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HEART DISEASE MORTALITY AMONG BRIDGE AND TUNNEL OFFICERS EXPOSED TO CARBON MONOXIDE[1]

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The authors investigated the effect of occupational exposure to carbon monoxide on mortality from heart disease in a retrospective study of 5,529 New York City bridge and tunnel officers employed between January 1, 1952 and February 10, 1981, at any one of nine major water crossings operated by the Triborough Bridge and Tunnel Authority of New York City. Among former tunnel officers, 61 deaths from arteriosclerotic heart disease were observed, as compared with 45 expected (standardized mortality ratio = 1.35, 90% confidence interval 1.09–1.68); expected rates were based on the New York City population. Using a proportional hazards model, the authors compared the risk of mortality from arteriosclerotic heart disease among tunnel officers with that of the less-exposed bridge officers. No association of arteriosclerotic heart disease with length of exposure was observed, but there was significant interaction of exposure with age. The elevated risk of arteriosclerotic heart disease among tunnel officers, as compared with that of bridge officers, declined after cessation of exposure, with much of the risk dissipating within as little as five years. The parallel findings of this study of occupational exposure to carbon monoxide and those studies showing the relation of cigarette smoking to cardiovascular mortality suggest that carbon monoxide may play an important role in the pathophysiology of cardiovascular mortality associated with cigarette smoking.

carbon monoxide; heart diseases; mortality

Carbon monoxide may contribute to cardiovascular disease through several accepted and potential mechanisms (1, 2), including binding to hemoglobin (3, 4) and reducing oxygen dissociation at the tissue level by shifting the oxygen-hemoglobin dissociation curve (5). Carbon monoxide in experimental animals also increases platelet stickiness (6), which may contribute to atherogenesis, and accelerates atherosclerosis either through altered lipid metabolism (7) or through increasing vessel permeability to cholesterol (7–9). In experimental studies, carbon monoxide has been shown to contribute to sudden death by reducing the cardiac threshold for ventricular fibrillation (10). Some of these mechanisms, such as those that would lead to transient hypoxia of the myocardium, would lead to increased risk of myocardial ischemia only

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when exposure is current, while other mechanisms, e.g., acceleration of atherogenesis, may lead to long-standing elevation of risk for cardiovascular disease.

The current Occupational Safety and Health Administration standard for carbon monoxide for the protection of workers allows exposure of 50 parts of carbon monoxide per million parts of air as a time-weighted average over an eight-hour work day (11). The margin of safety of this standard for workers with underlying cardiovascular disease is questioned by a series of studies of exposures averaging 50 parts per million (ppm) which have demonstrated a decrease in exercise necessary to induce angina in experimental subjects with underlying cardiovascular disease (12–14). The National Institute for Occupational Safety and Health recommends a standard for carbon monoxide exposure of 35 ppm as an eight-hour time-weighted average, with a ceiling limit of 200 ppm (15).

To assess the long-term effects of permissible levels of exposure to carbon monoxide on cardiovascular mortality in a working population, we studied a cohort of traffic control officers employed on bridges and in tunnels who were exposed to carbon monoxide as a constituent of vehicle exhaust. We show that, parallel to cigarette smoking, which is associated with cardiovascular mortality (16–23) and which causes a rise in carboxyhemoglobin on the same order of magnitude as permissible occupational carbon monoxide exposure (2, 24–26), occupational exposure to vehicular exhaust containing carbon monoxide also results in a significant excess of mortality from cardiovascular disease.

MATERIALS AND METHODS

Population identification

The study population consisted of all male bridge and tunnel officers employed between January 1, 1952, and February 10, 1981, at one of nine major water crossings (two tunnels and seven bridges) operated by the Triborough Bridge and Tunnel Authority of New York City. Officers were hired on the basis of the successful completion of a preemployment medical examination and training period and were then assigned to a particular facility dependent upon the needs of the Triborough Bridge and Tunnel Authority. Data available from personnel records of the Triborough Bridge and Tunnel Authority contained name, Social Security number, sex, date of birth, date of hire, date of separation, and specific work history information identifying the bridge(s) or tunnel(s) at which the officers had worked. We obtained information on race from the Social Security Administration. Bridge and tunnel officers who left employment prior to 1952 were excluded from the cohort since their Social Security numbers generally were not available from employment records, making vital status follow-up very difficult. The primary duties of the bridge and tunnel officers included toll collections from booths, traffic observation within and outside the tunnels, and direction of traffic within the tunnels and on the bridges when necessary (i.e., during rush hours or motor vehicle accidents).

