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The Importance of Autopsy and Injury Data in the Investigation of Fires

Jamie L. McAllister, Douglas J. Carpenter and Richard J. Roby, Combustion Science & Engineering, Inc., Columbia, MD, USA*
David Purser, Hartford Environmental Research, Hatfield, UK

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Abstract. Autopsy and injury data from victims of fire may provide the investigator with important, discriminant, scientific evidence to assist in origin and cause determination. Through the combined use of fire testing, fire modeling, and physiological modeling, the fire investigator may be able to test or further validate their origin and cause hypothesis(es), and other aspects of a fire incident based on the facts of the case and data collected during autopsy or hospital evaluation. As demonstrated in the case study, autopsy data was compared against carbon monoxide concentrations and temperature profiles for two competing origin and cause hypotheses. Only one of the fire scenarios produced toxicant doses and thermal conditions consistent with the victim's injuries. Hence, the evaluation of autopsy data in combination with the facts of the case and dynamics of the fire assisted in origin and cause determination.

Keywords: Fire investigation, Toxicology, Fire victims, Carbon monoxide, Hydrogen cyanide, Fire modeling

1. Introduction

The determination of the origin and cause of some fires proves challenging for fire investigators due to the limited physical evidence that typically remains especially after a post-flashover fire. A fire investigator's ability to test one or more origin and cause hypotheses, as set forth in the Scientific Method, is dependent on the evidence available to support one particular hypothesis and the factual nature of that evidence. The Scientific Method, the basic tenet of fire investigation, requires that the investigator identify the problem, define the problem, collect and analyze the data, develop and test hypotheses, and select a final hypothesis [1]. Because the collected data may lead to the development of multiple hypotheses, these hypotheses must be tested against all available data to ensure an objective analysis and to determine, through deductive reasoning, which hypothesis is most probable. The main objective of this paper is to emphasize the importance of autopsy and injury data in the context of origin and cause investigations, and to provide examples of how autopsy and injury data can be used to evaluate origin and cause hypotheses. Additionally, this paper is intended to emphasize the need for continuity between the autopsy and injury data and the origin and cause

* Correspondence should be addressed to: Jamie L. McAllister, E-mail: jmcallister@csefire.com

hypothesis. That is, the hypothesized fire scenario must be able to produce toxicant doses and thermal conditions consistent with the victim's injuries.

Autopsy and injury data from the victims of fire may provide the investigator with important, discriminant, scientific evidence to assist in origin and cause determination. The toxicological data also plays an important role in understanding the dynamics of the fire, such as the fire growth and spread and fire effluent development. While fatal fire victims may not be able to communicate their observations through words, they can communicate them through physiological and toxicological data that are collected during their autopsies. During experimental fires, fire scientists collect data on the burning of materials by utilizing thermocouples and radiometers to measure temperatures and fluxes in a fire compartment and by utilizing gas probes and calorimetry to measure fire effluents; during fire incidents fire victims collect these same data (temperature and flux) on their skin and (fire gases) in their respiratory systems. While measuring instruments are not present during actual fire incidents, victims often are present. Hence, the victims (both fatal and non-fatal) are important data collectors during the fire's development.

1.1. Fire Asphyxiants and Their Mechanism of Action

U.S. fire statistics show that the majority of all fire deaths are related to smoke inhalation. Carbon monoxide (CO) and hydrogen cyanide (HCN) are generally accepted as the two key toxicants in fire-related deaths. The importance of CO in the blood of a fire victim and the role of CO in cause of death have been well documented by numerous studies in both the medical and fire protection communities [2–7]. Studies indicate that 75%–80% of all fire victims suffer significant smoke inhalation and that this same percentage die as a result of smoke inhalation.

CO is very stable in postmortem blood, is measured toxicologically, is readily related to its level in the blood at time of death, and is easily predicted using an uptake model. CO is a powerful toxicant, because its affinity for the blood is ~250 times that of oxygen. As CO is absorbed into the blood via the lungs, the CO binds to the hemoglobin within the red blood cells and forms carboxyhemoglobin (COHb). As the body becomes oxygen-deficient, the functioning of the heart and other vital organs will begin to decrease. Continued exposure to CO will lead to incapacitation and, eventually, death. Incapacitation typically occurs between 30% and 40% COHb, but has been recorded at values as low as 25% for victims who were active prior to incapacitation. Lethal levels in fires typically range between 40% and 80% COHb [8, 9].

CO is endogenously produced in humans through the natural breakdown of hemoglobin to bile pigments; the resultant COHb levels due to endogenous sources in healthy adults are typically <1% [10]. Exogenous exposure to CO is found through environmental pollutants (e.g., high traffic urban areas) or from habits, such as cigarette smoking. Due to the combinations of endogenous and exogenous sources of CO, COHb levels in adult smokers are on the order of 3%–8%, with levels as high as 15% found in chain-smokers (e.g., 2 packs per day or more) [10].

COHb levels >1% have been found in non-smokers due to exogenous environmental exposure to pollutants [10]. The half-life of CO in surviving victims is described as the time period over which the quantity of CO in the blood decreases to half its original value. Typical literature values for the half-life of CO are 3–5 h when air (~21% O_2) is inhaled by the victim. Half-life values with 100% O_2 therapy are ~80 min [10, 11].

For these reasons %COHb in the blood (corrected for any smoking history) of a fire survivor or fatality provides an excellent record of the CO exposure dose (CO concentration × exposure time) experienced by a victim during a fire. For a fatality, the %COHb is essentially frozen at the time of death and represents the total exposure up to the time of death, because it is stable in blood. For a fire survivor, the %COHb in a blood sample taken on arrival at the emergency room can be used to back-calculate the concentration at the time of rescue and, hence, the CO exposure up to that time.

