THE UNIVERSITY OF CALGARY

AN EPIDEMIOLOGIC STUDY OF FATAL CARBON MONOXIDE POISONING IN THE PROVINCE OF ALBERTA, 1978-1983

by

MARGARET MURIEL WATSON

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE

DEGREE OF MASTER OF SCIENCE

DEPARTMENT OF MEDICAL SCIENCE

CALGARY, ALBERTA

JULY, 1986

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ISBN Ø-315-32766-9

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FACULTY OF GRADUATE STUDIES

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled, "An Epidemiologic Study of Fatal Carbon Monoxide Poisoning in the Province of Alberta: 1978-1983", submitted by Margaret Muriel Watson in partial fulfillment of the requirements for the degree of Master of Science.

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Abstract

Carbon monoxide poisoning (COP) is considered to be a largely preventable environmental health problem but despite preventive measures it remains a common cause of poisoning. Although much is known about carbon monoxide (CO), there is only sparse epidemiologic literature and there have been no Canadian studies.

The present investigation attempts to characterize fatal COP through the description of the demographics of the accidental, suicidal and undetermined/unclassified deaths during the time period selected for study and to describe the human activities preceeding death. Also the relationships between occupation, the presence of diseases or conditions, drug use and blood alcohol and the deaths are examined.

Victims in the different manner-of-death classifications were found to differ in their age structures, where the deaths occurred, in the occupations which were over-represented and in the mean fatal carboxyhemoglobin levels. Moreover, unlike the other classifications, the accidental deaths appear to be better described through further sub-division into three source-environment situations i.e., deaths from motor vehicle exhaust in the indoor setting (IMV), in the outdoor setting (OMV) and those deaths from a miscellany of other sources (MISC).

These three sub-groups show differences in the age, place of death, month, season, and weekly distributions of the deaths when these are compared with each other, with the entire group of accidental deaths and with the respective distributions found in the general population of the province. This suggests that there may be three distinct populations of accidental death from carbon monoxide.

Statistically significantly different mean fatal carboxyhemoglobin (COHb) levels were found in the results from the three main laboratories in the Province. Moreover, significant differences in the mean fatal COHb values were found between (and among) the accidental, suicidal and the undetermined/unclassified cases. This was confirmed in all three laboratory situations.

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Evidence of recent alcohol use was a common finding in all manners of death and the mean fatal carboxyhemoglobin value for suicidal victims with positive blood alcohol determinations was significantly higher than that found for non-drinkers.

The mean fatal carboxyhemoglobin value for those suicidal victims with atherosclerosis and/or heart disease was significantly lower that that of individuals with no health problems, indicating that people with these conditions may be more susceptible to the effects of CO.

Over all, the findings suggest that there likely are groups of people who may be more susceptible to death from carbon monoxide than are others; indicating that preventive measures should be designed accordingly.

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ACKNOWLEDGEMENTS

The author is grateful to Dr. Donald E. Larsen, Dr. John W. Markham and Dr Herb Buchwald, members of the graduate committee at the University of Calgary, who provided encouragement and valuable suggestions throughout this investigation.

The author also thanks the following people for their time and interest: Dr. M.J. Fritzler, Dr. T.D. Kinsella, Dr. K.L. MacCannell and Dr. W.A. Whitelaw all of the Department of Medicine the University of Calgary. A special thanks is also extended to Dr. John Butt, Chief Medical Examiner of the Province of Alberta, for invaluable discussion and for the permission to examine the files held by that office. The author also thanks Dr. A.W. Rademaker, the Department of Community Health Sciences, the University of Calgary, for his assistance in the statistical analyses of the data and a special thanks is also extended to the many other people who through their interest and support made this study possible.

This investigation was generously supported by the Government of Alberta Occupational Health and Safety Alberta Heritage Grant program.

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Chapter 1

Introduction

Carbon monoxide (CO) has been referred to as the "silent killer" (Shephard, 1983); so-named as it cannot be detected by the human senses and the symptoms it evokes are non-specific, with death often attributed to other agents. This gas is ubiquitous in western society; any process involving heat, combustion or oxidation can be a source, while the greatest hazard has been found to be associated with motor vehicle exhaust.

The toxic effects of carbon monoxide have been recognized for centuries; it was utilized by the Romans to execute prisoners and it is a component of the dreaded "whitedamp", greatly feared in underground mining operations. Yet, although there is extensive literature on CO, there is far from complete understanding of the full range of its physiologic effects.

The number of individuals potentially exposed to this compound is described as exceeding that of any other harmful physical or chemical agent. In the United States, Sopher and Masemore (1970) state, "If one excludes alcohol, carbon monoxide then represents the most frequent cause of fatal accidental poisoning in this country."

A wide range of occupations potentially expose a large number of workers to this compound (e.g. Murray, 1978). However, despite educational and precautionary measures, it remains a common cause of poisoning in the home as well as at the worksite.

There is no specific (pathognomonic) symptom for carbon monoxide poisoning (COP) and those which occur may be ignored or attributed to other causes, or if correctly assigned, may go unreported. These factors, coupled with the often difficult diagnosis by health care professionals, likely act to make existing morbidity and mortality statistics somewhat unreliable; therefore, the problem may be greater than these would indicate.

Although there is a large body of information on the chemical and physical as well as the biochemical and physiologic properties of carbon monoxide, there is only sparse epidemiologic literature. Early studies (e.g. Meigs and Hughes, 1952; Mant, 1960) indicate that accidental deaths from this agent were nearly exclusively from the CO found in the coal gas which was used for residential lighting, cooking and heating purposes. Some investigations show that this type of death eventually disappeared with the decreasing use of this dangerous fuel (Friedrich, 1979). More recent studies show that currently the most common setting for accidental deaths is also the residential environment (Shaplowsky et al., 1974; Lehr, 1969; Savage et al., 1976) and that many of these fatalities are caused by CO escaping from household appliances and heaters due to their poor maintenance, faulty installation or inappropriate use. However, the main cause of death is from exposure to carbon monoxide generated by vehicles.

Most studies report that deaths appear to occur more often to males than to females and that there appears to be a seasonal relationship, largely related to the human need for warmth. The age structure of the victims is reported in all of these investigations, but findings relating to this correlate are difficult to interpret without a more complete knowledge of the population structures from which the study samples were derived.

Another problem encountered in interpreting the results of some of the investigations into COP is that the fatal and non-fatal accidental and suicidal cases are not always clearly distinguished from one another. Moreover, the paper by Baker et al (1972) suggests that the accidental category of CO deaths might be better examined through further division into several sub-categories, depending on the source and the particular environment which characterized the accident.

In addition to these considerations, information regarding the data collection and decision making processes which eventually produced the statistics used in these studies is not generally available. This would aid in the assessment of the investigations.

A number of investigations into CO poisoning suggest that individuals with atherosclerosis (Anderson et al., 1973; Aronow and Isbell, 1973) or positive blood alcohol values (Baker et al., 1972) may be at higher risk to CO. And that altitude may be a contributing factor to the number of deaths which occur at higher elevations (Savage et al., 1976). These observations pose some interesting questions ábout the relationships between COP and other factors. Because of the size of the sample in present study it may be possible to investigate these relationships.

Existing epidemiologic information about COP has been derived from different types of data bases (e.g. hospital records, death certificates, medical examiner records) and different, sometimes very specific, population samples, depending on the particular research objectives. Unfortunately the distinction between accidental and suicidal deaths or between deaths and non-lethal exposures is not always clear. While these studies do give indispensible information on carbon monoxide poisoning, results generally cannot be translated to more general populations as found in Alberta.

There have been no epidemiologic studies of fatal carbon monoxide poisoning in Canada. Alberta, the site of the present investigation, may be a particularly interesting area in which to examine deaths from this agent, for it experiences severe winters and much of the province is at higher altitudes than the rest of the country. Cold affects the efficiency of combustion of fuels, and can determine some human activities; as well, altitude can affect the performance of the source and be a factor in the uptake of CO.

Although Alberta has fewer of the high risk industries, such as metal refining and smelting than may be found elsewhere, it does have petroleum refining and mining industries and a high number of motor vehicles per capita. This latter fact suggests the possibility of exposure in many settings, but is also indicative of an active automotive maintenance industry, considered a high risk occupation for exposure to CO.

Investigations into the relationship between occupation and fatal carbon monoxide poisoning are not reported in the epidemiologic literature, although this factor may be intimately related to certain lifestyle decisions. This along with acquired skills, may in some way contribute to death through this agent.

Since there is little epidemiologic research on this problem the present study is largely descriptive and exploratory and not hypothesis-testing. It is designed to investigate all deaths (except fire deaths) attributed to carbon monoxide in Alberta from 1978 up to 1983, a period during the tenure of the current Chief Medical Examiner. Its primary goals are to give an accurate description of these CO deaths, to determine the extent of the problem and to look for trends and also to analyse the data for evidence of relationships between the fatalities and presence of disease, drug and alcohol use. It is also the intention of this investigation to describe and discuss the data collection system of the chief medical examiner system in this province and finally to suggest other avenues for future research with respect to carbon monoxide poisoning.

Research Objectives

This study has five research objectives:

 to describe the trends and the sex- and area-specific death rates, to describe the communities where these deaths occurred and the demographics of age, marital status and race in the accidental, suicidal and undetermined/unclassified deaths.

2. to examine the CO deaths for relationships with the temporal and physical environment and describe the human activities preceeding death,

3. to determine if the three populations of deaths are better described through subdivision into three source-environment categories,

4. to examine the deaths for relationships to work, such as to the worksite, to occupation and to problems with work, and

5. to describe the frequencies of and the relationships between the presence of diseases or conditions, drug use and blood alcohol and fatal carboxyhemoglobin levels.

The first objective is examined in Chapter 4, the second and third in Chapter 5, the fourth in Chapter 6 and the fifth in Chapter 7. Each chapter will be concluded by a brief summary and discussion of the findings.

Chapter 2

Literature Review

Death from carbon monoxide is generally considered as a direct and logical consequence of exposure to lethal concentrations of this agent. However, it is possible that some individuals may be inclined to be more susceptible to this type of death because of personal factors. As well, certain sources of CO and certain settings may be more often involved than others in fatal exposures.

The five objectives of the present study are aimed at the description of the circumstances of fatal carbon monoxide poisoning and it would initially appear that this review of the literature be restricted to information related to the inquiry into the characteristics of person, place and time of fatal carbon monoxide poisoning. However, both the selection of variables and the interpretation of results were greatly influenced by investigations into how carbon monoxide affects humans (i.e., its toxicology) and that it is possible that this may vary, depending upon the particular situation. This information is accordingly fundamental to the study.

This review of the literature will therefore first discuss those publications which are representative of the somewhat controversial field of CO toxicology and physiology. This will be followed by a review of those descriptive and experimental studies which reveal the current knowledge of the circumstances of carbon monoxide poisoning and introduce variables which are considered to likely affect the course of an exposure to this agent, including age, presence of diseases/conditions, drug and/or alcohol use, acclimatization, and certain factors in the built and natural environment, such as the exposure setting, temperature and altitude. Finally, this review will discuss the existing epidemiologic literature on carbon monxide poisoning.

Carbon Monoxide Toxicology and Physiology

It is commonly held that the pathophysiology of CO is due to hypoxia (e.g. Mountcastle, 1980; Wyngaarden and Smith, 1982) and that this is caused by the decreased oxygen carrying capacity of hemoglobin (Hb). This is the result of the substitution on the hemoglobin molecule by CO to form carboxyhemoglobin (COHb), thereby reducing the number of combining sites available to oxygen (0_2) .

The rate of combination of carbon monoxide with Hb is approximately 20% slower than that of oxygen with hemoglobin (Coburn, 1970); however, CO has an estimated 200 to 250 times the affinity for hemoglobin as has O_2 , with a half-life of 4-5 hours (Stewart, 1975). Therefore, under normal conditions, any carboxyhemoglobin formed will remain in the human system for a prolonged period of time.

The substitution on Hb by CO not only reduces the number and kind of sites available for oxygen transport (Kernohan, 1961), but also renders any oxygen in combination with Hb less available to the tissues (through left-ward displacement of the oxyhemoglobin dissociation curve). These reactions are thought to explain why the degree of hypoxia produced by a given blood level of COHb is found to be clinically more significant than that which would be expected in an equivalent hemoglobin-related anemia (Mountcastle, 1980).

Carbon monoxide rapidly diffuses through the lungs, the combination of this compound with hemoglobin the rate-limiting reaction (Davies, 1982). Other variables which affect the uptake (and elimination) of CO are the partial pressure of both carbon monoxide and oxygen in the ambient air, blood volume, alveolar ventilation, pulmonary diffusing capacity as well as the general health of the lungs. Many equations have been derived for the prediction of the uptake of carbon monoxide. One of the more exact models is considered to be that developed by Coburn et al.(1965).

The symptoms of carbon monoxide poisoning (COP) are commonly held to be directly related to the concentration of COHb in the blood (Table 1). The classical but non-specific symptoms include

headache, nausea and vomiting at low concentrations which may progress from mild to severe, leading to coma and ultimately resulting in death as COHb levels increase to over 60% (Dolan, 1985). Others however report a wide overlap between COHb levels and symptoms (e.g., Lasater, 1986) It is also known that exposure to very high concentrations of CO, for example, 1.0% (10,000 ppm), may cause sudden unconsciousness with no intervening symptoms (Stewart, 1975).

% COHb	SYMPTOMS*
0-10	none in healthy individuals
10-20	headache, dyspnea on vigorous exercise
20-30	throbbing headache; dyspnea on moderate exertion
30-40	severe headache, dizziness, nausea, vomiting, trouble in thinking
40-50	confusion, syncope on exertion
50-60	collapse, convulsions
60-70	coma, frequently fatal

Table 1. Levels of Carboxyhemoglobin and Corresponding Symptoms_

*from Dolan, 1985.

Although COHb is generally accepted to be the index of CO toxicity, there is a body of literature which questions whether the blood level of this compound fully reflects the physiologic insult. Indeed, some publications emphasize that the toxicology of CO is not completely understood, and suggest that carboxyhemoglobin may not be the appropriate indicator of either the range or of the kind of pathologic processes which occur.

These publications can be thought of as addressing two broad questions regarding CO toxicity; 1. whether a particular toxic action is the result of the history of the formation of COHb in the organism, i.e., time and concentration, and 2. whether other toxic mechanisms are concurrent to the effects of COHb.

<u>Carboxyhemoglobin Formation</u>. While carboxyhemoglobin levels greater than 60% are generally thought to be consistant with death, fatalities are not uncommonly found at levels below

this level (Baker et al., 1972), and some deaths have been reported at levels as low as 20% (La Presle and Fardeu, 1967) and 10% (Somogyi et al., 1981). It is not clear whether these are true findings or are only a consequence of the action of individualistic factors such as illness, or are artifacts of either the timing of blood sampling or of poor assay technique.

There is some evidence to suggest that CO toxicity is not related in a simple fashion to the carboxyhemoglobin level and that the COHb concentration alone may not explain the resultant symptoms. Notably, the concentration of inhaled CO and the duration of the exposure (i.e., the course or history of the exposure) are generally considered to be more important in predicting the clinical manifestations than is the carboxyhemoglobin level alone (Plevova and Frantic, 1974; Sokal, 1975). For example, the effects of a long exposure on humans are shown to be more severe than those of short duration, even though the resultant COHb levels are similar (Mountcastle, 1980).

Also, Plevova and Frantik (1974) find twice the decline in running performance in rats exposed to brief, high concentration of CO (COHb=19.6%) than in rats exposed to long low concentrations (COHb=22.8%). On the other hand, Sokal (1975) reports that the biochemical disturbances in rats receiving the "longer-lesser" exposure to CO are more severe and of longer duration than in animals receiving the "short-intense" exposure, even though the resultant carboxyhemoglobin levels in both groups are approximately 50%. The conflicting findings may be due to different experimental designs, in particular, the different times and concentrations selected in the two studies. What does appear to be consistant is the observed dependence of the response on the concentration of the ambient CO, as well as on the exposure time.

Further evidence of the effect of other variables on the individual response is demonstrated by Winston et al (1974), in a study of alteration of CO-lethality in rats using various drug pretreatment conditions. They report that changes in CO-lethality occur without the direct correlation of carboxyhemoglobin concentration. They also find greater lethality for pretreatment with ethanol, reduced lethality with phenobarbitol pretreatment compared to controls, while the resultant COHb concentrations in all groups are not significantly different.

These studies suggest that the clinical response to carbon monoxide exposure may not be correctly predicted by COHb concentration alone, but rather it is the individual exposure history which determines the degree of impairment and consequently the outcome.

While it is generally accepted that long periods of hypoxia caused by COHb formation explains deaths at low COHb values, this may not be the only explanation. In two provocative studies (Goldbaum et al., 1977; Orellano et al., 1976) it is reported that dogs breathing carbon monoxide succumbed to its effects at COHb levels of about 60%, while other groups of dogs receiving either COHb by transfusion or CO through interperitoneal injection, are unaffected, although both groups of animals have resultant COHb levels between 45 and 80% (mean COHb 60%).

It is concluded that the markedly different response is a consequence of the different route of entry of CO and that the toxic action of carbon monoxide is not due to hypoxia caused by COHb formation, but rather that the mechanism is through direct action on cellular respiration. Experimental evidence of a toxic mechanism of carbon monoxide, independent of carboxyhemoglobin formation, which supports these findings, is demonstrated in a hemoglobin-free system (Ingenito et al., 1974)

Such evidence suggests that, while time and concentration of CO are important considerations in describing CO poisoning, these variables alone may not adequately explain why different exposure histories result in varying pathology. It is possible that the pathologic manifestations of different times and concentrations of exposures are the result of the action of one or more toxic mechanisms, (which may include COHb formation); the possible pathway(s) of action dependant upon the particular exposure history. As Goldbaum (1977) suggests, "COHb expresses the toxicity of inhaled CO only indirectly, and more meaningful parameters of its toxicity must be searched for". The next two sections will examine the evidence for these alternate toxic mechanisms.

<u>Alternate Toxic Mechanisms.</u> There are several other biochemical entities other than hemoglobin which can react with carbon monoxide. These include the muscle pigment, myoglobin, and cytochrome oxidase in the cell mitochondria. Of these, the reaction of myoglobin with CO is well described. It is thought that carboxymyoglobin formation lags behind that of carboxyhemoglobin in

acute exposure (Goldbaum et al., 1972) but is in combination several times that of COHb under conditions of hypoxia (Coburn et al., 1973). However, CO binding to myoglobin in heart and skeletal muscle has been demonstrated to be significant even at COHb concentrations below five percent.

The reaction of CO with the cytochrome system (which is intimately involved in bioenergetics and in a number of enzyme systems) is, on the other hand, a hotly debated subject and it must be emphasized that there is disagreement whether this reaction has any physiological significance whatsoever, particularly under normal equilibrium and resting conditions (Coburn, 1979; Murray, 1978). It has been suggested that the dose may determine the reaction pathway and that exposure to a bolus of carbon monoxide could promote this combination; the finite reaction with Hb and myoglobin allowing for a temporary attack upon the cytochrome system , in particular on cytochrome^a3, an important link in the enzyme system of cellular respiration (Goldbaum, 1977).

Biochemical and pathological investigations give supportive evidence for the inhibition of the cytochrome system by carbon monoxide. For example, pulmonary cells exposed to CO have reduced or absent cytochrome oxidase staining and also exhibit bizarre changes in the mitochondrial configurations (Rhodes; 1971). Somogyi et al (1981) report neuropathologic findings which are not consistent with hypoxia alone and conclude that "the inhibition of cytochrome oxidase activity can be regarded as a toxic effect of CO".

Some physiological studies propose that many of the effects of carbon monoxide differ from those of an equivalent hypoxia. For example, the pulmonary edema frequently in evidence might be better explained by direct cellular toxicity through damage to the normal oxidative pathways in the alveolar epithelium and capillary endothelium (Fein et al., 1980). The likelihood of active CO transport by cytochrome P-450 across the placenta and alveolar-capillary membranes is suggested by Ginsberg and Myers, 1977. Mendoza (1977) suggests that such biochemical mechanisms tend to more fully explain the irregular findings of several lung-diffusion studies.

These studies present support for the existence of additional toxic mechanisms of CO; however, other studies refute these findings and although the latter investigations are not included in the

present review it must be emphasized that they are frequently well argued. The evidence for the action of other toxic mechanisms appears to be strong enough however to warrant consideration in the assessment of any case of CO poisoning and in particular when a case is equivocal. Supportive evidence has been included for this reason.

Further support for the existence of alternate toxic mechanisms may be found in studies into the effects of very low levels (2-5%) of COHb. This information is largely the result of research in the relatively new field of behavioural toxicology.

Low Concentrations and the Behavioural Toxicology of CO. The heart and brain each have high oxygen requirements and each is considered highly sensitive to the effects of CO (Nardizzi, 1979). Therefore, much of the physiological and pathological investigations concentrate on the function and cellular changes of these organs. It is in the investigation of effects on the central nervous system (CNS);however, which also has high O₂ demands, where the existence of other modes of action of CO may eventually be confirmed.

Behavioural toxicology investigates these CNS changes through a variety of experiments. These can be thought of in two broad categories; a. the neurobehavioural tests which are designed to observe changes in function of visual and auditory response, vigilance, time estimation, learning and memory, reaction time and psychomotor response (Bender et al., 1971; Oliner et al., 1983; Horvath et al, 1971); b. those tests which concentrate upon measuring the electrical activity of the brain at different levels (e.g., Groll-Knapp et al., 1982).

Although some researchers show significant relationships between low levels of CO and some behaviours, many of the results of these investigations are inconclusive. Most human experimentation has been carried out on young, healthy adults who are frequently are selected for their performance in pretesting situations. These individuals may have a greater than average ability to compensate for small dimunitions in function, having the resources to employ spare mental capacity. They thereby may be able to maintain a state of arousal which might cancel the effects of small quantities of carbon monoxide: Furthermore, these subjects generally are nonsmokers and are not stressed by fatigue or recent alcohol use.

The general state of arousal of the subject is cited as a critical element in selection of a test situation, with the consensus that, if the task is interesting enough the state of arousal will be high enough to negate the effects of low levels of COHb (O'Hanlon, 1975). And it is suggested that it is the monotonous task, the one requiring little mental input, which might better serve to demonstrate the effects of CO and that it is most important to know the effects under realistic conditions where this level of attention can be distracted. Such a situation may exist in some vehicular accidents. Wright et al (1973) show that an exposed group (COHb=3.4%) have a highly significant (p<.005) deficit in driving skills, while Ray and Rockwell (1970) demonstrate increases in time to respond to tail light intensities, a decrease in precision of maintaining separation from a leading car, and a decrease in time interval estimation. Similarly, Ramsay (1973) shows an increase in reaction time which was more pronounced in older than in younger healthy subjects.

In most behavioural studies, COHb concentration is generally reported as the meaningful evidence of exposure, but the concentration-over-time, and the testing period is not always well described. Halperin et al (1959) report that the absolute amount of COHb in the blood at a particular time alone may not represent the entire situation, rather it is the course of the exposure which determines the effect (in this case, visual discrimination). The final effect is therefore more accurately predicted by the duration of the presence of COHb in the blood as well as its concentration and even at low concentrations. It would appear then that COHb levels alone may not represent the complete situation.

Understanding low level effects may prove to be helpful in the reconstruction of the chain of events which led to an injury or to a fatality. But more importantly, rather than considering CO poisoning as a somewhat static event, the view of it as an integrated process which may include other mechanisms of toxicity, time, and concentration may eventually lead to new ways of prevention.

Presently there are no methods available to the toxicologist for routine measurement of the effect of carbon monoxide on the cytochrome system. Therefore, only COHb values are available and can be examined in the present investigation.

<u>High Concentrations of CO.</u> In 1895 the celebrated English respiratory physiologist, J.S. Haldane, exposed himself and some of his friends to considerable quantities of carbon monoxide. He reports COHb levels as high as 45%. While these values may not be comparable to results of current assay techniques, the experiments remain an invaluable source of information on the behavioural effects of relatively high concentrations of CO on humans.

Subsequent human experimentation has largely been restricted to healthy young subjects in rigidly controlled experimental conditions. Exposure has been to high concentrations of CO for only brief intervals and to subjects who were fully cognizant of the hazards.

Little is known of the experience of individuals who succumb to the effects of a sudden and deadly concentration of this gas, or what transpires as an individual remains in a marginally toxic environment, but which in time overcomes him. The only available information is from animal investigations such as that of Purser and Berrill (1983) describing the behavioural incapacitation in monkeys due to acute exposure to high concentrations of CO.

While animal investigations suggest what may happen to people, extrapolation to the behaviour of healthy humans must be approched with caution. Individual variation, different chemical composition of the source, as well as certain environmental factors may also act to mediate the effects of an exposure. Many of these latter variables are available for study and the literature in these fields will now be reviewed.

Possible Mediating Factors in Carbon Monoxide Poisoning

Several environmental and physiological variables are important in the uptake of carbon monoxide. These are the elements in the Coburn equation, referred to on page six. They include; the total inspired gas pressure, the pulmonary oxygen pressure, the endogenous production of carbon monoxide, the total blood volume and hemoglobin content, pulmonary diffusing capacity and alveolar ventilation. These variables can be expressed as those; 1. representing the composition of the inspired air, and 2. the level of health, adaptation and activity of the individual at the time of exposure.

Some of the personal factors which are thought to affect these factors include age, disease, alcohol, drug use and smoking. The environmental factors include the parameters of the source and the setting which determine the composition of the ambient air and contribute to the individual response.

Personal Factors

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<u>Age</u>. Age plays a composite role in human response to carbon monoxide. It is related to physiologic paramenters such as body size, metabolic rate and hemoglobin level; but, it also has considerable influence upon individual activity level, mental and physical health and lifestyle decisions which can contribute to the circumstances which led to the exposure to CO and may in part have determined the fatal outcome.

Young children, with higher basal metabolic rate and lower hemoglobin values, have a higher respiratory minute volume and are more susceptible to a given concentration of CO (Shephard, 1983; Zimmerman, 1981). The fetus, while resistant to other forms of maternal hypoxia, is highly sensitive to CO which readily crosses the placenta (Ginsberg and Myers, 1977; La Presle and Fardeu, 1967).

The elderly may also be more liable to COP, the consequence of compromised physiologic functions resulting from disease and/or the result of decreased physical or mental capacity (Smith and Brandon, 1970). Moreover, Pace (1948) reports that carbon monoxide is eliminated more slowly from the body with increasing age. Each year of life extending the half-life of COHb by 1%, perhaps a critical period for survival if the time course of exposure is in fact an important factor in outcome.

<u>Diseases and Conditions</u>. The efficient transport and unloading of oxygen can be influenced by a number of conditions and disease processes and this can result in an altered response to CO exposure. Aronow and Isbell (1973) report a reduced exercise tolerance in angina patients with carboxyhemoglobin levels as low as 2.5 and 3.0%. Similarly, Anderson et al (1973) reduced the time to onset of angina with COHb levels of 2.9 and 4.5%. Jones and Knelson (1980), using results

from the Aronow findings, obtain a significant linear dose-reponse regression, and from this suggest that there is no threshold dose for angina; that is, there is an effect at every COHb level.