Historical exposures

Continuous (tape) monitoring of carbon monoxide levels within the tunnels operated by the Triborough Bridge and Tunnel Authority began in 1940 at the Queens Midtown Tunnel and in 1950 at the Brooklyn-Battery Tunnel, and measurements were first summarized in an internal memorandum in 1959 as showing peak concentrations exceeding 400 ppm (27). In 1961, at the request of the Triborough Bridge and Tunnel Authority, an investigation of the ventilation systems demonstrated 24-hour average carbon monoxide levels inside the tunnels of 53 ppm in the summer (with peaks of 200–300 ppm) and 49 ppm in the winter (with peaks of 100–200 ppm) (28). In 1968, 24-hour average carbon monoxide concentrations measured inside the tunnels were 35–40 ppm (29); carbon monoxide exposures measured during rush hour traffic were found to range from 120–165 ppm in the morning and 65–

145 ppm in the evening in the tunnel toll booths, and 15–45 ppm in the morning and 12–22 ppm in the evening in the bridge toll booths (30). In 1970, monitoring over 38 continuous days revealed mean carbon monoxide levels of 63 ppm in the tunnel toll booths and 13 ppm in the bridge toll booths (31). During the same year, fresh-air ventilation systems were installed in all toll booths. In 1971, an increase in electrical service to the ventilation fans in the tunnels yielded an increase of approximately 15 per cent in tunnel ventilation capacity. Also, starting in 1971, officers were allowed one half-hour "air break" for each day's work, which consisted of two two-hour tours inside the tunnel. In 1977, ventilation equipment for the tunnels was linked electrically to continuously reading carbon monoxide monitors. In 1981, daily sampling conducted over two weeks by National Institute for Occupational Safety and Health industrial hygienists using direct-reading portable Ecolyzers for area exposures and Draeger direct-reading long duration detector tubes for personal and area exposures found mean area levels of carbon monoxide of 38.3 ppm inside the tunnels and 23.0 ppm outside the bridge toll booths, and personal samples of 10.8 ppm collected on tunnel officers and 6.2 ppm collected on bridge officers (32). Peak carbon monoxide levels measured in the traffic lanes of both the tunnels and the bridges and on the tunnel catwalks were frequently greater than 100 ppm and occasionally greater than 400 ppm. Exposures to other contaminants were also measured in 1981 and were found to be well below the respective permissible or recommended exposure levels. Personal samples for tunnel and bridge were 0.3 and 0.1 ppm for nitrogen dioxide, 0.07 and 0.02 mg/m^3 for polycyclic aromatic hydrocarbons, 0.005 and 0.004 mg/m^3 for lead, and 0.06 and 0.02 fibers/cc for asbestos (32).

Smoking histories were collected during medical evaluations of active officers from 1972 to 1981 (33) and by National Institute for Occupational Safety and Health investigators in 1981 (32). Pipe and cigar smokers were classified as nonsmokers since they experience only a small increase in mortality from coronary heart disease above that of nonsmokers (22). Carboxyhemoglobin levels measured in 1970 (before ventilation systems were installed in the toll booths) averaged 2.12 and 3.90 per cent in nonsmokers and smokers, respectively, for bridge officers, and 2.93 and 5.01 per cent in nonsmokers and smokers, respectively, for tunnel officers (33). Post-shift carboxyhemoglobin levels measured by the National Institute for Occupational Safety and Health in 1981 were not found to be significantly different between bridge (4.9 per cent carboxyhemoglobin) and tunnel officers (4.5 per cent carboxyhemoglobin), with pre- and post-shift carboxyhemoglobin levels rising about 20 per cent in nonsmokers and 10 per cent in smokers (32).