While CO affects the ability of the hemoglobin to carry and deliver oxygen, HCN affects the ability of the cells to utilize oxygen. After inhalation, HCN is absorbed into the bloodstream via the alveolar capillaries in the lungs. Once in the blood, a portion is sequestered in the red blood cells and, more specifically, in the methemoglobin (MetHb), where an inactive compound CN-MetHb is formed [12]. The remaining cyanide is transported by the systemic circulation to target organs and tissues. In particular, cyanide targets the brain. Cyanide has an affinity for cytochrome c oxidase, and the brain has a high concentration of this oxidase. Due to its reliance on oxidative metabolism and its limited anaerobic ability, the central nervous system is particularly susceptible to the effects of cyanide.

Cyanide interferes with cytochrome c oxidase by binding heme iron, which disrupts the electron transport chain. This cellular interference hampers the transport of electrons necessary to form water and the resulting ATP molecules needed for energy production. As a result, the Kreb's cycle ceases, causing metabolic acidosis, histotoxic anoxia, paralysis of the respiratory center in the brain, and, ultimately, death [11–14]. Cyanide is detoxified in humans at an estimated rate of 0.017 mg CN/kg*min [12]. Cyanide is metabolized through enzymatic conversion to thiocyanate (SCN), which has an average half life of ~3 days [12]. This major route of detoxification occurs in the liver (about 80% of dose) and is driven by the mitochondrial enzymes rhodanese and, to a lesser extent, by β-mercaptopyruvate cyanide transulferase. Rhodanese acts to catalyze the transfer of the sulfane sulfur of thiosulfate to the cyanide ion to form SCN; therefore, the rate-limiting step in this process is the amount of available thiosulfate. After metabolism, the majority of the cyanide is eliminated via the kidneys. A small portion (1%–2%) is excreted via the lungs during exhalation, and the remainder reacts with cystine and hydroxocobalamin to form inactive metabolites [12].

Incapacitating values of cyanide are between 2.0 mg/L and 2.5 mg/L, and lethal values are reported to be 3.0 mg/L or greater in whole blood [15], although the relationship between blood concentrations and effects is complex and requires careful interpretation depending on the history of sampling in relation to exposure [16]. In terms of parts per million (ppm) in the inhaled air of humans, ~280 ppm exposure will cause loss of consciousness within ~1 min and death within a few

minutes [15]. At 100 ppm HCN, incapacitation will result after ~30 min and may result in death after ~1 h of exposure [11, 15]. Exposure to 60 ppm for up to an hour has been shown to cause no serious effects [17]. Hence, the cyanide dose necessary to cause toxic effects is orders of magnitude less than the toxic CO dose.

Endogenous blood cyanide levels of <0.26 mg/L are found in normal healthy adults due to the metabolism of Vitamin B12 (cyanocobalamin) [8]. However, the levels of cyanide found in fire victims are generally at least one order of magnitude higher (i.e., 2 mg/L–3 mg/L), but can decrease postmortem [8, 15]. Over the short exposure times in fires, the metabolism of HCN is limited, so that at the time of death the total body cyanide represents the majority of the dose inhaled during the fire. However, because only a proportion of the cyanide remains in the blood, the blood level is only a partial guide to the total inhaled cyanide dose. Another issue is that cyanide is unstable in a cadaver, the blood concentration decreasing by ~50% over 24 h, and is also less stable in stored blood than CO, unless the sample is treated with an appropriate preservative [18]. For this reason, although elevated blood levels can be related to HCN exposure levels in fires, care is needed in the interpretation of blood cyanide data, and the history of the sample needs to be taken into account. However, the finding of elevated blood cyanide levels does confirm that a subject has been exposed to fire effluent from fuels containing nitrogen (such as wool, nylon, polyurethane foam). Since essentially all fuels produce CO during fires (rate dependent on combustion conditions) the finding of COHb is more indicative of exposure to fire effluent from the generic fuels involved in a fire.

Some debate exists in the scientific literature regarding the combined effects of CO and HCN inhalation in fire victims. The state of the art in fire science today generally assumes that the effects of CO and cyanide are additive. This additive effect is reflected in the use of fractional dose models (i.e., a fractional dose of 100%) which assume, for example, if a victim has 60% of a lethal dose of CO and 40% of a lethal dose of cyanide, that the combination is borderline lethal. However, some researchers believe that CO and cyanide act independently because of their different mechanistic and toxic effects. Still other researchers believe that the combined effects of CO and cyanide are more than additive, such that a fractional dose of each of these two toxins can be fatal even when the fractions do not add up to one [15, 19].

1.2. Effects of Heat

Evidence concerning the nature of the fire environment in the vicinity of an exposed subject can also be obtained from the effects of heat exposure. Heat exposure can cause injury and death in fires by three main mechanisms:

1. Heat stroke (hyperthermia)
2. Body surface burns
3. Respiratory tract burns.

The effects occurring depend upon a number of factors, including the temperature and radiant heat levels (total incident heat flux to the body) and the duration of exposure. As with exposure to toxic gases, the effects are related to the heat "dose" (heat flux *times* exposure time) to which the subject has been exposed [15]. When air temperatures are below 121°C (water content <10% by volume) or radiant heat fluxes are <2.5 kW/m^2, the resultant temperature of the exposed skin will be <44°C. Burns do not occur at this skin temperature, so the main hazard from prolonged exposure to these temperatures/heat fluxes is hyperthermia. Above these limits burns occur to exposed skin; the extent and severity depend upon the exposure intensity and time [15]. From the extent and severity of burns it is therefore possible to determine the fire conditions to which the subject has been exposed. If the duration of exposure is known, it is possible to make some determination of the heat environment during the exposure in terms of temperature and heat flux. Upper respiratory tract burns usually occur only in the presence of burns around the mouth and nose, while burns to the deep lung are indicative of inhalation of air with a high water content (e.g., saturated air at an inhaled temperature above 60°C) [15].

1.3. Smoke Particulates

Another important indicator of exposure and fire conditions is the extent of soot deposits in the lungs (and also potentially the composition of the inhaled particulate). Since a large proportion of inhaled particles are retained in the lung, the amount deposited is directly proportional to the concentration in the inhaled smoke, the breathing rate (minute–volume) of the subject during the exposure period, and the duration of exposure. Smoke deposition is normally considered as a qualitative indication of significant fire effluent exposure, while evidence of this exposure is quantitated through the use of %COHb in the blood. A lack of soot deposition in combination with a lack of elevated %COHb typically indicates that the victim was dead prior to the start of the fire.