Aronow and co-workers (1977) further demonstrate the negative effect of low levels of CO on exercise performance in chronic obstructive pulmonary disease and to time of onset of intermittent claudication (1974), the lameness and pain caused by reduced blood supply due to narrowing of the arteries of the legs. Baker et al (1972) report deaths at COHb levels of 30%, and 40% in two individuals with severe arteriosclerotic changes, suggesting that these problems may make the individual more vulnerable to the effects of CO. In agreement with these observations, Pach et al (1979) show that people with heart disease died with lower COHb saturation than those without circulatory changes.

In the United States it has been estimated that 12% of the population over 40 has substantial atherosclerotic changes (Shephard, 1983). This rate would translate to over 62,000 (approximately)people with this condition in Alberta.

A high concentration of CO can cause cardiac arrhythmia before there is a substantial elevation in COHb, even in normal individuals (Ayres et al., 1970). The bolus of carbon monoxide is thought to produce very high momentary COHb levels which would be inadequately reflected by the analyses of blood drawn minutes after damage occurs. Some individuals experience this arrhythmia even in very low concentrations of CO. DeB ias et al (1976), in a study on monkeys, shows that exposure to CO increased the vulnerability of the heart to induced fibrillation. They also show that a predisposition to ventricular fibrillation due to organic heart disease or myocardial ischemia (reduced blood supply due to narrowing of the arteries) is enhanced with carbon monoxide exposure. Such people might be more susceptible to the effects of CO than are healthy persons.

Anemic individuals have a reduced hemoglobin to supply oxygen to the tissues and therefore will reach high COHb levels more rapidly than normal individuals given a similar exposure to CO. It is also thought those with anemia would likely be more vulnerable to any reaction dependant upon COHb concentration, such as the reaction with myoglobin and cytochrome P-450 (Shephard, 1983). Pregnancy, with the increased metabolic rate and concomitant 0₂ demands, places a woman

in a higher risk category than she ordinarily would be and her unborn child shares this risk (Ginsberg and Myers, 1977).

Although the function of the brain and the central nervous system is intimately related to oxygen as well as is the efficiency of the heart and lungs, there have virtually been no investigations into the effects of carbon monoxide on individuals with brain or CNS lesions, for example, patients with cerebral palsy, stroke and Parkinsonism Barbeau (1985) suggests that individuals with this latter condition may have highly sensitive cytochrome systems). Nor are there reports available on the effects of CO on various mental disorders.

<u>Drugs</u>. Some drugs have the potential to contribute to the effects of CO. The drug and carbon monoxide may each prolong or otherwise alter the metabolism of the other, as occurs when some side effects of a medication (e.g. drowsiness and confusion) are enhanced by exposure to CO. This may lead to faulty judgment during the initial stages of exposure and perhaps eventually contribute to the inability to escape from a lethal environment.

Information on the effects of drugs and carbon monoxide is limited to a few animal studies. Montgomery and Rubin (1971), in an in-vivo study on rats, report a prolonged response to the CNS depressant, hexobarbitol, and to the muscle relaxant, zoxozolamine. The extended pharmacologic response is a result of slowed metabolism in the liver, for a delaying the rate of disappearance in the blood, thereby prolonging the depressant effects.

Winston et al (1974), show a significant increase in CO-lethality in rats receiving a one-hour pretreatment with chlorpromazine (a drug which has been used in the treatment of COP). This increase in lethality results in significantly lower carboxyhemoglobin concentrations than in rats which did not receive pretreatment. Alternatively, one-hour pretreatment with phenobarbital significantly increased COHb levels, but had no marked effect upon lethality during the inital stages of exposure. While there is little information of these effects on humans, the evidence suggests that exposure to CO and the ingestion of some medication may alter the individual response.

<u>Alcohol</u>. Alcohol (EtOH) represents a special case of drug use because of: its prevalent use in society, the close association found between it and many accidental, suicidal and homicidal deaths and the frequency of use in both accidental and suicidal deaths from carbon monoxide (Baker et al, 1972; Fink, 1966). The relationship between vehicular accidents and alcohol has frequently been described; however, the possible interactive effects of CO and EtOH on driving performance are not well known. There is some evidence that smoking, with its concomitant CO production, in combination with alcohol consumption, may be responsible for many vehicular accidents (Rockwell and Weir, 1975). For example, the relative risks of a fatal crash for individuals with blood alcohol (BAC) levels between 20–99 mg./100 ml., and COHb levels less than 5%, compared to those with similar BAC levels and CHOb equal or greater than 5%, is 1.8 and 3.8 respectively.

The World Health Organization supports this finding and suggests that CO may enhance the effects of socially acceptable amounts of alcohol (WHO, 1979). It is possible that such levels enhance the effects of carbon monoxide as well. Mitchel et al (1979) find that EtOH significantly shortened the time to behavioural incapacitation of rats exposed to CO.

Finck (1966), in a review of 567 fatal cases of COP, reports evidence of alcohol use in 60% of both accidental and suicidal deaths from CO. Baker et al, (1972) observe that four of the five fatalities with COHb levels in the 40–49% range had been intoxicated at the time of death and it is suggested that blood alcohol levels may somehow contribute to death at COHb levels which are not usually considered lethal. Molenda (1984), in a study of CO toxicity on rats, reports that ethanol prolonged the survival time a higher concentrations of carbon monoxide, while shortening it at lower concentrations. Pach et al (1979), in an analysis of the predictive factors in acute COP, reports finding COHb saturation levels slightly but significantly higher in those human subjects with positive BAC levels.

Winston et al (1974) report that one hour pre-treatment with alcohol of rats exposed to CO significantly increased lethality without affecting COHb concentration, compared to controls. Pankow et al (1974) demonstrate an ethanol-CO interaction on the activities of two indicator liver enzymes only when COHb levels were over 50%, and when either EtOH or CO had been administered

in large doses. This article cites the work of Fazekas and Rengie (1967), who report finding lower fatal COHb levels in rodents with BAC >2.3 g./l. compared to controls receiving only CO; while higher fatal COHb levels were found in animals with BAC 1.16 to 1.29g/l.

The mechanism responsible for these findings could be changes in blood supply to the liver in response to CO, or changes wrought by CO on the rate of metabolism of alcohol in the liver, as is suggested to occur with carbon tetrachloride (Suarez et al, 1972)

<u>Smoking</u>. Smokers are frequently shown to have COHb levels between 5–10%; however, values over 15% are not infrequent. It is suggested that smokers, because of their chronic exposure to carbon monoxide in tobacco smoke, will have a different response to CO than non-smokers, perhaps due to an increased number of red blood cells, a decreased CO diffusion capacity, or because of their "head start" in COHb concentration. However, Burney et al (1982), in a study of 184 victims in a case of mass COP find no evidence of acclimatization of smokers when toxic levels of CO were encountered. Smokers did not differ significantly from non-smokers with regard to frequency of symptoms, nor did smoking delay the onset of symptoms but smokers recovered from the effects of CO exposure earlier than did non-smokers. This is attributed to an increased tolerance to low COHb levels.

These findings suggest that smoking does not appear to have a protective effect on a response to high concentrations of CO. Therefore, although it initially appears to be an important variable, smoking in fatal COP will not be included in the present investigation.

Acclimatization. Acclimatization to CO has been reported in both human and animal studies and is commonly thought to be due to polycythemia, an increase in the number of red cells (Clark and Otis, 1952; Gorbatow and Noro, 1948; Stupfel et al., 1979; Wilks, 1959). However, this may not be the only explanation. Otis (1970) demonstrates that, while increased tolerance was accompanied by polycythemia in animals exposed to 0.1 and 0.5% CO, the red cell count of a group exposed to 1% CO initially increased but then fell to starting levels. This work may support that of Killick, who in 1936, suggested a reverse secretory hypotheses; whereby it is proposed that the acclimatized lungs

secrete CO from blood to the gas phase. Although there is no concordance on the mechanism of acclimatization, it apparently does occur, the history of exposure again seen as highly relevant to the prediction of the individual response.

Environmental Factors

The nature of the source will to a large extent determine what other compounds are produced with CO. For example; while HCN is a common co-contaminant of CO in fires, in other situations carbon monoxide is accompanied by varying quantities of other combustion products such as NO_X , SO_2 , NH_3 , HBr, and HF (Cohen and Guzzari, 1983; Hartzel, 1983); the composition of engine exhausts can include such gaseous products as acrolein and other aldehydes, ketones, alcohols, phenols, partially burned hydrocarbons, oxides of nitrogen, sulfur dioxide organolead compounds and a complex array of particulates (WHO 1979). And a gas stove burning an alkane fuel (e.g., propane), which in the presence of air will burn to $CO_2 + H_2O$, can, in a closed situation, produce carbon monoxide and a mix of aldehydes (Hartzell and Packham, 1983).

Exposure then is rarely to CO alone and the physiologic response may in part be the result of the additive or synergistic impact of an unknown mixture of compounds. Moreover, the production of these compounds is almost always accompanied by the consumption of oxygen in air; a vital factor since the reduced partial pressure of O_2 is in itself a hazard, particularly so in combination with increased partial pressures of CO.

<u>Altitude</u>. Savage et al (1976), in a study of COP in Colorado and Wyoming, report that the CO poisoning rate in cities at altitudes over 1829 meters is over four times that seen in cities lower than 6,000 feet. It is suggested that high elevation is an important contributing factor but it was not clear if the two areas were comparable in other important variables such as; socioeconomic status, automobile age, or number and kind of industries. It is possible that the lowered partial pressure of oxygen at higher altitudes could aggravate the situation either through the physiologic efffects of hypoxic hypoxia and/or through vehicular performance, since most vehicles are designed

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for operation at sea level. However, colder temperatures which occur at high altitude likely are reflected in an increased need for heat, thereby increasing the chance for exposure to CO. Some researchers (Mitchell et al., 1979) conclude that the evidence of a relationship between carbon monoxide poisoning and altitude is strong enough to recommend the reappraisal of CO standards for altitudes over 1,500 meters.

<u>Monthly and Seasonal Variations in COP</u>. There is strong evidence of a relationship between the frequencies of CO poisonings and month (or alternately, season) of the year. Baker et al (1972), Finck (1966), Lisella (1978), Pach et al (1979), and Savage et al (1976) all report an increase in the number of deaths in the winter months, with the latter study indicating a different monthly distribution for accidental and suicidal CO deaths.

<u>Source and Exposure Environment</u>. There is an intimate relationship between the source and the environment in CO poisoning. While the source largely determines what the composition and concentration of the combustion products will be, the environment can modify the rate at which these are generated as is found in the cold starts of an engines; whereby, during the first few minutes, the catalyst is too cold to oxidize hydrocarbons (Bernson, 1983).

The physical dimensions and structural qualities of the built environment can in part determine the rate of collection of toxic levels which occur-for example, the contrast of what would occur in a well insulated single car garage versus that found in an open structure.

The physical environment may also act in concert with human activities to promote the fatal situation, such as the need for an individual to work on a vehicle in the winter-time inside a garage in order to keep warm. Climate (or temperature) may therefore influence the circumstances of COP at different times of the year.

The three environments, or settings where CO exposure most often occurs is in the home, in a vehicle or at the place of work.

1. Domestic Exposures. The home is reported to be the most common setting for accidental COP, with approximately 60% of deaths (Rench, 1976; Savage et al, 1976). The most common

household sources of CO are vehicular exhaust, defective flues or furnaces, and structural fires. In the case of fires, it is estimated that half of all of the fatalities are due to lethal concentration of COHb rather than the result of burns (Hartzell et al, 1983).

Other origins in the home of CO are various types of heating equipment and a miscellany of stoves, refrigerators, charcoal grills, keroscene or gas-powered camp lanterns, power mowers and other small equipment (e.g., Gorbatow and Noro, 1948; Hayman, 1982). This indicates that, although appliances are approved to meet standards, they will at times be operated in an unsafe manner.

In a study of 1,820 homes in the U.S. (Schaplowsky et al, 1974), nearly 17% are found to have C0 readings greater than 10 ppm. This supports the findings of McFee et al (1970)where positive (30-70) carbon monoxide readings (using paladium chloride detector tabs) are found in 27% of 460 homes, while an additional 5% of the homes had higher concentrations of C0. In another report, 25% of the home appliances which were inspected contribute to measurable amounts of C0 in the home (Lehr, 1969). It is been suggested that long-term low-level concentrations of C0 may be harmful to health, but the effects of this type of exposure on judgment, (which may have led to the fatal exposure) can only be guessed from some of the research in behavioural toxicology.

While these investigations into domestic exposures do not involve fatalities, they indicate the prevalence of levels of this gas in homes in various geographical areas; levels which prior to the investigations had gone unnoticed.

2. Vehicles. Exhaust from automobiles and other vehicles are responsible for many unintentional carbon monoxide deaths. A study (Baker et al., 1972) of fatal unintentional COP in motor vehicles, estimates that over 500 Americans die in this manner each year and that the principal reason for these deaths is because "their vehicles are defective due to deterioration, damage, or poor automative design". McFee et al (1970), found defects in 16 of 342 (4.7%) automobiles tested. The ages of these vehicles are not described; however, the reasons for the escape of CO are similar to those seen by Savage and co-workers.

Williams (1985), in a community program of screening and education for prevention of vehicular COP in a high risk population, finds that 18.6% of the vehicles tested exceeded the EPA standard for 8 hours of exposure (9 ppm), and that 2.6% exceeded the 1 hour standard (35 ppm). It is concluded that there are high risk populations for vehicular COP, these made up of individuals who require vehicles for transporation, but who have low incomes and are without adequate means (i.e., financial) to perform routine maintenance.

Among other factors, the concentration of carbon monoxide in vehicular exhaust depends upon the type and condition of the engine. Rose and Rose (1971) state that this could range from 1 volume percent (10,000 ppm) for an idling, well-adjusted engine, to 14 volume percent (140,000 ppm), for a badly-adjusted old engine at full throttle. Finck in 1966, states that "in general, the engine running in a closed single-car garage produces an amount of CO fatal to man within 5 minutes.

Over the years improved engine performance and the use of properly installed catalytic converters reduced the amount of CO in vehicular exhaust (Ninomiya, 1977). For example, 1980 U.S. emission standards represented an 96% reduction in CO emissions compared to uncontrolled emission levels in 1967. However, disconnection or fouling of converters (e.g, with misfueling Nebel, 1981), poor engine maintenence, cold temperatures and altitude are all factors which can effect the efficiency of any engine. Therefore each vehicle will likely differ from another and will likely vary itself over time and conditions.

3. Industrial Exposures. Carbon monoxide is frequently cited as one of the most common industrial poisons. The jobs with potential carbon monoxide exposure are varied and numerous (e.g., Michael, 1979); however, occupational exposure can occur from any process which at some stage involves the use of heat, fire, combustion or oxidation. At particular risk, for example, are workers in iron or steel foundaries, pulp mills, petroleum refining, coke production, arc welding, chemical processes, firefighting, transportation and automotive or garage work (Zenz, 1979).

In Alberta the number of mechanics, repairmen, firefighters and workers in the transportation industry would have been approximately 90,000 using 1981 Statistics Canada figures. Other occupations which may have some risk of CO exposure, such as farming, mining processing

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occupations, metal shaping and forming, bring this to nearly 190,000 workers. This number does not include an additional 72,000 people who are in occupations involved in food and beverage preparation, excavating, grading and paving, chemical, petroleum and rubber industries or stationary engineers, workers who also may be at some risk to carbon monoxide exposure. The actual number of workers with the potential for exposure cannot be easily determined, but the above numbers do suggest that, although Alberta is not a highly industrialized area, opportunities for exposure do exist for many persons in many occupations.

There have been several studies investigating the non-lethal exposure to CO in various occupations (e.g. Buchwald, 1969; Gothe et al., 1969; Jones et al., 1972). The objective of some of these studies is often to assess the exposure to CO with reference to the occurrence of atherosclerosis and little is known from these investigations about the actual number of fatalities from this agent in various industries or occupations.

Epidemiologic Literature on Carbon Monoxide Poisoning

Compared to the volume of literature on the biochemistry, the physiology or the behavioural toxicology of CO there have been few epidemiologic investigations into fatal CO poisoning. The studies which do exist generally use different populations and different data bases (e.g., death certificates, coroner or hospital files) and it is therefore difficult to compare results between studies. Although the relevant results of these studies have already been discussed, it is useful to briefly review these investigations and their particular emphasis.

Finck, in 1966, studied 567 accidental, suicidal and undetermined fatalities from CO from all sources (including fire) which were found in the files of the U.S. Armed Forces Department of Pathology from 1940–1960. A review of the pathology was of prime interest; however, this publication also includes information about the age, race, sex, COHb levels and alcohol use and also documents the source and the month of the year in which the deaths occurred.

Smith and Brandon in 1970 looked at 206 fatal and non-fatal episodes of COP known to the coroner of the city hospitals in Newcastle on Tyne between 1965-1967. While several

demographic variables such as age and sex, as well as the nature of the source and occupation are discussed, the emphasis of the study is to examine the nature and incidence of neuropsychiatric sequelae of CO poisoning and to suggest whether current patterns of management should be revised.

Baker et al in 1972 reviewed 68 cases of fatal accidental COP which occurred in vehicles, using data extracted from the files of the Chief Medical Examiner of the State of Maryland. This informative paper includes the age and sex of the victims, as well as the COHb and alcohol levels in this specific kind of accident. The objective of the study is determine the extent of the problem in the state, and to suggest preventative measures.

Savage et al, in 1976 reviewed 237 cases of fatal and non-fatal accidental cases of COP from all sources (including fire) which were in the records of 30 selected urban and rural hospitals in Colorado and Wyoming in the period 1971-1973. The objective of this study is to understand ways to prevent this occurrence. Variables of interest are; age, race, sex and month of year.

Lisella et al, in 1978 published a brief paper describing 804 fatal events in which CO was either a primary or a contributing cause of death. Data we're extracted from death certificates in the State of Georgia from 1961 to 1973. The objective of the study is to obtain certain epidemiologic information about CO deaths in the state. Variables of interest are; age, race, sex, marital status, and the month of death. All sources of CO including fire are included.

Pach et al, in 1979 compare 220 patients treated in the Toxicological Clinic with 101 cases from the Institute of Forensic Medicine, Warsaw, Poland in the period 1975–1976. The aim of the study is to search for the factors influencing the clinical picture and mortality. Variables of interest are; age, sex, month, alcohol and carboxyhemoglobin levels, as well as circulatory changes.

Chapter 3

Methods

The data for this study were extracted from files held by the Office of the Chief Medical Examiner (OCME) of the Province of Alberta. The validity of a descriptive study greatly depends upon the quality of the data from which it was derived; therefore, this chapter will introduce the topic of the source and quality of the data by describing in some detail the Medical Examiner system in this province, the reasons for the data collection, its completeness and reliability and its applicability to the objectives of the present research.

Other topics to be included in this chapter are: the retrieval of files, selection of the sample, data collection, design of the data forms, agreement of confidentiality, statistical procedures and definitions.

Medical Examiner System in Alberta

A medical examiner system is responsible for establishing the cause as well as the manner of death in certain well-delineated situations of sudden or unexpected deaths which may occur within its jurisdiction. In Alberta these include all deaths which have occurred unnaturally, unexpectedly or cannot be explained. These are:

- 1. unexpected death;
- 2. unexpected death when the deceased was in apparent good health;
- 3. death as a result of violence, accident or suicide;
- maternal death during or following pregnancy and that might reasonably be related to pregnancy;
- 5. death that may have occurred as a result of improper or negligent treatment by any

person;

- death that occurs within 10 days of an operative procedure or while under or during recovery from anesthesia;
- death that results from poisoning;
- 8. death of a person not under the care of a physician;
- 9. death resulting from any disease, ill health, injury or toxic substance arising from a person's employment or occupation now or in the past.
- 10. death of a person while in custody, for example:
 - i) in jail or in a correctional facility, or in custody of a peace officer,
 - ii) death of a formal patient in any facility under the Mental Health Act,
 - iii) death of a child in custody of the Director of Child Welfare, for example, foster children.

In Alberta the Chief Medical Examiner must be a medical pathologist and is appointed by the Lieutenant Governor in Council, while the Medical Examiners are physicians who have been appointed by the Attorney General. Many Medical Examiners have received medicolegal training, and they are frequently assisted by medical examiners' investigators. Full-time medical examiners' investigators work for the Chief Medical Examiner in the Edmonton and Calgary offices. The investigators are often graduate nurses with specialized training in medicolegal investigation; however, every member of the R.C.M.P. or a municipal police force is, by virtue of his office, a medical examiner's investigator. The police will assist the medical examiner; however, if foul play is suspected the investigation will be directed by the police, but police interest in the case frequently ends when it has been confirmed that no crime was committed.

<u>Reasons for Collection of the Data</u>. The medicolegal investigation into a death goes beyond the strictly medical interest in certitifying the nature of the disease or injury which caused the death, and inquires into the set of circumstances which led to this cause. The emphasis therefore is not only upon the cause, but also upon the identification of the victim and an explanation of the manner-of-death.

Every effort is made to ensure the accuracy and completeness of this information, since it has a bearing upon many aspects of our society. This information in some cases is vital to the administration of justice; most notably in situations where there is a potential for criminal prosecution, civil action, insurance benefits or workmen's compensation; moreover, the decision of the Medical Examiner may have significant consequences for survivors, such as financial considerations, social and psychological stigma, and the denial of certain religious rites.

Much of the investigation is directed to the understanding of the circumstances of death in order to prevent future occurrence; thereby acting as an often unrecognized form of preventive medicine. The system strives for complete and accurate death certification which in turn determines the validity of certain vital statistics upon which much research is based. There has been criticism regarding how, and how well (Atkinson, 1978), coroners arrive at their decisions, and there is much disparity between different systems (Barraclough, 1972; Nelson et al, 1978),but it is likely that the Medical Examiner System in the Province of Alberta represents the state-of-the-art in the investigation into sudden and unexpected death.

In the case of carbon monoxide poisoning the OCME is frequently called after the ambulance attendants, the police or an attending physician have abandoned resuscitative attempts. The medical examiner, or the medical investigator, generally thoroughly documents the death scene, inquires into the victim's medical background, recent behaviour, previous suicide attempts, drug and alcohol use, and obtains any other information which appears to be relevant to the case. Information is obtained from relatives, witnesses, personal physicians, and pharmacists, or any source which might possibly aid in qualifying the death. Every effort is made to reconstruct the events preceeding the death so that it may be understood and explained. Some of this information is reported on standardized forms; however, much of the detail is frequently found in the form of a hand-written narrative.

The system strives for a detailed record of all phases of the investigation, and eventually all information on the case is bound into a single case file. The variables considered to be of interest are then coded onto a form and attached to the file cover. This system of investigation is not static

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and the variables of particular interest are not fixed; rather, their inclusion has evolved over time. Changes are in part determined by the needs of the society the system serves, scientific knowledge, and as well they are conditioned by the experience and philosophies of the current Chief Medical Examiner.

The information found in the files of the Office of the Chief Medical Examiner can therefore be described as the result of a highly organized data-collection system, alternatively referred to as a "high profile system" since the data describe the victim and the circumstances in high relief, developing a detailed personal profile for each fatality.

<u>Completeness</u>, <u>Reliability and Applicability of the OCME Data to the Objectives of the Present</u> <u>Research</u>. Many deaths caused by carbon monoxide pose notable medicolegal problems. The physical properties of this compound, and the insidiousness and speed with which it can act, the availability and variability of the sources, the unreliability of witnesses, the modification of the scene by survivors and ambivalent personal histories or unclear circumstances frequently act to complicate and confuse the reconstruction of events and to obscure the intent of the victims.

Certain equivocal deaths due to carbon monoxide are, characteristically, exhaustively investigated until a decision can be made, or until no further evidence is forthcoming. If doubt still remains as to the manner of death, the case may be given an open verdict, which will be entered as "unclassified" or "undetermined", depending upon whether or not an excessive consumption of alcohol was found to have occurred before death. The depth of the investigation and the completeness of the data collection therefore will not be identical in every case. While this will not likely affect the routinely collected demographic variables such as age, sex and marital status death, it can influence the inclusion of variables such as drug use and the presence of diseases or conditions. This fact must be considered during the analyses and in the interpretation of the results.

The understanding and prevention of fatal exposure to carbon monoxide is the principal goal of the present research, encompassing all of the stated objectives. Although the data are primarily collected to aid in the determination of manner and cause of sudden death, they are also used for a secondary objective of the OCME, i.e., the prevention of death, an aim common to this investigation.

The rigorous manner in which categorization of manner of death is accomplished under the medical examiner system in this province likely minimizes the chances of incorrect assignment and suggests that a conscientious collection of information has taken place. The completeness and reliability of the OCME data and its applicability to the present study would appear to support the use of this data in the epidemiologic investigation of fatal carbon monoxide poisoning.

Selection of Sample

The selection of the cases to be included in the present investigation was influenced by three main factors: the tenure of the present Chief Medical Examiner, considerations of time and ease of access; and carboxyhemoglobin levels.

<u>Tenure of the Chief Medical Examiner</u>. The office of the Chief Medical Examiner was held by the same pathologist throughout the period of this study. He had assumed office in 1976, and at that time had initiated a new system of investigation.

<u>Time and Access Considerations</u>. Examination of the OCME records showed that there had been approximately 100 (non-fire) deaths ascribed to carbon monoxide each year since 1976. Estimation of the time required to examine each file and the ease of access to the files influenced the choice of the time period; as well, inspection of the the files earlier than 1978 indicated that these were not as detailed as those from 1978 onward.

<u>Carboxyhemoglobin Levels</u>. It is commonly held that levels of carboxhemoglobin of 60-70% are nearly always fatal, or consonant with death; however, there have been many fatalities at levels far below 60% (e.g. Baker et al, 1972), and some as low as 10 and 20% (Somogyi et al, 1981; La Presle and Fardeu, 1967) It was therefore decided that for a case to be included in this investigation the death must have been attributed to carbon monoxide, a COHb determination must have been reported and the level of COHb must have been equal to, or greater than, 20.0% COHb.

The physiologic action and the effects of CO on humans is likely similar irrespective of the intent which was assigned i.e., accidental suicidal or undetermined/unclassified; therefore, all categories

of intent are included in the study. It was felt that, not only would this would increase the number of cases and the breadth of situations which could be investigated, but also the different categories of · intent might prove useful contrast groups at different stages in the investigation. The undetermined and unclassified groups were small and were combined into one category, undetermined/unclassified, or "Undc".

Therefore, all accidental, suicidal, undetermined and unclassified carbon monoxide deaths (nonfire) found in the OCME log books from 1978-1983 with carboxyhemoglobin levels equal to, or

greater than, 20.0% COHb are included in this study.

Data Collection

All sudden and unexpected deaths which were accepted as cases by the Office of the Chief Medical Examiner are listed in the annual log books held by this office. Different medical examiners frequently certify death from carbon monoxide in different ways, and this cause of death is transcribed directly into the log book. Most causes of death were described through the agent itself (e.g., carbon monoxide poisoning/intoxication), through the source, (e.g., inhalation of exhaust fumes), while others were through the mechanism of the death (e.g., anoxia). Therefore, to ensure the inclusion of all deaths ascribed to COP, it was necessary to examine all files which referred to carbon monoxide, carbon monoxide sources, and to unqualified anoxia, in order to determine if the cases had indeed been attributed to CO. Also to discover if there was a reported carboxyhemoglobin equal or greater to 20.0% COHb. Each case which met these criteria was read in its entirety by the author for the purpose of coding relevant variables.