Ascertainment of vital status

Vital status of each officer was ascertained as of December 31, 1982, through the sources of the Social Security Administration, the Internal Revenue Service, and the National Death Index, among others. For deceased officers, death certificates were obtained from the appropriate state vital statistics offices. The underlying and contributory causes of death were coded by a qualified nosologist according to the rules of the revision of the *International Classification of Diseases* in effect at the time of death.

Statistical analyses

We initially analyzed the bridge and tunnel officer cohort by means of a modified life table analysis system (34) which computed expected number of deaths, using indirect adjustment, by multiplying the person-years at risk of dying among the officers by the corresponding age-, sex-, calendar time-, and cause-specific mortality rates of the New York City population. Death rates for New York City were obtained for the years 1950–1984. Accumula-

tion of person-years at risk and observed deaths began with the first day of employment at the Triborough Bridge and Tunnel Authority on or after January 1, 1952, and continued until either December 31, 1982, or the date of death of the officer, whichever occurred earlier. The relative risk estimates for cause-specific mortality among the cohort were calculated as standardized mortality ratios which were derived by dividing the number of observed deaths by the number expected. One-sided significance tests and 90 per cent confidence intervals were computed for the standardized mortality ratios assuming a Poisson distribution for the observed deaths (35). Since the a priori hypothesis was a positive association between vehicular exhaust, namely carbon monoxide, and cardiovascular mortality, standardized mortality ratios were considered to be statistically significant if the p values were less than 0.05 or, equivalently, if the lower confidence limit was greater than 1.00 (36). We examined the mortality experience of those officers employed only in tunnels and of those employed only on bridges separately, because previous environmental sampling had indicated that carbon monoxide levels had been substantially higher within and around the tunnels than on the bridges (see above). Officers who transferred permanently ($n = 472$) between the bridges and the tunnels were excluded from the cohort because their numbers were small and the results of their mixed exposures would be difficult to analyze. Because environmental sampling results for carbon monoxide were only available for a few years of the study, duration of employment was used as a surrogate for cumulative exposures. Two categories of less than 10 years and greater than or equal to 10 years employment were used in an attempt to ascertain the adverse effects from cumulative long-term vehicular exhaust exposures. For cancers only, an additional analysis by latency (time period from initial employment to death) was performed. In order to make *direct* comparisons of the mortality experience of bridge officers as compared with tunnel officers while controlling potential confounders and effect modifiers, we used the Cox proportional hazards model (37) with time-dependent covariables to analyze the mortality experience of the cohort. This analysis uses internal comparisons and therefore did not require the use of mortality rates from New York City. A recent modification of the BMDP2L survival analysis procedure permitted us to use general forms of the Cox model (38). This method produces relative risk estimates based upon incidence rates as a function of covariables such as age, exposure status, calendar time, and duration of employment which may change over the period of study.

RESULTS

Population identification

There were 4,317 bridge officers and 1,212 tunnel officers employed between January 1, 1952, and February 10, 1981, by the Triborough Bridge and Tunnel Authority (table 1). The cohort contributed a total of 103,900 person-years at risk. As of December 31, 1982, 88 per cent of the cohort were alive, 9 per cent were deceased, and 3 per cent were lost to follow-up. Death certificates were obtained for 97 per cent (460 out of 474) of all known deaths. The percentage of tunnel officers who died (13 per cent) was almost twice that of bridge officers (7 per cent). On average, the bridge officers and tunnel officers were very similar in racial composition and calendar year of birth (table 2). In addition, the bridge officers and tunnel officers started employment with the Triborough Bridge and Tunnel Authority and died at approximately the same ages and calendar time periods. However, on average, the tunnel officers had worked for five years at the Triborough Bridge and Tunnel Authority, while the bridge officers had worked there for only three years.

TABLE 1

Vital status of male bridge and tunnel officers employed by the Triborough Bridge and Tunnel Authority, New York City*

Vital status	No. of officers (%)		
	Bridge officers	Tunnel officers	Total
Alive	3,872 (90)	1,014 (84)	4,886 (88)
Deceased	314 (7)	160 (13)	474 (9)
Death certificates obtained	303	157	460
Death certificates outstanding	11	3	14
Lost to follow-up	131 (3)	38 (3)	169 (3)
Total	4,317 (100)	1,212 (100)	5,529 (100)
Person-years at risk	79,865	24,035	103,900

* As of December 31, 1982.

