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Interpretation of Carboxyhemoglobin and Cyanide Concentrations in Relation to Aviation Accidents

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16. Abstract Introduction: Carbon monoxide (CO) and hydrogen cyanide (HCN) are combustion products of organic material, but their production depends on material constituents and environmental conditions. Non-nitrogenous organic materials generate CO, whereas nitrogenous organic materials also produce HCN. For fire-involved aviation accidents, it is important to determine if the fire occurred during flight or after the crash and to establish the source(s) of the toxic gases. Therefore, this study was pursued. Methods: Bio-specimens from aviation accident fatalities (cases) are submitted to the Civil Aerospace Medical Institute for analyses. In blood, CO is analyzed as carboxyhemoglobin (COHb) and HCN as cyanide (CN⁻). These analytical data are stored in a database, and this database was searched for the period of 1990–2002 for the presence of COHb and CN⁻ in the submitted cases. Results: Out of 5945 cases, there were 223 (4%) cases wherein COHb was $\geq 10\%$. Of the 223 cases, fire was reported with 201, no fire with 21, and undetermined fire status with 1. CN⁻ concentrations were at or above 0.25 ug/mL in 103 of the 201 fire-related cases. None of the 21 non-fire cases had CN⁻, but nicotine was detected in 9 of the cases. All non-fire cases with COHb $>30\%$ (4 cases) were associated with exhaust leaks. Of the 223 cases, COHb-CN⁻ Fractional Toxic Concentration (FTCs) was lethal only in 31 cases with elevated CN⁻ levels. Conclusions: The presence of COHb and CN⁻ in elevated concentrations in the blood of victims who died on impact would indicate an in-flight fire. In the absence of fire and CN⁻, the elevated COHb concentrations would suggest an exhaust leak, particularly at COHb $>20\%$. Findings of this study also suggest that, in addition to COHb, CN⁻ contributes to the detrimental effects of fire-associated aviation accident fatalities.			
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INTERPRETATION OF CARBOXYHEMOGLOBIN AND CYANIDE CONCENTRATIONS IN RELATION TO AVIATION ACCIDENTS

INTRODUCTION

Carbon monoxide poisoning has been the cause of death in humans since the first human discovered the use of fire for warmth, protection, and cooking. It continues today as a cause of death in humans. The introduction of household products containing synthetic nitrogenous materials in the early 19th century has increased the chance of hydrogen cyanide (a toxic combustion gas) production in a fire. The Civil Aerospace Medical Institute (CAMI) became directly involved in combustion toxicology in 1970, when a Capitol International Airways DC-8 aircraft crashed on takeoff at Anchorage, Alaska, and blood specimens from the victims were found to contain cyanide (CN⁻) inhaled during the post-crash fire. The Federal Aviation Administration (FAA), as well as the public, wanted to know the source of the cyanide and what could be done to prevent a recurrence of cyanide exposures. At that time, little was known about the potential for aircraft interior materials to produce toxic combustion gases. Even more surprising was the lack of documented data on the toxicity of individual combustion gases. During the ensuing years, scientists at the FAA Technical Center and CAMI have systematically researched this issue to answer some important questions (2, 3, 9, 10).

MATERIALS AND METHODS

The CAMI Bioaeronautical Sciences Research Laboratory receives biological specimens from almost all aviation accidents that occur in the United States. Blood specimens received from aviation accidents were analyzed for carboxyhemoglobin (COHb) and cyanide using spectrophotometric methods. The results of these tests are maintained in a computerized searchable database. All aviation cases received between 1990 and 2002 were searched for positive COHb and CN⁻ findings using the "Bioaeronautical Sciences Research Laboratory, Forensic Case Management System" (copyright 1998-2004, DiscoverSoft Development, LLC, Oklahoma City, OK). The National Transportation Safety Board database was searched for information regarding the presence of fire during the study period. COHb levels at or above 10% and CN⁻ levels at or above 0.25 ug/mL were considered positive. COHb concentrations were determined using a spectrophotometric method (1). CN⁻ concentrations

were determined using a spectrophotometric method (4, 7). In this study, we did not investigate postmortem changes of COHb and CN⁻ prior to sample collection, or the stability of COHb and CN⁻ in stored biological specimens. The Fractional Toxic Concentration (FTC) was calculated using the equation $FTC = CN^-(ug/mL) / 3.0 ug/mL + COHb(\%)/70\%$, which is based on the Fractional Exposure Dose premise reflecting the additive effects of CN⁻ and CO (2, 6). FTC values at or above 1.0 are considered fatal. CN⁻ concentrations at or above 3.0 ug/mL are considered lethal concentrations (5). COHb concentration at or above 70% are considered to be lethal (8).