1.4. Comparing Burns with %COHb

Comparing the victims' burns with their %COHb levels can provide the investigator with useful evidence regarding the fire conditions at the time of exposure. For example, the finding of a fatality with severe burns in a fire enclosure and little to no %COHb can be indicative of death resulting from exposure to a rapidly growing, flaming fire. Such a fatality can also indicate that the victim was in a location containing little to no fire effluents (e.g. on the floor beneath a hot fire effluent layer); in this scenario, he/she would die from thermal exposure before being able to inhale a significant dose of CO [20]. This situation has been found to be typical of most victims of post-crash vehicle fires, in which rapid fire involvement of the passenger compartment, with high radiant heat fluxes, results in victims with severe burns and little CO in their blood [21]. Alternatively, the finding of a high %COHb in the absence of burns may suggest that the victim was in a location remote from the fire origin, but was exposed to considerable amounts of fire effluents which spread to that location. This finding also confirms how fire effluent plumes spreading beyond the immediate fire enclosure rapidly cool as they entrain

fresh air and lose heat to building surfaces away from the fire compartment. However, these plumes generally do not lose their CO.

More difficult to interpret are situations where both burns and high %COHb levels (40%–50%) are present. These injuries can occur when subjects are exposed in the enclosure of fire origin in situations where limited ventilation prevents flashover and limits effluent plume temperatures. For survivors, the finding of this combination is indicative of approximately simultaneous exposure to both heat and toxic smoke, typical of a subject exposed in the room of origin in a domestic fire incident where flashover has not occurred. The location of the burns may also provide an indication of the extent to which the subject was intimate with the fire (uneven distribution of burns, or more burns to the directly exposed areas) or exposed more remotely to the heated upper layers (more generalized burns, especially to face and head). For fatalities, a problem arises as to when the burns occurred relative to the inhaled CO, since it is not possible to reliably distinguish between antemortem and postmortem burns [22]. In these situations, the finding of a combination of COHb and burns may indicate a simultaneous exposure or a situation in which a victim has initially been exposed to relatively cool smoke with high toxic gas content, resulting in incapacitation or death; the subsequent spread of the fire then causes severe burning of the body after death. One piece of physical evidence that may help to resolve this issue is the extent of respiratory tract burns, which do not occur once the victim has ceased breathing. Such burns are evidence that the victim was exposed to very hot gases while still alive and breathing.

1.5. Other Indicators

A number of studies have been made attempting more sophisticated measurements of substances recovered from the lungs or blood of fire victims in order to establish what they were exposed to during fires [23, 24]. Although these studies have been rather limited so far, their use of increasingly sophisticated and sensitive analytical techniques could have considerable potential from an evidentiary perspective. During one incident, it was demonstrated that certain fatty acids measured in victims were evolved from painted surfaces that burned during a fire. Carbon fibers and other mineral fibers and dusts have been recovered from the lungs of some persons exposed during the World Trade Center incident [24]. The key point is that the body represents a continuous air sampling mechanism during such incidents, so that anything inhalable and deposited in the lungs is likely to remain in the body. Therefore, any substance that is chemically or structurally foreign to the body and is retained can potentially provide a source of evidence concerning the victim's exposure conditions.

2. Toxicology and Fire Origin and Cause Determination

2.1. The Relationship Between Toxic Gas Production and Fire Development

Substantial fire safety literature exists that relates the generation of heat and the generation of toxic gases during the various stages of the development of a fire

[15, 20, 25–30]. Thus, a relationship exists between the nature of the fire and the production of heat and toxic gases. Because of this relationship, it may be possible for fire investigators to determine the nature of a fire (e.g., smoldering, flaming, post-flashover) if they have reliable autopsy or injury data and can establish a timeline for exposure. Understanding the fire dynamics and correlating this information with victim autopsy or injury data can assist with origin determination as well as cause determination because the origin and cause of the fire are inherently linked. As part of the testing of an origin hypothesis, investigators must identify competent ignition sources within the origin. In instances where only one competent ignition source exists within the area of origin, the determination of an origin lends itself to the determination of a cause. There may also be instances where multiple hypothesized causes exist, and the autopsy or injury data assists in the evaluation of the most probably cause. For example, in a case where an accidental and incendiary cause are being evaluated, if the two hypothesized causes have different toxicant production and heat profiles (e.g. one-gallon gasoline pool fire versus a cigarette ignition of a sofa), and only one of these profiles can result in the injuries to the decedent, then it is possible to rule out one of the causes.

With regards to the nature of the fire, CO is produced in some quantity in every fire. The production of CO is affected by the quantity of air available for combustion. When an unlimited supply of air is available (such as in outdoor fires) and/or sufficient ventilation openings exist, fuel will control the rate at which a fire burns. This mode of combustion is referred to as a fuel-controlled fire. A compartment with ventilation openings that are not capable of supporting the rate at which fuels pyrolyze will produce a ventilation-controlled fire, and ventilated-controlled fires generate large quantities of CO [31].

In smoldering or non-flaming fires, the rate of CO production is low because of the low mass loss rate of the fuel; such fires generally create CO concentrations of no more than hundreds of parts per million. However, in some cases where the compartment is small and the smoldering continues for a long time, sufficient CO can be produced to create concentrations on the order of 1000 ppm–1500 ppm [15, 30]. During the development of a ventilation-controlled, flaming fire, but prior to flashover, CO production can lead to concentrations of 10000 ppm, and incapacitation can occur within minutes. In the fully developed phase or post-flashover state of a fire, CO concentrations on the order of 30000 ppm commonly occur [15].