<u>Retrieval of Files</u>. The current files and those of the previous two years are held in one of the two offices of the Chief Medical Examiner, with the Edmonton office holding the city's files and the North Rural documents. Similarly, the Calgary office holds its files and those of the South Rural area. Files of earlier years are held in the archives, in Edmonton. The out-of-town files were accessible by request using catalogue numbers which appear in the annual log books for the four

areas. All but 10 case files were retrievable. Six of these ten were suicidal, one was accidental and three were undetermined/unclassified cases.

<u>Design of the Coding Sheets</u>. The development of the coding sheets was guided by the literature, by variables used by the OCME and by the aims of the present study. This process was concomitantly conditioned by the limitations of the data base. It was possible to code the more common demographic variables such as age and sex in the same manner employed by the OCME; however, some variables such as drugs and diseases-conditions required the development of a coding scheme which would reflect the range and levels of these variables.

The first pretesting was done on one lot of fifty CO files which had been randomly selected from the provincial deaths over the six-year period. A second pretesting was done on another set of files randomly selected from the Calgary area. The forms were modified with each testing until the final design proved suitable. A reliability check on the coding was to have been carried out by having another investigator independently code some of the cases. However, permission to perform this step could not be obtained. Instead, the design of the coding sheets was thoroughly discussed with a senior faculty member of the Department of Community Health Sciences at the University of Calgary, and it was concluded that the sheets were appropriate for the purposes of this study (see Appendix A).

Statistical Procedures

This study is mainly descriptive and therefore will be largely enumerical: characterizing the person, place and time variables found in fatal carbon monoxide poisoning. Comparisons will be accomplished with the use of univariate statistical methods.

Additional analyses will explore this data-set for evidence of relationships between variables such as fatal carboxyhemoglobin levels and disease or alcohol consumption. These analyses require bivariate statistical procedures. A significance level of p <.05 is used in all analyses.

Agreement of Confidentiality

Each researcher using the data held by the Office of the Chief Medical Examiner must agree to keep all information confidential, not to contact the survivors of the victims, and to present the data so that the individual cannot be identified. This agreement is in the form of a signed document which is held by the OCME.

Definitions

In nearly all cases each study variable is clearly defined in the code book (see Appendix B). For clarity however, it is necessary to describe in greater detail what exactly is meant by the use of the two terms, area and community of residence.

<u>Area</u>. This refers to the geographic areas defined by the office of the Chief Medical Examiner. This office has divided the province into four areas of roughly equal population: 1. Calgary, 2. South Rural, 3. Metropolitan Edmonton, and 4. North Rural. The north-south rural boundary has been drawn east-west through Jasper, Hobbema and Provost.

While the two major cities are well described by the term "urban", the North and South Rural areas are not strictly rural; for they include the smaller cities of the province, as well as the towns, villages and other smaller communities. To differentiate between the populations of the two large cities of Calgary and Edmonton and that found in the rest of the province, this investigation will substitute the terms "metropolitan" and "non-metropolitan", to avoid confusion with the commonly accepted meanings of the terms urban and rural.

<u>Community of Residence</u>. This variable was developed to attempt to deal further with the problem of defining urban and rural areas. As well, it is used to describe the actual locale where the victim had resided immediately before death, rather than the area in Alberta where the death occurred.

Chapter 4

Description of the Time and Place of the Fatalies and the Demographics of Sex, Age, Marital Status and Race

This chapter will describe the findings relating to when and where the deaths occurred as well as enumerate the common demographic variables of sex, age, marital status and race which characterized the carbon monoxide fatalities. These findings relate to the first research objective, "to describe the trends and the sex- and area-specific death rates, to describe the communities where these deaths occurred and the demographics of age, marital status and race in the accidental, suicidal and undetermined/unclassified populations of deaths over the six-year study period.

Trends and the Sex- and Area-Specific Death Rates

Calgary and Edmonton had between 56-60% of the total provincial population in the period selected for study and approximately 51% of both the urban and rural population was male. While these figures changed very little over the six years from 1978-1983, the total population of the province rapidly increased by 20%, with the two major cities and the less densely populated areas sharing equally in this rapid growth.

In 1980-81 the economy took a sudden downturn, slowing the influx of people to the degree that, in 1983, the population of Calgary had a negative increase over the previous year. It is of interest to see if these dramatic population and economic changes are reflected in the trends in the accidental suicidal or undetermined/unclassified death rates over this period.

<u>Trends</u>. Comparison of the annual death rates (Table 2) does not demonstrate any overall consistent trend but rather shows a mixed pattern. The rates increased in 1982 and 1983 over those

of the previous year for the accidental and suicidal categories of deaths; however, these changes are not statistically significant.

Year	N	Accidental	N	Suicidal	N	Undc
1978	18	0.92	44	2.25	12	0.62
1979	14	0.70	77	3.82*	20	0.99
1980	16	0.77	80	3.84	3	0.14*
1981	16	0.72	69	3.08	5	0.23
1982	19	0.82	78	3.36	11	0.48
1983	21	0.89	81	3.45	· 5	0.23
Total	104		429		56	

Table 2. Annual Accidental, Suicidal and Undetermined/Unclassified (Undc) Death Rates (per100,000) from Carbon Monoxide Poisoning in Alberta, 1978-1983.

*significant , alpha = .05

Sex- and Area-Specific Death Rates

For the years 1978–1983 there were a total of 104 accidental CO deaths; 83 (80.6%) are male, 21 (20.2%) are female, indicating a preponderance of male accidental deaths from carbon monoxide. Examination of the mean sex-and area-specific death rates over the period (Table 3) shows that the rate of accidental deaths for males exceeds the female rate in both the metro (5.1 to 1) and the non-metro (3.5 to 1) settings. The rate of accidental death for both sexes is higher in the non-metropolitan areas, compared to that seen in the two large cities, (3.2 and 4.5 times higher for men and women respectively).

The suicidal CO deaths present a somewhat different picture. There are 429 deaths deemed suicidal in the 6-year period; 336 (78.3%) of these are male and 93 (21.7%) are female. As described for accidental deaths, a preponderance of male deaths is found in both the two large urban

centers as well as in the more rural communities. The sex-specific male rates are respectively 3.0 and 3.6 times the female rates in the metro- and non-metro regions. However; unlike accidental deaths, the metropolitan areas have higher suicide rates than the nonmetropolitan areas and this was true for both males and females (1.4 and 1.6 respectively).

Area	Sex	Accidental Rate (N)	Suicidal Rate (N)	Undc Rate (N)
Metro	Male	0.66 (24)	5.74 (215)	0.88 (33)
	Female	0.13 (5)	1.73 (63)	0.11 (4)
	M+F	0.40 (29)	3.76 (278)	0.50 (37)
Non-	Male	2.05 (59)	4.22 (121)	0.56 (16)
Metro	Female	0.58 (16)	1.09 (30)	0.11 (3)
	M+F	1.33 (75)	2.68 (151)	0.34 (19)
Province	Male	1.26 (83)	5.08 (336)	0.76 (49)
	Female	0.28 (21)	1.46 (93)	0.11 (7)
	M+F	0.80 (104)	3.30 (449)	0.44 (56)

Table 3. Mean Annual Accidental, Suicidal and Undetermined/Unclassified (Undc) CO Death Rates (per 100,000)* for the Population of Alberta, by Sex and Area: 1978-1983.

*populations derived from the 1981 Canadian Census and intercensal estimates adjusted for annual urban growth-rate as reported in the annual city census of Edmonton and Calgary.

Of the 56 undetermined/unclassified deaths in this period, 49 (87.5%) are male while 7 (12.5%) are female. As found in the accident and suicide groups, the male death rates predominate female rates in both the metropolitan (8.0 to 1) and non-metropolitan (5.1 to 1) settings. The number of metropolitan undetermined/unclassified male deaths is greater than that in the non-metropolitan areas (1.6 to 1), while the female rates are identical in both areas.

Table 4 presents a more detailed breakdown of the frequency of deaths in various sizes of communities of the province. The number of accidental deaths in the rural areas of the province shows that there are 1.3 and 2.0 times the number of urban deaths for males and females respectively. On the other hand the number of urban suicidal deaths are 3.9 and 4.6 times the number of rural suicidal deaths for males and females respectively. The number of undetermined/unclassified deaths are also more of an urban phenomenon, with 3.4 and 2.5 times the number of male and female rural deaths respectively in urban versus rural areas.

	Acc	idental	Su	icidal	U	ndc
Community	M	F	. M	F	М	F
Calgary and Edmonton	22	6	230	66	34	4
Cities<500,000	13	1	30	8	· 3	1
Towns	25	9	42	12	7	2
Villages	18	3	24	4	4	0
Reserves	4	2	1	0	0	0
Other/Unknown	1	0	9	3	1	0
Total	83	21	336	93	49	7

Table 4. Number of Male and Female Accidental, Suicidal and Undetermined/Unclassified (Undc) Deaths by Community in the Province of Alberta: 1978-1983.

It must be noted that the definition of type of community in this province is not strictly related to size and therefore the area-specific death rates can not be readily calculated for all the subcategories of communities. However, with the knowledge that there were similar proportions of males in the rural and urban areas, it is sufficient, for the purposes of this study, to note that the male rates are certain to be higher in every type of community. The percentage of male deaths ranging from 67.0 to 92.9% for accidental, 77.7 to 100% for suicidal and 75.0-100.0% for the undetermined/unclassified deaths, depending upon the particular community.

Although the under-16 age category made up about one-quarter of the population of the province (Statistics Canada, 1981) it experienced proportionately very few of the deaths in any manner-ofdeath group. Therefore the comparison of the age distribution in the three groups with the provincial population is performed on the 16 and over age categories. Even with this manoeuver the age distributions in all manner-of-death categories differed significantly from that of the province, i.e., accidental p<.001, suicidal p<.001 and Undc .02<p<.05.

Accidental deaths from CO occurred over a wider age range (Table 5) compared to suicidal deaths; however, a comparison of median and modal ages shows that a younger age group generally experienced the former fate. These findings are confirmed by the examination of the age- and sexspecific rates for accidental, suicidal and undetermined/unclassified deaths (see Table 6,7,8).

	Accidental			Suicidal	,	· Undc		
	M	F	М	F	М	F		
N	85	23	336	93	49	7		
Median*	28	22	36	40	38	54		
Mode	19	17	22	40	not u	nique		
Mean	35.3	36.0	38.8	38.8	40.2	45.7		
S.D	19.4	21.8	15.4	11.1	14.1	19.0		
Range	2-80	16-76	13-89	9 16-62	17-74	20-68		

Table 5. Measures of Central Tendency and Dispersion in the Ages of the Male and Female Accidental, Suicidal and Undetermined/Unclassified (Undc) Populations of Deaths from CO.

It can be seen that the age-specific accidental death rate for males (Table 6) is highest for the 76-85 age group. However, while the age specific rate is higher for the older group, the actual count of deaths experienced by the younger men is nearly six times that of the older men. The age-specific rate in part conceals the fact that there are more deaths (5.8 to 1) and more men (13 to 1)

in the younger age group in the general population. Females have lower rates than males in all age categories but similarly have the highest rate and the greatest number of deaths in the 16-25 group.

Age	Male N	Female N	M+F N	%M/%FAlta
<16	0.11 (2)	0.00 (0)	0.06 (2)	26.0/25.7
16-25	2.29 (35)	0.84 (12)	1.69 (47)	22.3/21.8
26-35	0.77 (10)	0.08 (1)	0.44 (11)	18.9/18.1
36-45	1.29 (10)	0.14 (1)	1.00 (11)	11.3/11.0
46-55	1.78 (11)	0.17 (1)	0.73 (12)	9.0/8.8
56-65	1.60 (7)	0.87 (4)	1.34 (11)	6.4/7.0
66-75	0.75 (2)	0.33 (1)	0.52 (3)	3.9/4.6
76-85	5.12 (6)	0.67 (1)	2.63 (7)	1.7/2.3
>85	0.00 (0)	0.00 (0)	0.00 (0)	0.4/0.7
Total	83	21	104	100.0/100.0

Table 6. Age and Sex-Specific Accidental Death Rates (per 100,000)* for Males, Females and Both Sexes and the Provincial Population (%) in these Age and Sex Groups.

*mean six-year rate.

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The suicide rates peak at ages 36-45 for both males and females (Table 7). However, each of the two younger male age categories (16-25 and 26-36) have more actual deaths. This is not the case for the female suicidal fatalities, which have the highest rate, as well as the greatest number of deaths in the 26-45 age category.

Age	Male N	Female N	M+F N	%M/%FAlta
<16	0.11 (2)	0.00 (0)	0.06 (2)	26.0/25.7
16-25	5.04 (77)	0.91 (13)	3.04 (90)	22.3/21.8
26-35	6.46 (84)	1.85 (22)	4.26 (106)	18.9/18.1
36-45	8.77 (68)	4.70 (34)	6.80 (102)	11.3/11.0
46-55	8.07 (50)	3.13 (18)	5.70 (68)	9.0/8.8
56-65	7.07 (31)	1.31 (6)	4.12 (37)	6.4/7.0
66-75	7.87 (21)	0.00 (0)	3.68 (21)	3.9/3.6
76-85	1.70 (2)	0.00 (0)	0.75 (2)	1.7/2.3
>85	3.48 (1)	0.00 (0)	2.70 (1)	0.4/0.7
Total	336	93 [′]	429	100.0/100.0

Table 7. Age and Sex-Specific Suicidal Death Rates (per 100,000)* for Males, Females and Both Sexes and the Provincial Population (%) in these Age and Sex Groups

*mean six-year rate.

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The undetermined/unclassified age- and sex-specific death rates (Table 8) peaks for males in the 56-65 year age group and for females in the 46-55 age group. Males in the ages 26-35, 36-45, each have thirteen deaths, over one and one-half times the number seen in the older age category (56-65).

The total number of observed accidental deaths exceeds the expected number (calculated from 1981 population statistics) in only the 16-25 year age group and the proportion in this age category is significantly different (p<.001) from the corresponding proportion of 16-25 year olds in the provincial population.

Similarly, only the 36-45 and the 46-55 year age groups have an excess number of the suicidal deaths. Both of these are significantly different compared to the corresponding proportion of people in this age group in the population of the province (ages 36-45, p<.001 and ages 46-55, p=.0146).

The undetermined/unclassified deaths have an excess of deaths in three age groups, i.e., 36-45, 46-65 and 56-65, but comparison of proportions shows that only the 36-45 group is significantly different from the corresponding provincial population proportion (36-45, p=.0036, 46-55, p=.2033 and 56-65, p=.0749).

Age	Male	N	Female	N	M+F	N	%M/%FAlta
<16	0.00	(0)	0.00	(0)	0.00	(0)	26.0/25.7
16-25	0.46	(7)	0.14	(2)	0.30	(9)	22.3/21.8
26-35	1.00	(13)	0.00	(0)	0.52	(13)	18.9/18.1
36-45	1.68	(13)	0.14	(1)	· 0.93	(14)	11.3/11.0
46-55	1.13	(7)	0.35	(2)	0.75	(9)	9.0/8.8
56-65	1.82	(8)	0.22	(1)	1.00	(9)	6.4/7.0
66-75	0.38	(1)	0.33	(1)	1.04	(2)	3.9/3.6
76-85	0.00	(0)	0.00	(0)	0.00	(0)	1.7/2.3
>85	0.00	(0)	0.00	(0)	0.00	(0)	0.4/0.7
Total .	•	49		7		56	100.0/100.0

Table 8. Age and Sex-Specific Undetermined/Unclassified Death Rates (per 100,000)* for Males, Females and Both Sexes and the Provincial Population (%) in these Age and Sex Groups.

*mean six-year rate.

Marital Status

Both populations of accidental and suicidal CO deaths have a lower proportion of married people in the fifteen-and-over age group compared to persons in this sub-group in the Province of Alberta in 1981 (Table 9). The population of accidental deaths has a higher proportion of single and separated individuals, while the victims of suicide differ in the proportion of divorced and separated people when compared to the general fifteen-and-over population of the province. The marital status proportions for the undetermined/unclassified deaths closely resembles those in the general population at that time, except for individuals who were separated or widowed. No statistical tests were performed on these findings since the definition of categories used by Census Canada is likely different from that used in the medico-legal investigation into sudden death. For example, "single" is defined by Statistics Canada as "never married", and this is not required on the OCME forms.

Pop'n	Single	Married	Separated	Divorced	Widowed	Other	8
Accidental	50.5	7.4	9.1	1.0	2.0	0.0	100.0
Suicidal	32.7	38.4	16.0	7.3	3.2	2.4	100.0
Undc	30.4	57.1	7.1	5.4	0.0	0.0	100.0
Alberta	28.1	61.7	2.2	3.3	4.7	0.0	100.0

Table 9. Marital Status in the 15-and-Over Age Group for Accidental, Suicidal and Undetermined/Unclassified (Undc) CO Deaths and the Population of Alberta (%).

Race

Racial categories used by the Medical Examiner were: Caucasian, Oriental, North American Indian, East Indian and Negro. These can not be related directly to those comprising the "Ethnic Origin" classifications used by Statistics Canada. Table 10, which depicts the frequencies of deaths in the various racial categories, must therefore stand by itself.

The most notable finding is an inordinately high number of North American Indian accidental deaths. The death rate (per 100,000) for this population is 17 for males and 10 for females, in contrast to the corresponding rates of 1.26 and 0.28 for the entire population of male and female accidental fatalities.

Race	М	idental F	Si M	licida) F	U M	indc F	Total
Caucasian	77	18	332	90	47	7	571
Oriental	0	0	1	0	1	0	2
N.A.Indian	5	3	1	1	0	0	10
East Indian	1	0	1	1	1	0	4
Negro	0	0	1	1	0	0	2
Total	83	21	336	93	49	7	589

Table 10. Racial Origin in the Population of Male and Female Accidental, Suicidal and Undetermined/Unclassified (Undc) CO Deaths.

Chapter Summary and Discussion

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This chapter has been concerned with the description of the year and place of the CO deaths as well as the common demographic variables of sex, age, marital status and race. It is seen that the swift population and economic changes which occurred in the province during the time of the study are not reflected by any well-defined trends in the number of deaths in any of the manners of death categories. The rates for both accidental and suicidal CO deaths did however increase each year from 1981. It is possible that these increases may have continued over the following few years as the effects of the economic downturn became more universal and long-term. This would have contributed to personal and financial problems which might relate to increased numbers of deaths in all categories.

There are more male than female deaths in all sizes of communities. This is true for all three manners-of-death categories. But, while accidental deaths occur more frequently in the nonmetropolitan (and rural) settings, the reverse situation is found for the suicidal and undetermined/unclassified groups.

There is a distinct peak in the number of accidental deaths occurring in the 16-25 age group. The suicidal and undetermined/unclassified deaths; however, show a broader peak, extending over several age groups. Analyses of the age distributions of the three populations of fatal COP suggest that they are distinct from one another. Tests of differences in proportion between the age groups with an excess of deaths and the corresponding group in the provincial population, show that significant differences are demonstrated in the accidental 16–25 age group, in the suicidal 36–45 and 46–55 age groups, and the undetermined/unclassified 36–45 age groups. These findings are reflected in the death rates expressed in deaths per 100,000.

There are proprtionately fewer married persons in the accidental and suicide death categories compared to the population as a whole. This finding could be due to the ages of the victims, a high incidence of marital problems, or may be in part a result of differing definitions of marital status, particularly in reference to the single which have been used to define the study group and that used by Statisics Canada. The comparison of the distribution of marital status between the accidental and suicidal victims shows that they are significantly different from one another. Undc deaths on the other hand resemble the distribution of marital status in the general population. Finally, the examination of the racial characteristics of the study population indicates that there is an inordinately high number of native people involved in accidental COP.

The analyses at this stage therefore suggests that the accidental, sucidal and undetermined/unclassified deaths do differ from each other in the distributions of several variables.

Chapter 5

Description of Three Source and Environment-Determined Sub-Populations of CO Fatalities in Terms of Month, Season, Day and Activity and the Re-Examination of the Demographic Variables in These Sub-Populations.

Human activities and environmental factors can interact. For example, the ambient temperature may in part determine a particular human activity, such as working inside a closed garage in the cold winter months. At the same time, poor maintenance and inappropriate use of sources of carbon monoxide (which may be considered the result of somewhat remote human activities) will have contributed to the eventual lethality of an environment.

Carbon monoxide poisoning has in part been described in Chapter 4 through the examination of the death rates, communities in which these deaths occurred and the more common demographic variables. The current chapter seeks to gain greater comprehension of the characteristics of these deaths through a more detailed account of the setting i.e., when and where the deaths occurred, what sources generated the CO and description of the human activities related to these deaths.

This stage of the study will also examine whether the characterization of fatal COP is aided by the division of the of accidental, suicidal and Unde groups of deaths into three source-environment subcategories. Baker et al (1972) allude to the fact that fatal unintentional deaths from CO from motor vehicles can be described in two groups depending on the source and where the death occurred (indoor or outdoor environments). Inspection of the data in the present investigation indicates that similar categories exist here, but also a third category, i.e., deaths from CO produced by a miscellany of sources other than vehicles must also be defined. These three categories will be referred to as deaths from CO; in motor vehicle exhaust in the indoor setting (IMV), in the outdoor setting (OMV) and from miscellaneous sources (MISC). The findings will relate to the second and third study objectives: 2. to examine the deaths for relationships with the temporal and physical environment and describe the human activities preceeding death; 3. to determine if the three populations of deaths are better described through subdivision into three source-environment categories.

Accidental Deaths

The identity of the source as well as the place of exposure was documented in 100 of the original 104 accidental CO deaths. Of these, motor vehicle exhaust was the major source of carbon monoxide, causing 76 (76.0%) of the accidental deaths, while the CO generated by faulty furnaces, space heaters and appliances accounted for 24 (24.0%) of the fatalities. None of the deaths was attributed to CO from chemical or industrial processess, nor was any caused by the careless or inapproprate use of machinery or of small engines such as lawn mowers, ski-doos or motorcycles.

<u>Month and Season</u>. Using a one sample Chi-square goodness-of-fit test, a statistically significant (p<.01) difference is found between the observed and the expected frequencies of deaths per month for the entire or "Total" group of accidental deaths (Table 11). This remains the case when the monthly frequencies are adjusted for the varying days in each month. Similarly testing the distributions of the three source-location categories (IMV, OMV, MISC); however, indicates that, while the monthly distribution of observed deaths in the MISC sub-category is significantly different from the expected distribution, those of the IMV and the OMV are not.

Through inspection of the contribution of the $(O-E)^2/E$ components of the significant MISC Chistatistic it is seen that the excess of deaths in January contribute heavily to the significant finding. In the "Total" group, the deaths in the months of December and January, as well as the month of June contribute the most to this significant statistic. It can be seen from the table that the monthly excesses in the entire population of accidental deaths can be explained by contributions of the frequencies of deaths in each of the three sub-categories; which, while not always notable in the sub-categories, combine to create high observed values in the "Total" population of accidental CO fatalities.

		MONTH											
	J	F	Μ	A	My	Jn	JI	Au	S	0	Ν	D	Total
IMV	4	5	3	3	2	3	0	0	1	2	4	8	35
OMV	2	3	4	2	1	9	5	2	5	4	2	2	41
MISC**	9	2	1	0	0	2	0	0	1	1	4	4	24
<u>Total*</u>	15	10	8	5	3	14	5	2	7	7	10	14	100
*p<.01	r	*											

Table 11. Frequencies of the Indoor Motor Vehicle Exhaust (IMV), Outdoor Motor Vehicle Exhaust
(OMV), Miscellaneous (MISC) and Total Populations of Accidental Deaths by Month

**p<.001

A Chi-square test of independence to compare the monthly distributions of deaths among the IMY, OMY and MISC sub-categories showed that these distributions are significantly different (.01<p<.02). The table shows that the distribution of OMV deaths differs from the other two groups with fewer OMV deaths in December and January in contrast to the pattern seen for both the IMV and MISC deaths.

The data were grouped seasonally (Table 12) and it is seen that the distribution of deaths in all groups, except the OMY, are significantly different (goodness of fit Chi-square) over season of the year. Once again, the OMV demonstrates a unique pattern. The IMV and MISC deaths appear more as winter phenomena, in contrast to the distribution of the OMV fatalities, which (although not statistically significant) is seen to peak in the summer and taper off into the fall months.

Туре	Dec-Feb	Mar-May	SEASON June-Aug	Sept-Nov	Total
IMV*	17	8	3	7	35
ΟΜΥ	7	7	16	11	41
MISC**	15	1	2	6	24
Total**	40	16	21	23	100

Table 12. Frequencies of the IMV, OMV, MISC and Total Populations of Accidental CO Deaths by Season.

*.001<p<.01

**p<.001

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<u>Day of the Week</u>. Examination of the daily distribution of the "Total" population of accidental deaths shows a somewhat bimodal pattern, peaking on Saturday, with a smaller peak on Wednesday (Table 13). This distribution is statistically significant (.001<p<.01); however, of the three subcategories, only the distribution of the observed OMV fatalities proved to be satistically different for day of the week (.02<p<.05).

Table 13. Frequencies of the IMY, OMY, MISC and Total Population of Accidental CO Deaths by Day of the Week.

	DAY							
Туре	Sun	Mon	Tue	Wed	Thur	Fri	Sat	Total
IMV	6	4	3	7	6	0	9	35
0MV*	3	2	4	8	7	4	13	41
MISC	3	3	2	4	1	6	5	24
Total**	12	9	9	19	14	10	27	100
*.02 <p<.05< td=""><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td></p<.05<>								

**.001<p<.01

It is possible that the twenty-four hour day might not correspond to how people actually live, and that the period from 12:00 midnight to 6:00 a.m., behaviourally, should "belong" to the end of the prvious day rather than to the beginning of a current calendar day. Therefore this early morning period was "shifted" backward and the altered day of the week examined (Table 14).

Туре	Sun	Mon	Tues	DAY (A/ Wed	S) Thur	Fri	Sat	Total
IMV	5/3	2/2	2/3	5/6	5/3	0/3	· 5/5	24 ·
OMV	2/3	2/1	3/8	8/6	5/2	0/4	8/4	28
MISC	1/1	1/2	2/0	3/3	0/1	2/2	2/2	11
Total	8/7	5/5	7/11	16/15	10/6	2/9	15/11	63

Table 14. Actual (A) and "Shifted" (S) Daily Frequencies of the IMV, OMV, MISC and Total Group of Accidental Deaths.

This manoever acts to eliminate the significant results in the "Total" and OMV groups and it can be seen from the table that shifting the early morning period changes the daily frequencies only slightly, with Wednesday and Saturday remaining high while the number of "shifted" Friday deaths increase over the actual numbers. Since the number of Saturday deaths remain high with the shifting procedure this possibly presents a more realistic picture of when these people died, one which might be expected if drinking and/or fatigue from late weekend hours contribute to the daily frequencies of deaths.

Unfortunately it is not always possible to determine the time of death in a medico-legal investigation and for this reason the total number of cases in which this was documented is considerably less than the numbers of accidents. This may act to bias the data on the distribution of deaths. Because of this and also because of the lack of significant differences in the distribution of the shifted day of the week frequencies, further analyses will not include this shift in time.