RESULTS

The laboratory found 223 cases (subjects) at or above 10% COHb in blood out of 5945 cases (4%) received. The laboratory received specimens from 4482 aviation accidents. Not all of the 5945 cases received were suitable for CO analysis. CN⁻ concentrations in blood ranged from 0 to 6.3ug/mL, and positive CO concentrations were from 10% to 60% carboxyhemoglobin. Fire was reported in 201 of the cases with blood COHb at or above 10% and no fire in 21 of these cases. The fire status was not designated in 1 case. None of the 21 blood specimens from non-fire cases contained CN⁻. The National Transportation Safety Board attributed 7 of the 21 non-fire fatalities to exhaust leaks. Nicotine or its metabolite was found in 9 of the non-fire cases, and smoking may have contributed to the elevated blood COHb levels. All non-fire fatalities with COHb concentrations at or above 30% (4 cases) were determined by the NTSB to be from exhaust leaks. In some situations, damage to the aircraft prevented an examination of the exhaust system for leaks. FTCs values at or above 1.0 were only reached in 31 of the 223 cases and all of those 31 cases had elevated blood CN⁻ levels. The correlation coefficient between FTC and CN⁻ was 0.92 (Figure 1); however, no such close correlation was observed between FTC and COHb with a correlation coefficient of 0.53 (Figure 2). Elevated blood CN⁻ was detected in 103 of the 201 elevated CO cases with fire. Generally the high blood CN⁻ concentrations were associated with the low COHb concentrations (Figure 3).

The National Transportation Safety Board reported ground fires in 1716 aviation accidents, no fire in 3183 aviation accidents, 71 in-flight fires, and fire unknown in 419 aviation accidents. Only 1 of the 71 in-flight fires had a person with a COHb at or above 10%. The 1 in-flight fire case had 11% COHb and no CN⁻ at or above 0.25 ug/mL.

DISCUSSION AND CONCLUSION

Knowing the concentrations of COHb and CN⁻ in blood is useful in evaluating the contribution of the combustion toxic gases, CO, and hydrogen cyanide (HCN) to the cause of aviation accidents. The presence of elevated COHb and CN⁻ would indicate that the person was exposed to a fire environment while alive. Elevated COHb and CN⁻ levels found in the blood of individuals who died on impact would lead an investigator to conclude that there was an in-flight fire, because CN⁻ is not produced from an exhaust leak. An exhaust leak would be suspected in those cases with no fire, no CN⁻, and blood COHb levels above 20% because there is no plausible explanation for this concentration other than an exhaust leak. The fact that cyanide was found in all fire-related cases with an FTC level at or above 1.0 would indicate that cyanide played an integral part in the fire-related fatal aviation accidents (Figure 1).

The fact that COHb at or above 10% was found in only 1 of the 71 in-flight fires investigated by the National Transportation Safety Board would indicate that in-flight fires are catastrophic events resulting in the death of the individual before COHb and CN⁻ reach elevated concentrations.

However, the mere presence of COHb and CN⁻ in blood, even in potentially toxic or lethal concentrations, does not per se indicate that exposure to CO and HCN caused the death. Fire-associated deaths are highly complex events. Flame, heat, and smoke are present in all fires, including CO and carbon dioxide. Other toxic gases are also generated in some fatal fires, including corrosive or irritant gases in others, in variable proportions. Fire-induced shock can contribute to mortality; reflex cardiac death from vagal inhibition has been associated with fires. In fire-associated fatal aviation crashes, there is also commonly blunt force and other forms of trauma such as fractures. Therefore, such deaths can involve asphyxia, blunt force trauma, chemical, thermal, and/or other injuries – sometimes in combination. Further, the combined effects of 2 or more irritant, corrosive, or toxic gases can be additive, or synergistic in some circumstances.