Flashover, as described in NFPA 921 [1], is "a transition phase in the development of a compartment fire in which surfaces exposed to thermal radiation reach ignition temperature more or less simultaneously and fire spreads rapidly throughout the space." The transitioning phase of flashover results in full room fire involvement of the compartment. Once fire involvement throughout the room is achieved, the compartment is said to be in post-flashover [1]. In the post-flashover state, temperature and thermal radiation within the room of origin are immediately lethal and produce 2nd and 3rd degree burn injuries within seconds to victims in the immediate vicinity of the room of origin [15, 32]. Lattimer et al. [33] found that the presence of wood in the upper layer of a compartment, such as in homes with wood-paneling on the walls and ceiling, can significantly elevate the

production of CO in a post-flashover compartment, resulting in levels on the order of 100000 ppm.

Any nitrogen-containing material, such as polyurethane, wool, acrylonitrile butadiene styrene, lauan plywood, particle board, and nylon, can produce HCN when thermally decomposed. As previously discussed with CO, a significant factor in HCN production is the combustion efficiency. The air available for combustion will affect the quantity of HCN produced, as seen with CO. HCN yields and HCN concentrations are low (<50 ppm) during well-ventilated combustion conditions. During vitiated pre- and post-flashover fires, when the fire is ventilation-controlled, HCN production can reach as high as 500 ppm and under certain conditions can exceed 1000 ppm [15]. Purser [15] distinguishes four main types of fires when discussing the production of fire toxicants: smoldering/non-flaming fires, well-ventilated (fuel-controlled)/flaming fires, small vitiated/under-ventilated (ventilation-controlled) fires, and fully developed/post-flashover fires. In the presence of sufficient oxygen, the production of HCN during smoldering is typically not significant enough to cause death in and of itself. When heated under non-flaming conditions, the flexible polyurethane foams typically used in furniture upholstery decomposes into an isocyanate component containing nitrogen, which is released as a particulate smoke (''yellow smoke'') and a liquid polyol, which remains near the heated item. If a transition to flaming occurs in the heated item, then no HCN is released by the polyol component; however, if the isocyanate smoke is retained in the fire enclosure and is subsequently exposed to flames or trapped in a high temperature upper layer, then secondary conversion to release HCN can occur.

Levin's [29] research identified an additional phase of combustion in polyurethane foam where smoldering transitions to flaming in an enclosed chamber. In this case, significant quantities of HCN were produced from the secondary combustion of the yellow smoke evolved during the smoldering phase. Woolley et al. [34] noted a similar phenomenon where nitrogen-containing, aerosolized, isocyanate particulates were released during the non-flaming decomposition of flexible polyurethane foams. Therefore, some HCN may be produced when the fire transitions from non-flaming to flaming and the particulates are burned. In most cases, however, the generation of high HCN concentrations occurs as a flaming fire transitions from a well-ventilated to a ventilation-controlled combustion state (equivalence ratio > 1, where the equivalence ratio is the actual fuel to air ratio divided by the stoichiometric fuel to air ratio) [35].

Unfortunately, even though autopsy and injury data may be useful to test origin and cause hypotheses, it is often neglected in the data collection process. This paper will provide insight into the use and application of toxicological data in fire investigation.

2.2. *The Application of Autopsy and Injury Data in Fire Investigation*

Autopsy and injury data from victims of fire can assist the investigator in addressing three main questions related to the origin and cause, and other aspects of a fire:

(1) The types of materials that were burning at the time the victim died
(2) The victim's location with respect to the room of origin
(3) The stage of the fire at the time the victim died.

With regard to the type of material(s) that was burning at the time the victim died, the presence of toxicologically significant quantities of HCN in the blood of a fire victim provides the investigator with an immediate indication that a nitrogen-containing fuel was burning. As previously discussed, endogenous levels of blood cyanide are typically less than 0.26 mg/L, and levels related to exposure in fires are typically an order of magnitude higher, between 2.0 mg/L and 3.0 mg/L. While nitrogen-containing fuels, such as polyurethane, wool, and nylon, are fairly ubiquitous in today's households, they may not be the first items ignited or one or more of the items burning prior to the victim's death. Hence, the presence of high levels of cyanide in a victim's system can assist in the development of a timeline of events and may lead to the determination or resolution of a potential fire cause. For example, where two cause hypotheses are present and only one hypothesis involves a nitrogen containing fuel, the autopsy data would allow the investigator to establish one more probable hypothesis. Care should be taken to ensure that the biological samples are preserved and tested in an appropriate manner to produce reliable data [18, 36, 37].

Toxicological data can also be used to test area of origin hypotheses, since a relationship exists between the victim's location with respect to the room of origin and the victim's COHb and blood CN levels. A study of fatalities in landmark fires, such as the 1990 Happy Land Social Club fire in New York City, the 1986 Dupont Plaza Hotel fire in Puerto Rico, and the 1977 Maury County Jail fire in Tennessee, has shown that those victims with lethal concentrations of CO and HCN in their systems are most often remote from the room of fire origin [2–5]. Victims within the room of origin typically die from thermal burns, and victims remote from the room of origin die from smoke inhalation [20]. This is because CO and HCN are products of incomplete combustion, hence, the greater the burning inefficiency (equivalence ratio > 1), the greater the yield of CO and HCN. In compartment fires, the efficiency of combustion is heavily reliant on ventilation conditions [31]. In the incipient and flaming stages, the fire is fuel-controlled. The rate of burning and products of combustion are controlled by the rate at which fuel can be pyrolyzed.