Activities. The previous findings indicate that the IMV, OMV and MISC sub-categories of accidental COP have different monthly, seasonal and possibly day of the week profiles. These groups also represent discrete source-environment situations and it is therefore likely that they will also differ in the human activities related to the deaths. Knowledge of these activities may help to explain the different distributions. The IMV, and OMV sub-categories will now be examined for the activities which occurred immediately prior to death, while the MISC group must be approached in a different manner.

1. Indoor Motor Vehicle Exhaust Fatalities (IMV). Thirteen (37.1%) of the IMV victims are reported to have been working on their vehicles, or at some other task indoors before being overcome by CO. Nine (25.7%) are described as having been found sitting in their vehicles, while 6 (17.1%) were found lying in them, perhaps sleeping. Another 7 (20.0%) IMV fatalities had been involved in some activity which either was different from these, or which could not be determined from the position of the body, or from other evidence at the scene of the accident.

The observed monthly or daily distributions of these deaths are not significantly different from the expected values; however when these fatalities are examined on a seasonal bases (Table 15)

Activity	Dec-Feb	Mar-May	SEASON June-Aug	Sept-Nov	Total
Working*	8	2	1	2	13
Sitting	5	1	1	2	9
Lying	2	2	> 0	2	6
<u>Other</u>	2	3	1	<u>l</u>	7
Total**	17	8	3	7	35

Table 15. Distribution of Activities of Victims of Indoor Motor Vehicle Exhaust by Season.

*.02<p<.05

**.001<p<.01

there is a significant difference between the observed and the expected cell frequencies. The observed values of the "working" activity in the various seasons differ significantly from the expected (Chi-square goodness of fit test). Inspection of the $(0-E)^2/E$ contributions to the statistic , suggests that it is the higher frequencies of deaths in the fall and winter months which contribute to this finding.

2. Outdoor Motor Vehicle Exhaust Fatalities (OMV). The activities immediately prior to death of these fatalities are categorized as: sitting, lying/sleeping and couples parked. The distinction between the latter category and the other two is necessary in order to better describe the activities in vehicles in the out-of-doors. This category also aids in clarifying the reasons for many of the multiple fatalities; events which are not found in the other two sub-categories of accidental death.

There is no statistical relationship between the number of OMV deaths and the month or the season of the year; however, there is a statistically significant difference in the number of deaths in this sub-category per day. Table 16 presents the frequencies of activities of the OMV victims by

	DAY							
Activity	Sun	Mon	Tues	Wed	Thur	Fri	Sat	Total
Sitting	1	1	0	2#/1	4#/2	4#	3#/2	20
Sleeping/Lying	1	1	3	1	0	0	2	8
Couples*	0	0	0	2#/2	0	0	2#/2#/2#	10
Other	1	0	1	0.	1	0	0	3
Total OMV	3	2	4	8	7	4	13	41

Table 16. Distribution of Activities of Victims of Carbon Monoxide of Outdoor Motor Vehicle Exhaust by Day

*p<.01 #one or more fatality

day of the week and shows the numbers of multiple fatalities. The distribution of the couples parked category by day was significantly different (p<.01), with all of these deaths occurring on Saturday or on Wednesday.

. 3. Miscellaneous Fatalities. While the deaths in the IMV and OMV sub-categories can be described in terms of human activities, this is not appropriate for those in the MISC sub-group. For, although these latter deaths are usually the consequence of some remote activity, they appear to be better represented by some condition of the source rather than through human activity.

The greatest number of MISC fatalities (13, or 54.0%) are due to carbon monoxide escaping from furnaces and heaters in the home, while four (16.7%) are described as occurring in a van or • a camper through the generation of CO from appliances. The deaths are therefore categorized according to the reason attributed to the escape of CO. These are: poor installation, inappropriate use and poor maintenance.

There is no statistically significant relationship between the number of MISC deaths and the day of the week; however, such a relationship is found for the monthly and seasonal frequencies (Table 17) depicts the cause of liberation of CO by month and also indicates if the fatality(ies) occurred in a van or camper.

Source	J	F	М	A	My	Mon Jn	пн Л	Au	S	0	N	D	Total
Inappropriate Use	1	1	0	0	0	0	0	0	0	0	2#	į#	5
Poor Maintenance	3	0	1	0	0	1	0	0	1	0	0	0	6
Other/Unknown	5	0	0	0	0	1	0	0	0	0	0	1	7
Total *	9	2	1	0	0	2	0	0	1	1	4	4	24

Table 17. Cause of Liberation of Carbon Monoxide from Miscellaneous Sources by Month.

*p<.001, # = number of fatalities in a van or camper.

It is readily seen that the frequency of the MISC accidental deaths increases with the onset of cooler weather, decreasing with the approach of the warmer summer months. This appears to be the case for both accidents in vans and campers as well as for those which occurred in the home.

An examination of the seasonal distribution of these deaths (Table 18) confirms that they are generally a winter phenomenon, with 87.5% occurring from September to February.

Source	Dec-Feb	Mar-May	SEASON June-Aug	Sept-Nov	Total
Poor Installation	2/1#	0	0	3	6
Inappropriate Use	2/1#	0	0	2#	5
Poor Maintenance	3	1	1	1	6
Other	6	0	1	× 0	7
Total*	15	1	2	6	24

Table 18. Cause of Liberation of Carbon Monoxide from Miscellaneous Sources by Season.

#in van or camper. *p<.001

The differences in the monthly, seasonal and day of the week distributions in the three subcategories of accidental CO deaths and the examination of the activities related to these accidents suggest that these are different types of accidents. It is possible that the age, sex, area and community of residence of the IMV, OMV and MISC victims may vary as well. Therefore it is of interest to re-examine these variables to determine if the analyses of the total population of accidental deaths which was described in Chapter 4 is fully representative of the sub-groups. Marital status is not included at this point because of the differences in definition already discussed, while race is omitted because of the high preponderance of Caucasians.

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The median ages of the IMV, OMV and MISC sub-categories is in the 26-35, 16-26 and 56-65 age groups respectively. Examination of the age structures (Table 19) suggests that the three groups likely differ from one another and there may be a difference between the age structure of each one of these and that of the provincial population (using 1981 Statistics Canada figures)

Since nearly all (98.0%) of the deaths occurred to those older than 15, the statistical tests are performed excluding the under 16 ages; 16 and over considered as the population at risk. Chi-square tests of independence on the distributions of each of the sub-categories shows that, while both the OMV and MISC groups are significantly different (p<.001) from the distribution of the general (16-and-over) population of Alberta, that of the IMV is not (.10<p<.20).

Age	IMV f/%	0MV 1/%	MISC f/%	Total f/%	Alberta %
<16	1/2.9	1/2.4	0/0.0	2/2.0	25.7
16-25	13/37.1	27/65.9	4/16.7	44/44.0	22.1
26-35	3/8.6	8/19.5	0/0.0	11/11.0	18.5
36-45	5/14.3	4/9.8	2/8.3	11/11.0	11.2
46-55	6/17.1	1/2.4	4/16.7	11/11.0	8.9
56-65	5/14.3	0/0.0	6/25.0	11/11.0	6.7
66-75	0/0.0	0/0.0	3/12.5	3/3.0	4.2
<u>76-85</u>	2/5.7	0/0.0	5/20.8	7/7.0	2.0
Total	35/100.0	41/100.0	24/100.0	100/100.0	100.0

Table 19 Age Structure of the IMY, OMY, MISC Accidental Carbon Monoxide Victims and the 1981 Population of Alberta, in Frequencies and Percent (f/%).

Examination of the components of the Chi-square statistics shows that, in the OMV sub-group, the

Age

16-25 age category which has 2.27 times the expected number of deaths, contributes the most to the significant statistic, the ages 46 and over also contribute to the significant Chi square statistic, but this was due to their lower rather than higher expected values.

The MISC sub-group shows an excess of observed deaths in all of the over 45 year age categories; however, only those over 55 contribute substantially to the significant Chi-square statistic. The 26-35 age groups, while also being a source of this significance, have a lower than expected number of deaths And the IMV sub-group, while not significantly different from the Alberta population, deserves some attention because of the lower than expected number of deaths in the 26-35 year age category.

These findings are summarized in Table 20 showing the $(0-E)^2/E$ components of each Chisquare statistic and whether each is due to excess of deaths (*) in that age category.

Age Oroup	IMY	OMV	MISC
16-25	0.9	18.8*	1.3
26-35	4.0	0.4	6.0
36-45	0.0	0.7	1.0
46-55	1.0*	3.2	0.3*
56-65	1.3*	4.0	8.0*
66-75	2.0	2.0	4.0*
76+	1.0	1.0	16.0*
Chi Square Statistic	10.2	30.0	36.6

Table 20. [(0-E) ² /E] Contribution of the Age Groups to the Chi Statistics in the Co	mparison of the
IMV, OMV and MISC Sub-Category Age Structures to that of the 1981 Alberta Popul	ation.

* observed number of deaths greater than the expected number.

Comparison of the age distributions in the three sub-groups (>15 years) shows that there is a significant difference (p<.001) among their distributions. The comparisons of the age distributions

of the OMV with the MISC and the IMV with the OMV sub-categories also shows they are different (p<.001), while the comparison of the IMV and OMV distributions, though approaching significance at alpha=.05 results in a Chi-square statistic which is not greater than the critical value.

<u>Sex</u> The analyses of gender in the total group of accidental deaths has shown that males consistantly out-number females. Division into the three sub-categories shows this same preponderance of male deaths in every case (Table 21) and it is seen that these male/female distributions in the sub-categories of accidental deaths are not significantly different from one other.

		SEX	
Category	Male	Female	Total
IMV	29(82.9%)	6(17.1%)	35(100.0%)
OMV	33(80.5%)	8(19.5%)	41(100.0%)
MISC	19(79.2%)	5(20.8%)	24(100.0%)
Total	81(81.0%)	19(19.0%)	100(100.0%)

Table 21. Male and Female Distributions in the IMV, OMV and MISC Accidental CO Deaths.

Table 22 presents the distribution of IMV, OMV and MISC sub-categories of accidental deaths in the metropolitan and non-metropolitan areas. A significant Chi square test of independence (.001<p<.01) among these three sub-groups suggests that deaths in these sub-categories are different in how they are distributed in the two areas.

Comparison of the metro and non-metro distibution of deaths in each of the three sub-categories with the numbers of people living in these areas of the province shows that while the distribution of deaths in the OMV and MISC sub-categories of deaths are significantly different from what would be expected(.001<p<.01), that of the IMV is not.

Category	/ Metropolitan n/%	NREA Non-Metropolitan n/%	Total
IMV	17/48.6	18/51.4	35
omv*	6/14.6	35/85.4	41
MISC*	5/20.8	18/79.2	24
Total*	28/28.0	71/71.0	100

Table 22. Distributions of the IMV, OMV, MISC and Total Accidental Deaths by Metropolitan and Non-Metropolitan Areas (number/percentage).

*.001<p<.01

<u>Community of Residence</u>. It could be argued that the number of reported metro and non-metro deaths are not valid since the assignment of a death to either of these areas may depend upon where the death occurred rather than on the victim's actual area of residence. It is therefore of interest to examine the data for the community of residence to determine if this alters the apparent excess of deaths in the non-metropolitan areas. Table 23 shows the distribution of IMV, OMV, MISC and Total populations of deaths in the various communities. These are not significantly different from each other. Comparison of the rural/urban proportions in these sub-categories with that of the province shows that of the IMV is not significantly different from it (p=.3974) but that that of the OMV and MISC populations are (p=.0294 and p=.0256 respectively).

Area	IMV(N/%)	0MV(N/%)	MISC(N/%)	Total
City > 500,000	13/36.4	8/18.0	8/33.3	29/30.0
City < 500,000	5/18.2	5/15.4	1/4.2	11/14.4
Town	7/21.2	11/28.2	11/45.8	29/32.2
Village	8/24.2	8/20.5	4/16.7	20/22.2
Reserve	0/0.0	1/2.5	0/0.0	1/1.1
Total	33/100.0	33/100.0	24/100.0	90/100.0

Table 23. Distribution and Percent in Communities of Residence of Victims of IMV, OMV, MISC and Total Accidental Carbon Monoxide Deaths.

Comparison of the rural/urban proportions in these sub-categories with that of the province shows that the IMV is not significantly different from it (p=.3974) while that of the OMV and MISC populations are (p=.0294 and p=.0256 respectively).

Suicidal Deaths

Motor vehicle exhaust was the source of carbon monoxide in 420 of the original 429 (97.9%) suicidal deaths. Of the former, 324 (77.1%) occurred in the indoor setting (Table 24). The household garage is the most common environment, with 308 (95.1%) of these indoor deaths. The remaining 16 (4.9%) IMV suicidal deaths occurred elsewhere in the home, or inside other buildings such as warehouses and commercial garages. Ninety (21.4%) of the suicidal deaths from motor vehicle exhaust occurred in the outdoor environment. There are 9 non-motor vehicle exhaust deaths and these were caused by CO from sources such as charcoal briquettes, or the source is not clearly identified.

The division of the accidental CO deaths into the three source-location sub-categories shows some interesting differences. It is therefore of interest to see if a similar division aids in the description of suicidal fatalities. While twenty-four percent of the accidental deaths are attributed to carbon monoxide from miscellaneous sources, nearly all (97.8%) of the suicidal deaths are due to CO from

Location	Frequency	Percent
Household Garage	308	. 73.3
House	. 4	1.0
Other Building	12	2.9
Lane or Highway	23	5.5
Parking Lot	17	4.0
Field	50	11.9
Other	6	1.4
Total	420	100.0

Table 24. Location of Suicidal Deaths from Motor Vehicle Exhaust in Frequency and Percent.

vehicles (IMV and OMV), with very few in the MISC group. Therefore the latter is excluded in this section of the investigation.

Month and Season. The distribution of the Total, as well as the IMV and OMV suicidal subpopulations (Table 25) are not significantly dependent upon the month of the year.

MONTH													
Туре	J	F	М	Α	MY	J.	JI	Au	S	0	N	D	Total
IMV	28	27	23	36	36	18	26	19	17	32	31	31	324
<u>omv</u>	2	4	6	7	8	10	6	9	8	12	9	9	90
Total	30	31	29	43	44	28	32	28	25	42	40	40	414

Table 25. Distribution of Indoor Motor Vehicle Exhaust (IMV), Outdoor Motor Vehicle Exhaust (OMV) and Total Suicidal Deaths by Month.

Similarly, examination of the distribution of these deaths shows no relationship in either the IMV, the OMV, or in the Total population distributions of suicidal deaths to season (Table 26).

SEASON									
Туре	Dec-Feb	Mar-May	June-Aug	Sept-Nov	Total				
IMV	86	95	63	80	324				
OMV		21	25	29	90				
Total	101	116	88	109	414				

Table 26. Distribution of IMV, OMV and Total Populations of Suicidal Deaths by Season

<u>Day of the Week</u>. The distribution of the suicidal deaths by day of the week (Table 27) shows that there is not a significant relationship between the the distribution of these fatalities and day of the week for the Total group, or for either of the two sub-categories.

Туре	Sun	Mon	Tue	DAY Wed	Thur	Fri	Sat	Total
IMY	41	60	46	48	38	41	49	323
OMV	11	11	12	16	14	13	12	89
Total	52	71	58	64	52	54	61	412

Table 27. Distribution of IMV, OMV and Total Population of Suicidal Deaths by Day of the Week.

<u>Age</u>. Comparison of the age structures (Table 28) in the two suicidal categories with that of the Alberta population shows that the two sub-groups are both significantly different from that of the province (with or without the inclusion of the under-16 ages); however, the distributions of the two categories are not significantly different from each other, with the age groups, 36-45 and 46-55, contributing to the excess of deaths in both of the sub-categories.

Age	IMV** f/%	omv* f/%	Total f/%	Alberta %
<16	1/0.3	0/0.0	1/0.3	25.7
16-25	61/18.8	22/24.4	83/20.0	· 22.1
26-35	77/23.8	25/27.8	102/24.6	18.5
36-45	79/24.4	23/25.6	102/24.6	11.2
46-55	53/16.4	15/16.7	68/16.4	8.9
56-65	32/9.9	3/3.3	35/8.4	6.7
66-75	18/5.6	2/2.2	20/4.8	4.2
76-85	2/0.6	0/0.0	2/0.5	2.0
85+	1/0.3	0/0.0	1/0.3	1.0
Total	324/100.0	90/100.0	414/100.0	100.0

Table 28. Age Structure of the IMV, OMV, Total Suicidal Deaths and the Population of Alberta, in Frequencies and Percent (f/\Re) .

*p<.001 **.02<p<.05

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<u>Sex</u>. The distributions of males and females (Table 29) in the two sub-categories of suicidal deaths are significantly different from each other, with fewer observed female deaths in the OMV sub-group than expected when compared to the IMV category of suicidal deaths.

Category	Male	SEX Female	Total
IMV	245	83	328
OMV	83		90
Total	328	90	418

Table 29. Frequencies of Males and Females in the IMY, OMY and the Total Populations of Suicidal Deaths

.001<p<.01

<u>Area</u>. The metropolitan areas have 271 suicidal deaths from motor vehicle exhaust. Thirty-one (11.4%) of these occurred in the out-of-doors, with 240 (88.6%) indoors. The North and South Rural (or non-metropolitan) areas experienced 143 suicides from exhaust during the study period. Fifty-nine (41.3%) of these occurred out-of-doors and 84 (58.7%) indoors. The metro/non-metro distributions of the IMV and OMV sub-groups of suicidal deaths are significantly different from each other (p<.001). Examination of the components of the significant Chi-square statistic indicates that the OMV contribute the most to the significant value, with an excess of non-metropolitan deaths.

<u>Community of Residence</u>. When the distributions in all the communities of residence are examined (Table 30), it is seen that the distributions of the two sub-types of suicidal deaths are not significantly different from one another; however, when comparison is made between the distribution of deaths in the rural and non-rural settings (i.e. combined cities versus combined rural communities), a significant difference is seen (.001<p<.01), with the OMV category of deaths contributing to a large portion of the significant Chi-square statistic.

Community	IMY	OMV	Total
City > 500,000	239	49	288
City < 500,000	29	9	38
Town	34	16	50
Village	18	9	27
Reserve	1	0	1
Other	.0		<u> </u>
Total	321	84	405

Table 30. The Distribution in Communities of the IMV, OMV and Total Populations of Suicidal Deaths.

Undetermined/Unclassified Deaths.

Fifty-five of the 59 (93.2%) undetermined/unclassified deaths are caused by carbon monoxide in motor vehicle exhaust. Fifty (90.9%) of these deaths occurred in the household garage, with two more in another indoor setting. Only three of the undetermined/unclassified cases occurred out-ofdoors. These fatalities then are almost exclusively caused by motor vehicle exhaust in the indoor environment; therefore, subdivision is not of great benefit in furthering an understanding of these fatalities. The description of the age, sex, area and community of residence has been described for this manner of death in Chapter 4; therefore, the present analyses will deal only with the examination of the monthly, seasonal and daily distributions of these deaths.

<u>Month and Season</u>. The distribution of the undetermined/unclassified deaths is not statistically significantly dependent upon month of the year (Table 31). However, the seasonal distribution of these deaths is significant different from what would be expected (Table 32), showing a lower than expected number occurring in the summer months and peaking in the winter.

Jan	Feb	Mar	Apr	May	June	MONTH July	Aug	Sept	Oct	Nov	Dec	Total
8	10	5	9	5	3	0	4	2	4	5	4	59

Table 31. Distribution of the Undetermined/Unclassified IMY Deaths by Month.

Table 32. Distribution of the Undetermined/Unclassified IMV Deaths by Season.

Dec-Feb	March-May	SEASON June-Aug	Sept-Nov	Total
22	19	7	* 11	. 59

*.02<p<.01

<u>Day of the Week</u>. The distribution (Table 33) of the undetermined/unclassified deaths is dependent upon the day of the week (.01<p<.02), with an excess of deaths on Saturday contributing to the significant Chi statistic.

Table 33. Distribution of the Undetermined/Unclassified IMV Deaths by Day of the Week.

			D	AY			
Sun	Mon	Tue	Wed	Thur	Fri	Sat	Total
8	3	8	4	11	9	16	59
<u> </u>	-	-	•		-		

*.01<p<.02

Chapter Summary and Discussion

2

<u>Accidental Deaths</u>. Sub-division of the population of accidental deaths into the three sourceenvironment situations was explored to further understand the circumstances of fatal accidental carbon monoxide poisoning. Table 34 summarizes the findings.

Variable	IMV	OMV	MISC	Total
Month	*	¥	<.001	<.01
Season	<.01	×	<.001	<.001
Day	×	<.05	×	<.01
Age	*	<.001	<.001	<.001
Metro/non-Metro	*	<.01	<.01	<.01
Urban/Rural	×	<.05	<.05	X

Table 34. Results (p-values) of the Analyses of the IMV, OMV, MISC and Total Populations of Accidental Death from Carbon Monoxide.

*not statistically significant

X = cannot be established

The results suggest that, although the analyses of the Total population of deaths are useful in the description of fatal accidental COP, the findings are not always representative of the sub-categories, each one having a different profile of significance in the variables which were chosen for investigation. These can be summarized as follows:

1. The MISC deaths are found to be largely a winter phenomenon, occuring to people over age 45 more often than would be expected when compared to the age structure of the fifteen and over population of the province. They also appear to be more of a non-metro, with no dependency upon the day of the week.

2. The IMV deaths appear to be more of a winter phenomenon as well, but they have only a weak difference in age structure compared to that of the (over-fifteen) provincial population. No dependency on day of the week is seen, but unlike the MISC group, these deaths occur almost as frequently in the metro as in the non-metro setting.

3. The OMV, while not significantly dependent upon month or season, have a distribution which is in contrast to those found for the IMV and MISC sub-categories; its numbers peaking in the summer months, rather than in the winter. These outdoor deaths have 2.27 times the expected frequency of deaths (compared to the over-fifteen population) in the 16-25 year age group, with no other ages showing an excess of deaths. The OMV is the only sub-group in which a dependency upon day of the week is seen; but it appears, as in the case of the MISC deaths, to be more of a non-metro type of death.

Investigation of the human activities and the condition of the source of carbon monoxide surrounding these three sub-types of accidental death indicates that, as well as representing different source-environment conjunctures, these categories also differ in the consignment of the main cause of the fatal set of circumstances. For, while the indoor motor vehicle accidents appear to be exclusively due to individuals remaining in an environment in which any vehicle was operated, the outdoor motor vehicle deaths are nearly all a result of sitting or lying in idling vehicles which were later shown to have had defective exhaust systems and/or had rusted, e.g. wheel wells and floor boards. Finally, the miscellaneous group of accidental deaths are seen as largely the result of the poor maintenance, faulty installation and the inappropriate use of heating appliances and furnaces; with no specific and concurrent human activity directly related to the immediate situation.

Therefore it is suggested that the IMV deaths are best described in terms of the human activities associated with them, rather than through some defect in the source and that, although the OMV deaths do have a relationship to the condition of the source, they are better described through several specific human activities. The MISC deaths, differing from the IMV and OMV categories; however, are seen to be a result of function of the source, its condition or the misuse of it. Therefore in the MISC situation, it is the source which more clearly represents the factor directly

65

responsible for the lethal situation. Although some human activity (or lack of it) generally accounts for the generation of CO, this usually is a somewhat remote and non-specific activity and is not seen as having much utility in further characterizing the death.

<u>Suicidal Deaths</u>. The division of the population of suicidal deaths into sub-categories contributed little to the characterization of these deaths. This procedure shows only that the suicidal deaths of individuals in rural communities from motor vehicle exhaust appear to occur more often in the out-of-doors than to individuals who live in an urban setting and that rural females are less likely than rural males to choose the outdoors as a setting for suicide. No relationship is seen between the IMV, OMV or Total distributions of suicidal deaths and the month, season or day of the week.

<u>Undetermined/Unclassified Deaths</u>. The undetermined/unclassified deaths are almost exclusively in the IMV situation; consequently they are virtually the same as the entire population of undetermined/unclassified fatalities. These deaths are more of a winter phenomenon, with an excess of deaths occurring on Saturday.

Analyses of the human activities and the condition of the source for the various sub-categories in this study is frequently restricted by the numbers of cases involved, but this approach appears to be useful in furthering the understanding of the circumstances of accidental death from carbon monoxide, while being of limited benefit when applied to the suicidal and undetermined/unclassified populations of deaths.

Chapter 6

Occupation

"(*A*) Man's work is one of the more important parts of his social identity of his self; indeed, of his fate in the one life he has to live, for there is something almost as irrevocable obout choice of occupation as there is about choice of a mate." (Hughes, 1951).

The previous chapters have explored a number of the variables which characterize the victims of fatal carbon monoxide poisoning. However the occupations of these people must also be studied, since the skills and the socializing force of the work they were trained to do may have contributed in some way to an increased susceptibility to this type of death.

The purpose of this stage in the analyses is therefore: to describe the types of occupations of the victims of fatal COP, to report the frequencies of job, alcohol and personal problems and also to describe any relationship of time, place, or responsibilities of the job to these fatalities. Work will be considered in its physical setting as well as a socializing factor with possible etiologic consequences in accidental death from carbon monoxide. This chapter relates to the fourth research objective, "to examine the deaths for relationships to work such as to the worksite, to occupation and to problems with work.".

Description of the Working Age Group

The men and women in the working ages (16-65) make up 90.4% of all of the carbon monoxide accidents (Table 35). This figure is 89.3% and 96.4% for the suicidal and undetermined/unclassified victims respectively; indicating that the bulk of the carbon monoxide deaths in all manners-of-death categories were people in the ages 16-65.

-	Acc	idental	Su	icidal	U	ndc	
Age Group	M	F	M	F	<u>M</u>	F	Total
16-25	35	13	77	13	7	2	147
26-35	10	1	84	22	13	0	130
36-45	10	1	68	34	13	1	127.
46-55	11	1	50	18	7	2	89 .
56-65	7	5	31	6	8	. 1	58
Total	73	21	310	93	48	6	551

Table 35. Frequencies of Male (M) and Female (F) Accidental, Suicidal and Undetermined/Unclassified (Undc) Deaths in Working-Age Categories.

The sex and area-specific death rates for the working-age populations of the three manners-ofdeath are shown in Table 36. The male 16-65 accidental, suicidal and undetermined/unclassified

Area	Sex	Accidental Rate	Suicidal Rate	Undc Rate
Metro	Male	0.91	7.88	0.64
	Female	0.21	2.60	0.17
	M+F	0.68	5.35	0.54
Non-	Male	2.66	5.86	0.52
Metro	Female	1.98	1.63	0.07
	M+F	1.10	4.66	0.23
Province	Male	1.69	7.00	0.74
	Female	0.49	2.19	0.13
. <u></u>	M+F	1.10	4.66	0.44

Table 36. Mean Annual Sex and Area-Specific Death Rates (per 100,000*) in the 16-65 Age Groups of Accidental, Suicidal and Undetermined/Unclassified (Undc) CO Victims.

*Population statistics are derived from the 1981 Canadian Census and intercensal estimates adjusted for annual urban growth rate as reported in the annual city census of Edmonton and Calgary. death rates are consistently greater than the corresponding female rates. However, the comparison of the rates of the entire male and female study population (Table 3) with those of the working age groups shows a decrease in the male/female ratio in the non-metro areas (from 3.53 for the total population of accidental deaths, to 1.34 for those in the 16-65 age category). This suggests that the CO deaths are more common in the working age groups and particularly so in the working age nonmetropolitan female accidental victims.