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¹This publication and all Office of Aerospace Medicine technical reports are available in full-text from the Civil Aerospace Medical Institute's publications Web site: <http://www.cami.jccbi.gov/aam-400A/index.html>

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FIGURES

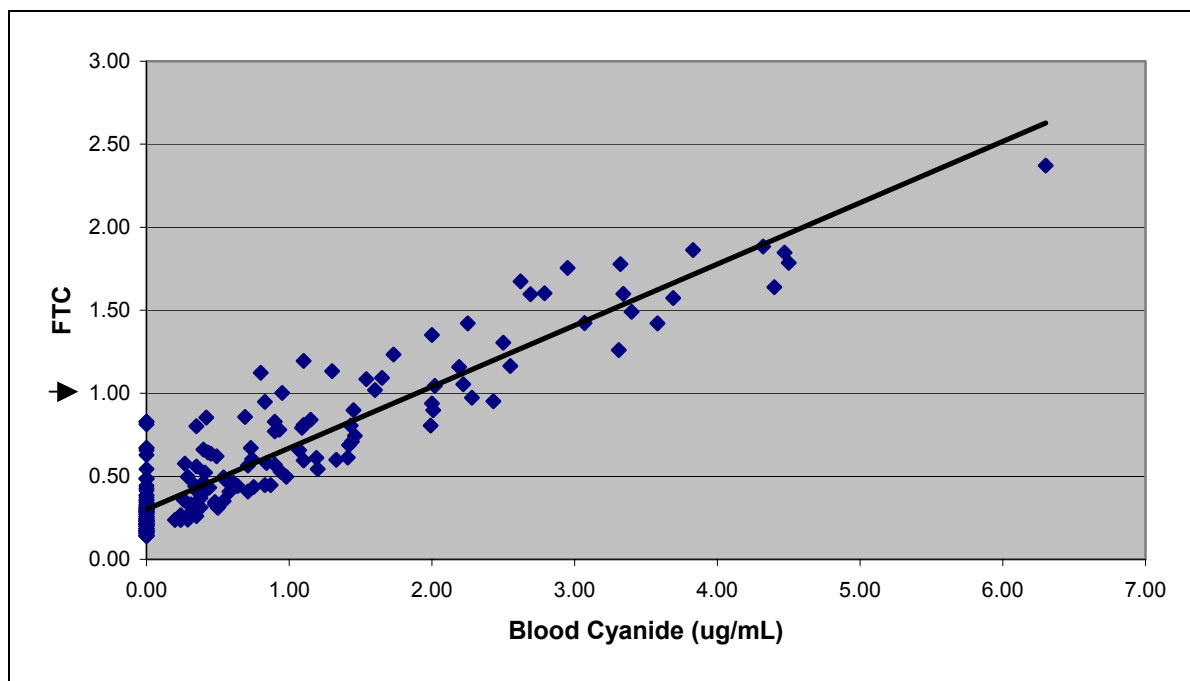


Figure 1. Fire cases showing relationship of FTC to cyanide concentrations.