The majority of flaming enclosure fires (such as fires in buildings) transition from fuel-controlled to ventilation-controlled combustion within a few minutes, as the upper layer fills down toward the burning fuels. In some cases, if the compartments are small and external doors and windows are closed, the fire remains relatively small and confined to the enclosure of fire origin, but large volumes of fire effluents containing high concentrations of CO and HCN spread throughout all open spaces. If exterior vents, such as doors and windows, are open or fail, or if sufficient air is available from other compartments in the building to support a sufficiently fast fire growth curve, then the burning and heat release rates increase rapidly, and the upper layer temperature becomes high enough to support flashover. At flashover, the pyrolysis rate of the fuel exceeds the rate of air entrainment into the compartment. Once

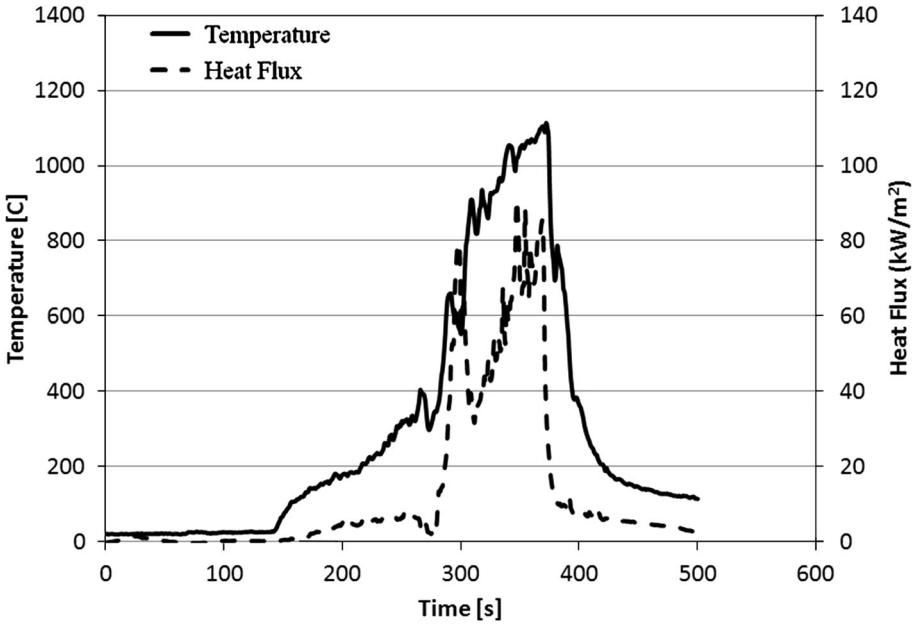

Figure 1. Temperature and heat flux within an experimental flashover compartment fire.

flashover occurs, there is a considerable increase in the mass production rate and spread of the toxic fire effluent plume throughout a building.

In fires that become ventilation-controlled before they reach a sufficient heat release rate to support flashover, victims in the enclosure of origin are exposed simultaneously to both increasing heat and increasing concentrations of CO and HCN. In these fires, incapacitation and death in the enclosure of origin can occur from the effects of either heat or toxic gases, depending upon the specific fire conditions and location of the victims. Victims remote from the enclosure of origin in these fires are exposed to toxic gases, but at reduced temperatures, so that incapacitation and death result from toxic gas exposure and not from heat.

If the fire in the enclosure of origin reaches flashover, then the mass flow rate, temperature, and heat content of the plume are likely to be such that fuels more remote from the area of fire origin may combust and the area over which victims may be subject to burns may increase. However, as with ventilation-controlled fires that do not reach flashover, the extent of a building affected by lethal toxic smoke is generally much greater than that affected by heat during the critical development phase of an incident. If surviving victims are in the enclosure of origin at the time of flashover, temperatures and thermal radiation within the room of origin are immediately lethal. Hence, a victim in the room of origin would die from thermal burns before sufficiently lethal concentrations of CO and HCN could be inhaled and absorbed into the systemic circulation. This concept is further explained in Figures 1 and 2 which shows temperature, flux, and gas data from testing of a wood crib inside a 2/5-scale model of the full-scale ASTM E1822 room. The wood

crib was sized to support flashover conditions in the compartment. The doorway was dimensioned based on the required minimum vent size needed to also support flashover conditions in the compartment. The compartment was 0.98 m (width) × 1.46 m (length) × 0.98 m (height) with a 0.48 m × 0.81 m doorway.

The onset of second degree burns to occupants will occur after ~1 min when the upper layer temperature reaches an average of 183°C or 2.5 kW/m^2 radiant heat flux at floor level [15, 32]. Radiant fluxes of 20 kW/m^2 are sufficient to cause severe burns and death within seconds [1, 32]. These fluxes are achieved at the onset of flashover (equivalence ratio of ~1). Figure 2 shows that CO concentrations rise when thermally lethal conditions are reached in the compartment (see Figure 1). Hence, the compartment of origin is not thermally survivable before sufficiently lethal levels of CO are produced.

Since the production of CO and HCN are heavily linked to the ventilation conditions within the compartment, the concentration of toxicants within the victim's system can aid the investigator in defining the stage of the fire at the time the victim died. The yields of CO at various fire stages, smoldering, flaming, post-flashover, etc., have been defined for various fuels. Since it is unlikely that sufficiently lethal concentrations of CO will be produced before a fire becomes ventilation-controlled, a high, lethal level of CO at autopsy would indicate that a victim was still alive and breathing at the time that the fire became ventilation-controlled. Depending on the subsequent fire development and size and the location of the victims, it may be possible to establish if the victim ceased breathing before or

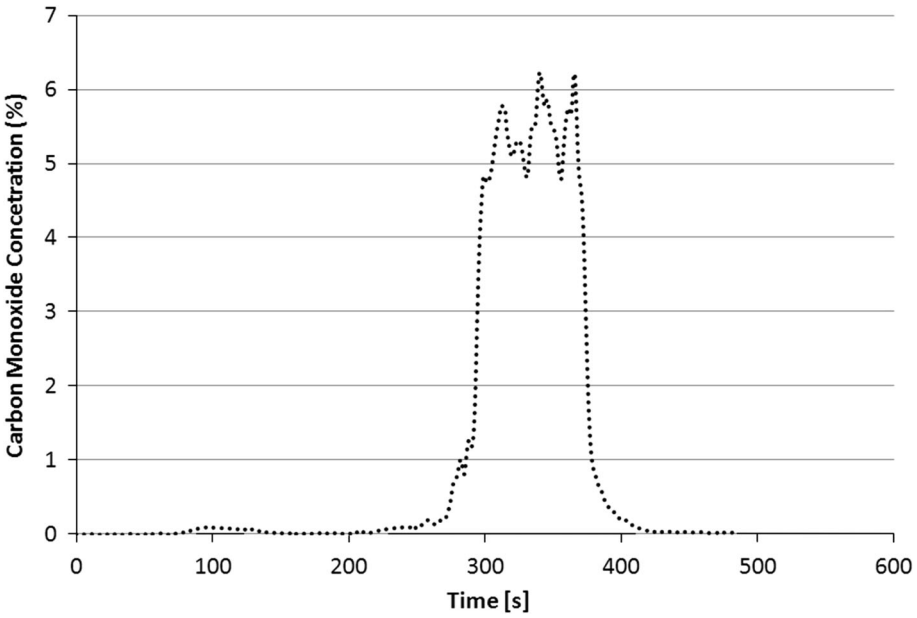

Figure 2. Carbon monoxide concentrations within the same experimental flashover compartment fire shown in Figure 1.

after flashover occurred. If other hard and soft times are available, these data could assist the investigator in ruling in or ruling out potential first fuels ignited based on common burning characteristics of those fuels as defined in the literature or through fire reconstruction.