Table 37 shows that 47 (64.4%) of the working age male accidental victims were fully employed, while 4 (19.0%) of the female victims shared this status. This latter figure is 33.3% if a housewife is considered to be fully employed. Full employment is found in 178 (57.7%) of the

		idental	Suic		Unde		
Work Status	M	F	<u>M</u> .	F	M	F	Total
Fully Employed	47	4	178	33	33	3	298
Part Time	3	1	2	2	2	0	10
Casual	1	0	4	1	0	0	6
Unemployed	2	2	80	10	5	0	99
Retired	5	1	8	1	1	0	16
Student	3	3	12	5	0	0	23
Housewife	0	3	0	27	0	t.	31
Sick-Leave	0	0	`7	1	2	0	10
Welfare	1	1	0	1	1	0	4
Workers' Comp.	. 2	0	2	0	1	0	5
Unknown	9	6	17	12	3	2	49
Total	73	21	310	93	48	6	551

Table 37 Working Status of the Male (M) and Female (F) Accidental, Suicidal and Undetermined/Unclassified (Undc) CO Victims.

woking-age male suicidal deaths, while corresponding figures are 33 (35.5%) for females, (64.5% if housewives are included). The undetermined/unclassified group have 33 (68.8%) fully employed working-age males, with 3 (50.0%) employed females (66.7% if housewives are included).

Occupations

In all manners-of-death categories, there is a much higher percentage of unknown occupations for females than for the corresponding male population. This may indicate that housewife, or homemaker, is not always considered to be a legitimate occupation, but also, it may reflect poor differentiation by an investigator of a woman's occupational training and/or status with her household responsibilities.

<u>Accidental Deaths</u>. Table 38 shows the breakdown of these working age accident victims in terms of the Blishen socioeconomic scale (Blishen and McRoberts, 1976). It can be seen here that occupational levels 1-3 have just 6,(10.3%) of the total group of accidental carbon monoxide

Level	N	× ×	Cumulative %
1 (Highest)	1	1.7	ĺ.7
2	0	0.0	1.7
3	5	8.6	10.3
4	9	15.2	25.5
5	22	37.3	62.8
6	19	32.2	95.0
7 (Lowest)	3	5.1	. 100.0
Total	59	100.0	100.0

Table 38. Socioeconomic Status (Blishen) of Accidental Victims of COP in the 16–65 Year Age Group in Frequencies, Percent and Cumulative Percent. deaths. Leviels 4,5 and 6 have 50 or nearly 85% of the total number of fatalities. These findings indicate that the CO fatalities were predominantly non-professionals with some formal skills as might be found in the building trades.

The occupations of working age accidental victims were categorized according to the 1981 Canadian Census. Table 39 depicts the number of victims in these occupational categories in the working-age population of accidental deaths, as well as the number of workers in these occupations in the entire 1981 labour force of the province. For comparison purposes, the annual occupational-specific death rates are also expressed in deaths per 10,000. The rates for the Labour Unclassified occupational classification were not determined as the data were not specific for the type of labour.

Further analyses are not performed on those occupational categories in which the calculated occupational proportion of the sample is less than that in the (1981) provincial population, or when there was only a single fatality.

A combination of the higher death rates and a greater number of observed than expected accidental deaths is found in the Construction Trades, Product Fabricating, Assembling and Repair and Machining and Related Occupations. In fact, occupations within codes 8310-8799, which represent 16.5% of the total Alberta labour force, experienced 44.1% of the accidental deaths from carbon monoxide.

Codes	Title of Occupation	N	n	n/10,000
2111-2189	Natural Science, Engineering	53,255	1	0.03
	& Mathematics			
4110-4199	Clerical Related	206,970	4	0.03
5130-5199	Sales	114,305	6	0.09
6111-6199	Service	122,620	5	0.07
7112-7197	Farming	82,395	2	0.04
7311-7319	Fishing, Hunting	175	1	9.52
7510-7519	Forestry, Logging	2,600	1	0.64
7710-7719	Mining, Oil & Gas	16,065	3	0.31
8100-8299	Processing	22,170	2	0.15
8310-8339	Machining & Related	24,775	6	0.40
8510-8599	Product Fabricating,	58,100	7	0.20
	Assembling & Repair			
8710-8799	Construction Trades	106,430	13	0.20
9110-9199	Transport Equipment Operating	48,285	4	0.14
	Labour Unclassified	**	4	
Total			59	

Table 39. Number of Workers in Alberta* (N) and in the Group of Accidental Deaths (n) by Type of Occupation and the Annual Occupation-Specific Accidental CO Death Rates (per 10,000).

*Total 1981 labour force = 1,145,100

It was seen in Chapter 5 that the accidental deaths do not appear to be a homogenous group but are better described in three source-environment sub-categories. It is therefore of interest to also examine socioeconomic status (SES) using these same sub-groups. Table 40 shows the distribution of the various socioeconomic strata of individuals in the three sub-categories for the accidental carbon monoxide poisoning victims. A Chi-square test of independence shows that there is no significant difference in the SES distributions of these three sub-categories of accidental death.

		IMV			OMV	,		MISC	<u>}</u>	
Level	N	8	Cum %	N	8	Cum %	N		Cum %	Total
1 (Highest)	0	0.0	0.0	0	. 0.0	0.0	1	12.5	12.5	1
2	0	0.0	0.0	0	0.0	0.0	0	0.0 ⁻	12.5	0
3	3	12.5	12.5	2	8.7	8.7	0	0.0	12.5	5
4	4	16.7	29.1	2	8.7	17.4	. 2	25.0	37.5	. 8
5	9	37.5	66.7	8	34.8	52.2	2	25.0	62.5	19
6	7	29.2	95.8	8	34.8	87.0	3	37.5	100.0	18
7 (Lowest)	1	4.2	100.0	3	13.0	100.0	0	0.0	100.0	4
Total	24		100.0	23		<u>`100.0</u>	8		100.0	55

Table 40. Socioeconomic Status (Blishen) of Indoor Motor Vehicle Exhaust (IMV), Outdoor Motor Vehicle Exhaust (OMV) and Miscellaneous (MISC) Sub-Categories of Accidental COP, Age 16-65 in Frequencies, Percent and Cumulative Percent.

The comparison of the occupational classifications among the three sub-categories of accidental death could not be made because of small numbers.

Suicidal Deaths

Table 41 shows the socioeconomic status of the suicide victims of COP in the working age group. Seventy (23.9%) of them are in SES levels 1-3. This is over twice the percent seen in the accidental CO deaths. Levels 4,5 and 6 have 73.0% of the suicidal deaths. These findings suggest that the SES structure of the suicide victims is similar to that of the accidental deaths in that it is predominantly made up of non-professionals, although to a lesser degree.

Level	N	8	Cumulative %
1 Highest)	9	3.1	3.1
2	13	4.4	7.5
3	48	16.4	23.9
4	51	17.4	41.3
5	74	25.3	66.6
6	89	30.4	97.0
7 (Lowest)	<u>`</u> 9	3.0	100.0
Total	293	100.0	100.0

Table 41. Socioeconomic Status (Blishen) of Suicidal Victims of COP in Frequencies, Percent and Cumulative Percent.

The occupations of 301 (74.7%) of the original 403 working age suicidal victims are known (Table 39). Table 42 presents the number of individuals in these categories for the group of suicidal deaths, the number in each category in the 1981 labour force of the province, and the occupation-specific annual suicidal death rates for each occupational category. In contrast to the accidental deaths, most of the occupational classifications are represented here.

A combination of the higher death rates and a greater number of observed than expected deaths is seen in the Construction Trades, the Machining and Related and the Transport Equipment Operating Occupations. These occupational classifications, with 15.7% of the provincial labour force, have 33.9% of the suicidal deaths.

Nearly all these deaths were due to the deliberate exposure to motor vehicle exhaust in the indoor and outdoor environments; therefore, the miscellaneous (MISC) sub-category is not included in the examination of SES or of occupation. Table 43 shows the composition of the various SES levels of individuals in the indoor and outdoor motor vehicle (IMV, OMV) sub-categories of suicidal death. Approximately 18% of the IMV suicidal deaths are in SES levels 1-3 while over 26% of the OMV

Codes	Title of Occupation	Ν	n	n/10,000
1130-1158	Non-Government Managers	55,925	6	0.18
1171-1179	Management Related	27,880	5	0.30
2111-2189	Natural Science, Engineering & Mathematics	53,255	10	. 0.30
2311-2339	Social Science & Related	17,150	1	0.10
2341-2349	Law & Jurisprudence	4,475	3	1.12
2511-2519	Religion	2,610	1	0.64
2711-2799	Teaching	42,415	4	0.16
3111-3159	Medicine & Health	43,860	12	0.46
3311-3379	Artists, Literary, Recreation	12,075	4	0.69
4110-4199	Clerical	206,970	30	0.24
5130-5199	Sales	114,305	36	0.52
6111-6199	Service	122,620	31	0.45
7112-7197	Farming	82,395	4	0.08
7510-7519	Forestry, Logging	2,600	3 7	0.31
8100-8299	Processing	22,170	7	0.52
8310-8339	Machining & Related	24,775	16	1.08
8510-8599	Product Fabricating, Assembling & Repair	58,100	18	0.52
8710-8799	Construction Trades	106,430	51	0.80
9110-9199	Transport Equipment Operating	48,285	35	1.21
9510-9599	Other Crafts & Equipment Operating	12,075	6	0.79
	Labour Unclassified		18	
Total			301	

Table 42. Number of Workers in Alberta* (N), in the Suicidal Deaths (n) by Type of Occupation and the Annual Occupation-Specific Suicidal Death Rates (per 10,000) from CO.

Total 1981 Alberta labour force = 1,145,100

are found in these categories. SES levels 4-6 had 75.8 and 71.4% of the fatalities for the IMV and OMV sub-groups respectively; suggesting that these two sub-groups have different SES distributions. A Chi-square test of independence indicates that they are significantly different from one another (p<.05).

		IMV			OMY	
Level	Ν	8	Cum %	N	8	Cum 🕱
1 (Highest)	1	1.6	1.6 .	8	3.7	3.7
2	6	9.7	11.3	7	3.2	6.9
3	4	6.4	17.7	42	19.4	26.3
4	10	16.1	33.8	41	18.9	45.2
5	15	24.2	58.0	55	25.3	70.5
6	22	35.5	93.5	59	27.2	97.7
7 (Lowest)	4	6.4	100.0	5	2.3	100.0
Total	62	100.0	100.0	217	100.0	100.0

Table 43. Socioeconomic Status (Blishen) of the Suicidal IMV and OMV Deaths from CO in Frequencies, Percent and Cumulative Percent.

p<.05

<u>Undetermined/Unclassified Deaths</u>. Table 44 shows the SES membership by the undetermined/unclassified group of deaths from carbon monoxide. The three SES levels 4–6 make up the bulk (73.8%) of this population of deaths, with the three highest levels experiencing 9 (21.4%) of the fatalities. This group therefore appears to more closely resemble the suicidal than the accidental deaths in terms of socioeconomic status, athough all of these groups have over 70% in levels 4–6.

Level	N	Percent	Cumulative %
1 (Highest)	0	0.0	0.0
2	5	11.9	11.9
3.	4	9.5	21.4
4	7	16.7	38.1
5	13	30.9	69.0
6 [•]	11	26.2	95.2
7 (Lowest)	2	4.8	100.0
Total	42	100.0	. 100.0

Table 44. Socioeconomic Status (Blishen) of Undetermined/Unclassified Victims of COP in the 16-65 Year Age Group, in Frequencies, Percent and Cumulative Percent.

Table 45 shows the occupational classifications of undetermined/unclassified deaths, the number of workers in each classification in the total labour force of the province in 1981, the frequencies of undetermined/unclassified deaths in these occupations, and the calculated annual occupation-specific death rates for each category.

Codes	Title of Occupation	N	n	Rate
1171-1179	Management Related	27,880	2	0.06
2111-2189	Natural Science, Engineering	53,255	1	0.03
•	& Mathematics			
2311-2339	Social Science & Related	17,150	1	0.10
3311-3339	Artists, Literary, Recreation	12,075	1	0.14
4110- 4199	Clerical Related	206,970	3	0.02
5130-5199	Sales	114,305	4	0.06
6111-6199	Service	122,620	4	0.06
7112-7197	Farming	82,395	2	0.04
7710-7719	Mining, Oil & Oas	16,065	2	0.21
8100-8299	Processing	22,170	3	0.22
8310-8339	Machining & Related	24,775	1	0.07
8510-8599	Product Fabricating,	58,100	5	0.14
	Assembling & Repair			
8710-8799	Construction Trades	106,430	5	0.08
9110-9199	Transport Equipment	48,285	3	0.10
	Operating			
9510-9599	Other Crafts and Equipment	12,075	2	0.28
	Operators			
	Labour Unclassified	······································	3	•
Total			43	

•

Table 45. Number of Workers in Alberta* (N) and in the Undetermined/Unclassified CO Deaths (n) by Type of Occupation and the Annual Occupation-Specific CO Death Rates (per 10,000).

Total 1981 Labour Force = 1,145,000.

Examination of the SES of the accidental, suicidal and undetermined/unclassified deaths indicates that, in each manner of death, most fatalities occurred in three of the lower strata (4–6). This could be simply because there are more people in these levels and not because these individuals are at greater risk of COP. However at this time it is not possible to compare the socioeconomic structure (Blishen) of the working population in Alberta with that of the CO fatalities.

Selection of the occupational classifications which have a greater proportion of deaths than proportion of workers (1981) in the entire working population of the province (excluding single fatality classifications), show that only a few of these had significantly different proportions of deaths than would be expected from the numbers of workers in that occupational classification (Table 46). The accidental and suicidal manner of deaths had a somewhat different pattern of significant findings, while the undetermined/unclassified fatalities do not show a significant difference of proportions in any occupational classification.

Occupatation	Accidental	Suicidal	Undc
Machining	.0212*	.0107*	
Prod Fab & Repair	.0537	.1601	.0901
Construction	.0080*	.0233*	-
Transport	.1977	.0023*	.2389

Table 46. Comparison of Findings (p-Values) in the Occupational Groups which Show any Statistically Significant Differences in Proportions for any Manner-of-Death.

*significant, alpha = .05

The analyses of the occupations of victims of fatal carbon monoxide poisoning suggest that certain occupational groups may be over-represented in the accidental and suicidal groups. These include Machining and Related Occupations, Product Fabrication Assembling and Repair, Construction Trades and Transport Equipment Operating. While reasons for these findings are unclear, it is recognized that some occupations carry with them an increased risk of exposure to CO, and several

of these are in the four over-reported categories. This section of the chapter will conclude with an examination of these CO-related occupations.

Carbon Monoxide-Related Occupations

All of the deaths in the Machining and Related Occupational classifications occurred to individuals working in occupations in which there is a potential exposure to carbon monoxide (Michael, 1979). These are: Sheet Metal, Welding and Flame Cutting, Metal Shaping and Forming Occupations.

Five of the 7 accidental (71.4%), 12 of the 18 suicidal (66.7%), and all five of the undetermined/unclassified fatalities in the Product Fabricating Assembling and Repair occupational classifications were in jobs considered to have a potential risk of exposure to CO. These were: Motor Vehicle Mechanics and Repairmen and Motor Vehicle Fabrication and Assembly.

Pipefitting or Plumber was the only occupation in the Construction Trades occupational classification which according to Michael (1979) is considered to be at potential risk of exposure to CO. There were two accidental (15.3%), 7 suicidal (13.7%) and no undetermined/unclassified deaths in this occupational category.

All of the accidental and undetermined/unclassified, 33 of the 35 (94.3%) suicidal deaths in the Transport Equipment Operating occupational classifications were in specific jobs which are considered CO-related. These are: Railway Transport, Bus, Taxi and Truck Drivers.

Alcohol, Life and Work Problems and Relationship of Work to the Death

This investigation into the relationship of occupation with fatal carbon monoxide poisoning will conclude with a description of the problems considered of consequence to the individual at the time of death and an examination of the relationship of the job to the death. Included in the former topic are alcohol problems, other life problems and work-related problems. The relationship of the death to three aspects of the job will be also considered, namely, the actual physical setting, time and job responsibilities.

<u>Problems with Alcohol</u>. It must be noted that alcohol problems include diagnosed alcoholics, where the individual either had recieved medical treatment for this condition, or s/he was referred in the file as a "known alcoholic" as well as "problem drinking", which is suggested if the victim's consumption of alcohol appears to have caused problems, e.g., rages or depression. However, it should not be overlooked that excessive drinking may have been a response to, rather than a cause of, other stressful life situations. There will therefore be some overlap of alcohol abuse with other life problems, yet the picture is not complete without the inclusion of alcohol as a problem in itself.

Abuse of alcohol was not unknown in any of the three manners of death, and is seen in 10.6, 30.5 and 40.7%, respectively, of the working-age accidental, suicidal and undetermined/unclassifed deaths.

<u>Life Problems</u>. Life problems include marital, other family, financial, legal, personal health or health of others, girl/boy-friend and other social problems. A total of eleven life problems are documented in the case files of the 94 working-age accidental victims, only one person had more than one problem. Eight (72.7%) of the life problems in this group were marital or personal health problems.

The working age suicidal victims have a total of 453 (single or multiple) life problems. Of these, 216 (47.7%) were either marital or personal health problems, the next two categories with the largest numbers are: concern with the health problems of a friend or family member, with 45 (9.9%) and girl/boy-friend problems with 44 (9.7%) of the total problems found in this group. These four types of life problems therefore account for 67.3% of the total number seen in the victims of suicide.

The working-age undetermined/unclassified victims have a total of 29 (single and multiple) life problems, 16 (55.2%) of these are marital or personal health problems, with difficulty with the health problems in others accounting for 4 (13.8%). These three types of life problems therefore account for 69.0% of the total in this group of deaths.

<u>Work-Related Problems</u>. Difficulties related to work are presented in Table 47. The particular problem which is coded is the one which most accurately describes the most prominant difficulty which the individual had been experiencing (within the year) prior to death. Other job-related problems may also have been present, but the one chosen is that problem which appears in the file as the one of main concern.

Problem	Accidenta)	Suicidal	Undc
Health Affecting Work	2	24	2
Job Loss	2	42	3
Pressures of Work	0	11	.4
Dissatisfaction With Job	1	13	2
Financial, Bankrupcy	0	12	0
Other	0	29	2
Total	5	131	13

Table 47. Work Problems in the Accidental, Suicidal, Undetermined/Unclassified (Undc) Victims of COP.

Five (4.8%) of the working-age accidental victims experienced some type of job problem, 131 (32.5%) of the suicidal and 13 (24.1%) of the undetermined/unclassified victims had experienced some problem related to work.

Table 48 represents the number of COP victims, by type, who had alcohol, work and other life problems which were documented in the files as being of some concern to the victims or to his/her survivor(s) within a year prior to the death.

The working age suicidal victims have the most problems (1.8 per person), followed by the undetermined/unclassified (1.2 per person) and then by the accidental victims (0.3 per person).

Problem	Accidental	Suicidal	Undc
Alcohol	11	123	22
Work Related	5	131	13
Other Life	11	453	29
Total Problems	27	707	64
Total Working-Age Cases	94	403	54

Table 48. Number of Alcohol, Work- Related and Other Life Problems in the Working Age Accidental, Suicidal and Undetermined/Unclassified (Undc) Victims of COP.

Relationship of Work to the Deaths

<u>Accidental Deaths</u>. There is one accidental CO death which occurred at the place of employment during regular working hours in the course of the victim performing the job. This accident was caused by carbon monoxide, generated by a vehicle in a garage at the work-site during working hours. This case was ruled compensatable and is the only one in the entire study which was.

There was some indirect association to work in the case of four other accidental deaths. One victim had arrived early for the first day of work and slept in his vehicle, while another, on his way to work, had slept in his car at the side of the highway. A third had lived in his van in the parking lot of the place of employment. The fourth, a welder, died in the course of a regular working day, and was found underneath his truck. No claims were filed for compensation in any of these four accidental deaths.

<u>Suicidal Deaths</u>. Two suicidal deaths occurred during weekly daytime hours at the place of employment, but were not otherwise work-related. Fifteen other suicidal deaths were associated with regular work, but not during regular hour, and not in the course of performing the job. The worksite in two of the latter cases appears to have been used solely as a place where a vehicle could be operated indoors, with convenience and in privacy. <u>Undetermined/Unclassified Deaths</u>. There is one fatality in this group with an association to usual employment. This death occurred in an automobile parked in the parkade at work, on a normal working day, but not in the course of performing the normal work tasks.

Two other undetermined/unclassifed deaths, one of a motor mechanic, and the other of an autobody mechanic, occurred while they worked privately in garages on vehicles owned by others. None of these Undc deaths was found in the files of Alberta Workers' Health Safety and Compensation.

Chapter Summary and Discussion

This chapter shows that almost all of the male and female CO fatalities in the three manners- ofdeath classifications occurred in the 16-65 year old, working age group. The percentage of the total number of deaths in this group is 89.3%, 90.4% and 96.4% in the suicidal, accidental and undetermined/unclassified categories respectively. By comparison, approximately 67% of the provincial population was between the ages 16-65.

The working age groups all show the same male/female and metro/non-metro distributions described for the entire populations of CO-deaths in Chapter 1. However, the working age female accidental death-rate in the non-metropolitan setting is 3.4 times that of the entire population of female accidental deaths in this setting, while the corresponding figure for males is 1.3. This suggests a concentration of working age female accidental deaths in the non-metro environment.

Full employment is found in 64.4% of the working age male accidental population, with 57.7% and 68.8% respectively in the male suicidal and undetermined/unclassified groups. The corresponding values for employed females are much lower, but these are comparable to the male values when housewife is included as a legitimate full-time occupation.

Findings relating to the socioeconomic status levels of the three manners-of-death suggest that the statuses of most of the victims are non-professional yet not without some formal skills. Examination of the distribution of victims in the occupational categories supports this finding.

Compared to the total labour force figures, a significantly greater proportion of victims of accidental COP were engaged in Machining and Related, Construction Trades Occupations, with a

borderline significant difference in proportions (p=.0537) in Product Fabricating, Assembling and Repair category. In regard to suicidal deaths, a significantly greater proportion of individuals worked in Machining and Related, Construction Trades and Transport Equipment Operating occupations. As for the undetermined/unclassified deaths, there are no occupational groups disproportionately represented when compared to the the proportion in these occupational categories in the total labour force of the province. These findings suggest that there may be certain occupations which in some way render an individual more vulnerable to this type of accidental death and also may point to the possibility that certain occupations may influence the "choice of weapons" in an intentional death.

Victims of accidental COP in Machining and Related, Product Fabricating, Assembling and Repair are people in those occupations with a potential for exposure to carbon monoxide, i.e. individuals who should have had some formal instruction in the hazards of carbon monoxide.

Examination of the specific occupations of the suicidal victims in the three occupational classifications which show a significant difference in proportions shows that the CO-related occupations make up the bulk of deaths, with the exception of the Construction and Trades classification. The underlying reasons for these findings remain unclear as it is not known if these occupational groups are also over-represented in other types of suicide (e.g. by firearms or drugs).

No occupational classifications are significantly over-represented in the undetermined/unclassified deaths. It must be recalled that this group is made up of individuals who are, in reality, accidental or suicidal in intent. This group then is not a discrete category, but rather is an unknown mixture of different manners-of-death. It was therefore expected that this group might have some of the occupational characteristics of the accidental and suicidal deaths; particularly, a significant proportion of individuals in the Machining and Related and the Construction Trades categories since these were common to both the accidental and suicidal groups, but this was not the case.

This could be in part explained if a Medical Examiner's knowledge of the victim's occupation plays some role in the assignment of manner of death. For example, if it is held by the Medical Examiner that individuals in certain occupations (such as automobile mechanics) should "know better" than to work in a closed environment on an operating vehicle, the death might more likely appear deliberate than if the victim was unschooled in such fields. Therefore, a case which might have otherwise been considered equivocal would be judged as suicidal in intent.

Sub-division of the deaths into the IMV, OMV or MISC sub-categories at this stage in the analyses is not generally useful in further characterizing accidental COP because of small numbers. However, it should be noted that all of the Product Fabricating Assembling and Repair accidental victims are in the IMV and OMV sub-categories and 5 out of 7 of these deaths were of individuals trained to work on motor vehicles. A larger study population might show similar relationships in other occupations.

An examination of the life problems (considered to have been of consequence to the individual prior to death) shows that the victims of suicidal have the highest number of problems per person while victims of accidental COP have the lowest rate. The accidental group also have the lowest number of of problems with alcohol, while the highest is found in the undetermined/unclassified deaths. This latter finding was anticipated since high alcohol levels can act to obscure the true intent and therefore directly contribute to the assignment of a death to the undetermined or the undecided category.

Problems related to work are noted in 5.3% of all of the accidental working age deaths. This figure is 32.5%, and 24.1% respectively for the suicidal and undetermined/unclassified deaths.

There was only one accidental CO death which had occurred on the job, in the course of the individual performing the usual work and during normal working hours. This was the only case in the study which received Workers' Compensation. Four other accidental deaths show an indirect relationship to the job, but none of them was considered to be an occupational accident.

Seventeen individuals who deliberately took their own life, did so at their place of employment and one undetermined/unclassified death has a similar indirect relationship to the work site, while two other undetermined/unclassified deaths occurred to freelance mechanics.

Chapter 7

Health Problems, Drug and Alcohol Use. Fatal Levels of Carboxyhemoglobin

The review of the literature in Chapter 2 suggests that certain diseases or conditions and drug and alcohol use must be considered when studying COP. These factors could directly contribute to a fatality, or in combination with CO, may act to make the individual more susceptible to the effects of this compound. For example, independent of any carbon monoxide in the environment, alcohol may alter judgment and reaction-time; some drugs have side effects which may include confusion, dizziness and fainting, while some diseases or conditions may produce similar symptoms. In some cases these effects may directly relate to a CO fatality through some action such as fainting, or they may contribute to it through an impaired assessment of a dangerous situation. CO may also act in concert with certain disease-states or with ingested alcohol or some medications to increase individual risk to this compound.

For the foregoing reasons, one purpose of this chapter is to further characterize the circumstances of fatal COP by describing the number and kinds of health problems and the prevalence of alcohol and drug use among the CO victims. A second purpose is to investigate the evidence of relationships between these variables and fatal COHb levels. This being the only dependent variable available for study. This analysis relates to the fifth objective of the study: "to describe the frequencies of and to demonstrate relationships between the presence of diseases or conditions, drug use and blood alcohol levels and fatal COHb levels".

Health Problems,

Of the 83 men who died accidentally from carbon monoxide intoxication, 15 (18.1%) had at least one diagnosed disease or condition at the time of the accident. Seven of these men had two ailments, while one man had three. Of the twenty-one females who died accidentally, only two (8.7%) are found to have had any disease or condition; none had more than one.

Of the 336 male CO suicide cases, 139 (41.4%) had at least one reported disease or condition at the time of death. Thirty-four of these had two health problems, and six men had three. Of the 93 female suicidal deaths 45 (48.4%) had at least one ailment, five of these had two disease/conditions, and one woman had three health problems.

Of the 49 male undetermined/unclassified (Undc) deaths, 21(42.9%) had at least one reported disease or condition, four had 2 and one man had three. Of the 7 female Undc deaths, four (57.1%) had one problem, and two of these women had two diseases or conditions.

