

Interaction between carbon monoxide and ethanol in fire fatalities

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Abstract

Impairment due to ethanol is clearly a risk factor in deaths due to fire. However, it is less clear whether there is a physiological interaction between ethanol and carbon monoxide (CO) that would alter the carboxyhemoglobin saturation level (COHb sat.) that accounts for death. In an attempt to explore this issue further, 196 fire fatalities investigated by the Office of the Chief Medical Examiner, State of Maryland over a 3-year period were examined. COHb sat. and blood ethanol concentrations (BAC) were tabulated. Twelve cases positive for therapeutic or abused drugs other than lidocaine or atropine were excluded; 184 cases were included. The data indicate that ethanol does not affect the COHb sat. that accounts for death, since the percentage of cases positive for ethanol at a given COHb range shows no trends. Therefore, we conclude that although ethanol remains a risk factor in fire fatalities, the risk appears to be related to the impairment that it produces as opposed to a direct interaction with CO. © 2001 Elsevier Science Ireland Ltd. All rights reserved.

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1. Introduction

Carbon monoxide (CO) and ethanol are two of the most common substances identified in a postmortem toxicology laboratory. It is not surprising to find ethanol in addition to carboxyhemoglobin (COHb) in fire fatalities. In a previous study of fire fatalities in Maryland [1], approximately 40% of the cases were associated with a blood ethanol concentration (BAC) greater than 0.01 g/dl. Although impairment due to ethanol is clearly a risk factor in deaths due to fire, it is less clear whether or not there is a physiological interaction between ethanol and CO. Such an interaction would alter the lethal COHb saturation level (COHb sat.). One British study concluded that the effect of ethanol was to increase the COHb saturation required to produce death [2]. They concluded that ethanol had a “protective effect” against CO intoxication since, although CO reduces the supply of oxygen to the tissues, the demand for oxygen is also reduced by the depressant action of ethanol. Therefore, individuals are able to survive higher concentrations of COHb. However, the Maryland study previously cited stated that there was no evidence to suggest that the presence of ethanol itself had any effect on the COHb sat. associated with death.

In an attempt to explore this issue further, all fire fatalities investigated by the Office of the Chief Medical Examiner, State of Maryland over a 3-year period were examined. The percent COHb sat. and BAC were tabulated and the compiled data were studied.

2. Experimental

Ethanol was quantitated in the blood by headspace gas chromatography [3]. Blood specimens were screened for COHb using a CO-Oximeter and confirmed and quantitated by gas chromatography [4]. In addition, comprehensive drug testing for therapeutic and abused drugs was performed.

3. Results and discussion

A total of 196 cases were identified over the 3-year period. Table 1 summarizes the ethanol and COHb data in these cases. Approximately 40% of the cases were associated with a COHb sat. greater than or equal to 50%. In the 1970s Maryland study, 61% of the cases were associated with a COHb sat. greater than or equal to 50%. One possibility to account for this observation is that other toxic substances are

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Table 1
BAC and COHb sat. in the compiled cases

BAC (mg/dl)	COHb sat. (%)					
	<10	10–19	20–29	30–39	40–49	≥50
<0.01	20	17	16	17	13	54
0.01–0.09	2	3	2	2	0	5
0.10–0.19	3	0	2	1	4	12
0.20–0.29	1	1	1	3	3	7
>0.30	1	0	0	3	2	1

present in more recent fires relative to the fires in the 1970s. In turn, these substances would contribute to death. However, a more likely explanation is the difference in analytical methodology used to measure COHb in the two studies. In the 1970s study, COHb was quantitated by spectrophotometry; in the current study, COHb was quantitated by gas chromatography. Our experience in using both methodologies over the past 20 years indicates that the gas chromatographic method generally gives slightly lower quantitative values than the spectrophotometric method on postmortem specimens. These differences would not be sufficient to alter the interpretation of the COHb values in the context of the case, but must be considered when comparing data collected using the different methodologies. The comparison of COHb values between the two studies is shown in Table 2.

In the present study, about 30% of the cases were positive for ethanol at a cutoff of 0.01 mg/dl. This compares to 40% of the cases that were positive for ethanol in the 1970s study. Moreover, in the current study, 77% of the cases that were positive for ethanol had a BAC greater than or equal to 0.10 g/dl. A similar percentage of cases with a BAC greater than or equal to 0.10 g/dl were observed in the earlier study. This indicates that ethanol intoxication remains a factor in deaths due to fire.

Twelve cases (6%) were positive for therapeutic or abused drugs. Drugs administered during resuscitation such as lidocaine or atropine were not considered as positive drug cases. This suggests that drugs do not play a major role in fire deaths and is consistent with observations of the 1970s study.

To assess any physiological interaction between ethanol and CO, the 12 cases positive for drugs were excluded.

Table 2
Comparison of COHb sat. between the present study and an earlier study

COHb sat. (%)	Percent of cases	
	1970s study	Current study
<10	9	14
10–19	8	11
20–29	7	11
30–39	7	13
40–49	8	11
≥50	61	40

Table 3
BAC and COHb sat. in the compiled drug negative cases

BAC (mg/dl)	COHb sat. (%)					
	<10	10–19	20–29	30–39	40–49	>50
<0.02	17	17	14	16	13	52
0.02–0.09	2	2	2	2	0	4
0.10–0.19	3	0	2	1	3	11
0.20–0.29	1	1	1	3	3	7
>0.30	1	0	0	3	2	1

Table 4
Detection of ethanol as a function of COHb sat.

COHb sat. (%)	% Cases positive for ethanol
<10	30
10–19	15
20–29	27
30–39	34
40–49	38
>50	31

Table 3 provides the distribution of BAC and COHb sat. excluding the drug positive cases. If there is an interaction between ethanol and CO, then as the COHb level increases, either an increase or a decrease in the percentage of cases positive for ethanol would be observed. Therefore, the data was reorganized to determine whether this in fact occurs (Table 4). From this table, it appears that ethanol does not affect the COHb sat. that accounts for death, since the percentage of cases positive for ethanol at a given COHb range shows no correlations. For instance, 30% of the fatalities with a COHb less than 10% saturation were positive for ethanol. Thirty-one percent of the fatalities with a COHb above 50% were positive for ethanol. Furthermore, 30% of the fatalities with a COHb sat. between 10 and 50% were positive for ethanol.

From this study, we conclude that although ethanol remains a risk factor in fire fatalities, the risk appears to be related to the impairment that it produces as opposed to a direct interaction with CO.

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