In order for this information to be useful, the investigator must be able to resolve the victim's location during the time that consciousness and breathing were still present. If the victim moved throughout the structure, it may be difficult to estimate exposure levels. If the victim, however, was found in bed, in a sleeping position, wearing night clothes, the investigator could reasonably infer that the victim remained in that area during the entire duration of exposure. The specific facts of the case must be considered in determining the relevance of the autopsy or injury data There may be some cases where insufficient information is present about the actions of the victim and movement of the victim to be able to determine where the victim inhaled the toxic gases or to correlate the exposure to a specific timeline of events related to the fire's development. Hence, the autopsy or injury data in those cases may not assist the investigator in any significant way. Another important aspect of the investigation relates to the timing and extent of opening of different exterior vents and interior doors. For example, if occupants take refuge in a room remote from a fire and are found to have suffered a high %COHb exposure within a short time, then this is an indication that the door of the room is likely to have been open during the key stages of the fire.

2.3. *Locally Under-Ventilated Fires*

There have been reported cases of victims with high, lethal levels of CO and HCN in their systems who are found within a hypothesized "room of origin." In these cases, the investigator must consider the potential for a "locally under-ventilated" fire. "Locally under-ventilated" fires occur in a compartment within the room of origin, which can be characterized as a "compartment within a compartment" or a "room within a room." Examples include the knee well of a desk, the void space above a ceiling or wall, an entertainment center or nightstand, etc. Icove et al. [38] provided one such case study of a fire that is characteristic of a locally under-ventilated fire. A woman was found dead outside of her bedroom door. Inside the bedroom, a localized fire was found to have occurred in a nightstand, as shown in Figures 3 and 4.

The victim was found to have extensive soot in her airway, non-lethal thermal injuries, and a 67% COHb concentration [38, 39]. In this case, the fire was able to undergo a localized flashover in the smaller compartment which produced copious amounts of incomplete products of combustion composed of high levels of CO. The physical evidence at the scene indicated that she was in the room of origin when the fire was discovered; however, she was not within the compartment of origin. While the spread of the fire was limited to the nightstand, investigators should consider the potential for the locally under-ventilated fires to spread beyond the compartment of origin to the room containing the compartment. Hence, fire investigators must consider that this type of fire scenario, and the accompanying physical evidence, may be masked by fire extension and spread.

Figure 3. Locally under-ventilated fire in a nightstand resulting in one fatality. Photo courtesy of Ross Brogan AFSM, MA, IAAI/CFI, GWBFORENSIC, NSW, Australia.

2.4. The Coupling of Fire and Physiological-Based Modeling

Fire models such as Fire Dynamics Stimulator (FDS) and CFAST can be useful in deriving information related to CO and HCN production in a compartment. When accurate information is known about the fuel(s) in question, e.g., heat release rate, smoke yields, burning duration, etc., or when this information can be measured through fire testing, information regarding the distribution of smoke and toxic gases throughout a compartment can be modeled. Since CO concentrations can be derived in the fire model, these outputs can be utilized as inputs for the physiological-based models, such as the Coburn Forster Kane equation [15].

One main reason why the effects of CO are so well established is because CO is inherently stable in the blood due to its high affinity for hemoglobin. Blood and tissue specimens from fire victims are routinely measured for COHb levels to assist the Medical Examiner in determining the cause and manner of death. Additionally, investigators can more easily garner conclusions related to CO, since only a small variation exists between the measurement of CO taken during autopsy of a fire victim and the quantity of CO in the blood at the time of death [4, 40, 41].

The Colburn–Forster–Kane (CFK) equation describes the uptake of CO in humans and can be used as an investigative tool to estimate the quantity of CO that a victim inhaled prior to death [15, 42, 43]. The CFK model requires an input of the concentration of CO produced by the fire over time, in addition to the victim's weight and respiratory-minute-volume. The CFK model outputs a

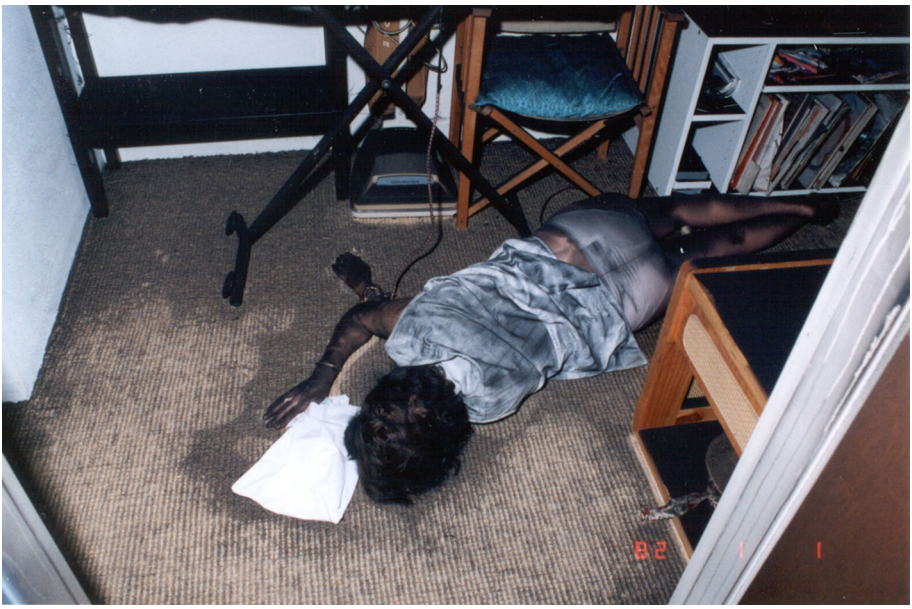

Figure 4. Partially undressed woman found dead on the landing outside of the doorway to the bedroom of origin. Photo courtesy of Ross Brogan AFSM, MA, IAAI/CFI, GWBFORENSIC, NSW, Australia.

