Lactate levels.** The diagnosis of acute CN- poisoning, even in "pure" CN- poisonings, has always been difficult in the absence of a credible exposure history because there are no pathognomonic signs and symptoms of CN- poisoning.(29) This dilemma is exaggerated in smoke inhalation victims where there may or may not be a significant CN- poisoning component. One of the more readily obtainable screening laboratory tests that has shown to have a good correlation with a CN- poisoning component is plasma lactate. In smoke inhalation victims, a plasma lactate level of greater than or equal to 10 mmol/L was a sensitive and specific indicator of the presence of a blood CN- concentration greater than or equal to 39  $\mu\text{mol/L}$ , which is generally agreed to be a toxic blood level.(29,38)

Obtaining a plasma lactate level is, however, restricted to the emergency department/hospital environment. A promising prehospital test that could be obtained in the field to aid in the suspicion of a significant CN- poisoning component in smoke inhalation victims is the potential use of portable lactate meters, which can be used with a finger-stick blood sample (similar to finger-stick blood glucose meters now in common prehospital use). Originally developed as training tools for high-performance athletes, such meters have been used to monitor type I glycogen storage disease and were found to

have good correlation with the standard laboratory plasma lactate assay.<sup>40</sup> They have also been used in obstetrics as a replacement for pH measurements in fetal monitoring during labor and for hyperlactatemia (elevated plasma lactate) screening during antiretroviral therapy in Africa. Such point-of-care devices have not yet been studied in smoke inhalation victims but hold out the potential for rapid field screening that could increase clinical suspicion of a significant CN- poisoning component and support prehospital administration of a specific CN- antidote.

**Exhaled Breath CN- Meters.** A meter is being developed that has the potential to measure both CO and CN- in exhaled breath. The developer anticipates that it would be adaptable to either collecting samples exhaled by voluntary effort, or that it could be placed in-line between an endotracheal tube and a ventilation bag. If this method proves to be accurate, reproducible, and well-correlated with blood CN- concentrations, it would provide a very rapid means for the prehospital diagnosis of a significant CN- poisoning component in smoke inhalation victims.

#### **Cobinamide Colorimetric Quantitative/Qualitative Blood CN- Measurements.**

Cobinamide is an hydroxocobalamin precursor under development as a CN- antidote. It has the properties of binding two CN- ion molecules per cobinamide molecule and also undergoes a color change and a change in its ultraviolet/visible light spectrophotometric spectrum. Both a quantitative method using this color change for rapid laboratory measurement of blood CN- concentrations in the emergency department/hospital setting and a qualitative finger-stick method with cobinamide-impregnated paper have recently been described.<sup>41</sup> Further development of this method has the potential for a rapid finger-stick test to determine blood CN- concentrations in the prehospital setting, which would provide further information to support field administration of a specific CN- antidote.

#### **MORE FACTS**

In experimental animals, inhalation of polymer pyrolysis products including HCN in smoke has been shown to result in cardiotoxicity with elevated creatine phosphokinase activity and an increased number of ectopic heartbeats.<sup>42</sup>

The production of HCN during combustion and pyrolysis is both material- and temperature-dependent.<sup>43</sup> CN- is released from nitrogen-containing material, and relatively high temperatures are required. (43) In one older study of ambient atmospheric

concentrations of CN- and other gases at structural fires, HCN was found in only 12 percent of studied fires; the maximum measured concentration was 40 parts per million.<sup>44</sup> This may not be true in today's fires.

HCN and CO in fire smoke are at least additive toxicants (43)<sup>45,46</sup> and may indeed be synergistic (having greater toxicity than predicted from the concentrations of either toxicant alone).<sup>47-51</sup> Clinically, this was observed in smoke inhalation victims in the classic Paris, France, study, where some fatalities were associated with blood CO and CN- concentrations, neither of which were predicted to cause death. (38)

Firefighting involves strenuous physical activity. One animal study showed that the time to lethality from breathing pyrolysis products of polyacrylonitrile was decreased with increased physical activity.<sup>52</sup> Among a group of 479 Baltimore firefighters, exposure to HCN in fire atmospheres was sufficient to increase their serum thiocyanate (a metabolite of CN-) concentrations above those of a control population.<sup>53</sup>

Victims of smoke inhalation in enclosed-space house fires have been noted to have soot in the nose or throat and carbonaceous sputum and to have alterations of consciousness including coma.<sup>54</sup> Compared to a control group of critically ill patients without smoke inhalation exposure, 66 smoke inhalation survivors had significantly lower mean blood CN- levels than 43 such victims who died. (38) Specific CN- antidotes were not administered to smoke inhalation victims in this study.

Potentially fatal blood CN- levels have been documented in smoke inhalation victims who have survived with supportive treatment and administrations of specific CN- antidotes. (54) It is also relevant that, because inhaled HCN has a rather short half-life, blood samples must be obtained close to the time of extrication from the fire scene to accurately reflect the degree of impairment because of CN-.<sup>55</sup> Two Boston fire companies participated in personal toxic gas monitoring in the ambient fireground atmosphere; it was found that low levels of HCN were detected in half the collected samples.(32)

Higher yields of HCN occur in small oxygen vitiated flaming fires in closed compartments and in fully developed, postflashover fires in open compartments at high temperatures. (55) In such fires, the systemic asphyxiant effects of CO, HCN, and low ambient oxygen

levels together with dense, irritant smoke (which impedes escape attempts) are the greatest hazards. (55)

Approximately 80 percent of fire victims die from smoke inhalation rather than burns, as was seen in the MGM Hotel fire in Las Vegas, Nevada; the Biloxi, Mississippi, jail fire; and the Westchester Hilton Hotel fire in Houston, Texas.<sup>56</sup> In analysis of carboxyhemoglobin and blood CN- concentrations from fatalities in the 1986 Dupont Plaza Hotel fire in Puerto Rico, both toxicants were found in concentrations generally lower than those usually associated with death.<sup>57</sup> These authors concluded that in non-burned fatalities, the combination of CO and CN- resulted in incapacitation and death. (57)

In nonhuman primates exposed to the pyrolysis products of polyacrylonitrile, HCN was considered to be the major toxic product. (33) Effects of inhaling polyacrylonitrile pyrolysis products were hyperventilation, loss of consciousness within five minutes, bradycardia, cardiac arrhythmias, and T-wave abnormalities.(33) Smoke inhalation is also a major factor in nontrauma-related deaths in aircraft crashes; CN- plays a detrimental role in fatalities from aviation accident fires.<sup>58-62</sup>

In an Australian study of 178 fire-related deaths, blood CN- levels were measured in 138 victims (17); there was no measureable blood CN- in 52 of these 138 cases. The remaining 86 cases had a mean whole blood CN- level of 1.65 mg/L (greater than the 1.0 mg/L generally accepted toxic level); in 11 cases, the level was less than 3.0 mg/L (generally considered lethal). (17)

CN- poisoning must always be considered in any victim of smoke inhalation. The availability of an FDA-approved, relatively safe, specific CN- antidote that can be administered in the prehospital setting means that, once recognized or suspected, cyanide poisoning can effectively be treated in the prehospital setting.

Author's note: For more information, visit [www.firesmoke.org](http://www.firesmoke.org).

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