Table 49 depicts the frequency of occurrence of 18 of the more common diseases/conditions in the three population of deaths. The miscellaneous category includes those health problems which occur infrequently. Although there are more than four times the number of suicidal as there are accidental deaths, the former group has over 9 times the number of diseases/conditions as the latter. The Undc group, with slighly over half the number of accidental victims, have slightly more health problems than the accidental group. The suicidal and undetermined/unclassified deaths therefore having more than two times the prevalence of diseases and conditions than is found in the group of accidental fatalities from CO.

This same table shows an absence of depression and other mental disorders in the fatal CO accidental victims and just one case in the Undc group. In strong contrast, there are 80 such cases in the population of suicidal deaths. The latter finding is supported by the fact that 92.0% of the men and women who committed suicide had some form of depressive episode in the past which either required professional treatment, or which was reported by survivor(s). In contrast, only 10% of those who met an accidental death with CO have such a history. Table 49 also shows a high number

of cases of alcoholism or cirrhosis, hypertention, heart disease and cancer in the suicidal cases,

compared to the accidental fatalities.

Disease/Condition	Accidental	Suicidal	Undc
Depression	0.	59	0
Other Mental Disorders	0	21	. 1
Parkinsonism	Ō	2	Ó
Epilepsy	Ō	5	2
Hypertension	1	11	2 5
Stroke	2	4	0
Atherosclerosis	4	7	2
Emphysema	0	5	0
Cancer	1	11	0
Arthritis	0	5	3
Ulcers	3	9	. 1
Alcoholism/Cirrhosis	2	20	3
Thyroid Problem	0	4	2
Diabetes	2 2	8	2
Back Pain	2	8	0
Blindness (full or partial)	0	5	1
Pregnacy	0	3	0
Miscellaneous	5	24	7
Total	25	230	30

Table 49 Frequency of the Eighteen Most Commonly Found Diseases and Conditions in the Accidental, Suicidal and Undetermined/Unclassified (Undc) CO Deaths.

Drug Use

The pattern of prescription drug-use in the three groups, while similar to that for health problems, is somewhat more pronounced. Five (4.8%) of the 104 men and women who died accidentally had some prescribed medication available to them; none of these was issued more than one drug.

Of the 336 male suicides, 89 (26.5%) had at least one prescribed medication, 43 of these may have been using two drugs, while 23 may have been using three different medications. Of the 93 female suicides, 38 (40.9%) had at least one prescribed medication; 16 of these may have been using two drugs, while 11 could have been using three different medications. Of the 49 male Undc deaths, 7 (14.3%) had one medication, while three had three. Of the 7 female Undc, two (28.6%) had one medication, two had two and one of these women had three drugs prescribed to them.

Table 50 shows the frequency-of-use of the more common drug families. Somewhat paralleling the findings of diseases/conditions, the suicidal group had 44 times the number of prescribed medications as did the accidental group and the range of drugs, as well as the frequency of prescriptions was in excess to that seen for the accidental victims. This represents over ten times the number of drugs per person prescribed to the suicidal victims as to the accidental cases, while the Undc have roughly five times the number of medications prescribed as did the accidental cases. Almost entirely absent (1) is the reported use of psychotrophic drugs by the 108 accidental CO fatalities; whereas the 429 suicidal victims had 132 prescriptions for these medications on hand at the time of death with the 56 Undc cases having five.

Class of Medication	Accidenta1	Suicidal	Undc
Anxiolitic	1	33	. 3
Antidepressant	0	53	. 0
Antipsychotic	0	22	0
Hypnotic	0	24	2
Antihypertensive	0	11	4
Analgesic	0	9	1
Bronchodilator	0	3	1
Skeletal Muscle Relaxant	0	5	0
Anticonvulsant	0	5 5	1
Antigiotic	0	5	0
Antispasmotic	0	· 4 '	0
Antiparkinsonian Agent	0	7	0
Coronary Vasodilator	0	4	0
Hist-H2-Receptor Antag.	0	6	0
Miscellaneous	3	29	3
Total	5	220	15

Table 50. Frequency of Prescribed Medications in the Accidental, Suicidal and Undetermined/Unclassified (Undc) CO Deaths.

Alcohol, or EtOH, although itself a drug, requires separate analysis. It is of particular interest in this investigation for two reasons; first, the physiologic effects of alcohol may have contributed to the accidental situation, particularly if this compound was consumed in combination with medications and second, the literature suggests that ethanol may have an additive or alterately a protective action in conjuction with carbon monoxide. Table 51 presents the alcohol levels in the accidental, suicidal and undetermined/unclassified deaths.

BAC	Accidental		Suicidal		Undc	
mg./100m1. 	Male	Female	Male	Female	Male	Female
* <11	38	13	158	40	14	2
11-40	6	1	31	5	0	0
41-80	7	2	⁻ 20	12	1	2
81-120	11	4	19	2	7	0
121-160	7	0	35	7	3	0
161-200	9	1	31	12	7	0
201-240	4	0	14	5	9	2
241-280	2	0	13	8	4	1
281-320	0	0	4	2	3	0
321-360	0	. 0	6	0	1	0
361-400	0	. 0	3	0	0	0
>400	0	0	2	0	0	0
Total	83	· 21	336	93	49	7

Table 51. Frequencies of Levels of Blood Alcohol (BAC) in the Population of Male and Female Accidental, Suicidal and Undetermined/Unclassified (Undc) CO Deaths.

*BAC <11mg/100ml is not considered to necessarily be due to the ingestion of alcohol.

There are 46 (55.4%) male and 8 (38.1%) female accidental carbon monoxide deaths with BAC>10 mg./m1.; 70.4% of these 54 individuals have levels greater than the present legal limit in Alberta (80 mg./100 ml.). One hundred and eighty (53.0%) of the male and 53 (57.0%) of the female suicidal CO deaths have BAC>10 mg./100 ml.; 84.5% of these 233 individuals have levels over the legal limit. Corresponding values for Undc were 71.4% of both males and females with BAC >10 mg./100 ml., and 92.5% of these individuals are over the legal limit.

The mean blood alcohol for accidental deaths with BAC >10 mg./100 ml. is 125.7 for men and 92.5 mg./100 ml. for women. Corresponding values for suicidal and Undc deaths are 183.0, 149.4 and 203.2, 187.2 mg./100 ml.

Some alcohol consumption occurred in 51.4% of all of the accidental, 53.8% of all the suicidal and 71.4% of all the undetermined/unclassified victims at the time of death. The ingestion of alcohol therefore appears to be a frequently occurring factor in all manners-of-death and the mean BAC levels for those who had been drinking (BAC >10 mg./100 ml.) are well above the legal limit, this being particularly true in the suicidal deaths.

Alcohol and Drugs

Many drugs are contraindicated in conjuction with alcohol consumption. These include most of the antipsychotic, antidepressant, hypnotic, anxiolytic, anticonvulsant and antispasmotic medications. Alcohol is a central nervous system depressant and can act to enhance and/or prolong many of the pharmacologic properties of these compounds (which frequently include dizziness, mental confusion, drowsiness, fatigue and possibly unconsciousness). Any one of these symptoms could be relevant to an exposure to CO, itself a CNS depressant. The data were therefore examined for the concurrent use of alcohol with one or more of these compounds.

None of the accidental or Undc fatalities who had prescribed medications available to them and on whom a blood alcohol determination was done, have blood alcohol levels greater than 10 mg./100 ml. Therefore, in these populations of accidental and Undc carbon monoxide deaths, the interaction of alcohol with drugs is not shown to be of consequence to the circumstances of CO poisoning.

Fatal Carboxyhemoglobin Levels

The final topics to be investigated in this chapter are the relationships between particular diseases or conditions, drugs or alcohol consumption, age and sex with fatal COHb values.

Carboxyhemoglobin is used as the dependent variable in this study, for although there is evidence in the literature to support the existence of other toxic mechanisms in COP, no routine assays can quantify these reactions. Before proceeding with the investigation into the relationships with COHb, the distribution of the fatal levels of this compound will first be considered.

More than ninety-nine percent of the carboxyhemoglobin determinations were performed by three main laboratories in the province. Analysis of variance on these values is significant (Table 52); moreover, using Bonferroni tests, significant differences (p=.05) are found between the mean COHb values in all combinations of laboratories. These differences could not be explained through different proportions in the age, sex or the suicide/accident/Undc case-structures in the three labs. They may be the consequence of other factors, in particular, various assay methods, or different instrumentation.

Lab	N	Range	Mean COHb	S.D.
1*	195	. 24-99	79.53	12.81
2*	282	30-96	75.50	12.44
3*	79	22-95	67.94	12.65

Table 52. Measures of Central Tendency of Fatal COHb Values Reported by the Three Main Laboratories on all Victims of COP.

*Bonferroni tests. lab 1/lab 2 p=.05 lab 1/lab 3 p=.05 lab 2/lab 3 p=.05 Since the source of the variation in the mean carboxyhemoglobin values between the laboratories is not known, the results from each laboratory must be examined separately. Only the results from the two laboratories with the largest number of COHb values were selected for use in the subsequent analysis of the relationships between age, sex, certain diseases/conditions and drugs and blood alcohol concentration with fatal COHb levels. The third laboratory, with 79 COHb values, was not included because of small numbers and because the calculated mean fatal COHb value (67.94) differed the most from the other two mean values (75.50 and 79.53). Further investigation into the source of the variation may show if this exclusion was necessary.

<u>Accidental, Suicidal and Undetermined/Unclassified Exposure</u>. The mean fatal COHb of the suicidal deaths is significantly higher than that of the accidental victims and this finding is duplicated both labs (Table 53). The mean COHb determinations from both laboratories for the

Lab	• Туре	N	Range	Mean COHb	S.D.	Median	Mode
1	Acc	25	24-94	73.60	17.53	76.00	86.00
2	Acc	45	45-92	68.13	12.45	69.00	not unique
							•
1	Suic*	148	39-99	80.94	11.03	82.00	88.00
2	Suic**	210	30-96	77.12	12.10	80.00	80.00
1	Undc	22	26-99	76.82	15.83	78.50	86.00
2	Undc***	27	53-92	75.22	10.83	78.00	not unique

Table 53. Measures of Central Tendency of COHb Values for Accidental (Acc) Suicidal (Suic) and Undetermined/Unclassified (Undc) CO Deaths, Lab 1 and Lab 2.

* .001<p<.01 Suic/Acc Lab 1.</p>

** p<.001 Suic/Acc Lab 2.

*** .01<p<.02 Undc/Acc Lab 2.

undetermined/unclassified deaths lie between those of accidental and suicidal cases and these are significantly different from the mean accidental fatal COHb values in only the Lab 2 situation.

The reason for differences in the mean levels of fatal COHb in the three manners-of-death is not readily understood; however this must be considered in the analyses. Since the suicidal deaths have the greatest number of cases and also may be more homogeneous because of the common intent (as evidenced by the generally smaller variances associated with these values), the analyses of the relationship of variables with fatal COHb levels is restricted to only those cases in which the manner-of-death was deemed to be suicidal.

<u>Distribution of COHb Values</u>. Table 54 shows the frequencies, percent and cumulative percent of the the fatal carboxyhemoglobin values in the suicidal deaths in both laboratories. Examination of

СОНЬ	Freq.	Lab 1 %	Cum %	Freq	Lab 2 %	Cum %
0-30	0	0.0	0.0	1	0.5	0.5
31-40	1	0.7	0.7	3	1.4	1.9
41-50	2	1.4	2.1	6	2.9	4.8
51-60	4	2.7	4.8	10	4.8	9.6
61-70	14	9.4	14.2	25	11.9	21.5
71-80	45	30.4	44.6	72	34.3	55.8
81-90	56	37.8	82.4	75	35.7	91.5
<u>91-100</u>	26	17.6	100.0	18	8.5	100.0
Total	148		100.0	210		100.0

Table 54. Frequencies, Percent and Cumulative Percent of Fatal COHb Values Reported by Lab 1 and Lab 2 for Victims of Suicidal COP.

the distribution of fatal COHb values shows that in Lab 1, 7 (4.8%) and in Lab 2, 20 (9.6%) of these deaths occurred at or below 60% COHb, while 1 (0.7%) and 4 (1.9%) occurred at or below 40% COHb. The table shows that although the actual numbers of deaths appear to rise sharply above

60% COHb (the level which is generally considered to be consistent with death), the medians are 82 and 80% COHb for the two labs and the modes, 88 and 80%. This indicates that there were a large number of deaths at values much higher than 60% COHb, in fact 55.4% and 44.2% of the deaths were over 80% COHb for Lab 1 and Lab 2 respectively.

<u>Age and Sex</u>. Neither of these variables shows a relationship with the mean COHb; both males and females have mean values within that which could be attributed to normal variation. Similarly, the mean fatal COHb values in the three age groups: under 16,16–65, and over 65 are not significantly different from one another. These findings are identical in both laboratory settings.

<u>COHb and Blood Alcohol</u>. The effect of alcohol on the human response to carbon monoxide is not well understood, as discussed in the review of the literature. Conflicting evidence from animal studies suggest either an enhancement or a protective effect of blood alcohol on the physiologic response to carbon monoxide (as measured by COHb levels).

The nature of data used in the present investigation restricts the analyses to two fundamental questions; 1. whether positive blood alcohol (>10 mg./100 ml.) values coincide with significantly different mean COHb values than those from cases with negative BAC (<10 mg./100 ml.), and 2. whether there is evidence of a dose-response relationship between the blood alcohol levels and fatal COHb concentrations.

1. Mean COHb and BAC.

Comparison of the mean COHb between deaths with BAC less or equal to 10 mg./100 ml., and those with blood alcohol levels >10 mg./100 ml., reveal that the latter group have a lower mean fatal COHb. This difference is statistically significant (Table 55) and this finding is confirmed in both laboratory situations.

Lab	N	<11mg/100m1	<u>S.D.</u>	<u>N</u>	>10mg/100m1	<u>S.D.</u>
1*	56	78.46	11.95	85	82.58	10.46
<u>2**</u>	100	75.60	13.79	106	78.79	9.66

Table 55. Lab 1 and Lab 2 Mean COHb Values in Suicidal Victims with Blood Alcohol Levels Greater than, or Less or Equal to 10 mg./100 ml.

p=.05** n=.03**

2. Evidence of a Dose-Response Relationship.

The Pearson correlation coefficient (r) between fatal COHb levels and blood alcohol values in suicidal cases with BAC>10 mg./100 ml. is not statistically significant in either laboratory situation. However, the correlation coefficient between COHb and BAC in cases which had BAC greater than 10 mg./100 ml. is significant, but only for Lab 1 values, with r = 0.1844, (.02<p<.05).

The weakness of the correlation found in Lab 2 (the laboratory with the greater number of cases) suggests that a linear association between increasing levels of BAC and fatal COHb values is in question. Some investigations have suggested that the relationship between COHb and BAC may not be linear (Fazekas, Rengei, 1967; Pankow, 1974). The mean fatal COHb values were compared using the same blood alcohol intervals suggested by Fazekas, i.e. 115–129 and >230 mg/100 ml. Using these intervals, the COHb values increased with increasing BAC and there is some suggestion that this relationship reverses at BAC levels over 130 mg./100 ml. (similar to what was reported by Fazekas). A non–linear relationship does aid in explaining the conflicting findings of other investigations, but no firm conclusions can be made without further investigation.

<u>Carboxyhemoglobin Level and Disease</u>. Past investigations into the effect of of carbon monoxide on individuals with certain diseases or conditions has been particularly concerned with the relationship between CO exposure and heart disease. But exposure of humans with these health problems has understandably been for only very brief periods and at low concentrations of carbon monoxide. In some animal experimentation; however, such symptoms such as cardiac arrhythmia in healthy animals is elicited using high concentrations of CO.

It was therefore questioned whether those cases in the study population with either heart problems or the closely related atherosclerosis, might die "earlier" (as indexed by lower fatal COHb values) than those with no disease. Comparison (Table 56) of the mean fatal COHb from

	Heart Disea	No Disea	No Diseases/Conditions			
<u>Laboratory</u>	COHb	(n)	<u>S.D.</u>	СОНЬ	(n)	<u>S.D.</u>
Lab 1 *	65.75	12	16.14	78.76	59	11.31
Lab 2**	62.73	11	17.47	75.39	92	14.54

Table 56. Comparison of Mean COHb Values in Suicidal Victims with Heart Disease or Atherosclerosis and those with No Diseases/Conditions, Lab 1, Lab 2 (BAC <11 mg./100 ml.).

*p<.001 **.001>p>.01

suicide cases with either one of these two health problems with those cases with no diseases or conditions (controlling for alcohol), shows a significant difference in the mean fatal COHb values, with the lower value in those cases with either (or both) of the two health problems. This finding is duplicated in both laboratories.

<u>Carboxyhemoglobin Level and Medications</u>. Medications which may have some physiologic effect in combination with carbon monoxide are those which are in themselves central nervous system depressants. These include many of the antidepressants, anxiolytics, antipsychotic and hypnotic formulations. Since these drugs were frequently used in the sub-population of suicidal deaths, a comparison is made in this group between the fatal mean COHb of those who were prescribed one or more of these medications with that of those with no reported disease, again controlling for BAC, as well as heart disease or athersclerosis (Table 57).

		Drug Use			ises or Cond	
Laboratory	СОНЬ	(n)	S.D.	СОНЬ	(n)	S.D.
Lab 1	79.00	24	10.78	78.76	59	11.31
Lab 2	76.78	36	15.80	75.39	92	14.54

Table 57. Comparison of Mean COHb for Suicidal Cases with Reported Use of Antidepressants, Anxiolytics, Antipsychotics or Hypnotics and for those with No Diseases or Conditions, Lab 1 and Lab 2 (BAC < 11 mg./100 ml.).

While the mean fatal COHb values are lower for those with drug use compared to those with no disease, neither of the Lab 1 or Lab 2 comparisons is statistically significant (alpha =.05). This suggests that these drugs may not have an effect upon the level of COHb at death; however, there is no certainty that therapeutic levels were present at the time of death. Moreover, only certain preparations, within certain drug categories may have an effect on the physiologic resonse to CO.

Chapter Summary and Discussion

This chapter has described the frequencies of health problems and drug and alcohol use in fatal COP and investigated the relationships between these variables and fatal COHb levels and the distribution of fatal COHb values.

The proportions of people with health problems are highest in the suicidal and undetermined/unclassified deaths, with over twice that of the accidental cases. Many health problems in the suicidal victims might be anticipated; for example, mental problems, alcoholism and cancer. However, these deaths also include a large number of individuals with heart disease and hypertension compared to that seen in the accidental group. Although the reason for this is not known, it could be because of the older mean age of the suicidal victims. This finding is of use in the examination for an interaction between certain health problems and CO.

The use of drugs paralleled the health problem situation, with 220 medications prescribed to the suicidal compared to five to the accidental victims. There is no evidence of a chance for interaction

between the ingestion of alcohol and the use of certain CNS depressant medications in the accidental and the Undc groups; however, 37 (8.6%) of the suicidal cases with positive blood alcohol readings had access to at least one of these drugs. Recent alcohol consumption, as evidenced by BAC >10 mg./100 ml., is seen in approximately 52% of the accidental, 54% of the suicidal and 71% of the Undc deaths, with over 70% of all of these cases with BAC over 80 mg./100 ml.

The investigation into the distribution of fatal COHb levels shows that the mean values from the three main laboratories in the province (which performed over 99% of the determinations) were all significantly different from each other. This could not be explained by different age, sex or manner-of-death structures in the populations of cases and is possibly due to some other sources of variation, notably some aspect of the testing itself. It was therefore necessary to examine the lab results separately. The two laboratories with the most similar results and the greatest number of cases were selected for study. These proved to be useful in that results could be contrasted.

Examination of the distributions of fatal COHb values of the suicidal deaths shows that there are five below 40% COHb and a total of 27 fatalities below 60% COHb (in the two laboratory situations). Although the actual numbers of deaths appear to rise sharply in the 60–70% COHb category, the medians and modes for COHb in both laboratories indicate that most of the deaths occurred well above these levels and in fact 44–55% of the deaths were over 80% COHb. This suggests that many individuals, while incapacitated by CO at levels around 60% COHb, frequently live long enough to take a final few breaths, thereby attaining elevated COHb values before their breathing stops and death occurs.

Purser and Berrill (1983) describe that monkeys breathing carbon monoxide attain high COHb levels before being suddenly overcome. This finding might partially explain why it is not unusual to find that investigations into CO deaths will frequently describe discovering many of the victims in body-positions which are suggestive of a sudden awareness of danger and need to escape. Fear, coupled with the energy requirements for flight could act to increase the CO-uptake at a time when some threshold may already have been reached. Alternatively, other toxic mechanisms may play a significant role in many of these deaths, depending upon the manner in which (time, concentration and the individual variation) this gas was encountered by individuals. These other toxic mechanisms may better account for the deaths at low COHb levels and for the large number of deaths well above 60%, with COHb-formation perhaps even a secondary phenomenon.

Different ages or sex do not show statistically significant relationships to fatal COHb levels; however, from the data it is not possible to determine if the younger (or older people), and females (with generally lower body weight and less hemoglobin with which CO can combine), do not reach fatal levels faster.

Levels of blood alcohol greater than 10 mg./100 ml. appear to be related to death at higher COHb levels than if EtOH is not ingested. This could possibly be due to lowered oxygen demands by the brain in the presence of alcohol permitting more CO to be inspired before breathing ceases. Alternately, ethanol in the system might possibly alter cell-permeability to CO and consequently affect its reaction with the cytochrome system.

The relationship between increasing blood alcohol and fatal carboxyhemoglobin levels is unclear, with only one laboratory showing a statistically significant linear relationship. Weak support is found for the suggestion that this relationship may not be linear over all values of BAC. It remains possible that different physiologic mechanisms predominate at different blood alcohol concentrations and may account for some of these findings.

Individuals with the diagnosis of heart disease and/or atherosclerosis died at a significantly lower COHb than did individuals with no health problems (controlling for blood alcohol). This finding is supported by the works of Purser and Berrill, 1983 and Aronow et al., 1978 which show that not only does CO reduce the time to onset in animals with a predisposition to fibrillation but also it induces fibrillation in healthy animals. The effect of CO on other disease states, e.g. anemia, pregnancy could not be investigated because of small number of cases with these conditions.

No relationship between certain CNS depressant drugs and fatal COHb levels is seen, but it is not known if therapeutic levels of these drugs were present at the time of any of these deaths. It is also possible that not all CNS depressants have an identical effect at all levels of COHb.

Chapter 8

Final Discussion, Limitations of the Study and Reccomendations for Future Research

Each of the four analysis chapters include a summary and discussion of results. Much of this information will not be re-introduced in this final discussion section. Here the emphasis will be upon the structure of the study, the over-all findings, comparisons with other investigations and the limitations of this research. This chapter will conclude with recommendations for future investigation into carbon monoxide poisoning.

This epidemiologic investigation has been in part exploratory and certain phases of the analyses produced information which could not have been anticipated beforehand. This information influenced the final structure of the study and this is evidenced by the use of various sub-groups at different stages in the analyses. In addition to the investigation of the entire population of fatalities the examination of these sub-groups aided in the characterization of deaths from carbon monoxide. These groups were defined according to: three source-environment situations, different laboratories and manners-of-death and finally, the working ages, 16-65.

The Entire Population of Fatalities from Carbon Monoxide

Examination of the entire population of deaths from CO indicates that there were no over all trends during the study period. The mean annual accidental rate of 0.8 per 100,000 compares well with 0.9 per 100,000 in a Georgia study (Lisella et al, 1978) and approximately 0.6 per 100,000 in the United States (Morrison, 1967), but contrasts with the rate of 2.8 per 100,000 reported in England by Smith and Brandon (1970). True comparisons are difficult as both the Lisella and the Smith studies include fire deaths in the rate calculations. If this type of death is

included in the calculation of accidental rates in Alberta the annual accidental death rate from CO approaches 2.0 per 100,000.

Using population estimates at the time of the investigation or rates reported in the particular study, none of the corresponding annual suicidal death rates agree. The calculated 3.3 per 100,000 in the present study is much larger than the 0.6 per 100,000 in the Georgia paper and the estimated 1.0 per 100,000 in the U.S. publication, while it is less than the 4.2 per 100,000 found by Smith and Brandon in 1970 (no comparative rates are available for undetermined/unclassified cases.) Differences may be a result of different coroners' systems, different social factors, different climatic conditions, or a consequence of the use of various data bases.

Males out-number females in all manners-of-death categories, with the male to female ratios of 4.0, 3.5 and 6.9 for the accidental, suicidal and undetermined/unclassified groups respectively. The corresponding ratios for accidental and suicidal deaths reported by Smith and Brandon are 1.4 and 1.8, and 2.0 for the acccidental CO deaths in the Lisella study. Assuming a similar male to female composition in these different study populations, Alberta appears to have a greater proportion of male deaths from CO (in the accidental and suicidal deaths) than is found elsewhere.

Accidental death rates are highest in the non-metropolitan settings, while the reverse is true in the suicidal and the undetermined/unclassified deaths (Undc). None of the comparative studies on fatal COP makes this differentiation, or a rural/non-rural comparison.

Sub-Populations of Fatalities from Carbon Monoxide

<u>The three Source-Environment Situations</u>. The characterization of the entire populations of accidental, suicidal and Undc fatalities is described in Chapter 4. This information is expanded in the following chapter with a more detailed investigation into the fatal settings; whereby, three apparently distinct source-environment situations emerge. The first two, indoor and outdoor motor vehicle exhaust (IMV,OMV), are those alluded to by Baker et al (1972); while the third group, MISC, is comprised of those individuals who died in miscellaneous situations which differ from the other two in the source and in the way human activity contribute to the fatality.

The division into three sub-categories is of marginal utility in better characterizing the suicidal deaths and of none in the undetermined/unclassified situation. The reason for this is because the latter fatalities are nearly exclusively from vehicular exhaust in the indoor setting. On the other hand, the suicidal deaths, although (almost all) due exposure to exhaust in the indoor or the outdoor setting, show few difference between these groups in any of the variables which were studied.

Division into the three source-environment categories, however, appears to be of considerable benefit in the characterization of accidental deaths from CO. These groups frequently demonstrate significant differences when they are compared among themselves, between each other, with the entire group of accidental deaths or with provincial population; the greatest contrasts seen in age and month, or season of the year (Table 34, page 64). This suggests that different groups of people may be vulnerable to different types of accidents.

1. Age

The Baker study shows a median and mean age of (IMV + OMV) accidental COP of 20–29 and 32. The corresponding parameters in this study are 16–25 and 31. While the medians are not comparable because of different age classifications, they consistently relate to a younger age group. And the mean ages, although not the best measure of central tendency in this situdation, are very similar. Smith and Brandon (1970) show an increase in the age-specific mean annual prevalence rate in the 20–30 year age group and also in the 70–80 age category. This agrees with the present findings which shows the highest age-specific death rates for all the accidental deaths in the 16–25 and also in the 76–85 age groups. The Smith and Brandon study also suggests that the two age groups differ in the type of accident, with accidents in the younger group principally due to occupational exposure, while accidents in the older ages are due to advancing age and mental deterioration. Similarly, the present investigation has supported the idea that different ages are involved in different types of accidents, i.e., IMV,OMV and MISC.