COHb value; therefore, through iterations it is possible to determine the CO exposure needed to achieve the COHb level found at autopsy. This CO exposure quantity can be related to a particular type of fire when compared with all the known facts of the case. Additionally, the victim's COHb level in combination with burn injuries can provide details as to the location of the victim during the fire and/or the proximity of the victim to the area of origin.

Due to the inherent instability of cyanide in postmortem cadavers and samples, this toxicant is not routinely measured in fire-death cases. Numerous studies have shown that cyanide can decrease over time in postmortem victims and stored blood samples [7, 15, 44]. As a result, the original value of cyanide at the time of death is not reliably comparable to the value at the time the blood is analyzed. Purser states in *The SFPE Handbook of Fire Protection Engineering* that "cyanide decomposes rapidly in cadavers by ~50% in 1–2 days, and may subsequently decrease further, or even increase slightly in stored blood" [15].

Recent studies completed by McAllister et al. [18, 36, 37] have developed a methodology for the stabilization of cyanide in blood collected from fire victims and have produced an uptake model for HCN. Based on these recent advancements in the field, it is possible to acquire accurate HCN blood measurements from victims under certain circumstances and to use the HCN uptake model to assess cyanide exposure for fire victims. While a HCN uptake model does now exist, it has not been heavily validated and, therefore, should be used with caution in the field at this time.

2.5. Historical Case Studies

The Dupont Plaza Hotel fire and the Happy Land Social Club fire have been extensively studied and are two well-documented cases in which toxicological data was utilized to better understand the fire and its effects. In December of 1986, the Dupont Plaza Hotel fire in Puerto Rico claimed the lives of 97 people [4]. The fire was determined to have started in the hotel ballroom in a large stack of corrugated boxes containing new furniture (dressers and sofa beds); the cause was determined to be incendiary [4].

The blood from 78 of the victims of the Dupont Plaza Hotel fire was tested for COHb by both the Institute for Forensic Sciences in Puerto Rico and the National Bureau of Standards [now the National Institute of Standards and Technology (NIST)] in Gaithersburg, Maryland. When the blood levels were compared with the victims' locations, a correlation was found. Those close to where the fire broke through had sub-lethal, but significant %COHb levels. On the other hand, those progressively further away from the origin, such as the many victims in different parts of the hotel casino, had progressively higher %COHb levels, although all eventually received thermal injuries. From the results obtained it was possible to establish during the trial that sufficient COHb and blood cyanide (corrected for post exposure changes during storage) were present in the victims to have caused incapacitation at the fire scene before significant heat exposure occurred.

The data also made it possible to establish that those close to the fire origin most likely died from thermal exposure, while those in the more remote locations were exposed to toxic smoke for a significantly longer period before severe heat exposure as the flames spread through the casino after windows on the far side were broken. In more protected locations, especially some offices, lethal %COHb levels were measured. In the cases of victims with significant, lethal thermal injuries, Levin et al. [4] stated that "blood data indicated that CO and HCN, singly or combined, were probably not responsible for the majority of the deaths." However, "significantly higher COHb" levels were found in victims with non-lethal thermal injuries (remote from the area of origin), which indicated that "CO alone or combined with HCN probably played a major role in the cause of their deaths." Three victims with lethal %COHb levels were found in an elevator in the basement after the fire, indicating that the elevator had opened on the fire floor above for a period sufficiently long to enable a lethal CO exposure to occur before the doors closed and the elevator continued down to the basement.

The Happy Land Social Club fire occurred on March 25, 1990, in New York City [2]. This fire was determined to have started when a disgruntled ex-boyfriend of a club employee threw gasoline into the club entrance and lit it with matches [2]. A total of 69 of the 87 fatalities were found on the second floor with smoke inhalation; 11 fatalities were found in the area of the first floor rear restroom with smoke inhalation and some thermal burns [2, 3]. The location of the remaining 7 victims is unknown; however, there is no indication that anyone was found in the area of origin [2, 3]. According to autopsy findings, all 87 fatalities died from smoke inhalation, and with the exception of three individuals, all the victims had COHb concentrations of $>50\%$ and as high as 93%. Only 26 of the victims had

thermal injuries, with only three of those 26 victims having body surface area burns exceeding 20%.

NIST was tasked with utilizing various fire models (HAZARD 1, FPETOOL, FAST) to determine what, if any, mitigation strategies could have been employed to prevent the tragic incident [3]. The NIST models showed that the victims located remote from the fire on the second floor would have succumbed to incapacitation due to smoke inhalation and that temperatures and fluxes at the entryway (fire origin) became incapacitating before sufficiently incapacitating levels of toxicants were produced [3]. Hence, the data collected from the Dupont Plaza Hotel and Happy Land Social Club fires further solidifies the relationship between victim toxicology and the area of fire origination.

2.6. Recent Applications

A fire occurred in May of 2008 in a trailer home in Brownsville, TX. An emergency call was placed by neighbors reporting the fire shortly after 11:00 p.m. and the fire department arrived ~10 min after the call was placed. The trailer home had two bedrooms, one bathroom, a living room, and a kitchen. The main entrance to the trailer was through the living room, and the kitchen was attached to the living room through a standard sized doorway with a lintel. A deceased 10-year-old boy was found on a couch in the living room after the fire was extinguished. There was no evidence that the victim was aware of or responded to the fire, and no smoke detectors were found in the home. An autopsy report documented a lethal COHb level of 77% in the boy's blood and soot in his airways. Based on fire department observations upon arrival and post-fire damage, the trailer was engulfed in flames in the area of the kitchen and living room. Due to full room involvement in the kitchen and living room, damage was too extensive for investigators to reliability utilize fire patterns. Two potential origin and cause hypotheses were developed based on witness observations and physical evidence at the fire scene: a fire originating in the living room due to the failure of a window air conditioning unit, and a fire originating in the kitchen due to cabinetry that was ignited by unattended food left on the stove.