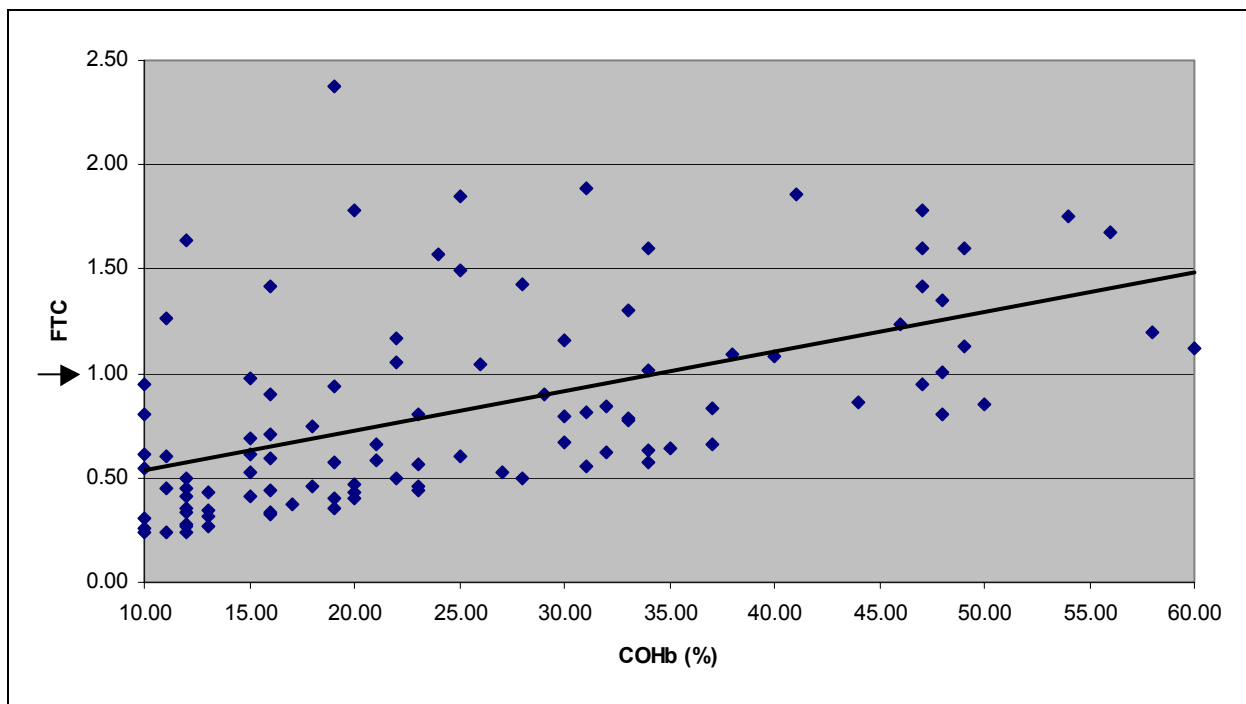


Figure 2. Fire cases showing relationship of FTC to COHb concentrations.

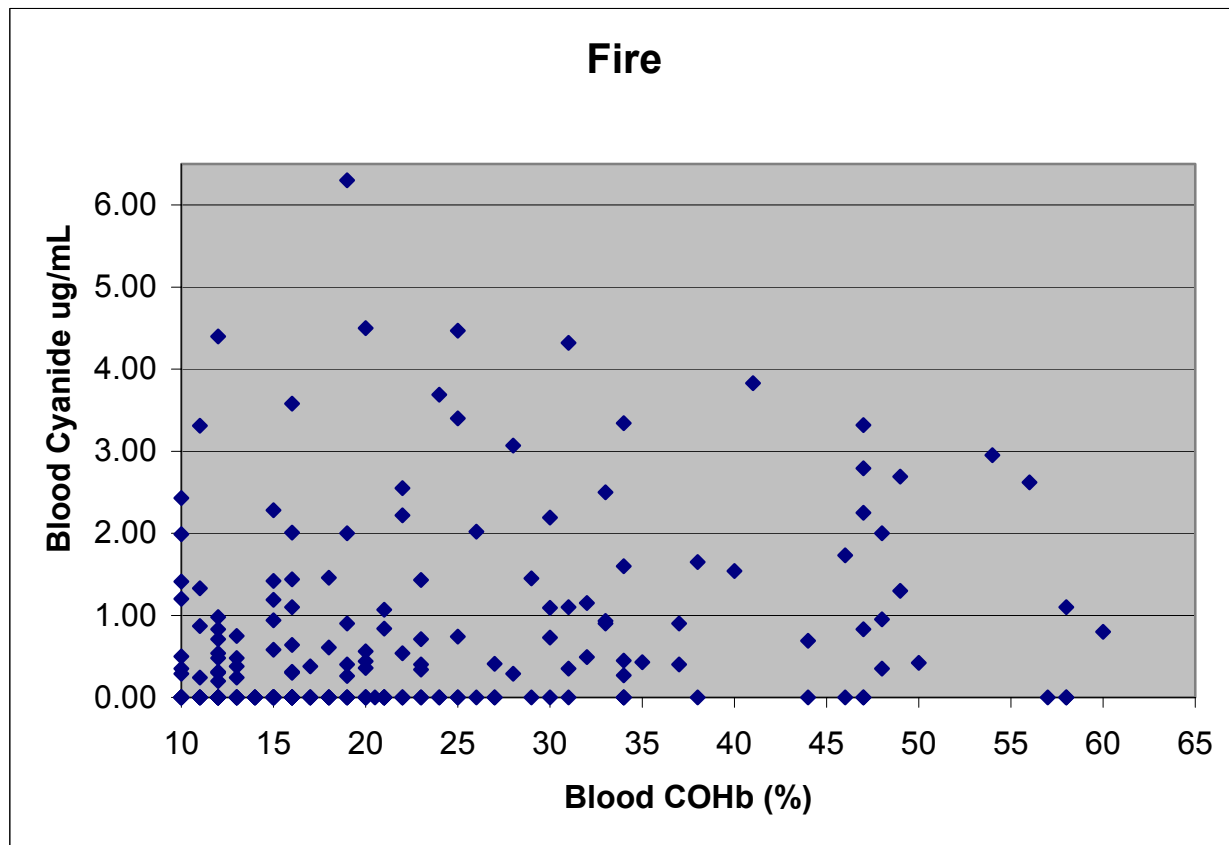


Figure 3. Fire cases showing relationship of carboxyhemoglobin to cyanide concentrations.