2. Month and Season.

Lisella (1978), Fink (1966) and Baker (1972) all indicate that the highest number of

accidental deaths occur in January and December. The "Total" group of accidental deaths and the IMV and MISC sub-categories in this study show the same pattern, however a different distribution is seen in the OMV sub-category, with the summer months of June and July having highest number of deaths while the winter months have the lowest.

Comparison of the distributions of the Baker (1972) IMV+OMV accidental deaths with the present results show that the two are significantly different from each other (p<.01), with the greatest differences seen in the deaths in June, July and August, the Baker group having far fewer deaths than expected in this season and the Alberta group having more. Since the OMV contribute heavily to the number of fatalities in this seasonal period it was anticipated that the proportion of this type of death in the Baker study would be greater than in the study population. However, the reverse situation is true. This seemingly conflicting finding may be the result of the different climates in the two areas The climate in Maryland may be warm enough in the summer months enabling people to sit in a vehicle without warming it with the engine, while the climate in Alberta is such that heat is often required at night even in the warmest months of the year. Faulty vehicles in warmer climates may not be discovered until later on in the year.

Finck (1966) also shows that the greatest number of deaths occur in January and December and the lowest in the late spring and in the summer. This investigation also includes fire deaths and the study population is largely military personel. Both of these may be factors which contribute to this distribution of deaths.

<u>Different Laboratories and Different Manners-of-Death</u>. Significant differences are found among the mean fatal carboxyhemoglobin values obtained from the three main laboratories and between the mean fatal COHb values in the accidental and suicidal deaths. The source(s) of variation could not be identified and may be due to some element in the sampling procedure or in assay techniques. Finck (1966) also shows a similar difference in the mean fatal COHb between suicidal (77.0%) and the victims of non-fire accidental (64.6%) COP victims. The accidental COHb values are lower than those in the suicidal victims, comfirming the results of the present investigation. Baker et al

(1972) find a mean fatal COHb of 62.8% in the accidental group. This compares well with the Finck values, but is somewhat lower than the 68.1 and 73.6% determined by the two largest Alberta laboratories.

The distribution of fatal COHb (in the suicidal cases) shows that deaths under 40% COHb are rare, with 5 (1.4%) of the total number. These values may represent individuals who are highly susceptible to the effects of CO, possibly through a highly sensitive cytochrome system as suggested by Barbeau et al (1985), or simply are the result of other sources of individual variation or of resuscitative attempts which reduced the level of COHb in the system before the blood sample is drawn for analysis.

Purser et al (1983), in a study of the effects of brief exposures of monkeys to high concentrations of CO, report that the animals exhibit a dramatic change in behaviour over a 1-2 minute period, changing from "a seemingly normal state to one of severe intoxication", passing very rapidly from this stage to one of deep coma.

It is considered likely that this same "threshold effect" may also occur in humans. Therefore, the high (90-95%) proportion of deaths over 60% COHb may indicate that, for most people, the critical point for incapacitation lies below this point, but death occurs at higher carboxyhemoglobin levels since this will continue to increase until respiration stops.

A similar distribution of fatal COHb levels is seen in accidental victims; however, there is a larger proportion of these deaths below 60% COHb than seen in the suicidal deaths. Early discovery and resuscitative attempts may be responsible, but as has been discussed earlier, Purser demonstrates that active animals are affected earlier and much more seriously than are the chair-bound ones. The active animal appearing to be unable to sustain a normal state of behaviour at COHb levels which have little effect on the inactive ones.

With the thought that it is the rate of uptake of CO, i.e., the concentration over time and perhaps not just the COHb level, which is a vital determining factor in fatal carbon monoxide poisoning, it is possible that activity may lead to earlier incapacitation and possibly to an earlier death (indexed by lower mean COHb values). That is, because an individual is affected faster (over a shorter period) s/he dies sooner, before acquiring a more highly elevated COHb.

This action may help to explain why accidental victims of COP who frequently are moderately active (for example, working on the car or in the garage), have a significantly lower mean fatal COHb than do suicidal victims who generally have been inactive (sitting or lying) in the vehicle.

The suddeness of onset of incapacitation as described in monkeys may explain why it is not uncommon for victims of COP to be found in positions which suggest that they tried to escape, but their awareness came perhaps suddenly and too late. Delayed awareness of what was happening to them, coupled with an even slight increase in energy requirements for flight, makes escape impossible.

The relationship between alcohol consumption and fatal COHb values suggests that alcohol does have an effect on fatal COHb concentrations. This could be due to decreased O_2 demands in the presence of alcohol, thereby increasing the time period over which CO is inspired before death. Alternately, it is possible that different concentrations of EtOH may vary the permeability of the cell by CO and therefore determine the rate of reaction with other biochemical processes.

An inverse linear relationship between increasing blood alcohol levels and decreasing fatal carboxyhemoglobin concentrations is not confirmed, but the data suggests that the relationship may not be linear over all values of BAC, possibly reversing at levels greater than 130 mg./100 ml.

Mean fatal carboxyhemoglobin values are often quite variable in and between other investigations studies. They may depend to a great degree upon the manner of death structure of the population of deaths and on the use of different techniques in measurement; accordingly they must be used and interpreted with caution.

<u>The Working Ages 16-65</u>. No other studies of fatal COP have been found to investigate this subgroup of individuals or the relationships of the deaths to occupation. The findings of the present research suggest that the working age sub-group is the population at risk to COP and that individuals trained in certain occupations may be at greater risk to this type of death than are others. Accidental deaths from carbon monoxide to those with the skill to work on vehicles is not surprising since these people likely have a greater opportunity for exposure to exhaust than do those who do not share this skill. However, the relationship of Machining occupations to accidental death from CO is more difficult to understand., but perhaps these people have a skill which is related to that which is required to install and maintain appliances and heating equipment. Similarly, the deaths of those involved in the construction trades may represent people with a willingness (or "handiness") to attempt these same activities, but perhaps with marginal ability and a limited awareness of the hazards.

The suicidal cases, while showing a similar significant difference in proportions for the Machining and Construction occupations, also show this difference in the Transport Equipment category. This may be due to factors which enter into the decison into the choice of a weapon for suicide, i.e., those who are so-involved with transportation, for example truck drivers and cab drivers, may have a special relationship to their vehicles. These kinds of jobs also may be inherently stressful and/or they may attract people with more personal problems which eventually contribute to death by suicide.

Another factor which may closely relate to the findings in both the suicidal and accidental deaths is socioeconomic status. This may determine the financial situation and concurrently influence certain lifestyle choices. Many of the accidents occurred to people who owned defective (often old) vehicles (OMV), possibly an indication of low income. Many others were working of their vehicles (IMV), or they were responsible for the poor installation and maintenance of various appliances and heaters (MISC). The financial necessity to operate an older model vehicle or to do one's own maintenance, coupled with a marginal level of expertise, may act to increase the risk of COP to lower-income, semi-skilled individuals.

Since the Undc fatalities are in actuality either accidental or suicidal, with the true intent frequently obscured by alcohol use, it would be expected that this group would share some of the over-represented occupations of the other two manners of death, but this is not the case. This may be because what a person "does" may be of some consequence to the medical examiner in the

decision-making process involved in determining intent, acting in some way to make the death more "explainable". Certain occupations therefore, may be less likely to appear in the undetermined/unclassified category since individuals with such training or experience are in some way, more convincingly, either accidental or suicidal in intent.

Work-related problems are discussed together with other life problems and problems with alcohol. The reasons for inclusion of the two latter sources of stress in the chapter on occupation is to emphasize the number of distressing life situations occurring to these groups of people and to suggest the possible impact of this on their working and personal lives. Life problems, whether due to the job situation, to alcohol or to those in private life, do not begin or end at the door to the home or at the place of employment. It is seen that the suicidal deaths have more of every type of problem except alcohol (which is more common in the Undc deaths). Marital problems, problems with personal health and job loss are commonly seen in each group of deaths.

There was one compensatable accidental CO death in the six year study period. Four other accidental deaths show some relationship to work, but they were not directly associated to it in time, place or activity. Two Undc deaths occurred to mechanics while working on vehicles, but privately in their own garages; while 17 suicidal deaths appeared to have used the work environment because of its remoteness and privacy.

The need for seclusion is not exclusive to suicidal situations, but it is also frequently encountered in the circumstances of accidental and Undc deaths. Vehicles, in or out of a garage and the garage itself, are often used as an extension of the living space-a place to which one can retreat for various activities.

Baker et al (1972) discuss the wide variety of uses to which automobiles can be put. Unfortunately this investigation can not be used for comparison because of fundamental differences in selection criteria; however, many of the situations are common to what has been observed in the present study. These include: going to the car/garage to end an argument, to drink, to listen to music or to think, to sleep, to be with a sexual partner, or to work on the vehicle. These more or less isolated environments, coupled with the human need for warmth often set the scenes for accidental exposure to carbon monoxide.

The epidemiologic approach gets its power from the aggregation of data; whereby events, rather than viewed as unique occurrences are better studied in their unifying conditions. In the case of fatal carbon monoxide poisoning, it appears that these conditions must be defined by several sets of circumstances. Without these partitions the collection, or aggregate, of cases is too wide-reaching for meaningful study and interpretation.

Limitations of the Study

Interpretation of the findings of this investigation must be with the full understanding of certain limitations of the data. Comparison with other studies must be with acknowledgement of the variability in different coroners' systems. Both of these topics were introduced in Chapter 3 with regard to the completeness, reliability and applicability of the data for the objectives of the study. Here, they are reconsidered in respect to the interpretation of the results.

Sackett (1979) catalogued 35 biases which can arise in sampling and measurement. Two of these are possible sources of bias in the present investigation. The first is, " diagnostic suspicion bias"; whereby, "knowledge of the subject's prior exposure to a putative cause (ethnicity, taking a certain drug, having a second disorder, being exposed in an epidemic) may influence both the intensity and the outcome of the diagnostic process.".

Atkinson (1978) explores the question of how coroners arrive at their decisions and suggests that certain characteristics, because they are commonly believed to occur more frequently in suicidal deaths may act as a self-fulfilling prophecy. For example, deaths of individuals with certain life problems may more likely be judged as suicidal than those without such problems.

Not only will suicidal (and accidental) death rates reflect this bias, but importantly here, since the medico-legal investigations are conducted until the cause and manner-of-death are decided or no further evidence is forthcoming, the investigation may be prematurely concluded when such "putative causes" are discovered.

Cases therefore may vary in the way in which they are investigated, with "obvious" cases likely investigated in lesser depth than those more equivocal. Allocation to a specific category therefore may in part depend upon "knowledge of the subjects prior exposure". This will likely have less effect upon the description of the common demographics such as age and sex, since these are routinely collected, but will be more likely to affect the collection of data on the more "esoteric" variables such as drug and alcohol use or state of health.

The second source of bias which must be considered is "recall bias" since information is almost exclusively obtained through interview(s) with survivor(s) (who are frequently in a highly emotional state). This may affect every aspect of an investigation, although it is likely countered by the experience and training of the medical investigators and examiners.

Since understanding and prevention of COP is the principal goal of this study and not to compare the characteristics of suicidal versus accidental (or undetermined/unclassified) deaths, the first source of bias should not seriously affect the quality of the study if the results are considered with the data limitations in mind.

Although it is interesting to compare the frequency of certain health problems in the various manners-of-death, any conclusions regarding these comparisons must arrived at with caution. But for the purposes of this study, information on health, drugs and alcohol is mainly for the identification of cases with certain qualities, such as heart disease or drug and alcohol use; variables which are considered to likely interact with CO to make the subject more susceptible to its effects.

Conclusions from this investigation must also consider the level of analysis which has been used. The analyses are exclusively through the use of descriptive and bivariate statistics. This is for two reasons:

 the bivariate methods are appropriate for the intended level of investigation of this study, which is largely exploratory and descriptive, not hypotheses-testing. The results of these statistical methods are also considered to be more easily represented and discussed. 2. Multivariate analysis at certain stages (for example, multiple regression with COHb as the dependent variable) produces very little additional information about the relationships than what is obtained using bivariate statistical methods (controlling for such confounders as alcohol).

<u>Comparability</u>. Comparison with other studies is constrained by the limited information about the other study populations. Where at all possible, contrasts are introduced for improved interpretation; however, more comparisons would help to substantiate the findings.

<u>Reproducibility</u>. Factors which might affect the reproducibility of this epidemiologic study are: differences in the coroner (or medical examiner) system in place at the time the population is studied, other characteristics of the study population, such as religious, economic and cultural features and finally, the climate, the availability of vehicles and the use of fossil fuels in the study area.

Recommendations for Future Research

This exploratory study has attempted to characterize fatal COP in the province of Alberta during the 6 year time period, 1978–1983 through investigation into the demographic, temporal, occupational and other individual characteristics of the population of CO deaths. This research has supplied evidence which suggests that certain sub–groups of the general population may be more susceptible than are others to death from this agent because of certain characteristics; unfortunately, contrast and verification of the findings is limited at this time because of lack of comparative information.

While the description of the common demographic variables in the population(s) of deaths indicates that certain sub-groups may be at greater risk to COP, it is not know whether these are characteristics which are common everywhere, or whether regional differences in social, economic and cultural factors and variations in climate, would act to present a different picture. Therefore, the universality of the findings require confirmation through other epidemiologic studies in similar as well as in different settings.

This same problem occurs in the interpretation of the occupational characteristics of the study population. Although there is an indication that certain occupations are over-represented in the CO deaths, it is not known if this occurs elsewhere. It is also important to know if the apparent excess in deaths in some occupations are specific to CO or whether workers in these types of jobs are also more susceptible to other types of accidents and to other ways of committing suicide. More investigations into occupation-specific fatalities/suicides would help to clarify these questions.

The one occupational death (for which compensation was received) which occurred in the sixyear period was due to exposure to vehiclular exhaust and not the result of an industrial process. This may indicate that excellent preventive measures are in effect in this province; however, it may also reflect the relative absence of those industries which are very high risk to CO exposure. Although many publications state that CO is a common industrial poison, no information was available which lists the frequencies of the occupational CO deaths in various industries in Canada or in the United States. Such information is necessary to confirm whether CO is less of an occupational hazare in Alberta than elsewhere.

The investigation into the "personal" characteristics describe the presence of diseases/conditions, alcohol and drug use in the accidental, suicidal and undetermined/unclassified populations of deaths. While the data collection process may affect these figures interpretation of these results also requires knowledge of the age-specific prevalence of these variables in the general population.

The analyses of fatal COHb values indicates that in this study it is necessary to stratify by laboratory and by manner-of-death. Possible reasons for the between-group differences are suggested, but further investigation is necessary to confirm the source(s) of variation of the mean COHb values.

This investigation shows a significant difference between mean fatal COHb levels of those with positive blood alcohol readings and those with BAC less or equal to 10 mg./100 ml. The reason for this difference is not understood, but this may be because alcohol in the system may in some way decrease oxygen requirements; therefore, more COHb can be formed before death occurs. However,

it is not well established whether alcohol in fact modifies the action of CO on the body. The relationship of fatal CQHb levels to increasing BAC could not be conclusively demonstrated. Further investigation into this might yield further evidence of the mechanism(s) of carbon monoxide toxicology

It is shown that individuals with atherosclerosis and/or heart disease die at significantly lower mean fatal COHb than do those with no health problems. Other conditions which are thought to increase individual susceptibility such as Parkinsonism and anemia, were not similarly investigated because of the small numbers of cases with these conditions. Further investigation into the susceptibility of those with certain health problems to exposure to CO might aid in identifying those people who are more sensitive to this agent. This knowledge also might increase the understanding of CO toxicology, since some of these disease-processes are understood. And, convincing evidence of the action of other toxic mechanisms and the different "scenarios" of death which might occur if these contributed to the incapacitation of an individual could potentially aid medical examiners in the often difficult task of certifying intent in a sudden death from CO.

While the interpretation of some of the findings is constrained by the lack of confirmational data, this study does suggest that there may be certain groups in the Alberta population which are, at times, at risk to fatal COP and that these groups can be identified by their age, sex, socioeconomic status, occupation and residence characteristics. The over-representation of these characteristics in the accidental group likely exists because of the various lifestyle decisions of these individuals, frequently evidenced by particular human activities.

The accidental deaths caused by CO in motor vehicle exhaust in the indoor environment (IMV) appear to be mostly to individuals who work on vehicles in an enclosed space or use the vehicle and/or the environment as a place of refuge or of privacy. This group is similar in age structure to the over 15 population of the province. These deaths occur more often in the rural than in the urban environment when compared with the other two sub-categories of accidental deaths and about equally often in the metro as in the non-metro environment when compared with the provincial population distribution. These deaths occur more often in the cold winter months than in other

seasons of the year. This finding does not preclude the fact that similar activities may go on all year, but may indicate only that it is during the coldest months when certain activities may be more likely to lead to fatal accidents. These accidents are not generally found to be the result of a faulty vehicle, but are more clearly seen to be the consequence of remaining in a closed environment with a vehicle which is operated perhaps for only brief periods of time. These facts suggest specific educational measures aimed at prevention of this kind of accident, designed to reach this group of individuals, perhaps at certain times of the year.

On the other hand the accidental deaths caused by CO in motor vehicle exhaust in the outdoor environment (OMV), with 2.27 times the expected frequency of deaths in the 16–25 year age group, appear to be a group which has a higher proportion of younger individuals than found in the general population of the province. This type of accident appears to be more of a non-metropolitan than metropolitan phenomenon when compared with the population distribution in the province, but it also happens more frequently to individuals who live in the rural areas than seen in the IMV type of death. Although not significantly dependent upon month or season of the year, the distribution of these deaths peaks in the summer months in contrast to the situation found for the IMV (and MISC) situation. While the IMV accidents appear to be a consequence of individuals remaining in an environment in which any vehicle was operated, the OMV deaths are nearly all the result of sitting or lying in vehicles which were (useually later) shown to have defective exhaust systems and/or rusted body parts. These findings suggest the need for a system of inspection, particularly for older vehicles and also for those vehicles which rust more quickly than others because of poor design (such as muffler placement), or inadequate rust prevention at the time of the manufacture.

The accidental deaths from CO sources other than vehicles (MISC) are more clearly seen to be the result of the poor maintenance, faulty installation or the inappropriate use of heating appliances and furnaces. These deaths occur to people over age 45 more often than would be expected when the age structure of this group is compared with the over 15 population of the province. They, like the IMV are more of a winter phenomenon, but like the OMV are more of a non-metro (and rural) event. Preventive measures should therefore be aimed at a more mature audience than for the other two

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sub-groups of accidental death, with emphasis upon the specific problem. For example, older people in rural communities and in particular those where conversions from one kind of fuel to another are common (e.g., propane to natural gas) might make up appropriate target areas. Since male deaths consistently outnumbered female fatalities in all sub-categories of accidental deaths as well as males rather than females more often being responsible for the condition of the source in the OEV and MISC fatalities, all preventive efforts should be designed with the gender of the target group in mind.

Unlike other toxic gases such as H₂S, carbon monoxide itself does not have an identifiable odour and in all three sub-categories of fatal accidental carbon monoxide poisoning individuals appear to have remained in the toxic enviroment without consciousness of the presence of this toxic compound, or awareness of its build up or of their own slow or sudden debilitation by this gas. Moreover, these deaths may represent only a fraction of the number of dangerous exposures in the private sector. This suggests the need for the technological development of a warning device which is sensitive to carbon monoxide (e.g., Bertin, 0; 1986); one which is both affordable and designed for effectivness in these settings. Better understanding of the circumstances of poisoning might aid in defining the specifications for such a device. For example, it should not be so sensitive to sound or flash at initial low concentrations of CO (such as might occur when a vehicle is first driven into the garage, at an intersection or in heavy traffic), but rather might be designed to be activated by high concentrations or very importantly, by concentration of CO over time.

References Cited

- Anderson, E.W., Andelman, R.J., Strauch, J.M., Fortuin, N.J., Knelson, J.H.: Effect of Low-Level Carbon Monoxide Exposure on Onset and Duration of Angina Pectoris. A Study in Ten Patients with Ischemic Heart Disease. Ann. Intern. Med., <u>79</u>: 46-50, 1973.
- Aronow, W.S., Isbell, M.W.: Carbon Monoxide: Effect on Exercise-Induced Angina Pectoris. Ann. Intern. Med., <u>79</u>: 392-395, 1973.
- Aronow, W.S., Ferlinz, J., Glauser, F.: Effect of Carbon Monoxide on Exercise Performance in Chronic Obstructive Pulmonary Disease. Am. J. Med., <u>63</u>: 904–908, 1977.
- Aronow, W.S., Stemmer, E.A., Isbell, M.W.: Effect of Carbon Monoxide Exposure in Intermittent Claudication. Circulation, <u>49</u>: 415-417, 1974.
- Aronow, W.S., Stemmer, E.A., Wood, B., Zweig, S., Tsao, D., Ragiio, L.: Carbon Monoxide and Ventricular Fibrillation Threshold in Dogs with Acute Myocardial Injury. Am. Heart J., <u>95</u>: 6, 754-756, June, 1978.
- Atkinson, J.M.: Discovering Suicide: Studies in the Social Organization of Sudden Death, Pittsburgh: University of Pittsburgh Press, 1978.
- Ayres, S.M., Giannelli, S., Mueller, H.: Myocardial and Systemic Responses to Carboxyhemoglobin. Ann. N.Y. Acad. Sci., <u>174</u>: 268-293, 1970.
- Baker, S.P., Fisher, R.S., Masemore, W.C., Sopher, I.M.: Fatal Unintentional Carbon Monoxide Poisoning in Motor Vehicles. Am. J. Public Health, <u>62</u>: 11, 1463-1467, 1972.
- Barbeau, A., Roy, M., Paris, S., Cloutier, T., Plasse, L., Poirier, J.: Ecogenics of Parkinson's Disease: 4-Hydroxylation of Debrisoquine. Lancet, 1213-1216, Nov. 30, 1985.
- Barraclough, B.M.: Are the Scottish and English Suicide Rates Really Different? Brit. J. Psychiatry, <u>120</u>: 267-273, Mar., 1972.
- Bender, W., Gothert, M. et al.: Effects of Low CO Concentration on Man. Arch. Toxikologie, <u>27</u>: 142-153, 1971.
- Bernson, V.: A Comparison of the Cellular Toxicity of Exhausts from Cars Driven on Present and Future Fuels. Toxicol. Lett., <u>19</u>: 119-126, 1983.

Bertin, Oliver.: "Starting Out." Report on Business Magazine, pp. 26-32, August, 1986.

- Blishen, B.R., McRoberts, H.A.: A Revised Socio-Economic Index for Occupations in Canada. Can. Rev. Sociol., <u>13</u>: 71-79, 1976.
- Buchwald, H.: Exposure of Garage and Service Station Operators to Carbon Monoxide. Amer. Ind. Hyg. Assoc. J., <u>30</u>: 570-575, 1969.
- Burney, R.E., Wu, S., Nemiroff, M.J.: Mass Carbon Monoxide Poisoning. Clinical Effects and Results of Treatment in 184 Victims. Ann. Emerg. Med., <u>11</u>: 8. 394-399, 1982.
- Chance, B., Erecinska M., Wagner, M.: Mitochondrial Responses to CO Toxicity. Ann. N.Y. Acad. Sci., <u>174</u>: 193-204, 1970.
- Clark, R.T., Otis, A.B.: Comparative Studies on Acclimatization of Mice to Carbon Monoxide and to Low Oxygen. Am. J. Physiol., <u>196</u>: 285-294, 1952.

Coburn, R.F.: Mechanisms of CO Toxicity. Prev. Med., 8: 3, 310-322, May, 1979.

- Coburn R.F., Forster, R.E., Kane, P.B.: Considerations of the Physiological Variables that Determine the Blood COHb Concentrations in Man. J. Clin. Invest., <u>44</u>: 1899-1910,1965.
- Coburn, R.F., Ploegmakers, F., Gondue, P., Abbaud, R.: Myocardial Myoglobin Oxygen Tension. Am. J. Physiol., <u>224</u>: 870-876, 1973.
- Cohen, M.A., Ouzzari, L.J.: Inhalation Products of Combustion. Ann. Emerg. Med., <u>12</u>: 628-632, Oct., 1983.
- Davies, N.J.H.: Does the Lung Work? 4. What does the Transfer of Carbon Monoxide Mean? Br. J. Dis. Chest, <u>76</u>: 2, 105–124, April, 1982.
- De Bias, D.A., Banerjee, C.M., Birkhead, N.C., Breene, C. H., Scott, S.D., Harrer, W.V.: Effects of Carbon Monoxide Inhalation on Ventricular Fibrillation. Arch. Environ. Health, <u>31</u>: 42– 46,1976.
- Dolan, M.C.: Carbon Monoxide Poisoning. Can. Med. Assoc. J., <u>133</u>: 392-399, Sept., 1985.
- Fazekas, J.G., Rengei, B: Ethyl Alcohol and Carbon Monoxide Intoxication, Their Simultaneous Effect on Men and Rats. Orv. Hetil., <u>108</u>: 1503-1506, 1967.
- Fein, A., Grossman, R.F., Jones, J.G., et al.: Carbon Monoxide Effect on Alveolar Epithelial Permeability. Chest, <u>78</u>: 5, 726-732, Nov., 1980.

Finck, P.A.: Exposure to Carbon Monoxide: Review of the Literature and 567 Autopsies. Milit. Med., <u>131</u>: 1513–1539, Dec., 1966.

Forbes, W.H.: Carbon Monoxide Uptake via the Lungs. Ann. N.Y. Acad. Sci., <u>174</u>: 1, 72-75, 1970.

- Friedrich E., et al: Review of CO poisoning in the Years 1967-1976. Beitr. Gerichtl. Med., <u>37</u>: 39-43, 1979.
- Ginsberg, M.D., Myers, R.E.: The Biological Effect of CO on the Pregnant Woman, Fetus and Newborn Infant. Am. J. Obstet. Gynecol., <u>129</u>: 69-103, 1977.
- Gliner, J.A., Horvath, S.M., Mihevic, P.M.: Carbon Monoxide and Human Performance in a Single and Dual Task Methodology. Aviat. Space Environ. Med., 54, 714-717, Aug., 1983.
- Godin, G., Shephard, R.J.: On the Course of Carbon Monoxide Uptake and Release. Respiration, <u>29</u>: 317-329, 1972.
- Goldbaum, L.R.: Is Carboxyhemoglobin Concentration the Indication of Carbon Monoxide Toxicity? Leg. Med. Ann., <u>77</u>: 165-170, 1977.
- Goldbaum. L.R., Orellano, T., Dergal, E.: Studies on the Relation Between Carboxyhemoglobin Concentration and Toxicity. Aviat. Space Environ. Med. <u>48</u>: 10, 969-970, Oct., 1977.
- Gorbatow, O., Noro, L.: On Acclimatization in Connection with Acute Carbon Monoxide Poisonings. Acta. Physiol. Scand., <u>15</u>: 77-87, 1948.
- Gothe, C.M., Frisfedt, B., Sundell, L., et al. Carbon Monoxide Hazard in City Traffic. Arch. Environ. Health, <u>19</u>: 310-314, 1969.
- Groll-Knapp, E., Haider, M., Jenkner, H., Liebich, H., Neuberger, M., Trimmerl, M.: Moderate Carbon Monoxide Exposure During Sleep: Neuro- and Psychophysiological Effects in Young and Elderly People. Neurobehav. Toxicol. Teratol., <u>4</u>: 709-716, 1982.