In order to access the level of CO produced in each scenario, a zone fire model (CFAST) was utilized. Inputs for the model were gathered from fire testing on an exemplar air conditioning unit and literature on CO production from kitchen fires in mobile homes. CO concentrations derived from the model were then utilized as inputs in the CFK equation to determine which scenario was most consistent with the level of CO found in the victim's blood. Based on the two scenarios, it was found that a fire originating in the living room would have reached upper layer temperatures of 750°C prior to the victim reaching a 77% COHb level. Therefore, the victim would have died from thermal conditions within the living room prior to reaching a 77% COHb concentration. The upper layer temperature within the living room during the kitchen fire scenario only achieved 120°C by the time the victim reached a 77% COHb level. Since temperatures in the kitchen scenario were well below the onset of pain (121°C–183°C) at the time the victim would have been incapacitated by CO (around 40% COHb), it is probable that the

occupant would not have awakened due to pain from exposure to heat. Thus, the use of autopsy data was a determining factor in establishing the origin of the fire. Since the origin and cause of the fire are inherently linked, the determination of origin in this case also allowed for the determination of cause.

2.7. Limitations

The application of autopsy or injury data in origin and cause determination may be limited by the investigator's background and training, the completeness and accuracy of the autopsy or injury data, and, when used, the limitations of fire models and physiological models.

In some cases, the medical examiner or coroner may not conduct a full autopsy. While measurement of COHb concentrations in fire victims is a common procedure in most jurisdictions, it may not take place in all jurisdictions, especially those with limited resources. There also may be instances where the measurement of toxicants is limited by the condition of the body and body fluids, e.g. significant body consumption from fire or severe decomposition. Additionally, when relying on toxicant measurements in the development of opinions, investigators should have some understanding of the accuracy of the data reported by the toxicological laboratory. The accuracy of the data will be dependent on the apparatus and method utilized by the laboratory, e.g. microdiffusion, CO-oximeter, chromatography). In cases where analysis of the autopsy or injury data falls outside of the investigator's area of expertise, the investigator may need to seek a special expert or additional training.

Fire modeling may not be necessary in every case that involves the application of autopsy or injury data. There may also be some cases where insufficient data on the primary fuel package (e.g. heat release rate, toxic gas yields) and/or the building or compartment may limit the investigator's ability to perform modeling. In those cases, it may be necessary to conduct fire tests. The limitations of fire models such as CFAST and FDS have been well documented, however, an in-depth discussion on this topic is outside of the scope of this paper [45–47]. When conducting fire modeling, the investigator should be aware of the limitations of the model, and ensure that the model is capable of accurately simulating the fire conditions that existed during the period of interest, e.g. while the victim was exposed to toxicants.

With regards to physiological models, the Stewart–Peterson equation and CFK equation are commonly used to evaluate the uptake of CO. The Stewart–Peterson equation is intended for use when exposure periods are short (1-hour or less), and exposure concentrations are high (on the order of 1000 ppm or greater) [48–50]. The Stewart–Peterson equation assumes linear uptake; however, the uptake of CO becomes non-linear under low concentration/long duration exposures. The Stewart-Peterson equation requires inputs of CO exposure concentration, respiratory minute volume, and exposure duration, and provides a %COHb output. The Stewart–Peterson equation assumes exposure is occurring in a normal, healthy adult which may limit its applicability in certain cases.

The CFK equation is a non-linear model that allows the user to vary a variety of physiological parameters such as body weight, blood volume, respiratory frequency, partial pressures, endogenous CO concentrations, etc. While the typical values utilized in the CFK equation assume a normal, health adult, physiological parameters can be adjusted to account for younger subjects and certain medical conditions. Investigators should review the autopsy or hospital records for information related to respiratory or cardiac deficiencies which may interfere with the normal absorption, distribution, metabolism, or excretion of CO, and may affect the predication accuracy of the model. Unlike many other physiological models for highly toxic substances, the CFK equation has been heavily validated using human subject. The prediction capabilities of the model have been evaluated by numerous researchers [51–54]. The model has good prediction capabilities under transient exposure conditions as well as low and high range exposure concentrations. Benignus et al. [51] found a deviation of ~4% between the CFK model and experimental %COHb concentrations measured in exposed subjects. As with any model, the largest limiting factor is the ability of the user to appropriately assign inputs and reasonably apply outputs in their analysis. Therefore, the user should have a sufficient understand of the models (both fire and physiological) to ensure their proper application.

3. Summary

Autopsy and injury data may provide the fire investigator with important scientific evidence to make a determination of the origin and cause of the fire. The overall goal of this paper was to expand the fire investigator's "tool box" by improving techniques available for the analysis of blood CO and cyanide levels in fire victims. Cyanide is inherently unstable in postmortem specimens; however, if appropriate procedures are followed to preserve samples, fire victims' cyanide and COHb levels can provide investigators with objective data for use in hypotheses development. Fuels containing nitrogen as well as carbon produce high concentrations of both CO and HCN under vitiated combustion conditions occurring in many enclosure fires (such as many fires in buildings or transport vehicles), while smoldering or non-flaming fires produce mainly CO, and rapidly growing well-ventilated fires produce low yields of both CO and HCN. HCN can produce incapacitation more rapidly than CO is able to at concentrations typical of vitiated fires. For these reasons, the levels of both cyanide and COHb in the blood of fire victims can provide important information relating to the materials involved during critical phases of fire development, and can contribute to evaluating the time course and causes of incapacitation and death in fatal fire incidents, especially when used in conjunction with fire experiments or fire and physiological modeling.

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