Haldane, J.S.: The Action of Carbonic Oxide on Man. J. Physiol., <u>18</u>: 430–462, 1895.

Halperin, M.H., McFarland, R.A., Niven, J.I., Roughton, F.J.W.: The Time Course of the Effects of Carbon Monoxide on Visual Thresholds. J. Physiol., <u>146</u>: 583-593, 1959.

Hartzell, O.E., Packharn, S.C., Switza, W.G.: Toxic Products from Fires. Am. Ind. Hyg. Assoc.J., <u>44</u>: 4, 248-255, 1983.

- Hayman, J.A.: Carbon Monoxide Poisoning from a Propane Lamp. Med. J. Aust. 2: 11,505-506, 1982.
- Horvath, S.M., Dalmos, T.E., O'Hanlon, J. F.: Carbon Monoxide and Human Performance. Arch. Environ. Health, <u>23</u>: 343-347, 1971.
- Hughes, E.C.: "Work and the Self", in Social Psychology at the Crossroads. eds. John H. Rohrer and Muzafer Sherif. New York: Harper, 1951.
- Ingenito, A.J., Fiedler, P.C., Procita, L.: Negative Inotropic Action of Carbon Monoxide (CO) on the Isolated Isovolumic Heart with a Hemoglobin (Hb)-Free Perfusate. Fed. Proc., <u>33</u>, 503, 1974.
- Jones, R.D., Commins, B.T., Cernik, A.A.: Blood Lead and Carboxyhemoglobin Levels in London Taxi Drivers. Lancet, 302-303, 1972.

Jones, K.H., Knelson, J.: 1980. Cited in Shephard, J.H., pg 105, 1983.

- Kernohan, J.C.: Kinetics of the Reactions of Two Sheep Hemoglobins with Oxygen and Carbon Monoxide. J. Physiol., <u>155</u>: 580-588, 1961.
- Killick, E.M.: The Acclimatization of the Human Subject to Atmospheres Containing Low Concentrations of Carbon Monoxide. J. Physiol., <u>87</u>: 41-55, 1936.
- La Presle, J., Fardeu, M. The CNS and Carbon Monoxide Poisoning. II Anatomical Study of Brain Lesions Following Intoxication with Carbon Monoxide. In: Carbon Monoxide Poisoning. Progress in Brain Research. Vol. 24, pp. 31–74. Eds. Bour, H. and Ledingham, I., McA. New York: Elsevier, 1967.
- Lasater, S.R. Carbon Monoxide Poisoning. C.M.A.J., <u>134</u>: 991–992, May, 1986.
- Lehr, E.L.: Better Standards for Home Heating Needed. Editorial Summary. Pub. Health Rep., <u>84</u>: 286, March, 1969.
- Lisella, F.S., Johnson, W., Holt, K.: Mortality From Carbon Monoxide In Georgia 1961-1973. J. Med. Assoc. Georgia, <u>67</u>: 98-100, 1978.
- Mant, A.K.: Accidental Carbon Monoxide Poisoning: A Review of 100 Consecutive Cases, Medicoleg.J., <u>28</u>: 30–39, 1960.

- McFee, D.R., Lavine, R.E., Sullivan, R.J.: Carbon Monoxide, A Relevant Hazard Indicated by Detector Tabs. Am. Indust. Hyg. Assoc. J., 31: 749-753, 1970.
- Meigs, J.W., Hughes, J.P.W.: Acute Carbon Monoxide Poisoning An Analysis of One Hundred Five Cases. Arch. Indusr. Hyg., <u>6</u>: 344–356, 1952.
- Mendoza, C.: Saturation Kinetics for Steady State Pulmonary CO Transfer. J. Appl. Physiol., Respirat. Environ. Ex. Physiol., <u>43</u>: 880-884, 1977.
- Michael, L.W.: Compliance with the Carbon Monoxide Standard in the Workplace. Prev. Med., <u>8</u>: 3, 289-294, May, 1979.
- Mitchell, D.S., Packham, S.C., Fitzgerald, W.E.: Effects of Ethanol and Carbon Monoxide on Two Measures of Behavioural Incapacitation in Rats. Proc. West. Pharmacol. Soc., <u>21</u>: 427– 431. 1978.
- Mitchell, R.S., Judson, F.N., Moulding, T.S., Weiser, P., Brock, L.L., Kelble, D.L., Pollard, J.: Health Effects of Urban Air Pollution. Special Considerations of Areas at 1,500 m. and Above. J.A.M.A., <u>242</u>, 11, 1163-1168, 1979.
- Molenda, R.: A Dependence of Acute Toxicity of Carbon Monoxide upon Temperature of Environment and Blood Ethanol Level. Pol. Tyg. Lek., <u>39</u>: 4, 101–105, 1984.
- Montgomery, M.R., Rubin, R.J.: The Effect of CO Inhalation in "In Vivo": Drug Metabolism in the Rat. J. Pharmacol. Exp. Ther., <u>179</u>: 465-473, 1971.
- Mountcastle, V.B., ed.: Medical Physiology, Vol 2. 14th ed. Toronto: C.V. Mosby Co; pp. 1920-1924, 1980.
- Murray, T.J.: Carbon Monoxide in the Modern Society. C.M.A.J., 118: 7, 758-760, April, 1978.
- Nardizzi, L.R.: Computerized Tomographic Correlates of CO Poisoning. Arch. Neurol., <u>36</u>: 1, 38-39, Jan., 1979.
- Nebel, G.G.: Effect of Misfueling on Aldehydes and Other Auto Exhaust Emissions. JAPCA, <u>31</u>: 8, 877-879, 1981.
- Nelson, F.L.: The Certification of Suicide in Eleven Western States: An Inquiry into the Validity of Reported Suicides. Suic. Life Threat. Behav., <u>2</u>: 75- ,1978.

- Ninomiya, J.S.: The Effect of Regulatory and Legislative Requirements on Automotive Emissions and Fuel Economy. J.A.P.C.A., <u>27</u>: 9, 841–842, Sept., 1977.
- O'Hanlon, J.F.: Preliminary Studies of the Effects of Carbon Monoxide on Vigilance in Man. In: Behavioral Toxicology. Eds. Weiss, B., and Laties, V.O. New York: Plenum Press, 1975.
- Orellano, T., Dergal, E., LiJani, M., Briggs, C., Vasquez, J.: Studies on the Mechanism of Carbon Monoxide Toxicity. J. Surg. Res., <u>20</u>: 485-487, 1976.
- Otis, A.B.: The Physiology of Carbon Monoxide Poisoning and Evidence for Acclimatization. In: Biological Effects of CO. Ann. N.Y. Acad. Sci., <u>174</u>: 242–245, 1970.
- Pace, N.: Influence of Age on Carbon Monoxide Desaturation in Man. Fed. Proc., 7: 89, 1948.
- Pach, J., Cholewa, L., Marek, Z., Bogusz, M., Groszek, B.: Analysis of Predictive Factors in Acute Carbon Monoxide Poisoning. Vet. Hum. Toxicol., <u>21</u>: 158-159, 1979.
- Pankow, D., Ponsold, W., Fritz, H.: Combined Effects of Carbon Monoxide and Ethanol on the Activities of Leucine Aminopeptidase and Glutamic-Pyruvic Transaminase in the Plasma of Rats. Arch. Toxikol., <u>32</u>: 331-340, 1974.
- Plevová, J., Frantik, E.: The Influence of Various Saturation Rates on Motor Performance of Rats Exposed to Carbon Monoxide. Act. Nerv. Super., <u>16</u>: 101–102, 1974.
- Purser, D. A., Berrill, K. R.: Effects of Carbon Monoxide on Behaviour in Monkeys in Relation to Human Fire Hazard. Arch. Environ. Health, <u>38</u>: 308-315, 1983.
- Ramsay, J.M.: Effects of Single Exposures of Carbon Monoxide on Sensory and Psychomotor Response. Am. Ind. Hyg. Assoc.J., <u>34</u>: 212–216, 1973.
- Ray, A.M., Rockwell, T.H.: An Exploratory Study of Automobile Driving Performance Under the Influence of Low Levels of Carboxyhemoglobin. Ann. N.Y. Acad. Sci., <u>174</u>: 396-407, 1970.
- Rench, J.D., Savage, E.P.: Carbon Monoxide in the Home Environment: A Study. J. Environ. Health, <u>39</u>: 2,104–106, 1976.
- Rhodes, M.L.: The Effect of Carbon Monoxide on Mitochondrial Respiratory Enzymes in Pulmonary Tissue. Am. Rev. Respir. Dis., <u>103</u>: 906, 1971.
- Rockwell, T.J., Weir, F.W.: The Interactive Effects of Carbon Monoxide and Alcohol on Driving Skills. Ohio State University Research Foundation. (R.F. Project 332), 1–112, 1975.

Rose, E.F., Rose, M.: Carbon Monoxide: A Challenge to the Physician. Clin. Med., <u>78</u>: 12-18, Aug., 1971.

Sackett, D. L.: Bias in Analytical Research. J. Chron. Dis., <u>32</u>: 51-63, 1979.

- Savage, E.P., Malberg, J.W., Wheeler, H.W., Tessari, J.D.: Accidental CO Poisonings in Colorado and Wyoming, 1971-1973. Pub. Health Rep., <u>91</u>: 6, 560-562, 1976.
- Schaplowsky, A.F., Oglesbay, F.B., Morrison, J.H., Gallagher, F.E., Berman, W.: Carbon Monoxide Contamination of the Living Environment – A National Survey of Home Air and Childrens' Blood. J. Environ. Health, <u>36</u>: 569–573, 1974.
- Shephard, R.J.: Carbon Monoxide, The Silent Killer. Charles C. Thomas Springfield, Illinois, 1983.
- Smith, J.S., Brandon, S.: Acute Carbon Monoxide Poisoning 3 Years Experience in a Defined Population. Postgrad. Med. J., <u>46</u>: 65–70, 1970.
- Sokal, J.A.: Lack of Correlation between Biochemical Effects on Rats and Blood Carboxyhemoglobin Concentrations in Various Conditions of Single Acute Exposure to Carbon Monoxide. Arch. Toxicol., <u>34</u>: 331-336, 1975.
- Somogyi, E., Balogh, I., Rubányi, O., Sotonyi, P., Szegedi, L.: New Findings Concerning the Pathogenesis of Acute Carbon Monoxide (CO) Poisoning. Am. J. Forensic Med. Pathol., <u>2</u>: 1, 31–39, March, 1981.
- Sopher, I.M., Masemore, W.C.: The Investigation of Vehicular Carbon Monoxide Fatalities. Traffic Digest and Review, 1-11, Nov., 1970.
- Stewart, R.D: The Effect of Carbon Monoxide on Humans. Ann. Rev. Pharmacol. Toxicol., <u>23</u>: 409-, 1975.
- Stupfel, M., Mordelet-Dambrise, M., Vauzella, A.: Animal Models and Acute Long Term CO Intoxication. Prevent. Med., <u>8</u>: 333-343, 1979.
- Suarez, D.A., Carlson, G.P., Fuller, G.C.: Effect of Carbon Monoxide or Hypoxia on CCl₄ Hepatotoxicity. Toxicol. Appl. Pharmacol., <u>23</u>: 789-791, 1972.

- Wilks, S.S., Tomashefski, J.F., Clark, R.T.: Physiobiological Effects of Chronic Exposure to Carbon Monoxide. J. Appl. Physiol., <u>14</u>: 305–310, 1959.
- Williams, R.L.: Vehicular Carbon Monoxide Screening: Identification in a Cross-Cultural Setting of a Substantial Public Health Risk Factor. A.J.P.H., <u>75</u>: 1, 85-86, 1985.
- Winston, J.M., Creighton, J. M., Roberts, R. J.: Alteration of Carbon Monoxide and Hypoxic Hypoxia–Induced Lethality Following Phenobarbital Chlorpromazine or Alcohol Pretreatment. Toxicol. Applied Pharmacol., <u>30</u>: 458–465, 1974.

World Health Organization. Environmental Health Criteria 13. Carbon Monoxide. Geneva, 1979.

- Wright, G. R., Randell, P., Shephard, R.J.: Carbon Monoxide and Driving Skills. Arch. Environ. Health, <u>27</u>: 349–354, 1973.
- Wyngaarden, J.B., Smith, L.H., Beeson, P.B., McDermott, W., eds.: Cecil Textbook of Medicine. 17th ed. Toronto: W.B. Saunders, pg. 2291, 1985.
- Zenz, C.: The Epidemiology of CO in Cardiovascular Disease in Industrial Environments. A Review. Prev. Med., <u>8</u>: 3, 279-288, May, 1979.

Zimmerman, S.S.: Carbon Monoxide Poisoning. Pediatrics, 68: 2, 215-224, 1981.

APPENDIX A

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CODING SHEET

		120
Variable Code	Variable Code	
card no 🛛	suicide note 🛛 🛛	
person ID 🛛 🗆 🗆 🗆 🗠 🛛 🗠	reason 🛛	
case ID 🛛 🗆 🗆 🗠	body position 🕺 🛛	
area ID 🛛	head position 🛛	
locale 🛛	device(s) 🛛	
year of death 🛛	clothing type 🛛	
month of death 🛛 🗠	camping 🗍 🛛	
day of the month 🛛 🖛	electric door 🛛	
near birthday 🛛	garage door(s) open 🛛	
day of week	garage doors locked 🛛	
time of death 🛛	car doors locked 🛛	
intent 🛛	car door(s) open	
cause of death	window(s) open	
	radio/tape on 🛛	
sex 🛛	heater on	
marital status	key position	
height DDD	battery charged	
weight DD	gas tank	
race D	attempt at privacy	
dwelling type 🛛	source of CO	
how long	fuel	
who lived with	age of source	
where died	maint/condition	
when died	type of death	
who found D	if fire, cause	
when found	B.P. low	
medical examiner	temp Hi DD	
dispute of intent	+/- 0	
autopsy	temp Lo DDD	
card no	+/-	
person ID 0000	history of alcohol 🛛	
laboratory D	history of substance	
СОНЬ 🔲	impairment D	
hemoglobin 000	history of depression	
blood alcohol	hospitalized depression	
drug levels	psychiatrist	
dis/con 🗌	other physician	
	antidepressives 🛛	i -
	recent treatment	
2 000 3 000	recent actions/stmnts	
medications D	prev suicide threats	
1 00	recent suicide threats	
	previous attempts	
2 DD 3 DD	recent attempts	
occupational status 🔲	physician last seen	
time not working	discharge from hosp.	
time at present job	adverse life events	
recent change	1 DD	
reg occupation DDDD		
occupation (CO)	3 00	
occ socioeconomic DDD	4 00	
work-related death		
work-rel impairmnt []		
work-rel distress		

APPENDIX B CODE BOOK

Var. No.	Variable Name	Column	Variable Description	Variable Values
		1	card number	
1	ID	2-4	sequential numbers	1-999
2	CASID	5-8	OCME ID numbers	1-9999
3	ARID	9	four areas of province defined by OCME	1=Calgary 2=south rural 3=Edmonton 4=north rural
4	LOC	10	community	1=city 5x10-5th 2=city <5x10-5th 3=town 4=village 5=reserve 6=out of province 9=U
5	YOD	11	year of death	0-9
6	MOD	12-13	month of death	1-12
7	DOM	14-15	day of month	1-31
B	BDA	16	+/- '3 wks of birthday	1=yes 2=no 9=U
9	DOW	17	day of week	. 1-7
10	TOD	18	time of death	1= 0:01- 6:00 2= 6:01-12:00 3=12:01-18:00 4=18:01-24:00 9=U
11	INT	19	manner of death	1=suicide 2=accidental 3=homicide 4=undetermined 5=unclassified 9=U
12	COD	20	cause of death	1=cmp 2=asphyxia cmp 3= inhalation of exhaust

4=other

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13	AGE	21-22	age	1-99
14	SEX	23	Sex	1=female 2=male 9=U
15	MAR	24	marital status	1=single 2=married 3=separated 4=divorced 5=widowed 6=common law 7=other 9=U
16	HGT	25-27	height (cm)	1-999 -99=U
17	WGT	28-29	weight(kg)	1-99 -9=U
18	RACE	30	race	1=Caucasian 2=Oriental 3=N.A. Indian 4=East Indian 5=Negro 6=other 9=U
19	DWL	31	type of dwelling	1=single family 2=apartment 3=duplex/twnhse 4=room 5=mobilehome/ trailer/van 6=motel/hotel 7=aux hosp/lodge 8=other 9=1)
20	LNG	32	how long there	1= =1 year<br 2=1-2 years 3=>2-5 years 4=>5 years 9=U
21	WHO	33	who lived with	1=lived alone 2=spouse/cl 3=spouse/cl and family 4=family 5=friend(s) 6=other 9=U

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2	22	WER	34-35		1=garage 2=house 3=apartment 4=duplex/twnhse 5=room 6=mobilehome/ trailer/van 7=building-other, (not work) 8=building (work) 9=lane or street 10=hwy or road 11=en route hosp 12=hosp 13=parking lot 14=other outdoor 15=motel-hotel 16=other -9=U
2	23	SMRES	δ	death site same as residence	1=yes 2=no 9=U
2	24	WFND	37	who discovered body	1=family member 2=friend 3=neighbor 4=co-worker 5=police/fireman/ amb attendant 6=passerby 7=landlord 8=other 9=U
2	25	wnfnd ,	38	when body found	1=24 hours or less 2=more than 24 hr 9=U
2	26	ME	39-40	medical examiner	1-99 -9=U
2	27	DIS	41	dispute of intent	1=yes 2=no
2	28	TPSY	42	autopsy performed	9=U 1=yes 2=no 9=U
2	29	NOTE	43	suicide note	1=yes 2=no 9=U
Ţ	30	RES	44	reason for suicide	variable deleted

31 .	BDPO	45 .	body position	1=sitting, front 2=lying, front 3=partly out 4=lying on garage floor near exhaust 5=lying garage floor other 6=in back seat 7=sitting in garage 8=other 9=U
32	HDPO	46	head position	1=drivers side,front 2=passenger side 3=back seat 4=under exhaust 5=lying in trail/camper 6=other 9=U
33	DVC	47	use of devices	1=yes 2=no 9=U
34	CLO	48	clothing type	1=fully outdoor 2=fully indoor 3=partial outdoor 4=partial indoor 5=none 6=nightclothes 7=other 9=U
35	CMP	49	evidence of camping	1=yes 2=no 8=likely no 9=U
36	LECD	50	electric garage door	1=yes 2=no 9=U
37	GRDR	51	garage door(s) open	1=yes 2=no 8=1ikely no 9=U
38	GRLK .	52	garage door(s) locked	1=yes 2=no 8=likely no 9=U
39	CRLK	53	car doors locked	1=yes 2=no 8=1ikely no 9=U

40	CROP	54	car door(s) open	1=yes 2=no 8=1ikely no 9=U
41	WNOP	55	window(s) open	1=yes 2=no 9=U
42	rdon	56	radio/tape on	1=yes 2=no 9=U
43	HTON	57	heater on	1=yes 2=no 9=U
44	күро	58	key position	1=on 2=off 3=intermediate 4=other 9=U
45	BACHG	59	battery charged	1=yes 2=no 9=U
46	GAS	60	gas tank level	1=full 2=empty 3=partly full 9=U
47	PRIV	61	attempt at privacy	1=yes 2=no 9=U
48	SOR	62-63	source of CO ,	1=auto 2=light truck 3=heavy truck 4=camper/van 5=furnace 6=space heater 7=appliance 8=machinery 9=process 10=str'l fire 11=vehc'l fire 12=mattress 13=other fire 14=combination 15=BBQ -9=U

49	FUEL	64	type of fuel	1=gasoline/diesel 2=propane 3=keroscene 4=charcoal 5=wood 6=comb prod. str'l fire 7=comb prod. vehicular fire 8=other 9=U
50	SAGE	65 ,	age of source	1=<6 months 2=>6 months-1yr 3=>1 yr-5 years 4=>5 yrs-10yrs 5=>10 years 6=older model 9=U
51	MAINT	66	maintenance/condition	1=good 2=poor 3=suspected poor 9=U
52	TYPE	67	type of death	1=exhaust garage 2=exhaust outdoor 3=exhaust-bldg. 4=fire-building 5=fire-vehicle 6=fire-other 7=CO-other 8=motor vehicle accident 9=U
53	CAUS	68	if fire, cause	1=careless smoking 2=careless cooking 3=gas leak, faulty furnace 4=MVA/aircraft 5=electrical short 6=children playing 7=arson 8=stove/fireplace 9=other/unknown
54	BPL.	69-71	barometric pressure, low	່ 1-999 -99=ປ
55	THI	72-73	temperature high (Celcius)	1-99 -9=U
56	PM	74	plus or minus degree	1=+ 2=- 9=U

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57	TLO	75-77	temperature low (Celcius)	1-99 -9=U
58	PLMI	76	plus or minus degree	1=+ 2=- 9=U
		t	card two	
	PERSID	2-3	person id	1-999 -9=U
59	LAB	4	laboratory	1=foothills hosp 2=provincial analyst 3=crime detection lab 4=other 9=U
60 61	сонв Нв	5-6 7-8	carboxýhemoglobin (%) hemoglobin	1-99 1-999 -99=U
62	BAC	9-11	blood alcohol (mg./100 ml.)	1=trace 2-600 -99=U
63	DRUG	12	drug levels	1=yes 2=no 9=U
64	DIS	13	existing disease. or condition	1=yes 2=no 9=U
65	DI	14-16	first disease/condition	1=depression 2=other mental problem 3=anxiety 4=insomnia 5=multiple sclerosis 6=Parkinsonism 7=epilepsy 8=blindness (total/partial) 9=heart 10=hypertension 11=atherosclerosis

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12=sinusitis 13=bronchitis 14=lung problem 15=ásthma

16=diabetes 17=ulcers 18=liver 19=haematuria 20=skin 21=arthritis DI

22=back 23=dizziness 24=chest pain 25=abdominal pain 26=weight loss 27=malaise 28=fracture 29=other injury 30=pregnancy 31=cancer/severe dysplasia 32=migraine 33=alcoholism 34=stroke 35=nervous disorder 36=cerebral palsy/dysfn. 37=hypoglycemia 38=scleroderma 39=viral/bacterial infection 40-avascular necrosis 41=ulcerative colitis 42=ear 43=kidney 44=pancreas 45=pituitary 46=thyroid 47=premature dementia 48=mental retardation 49=ammenorhea 50=pulmonary embolism -9=Ù

66	DII	17-19	second disease/condition
67	DIII	20-22	third disease/condition
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68	MEDS	2 <u>3</u>	medications prescribed
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	.	~	<i></i>
69	MI	24-25	first medication

1=anxiolitic 2=antidepressant 3=antipsychotic 4=hypnotic 5=antihypertensive 6=analgesic 7=bronchidilator 8=skeletal muscle relaxant 9=anticonvulsant 10=antihistaminic 11=antigiotic 12=antispasmotic 13=antiparkinsonism 14=antianginal

as DI as DI

1=yes 2=no 9=U

69	MI	24-25	first medication	1=anxiolitic 15=antiarrhythmic 16=hyperthyroidism 17=platelet inhibitor 18=CNS stimulant 19=coronary vasodilator 20=antialcoholic 21=ulcerative colitis therapy 22=histamine H2-antagonist 23=antiemetic 24=anorexiant 25=glucocoricoid 26=antidiarrheal 27=diabetes treatment 28=antihyperglycemia 29=gout therapy 30=vitamin therapy 31=miscellaneous -9=U
70	° MII	26-27	second medication	same as MI
71	mili	28-29	third medication	same as MI
72	OCSTAT	30-32	occupational status	1=fully employed 2=permanent part-time 3=casual 4=unemployed 5=retired 6=student 7=housewife 8=sick leave 9=welfare 10=workers' compensation 11=other -9=11
73	NTWK	33	time not working	1=lwk or less 2=>lwk-1month 3=>month-6mos 4=>6months-1yr 5=>1yr-2yrs 6=>2yrs-5yrs 7=>5yrs 9=U
74	TPREJB	34	time at present job	1=1wk or less 2=>1wk-1month 3=>month-6mos 4=>6months-1yr 5=>1yr-2yrs 6=>2yrs-5yrs 7=>5yrs 9=U

75	CHNG	35	change in job in last yr	1=yes 2=no 9=U
76	000	36-39	regular occupation	-999=U
. 77	OCCCO .	40	carbon monoxide related	1=transportation 2=automotive repairs 3=other machinery 4=welding, plumbing, fireman 5=oil rig, miner, metalwork 6=possible 7=laborer, maintenance 8=no 9=U
78	OCSOEC	43-45	Blishen socioeconomic scale	1-8 -9=U
79	WKDTH	46	relationship of work to death	1=not at work 2=during working hours 3=associated with work, but not during regular working hours 4=not associated with regular work, but during working hours 5=other 6=not associated with reg. work, not during working hours 9=U
80	WKIMP	47	work related impairment	1=yes 2=no 9=U
81	WKDIS	48	distress from work	1=previous health problem(s) affecting ability to work 2=job loss (within year) 3=pressures from job 4=job dis-satisfaction 5="problems" at work 6=bankrupcy or financial problems at work 7=other 8=no problems mentioned

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8=no problems mentioned 9=U

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82	ЕТОН	49	history of alcohol use	1=known alcoholic 2="problem" drinker 3=social drinker 4=did not drink 5="closet" drinker 6=not mentioned 9=U
83	SUBST	50	history of substance abuse	1=yes 2=no 9=U
84	IMP	51	physical impairment	1=yes 2=no 9=U
85	DEP	52	history of depression	1=yes 2=no 9=U
86	HOSP	53	ever hospitalized for depression	1=yes 2=no 9=U
87	PSYCH	54	ever treated by psychiatrist for depression	1=yes 2=no 9=U
88	PHYS	55	ever treated by physician for depression	1=yes 2=no 9=U
89	ANTI	56	ever prescribed anti-depressants	1=yes 2=no 9=U
90	RETRE	57	recent treatment for depression	1=yes 2=no 9=U
91	RECST	58	recent statements or actions of depression	1=yes 2=no 9=U
92	PRETH	59	previous suicide threats	1=yes 2=no 9=U
93	RETH	60	recent (6 mos) suicide threats	1=yes 2=no 9=U

94	PRETM	61	previous suicide attempts	1=yes 2=no 9=U
95	RETM	62	recent (6mos) suicide attempts	1=yes 2=no 9=U
96	SALST	63	when last saw physician	1=1-2 days 2=>2 days to 1 wk. 3=>1 wk. to 1 mo. 4=>1 mo. to 6 mos. 5=>6 mos. to 1 year 6=>1 year 9=U
97	DISCH	64	when last discharged from hospital	1=1-2 days 2=>2 days to 1 wk. 3=>1 wk. to 1 mo. 4=>1 mo. to 6 mos. 5=>6 mos. to 1 year 6=>1 year 9=U
98	LIVENT	65	any adverse life event(s)	1=yes 2=no 9=U
99	ONE	66-67	main life event.	1=girl/boy friend problem 2=marital argument 3=marital sep'n. 4=divorce 5=financial problem 6=occupational 7=health problem 8=illness/death of other 9=legal problem 10=social maladjustment 11=other social problem 12=other family problem 13=alcohol problem 14=other problem -9=U
100	TWO	68-69	second life event	coded as ONE
101	THREE	70-71	third life event	coded as ONE
102	FOR	72-73	fourth life event	coded as ONE

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