TABLE 2

Demographic characteristics of male bridge and tunnel officers, Triborough Bridge and Tunnel Authority, January 1, 1952–February 10, 1981, New York City

Demographic characteristic	Bridge officers				Tunnel officers			
No.	4,317				1,212			
No. deceased	314				160			
% deceased	7.3				13.2			
% white	83.1				80.1			
Year of birth	1936*	12†	1936‡	1893–1962§	1932	12	1930	1894–1961
Year first employed	1963	10	1963	1936–1981	1961	11	1957	1936–1980
Year last employed	1966	11	1967	1952–1981	1966	11	1966	1951–1981
Age (years) first employed	26	8	25	18–70	29	7	28	17–59
Duration of employment (months)	35	75	7	1–459	61	99	12	1–482
Age (years) deceased	49	11	50	19–81	51	10	52	23–79
Year deceased	1974	6	1976	1953–1982	1974	7	1975	1953–1982

* Mean.
† Standard deviation.
‡ Median.
§ Range.

Standardized mortality ratio analyses

In the 31-year period between January 1, 1952, and December 31, 1982, the overall mortality among bridge officers was less than expected—314 deaths observed versus 409 expected (standardized mortality ratio (SMR) = 0.76)—when compared with the mortality experience of the New York City population (table 3). The overall mortality among tunnel officers was approximately equal to that expected—160 deaths observed compared with 153 expected (SMR = 1.04). Heart disease mortality among tunnel officers was the only cause of death that was statistically significantly elevated among the entire bridge and tunnel officer cohort. There were 67 deaths among tunnel officers from heart disease (*International Classification of Diseases* (ICD), Sixth and Seventh Revisions, codes 400–468, and Eighth and Ninth Revisions, codes 390–458, excluding codes 430–438) compared with 54 expected, an excess of 24 per cent (table 3). This excess was even more pronounced among tunnel officers for deaths due to arteriosclerotic heart disease (ICD-6 and ICD-7, code 420, and ICD-8 and ICD-9, codes 410–414)—61 deaths observed compared with 45 expected, a 35 per cent increase. The mortality from arteriosclerotic heart disease increased to 88 per cent over that expected among tunnel officers

TABLE 3

Mortality (1952–1982), by duration of employment, among male bridge and tunnel officers, Triborough Bridge and Tunnel Authority, New York City *

Cause of death	Duration (years) of employment											
	<10				≥10				Total			
	Obs	Exp	SMR	90% CI	Obs	Exp	SMR	90% CI	Obs	Exp	SMR	90% CI
Bridge officers												
All heart disease	78	96	0.82	0.67–0.99	30	33	0.91	0.65–1.23	108	129	0.84	0.71–0.99
ASHD	66	76	0.87	0.70–1.07	23	28	0.81	0.56–1.15	89	104	0.85	0.71–1.02
Lung cancer	13	16	0.83	0.49–1.32	5	6	0.91	0.36–1.91	18	21	0.85	0.55–1.26
All other causes	154	223	0.69	0.60–0.79	34	37	0.93	0.69–1.24	188	259	0.73	0.64–0.82
All causes	245	334	0.73	0.66–0.82	69	75	0.92	0.75–1.12	314	409	0.76	0.70–0.84
Tunnel officers												
All heart disease	35	36	0.98	0.72–1.30	32	19	1.72‡	1.25–2.31	67	54	1.24†	1.01–1.51
ASHD	31	29	1.07	0.77–1.44	30	16	1.88‡	1.36–2.56	61	45	1.35†	1.09–1.68
Lung cancer	5	6	0.83	0.33–1.75	4	3	1.29	0.44–2.95	9	9	0.97	0.52–1.73
All other causes	69	68	1.01	0.82–1.23	15	21	0.72	0.44–1.11	84	89	0.94	0.78–1.13
All causes	109	110	0.99	0.84–1.16	51	43	1.20	0.94–1.52	160	153	1.04	0.92–1.20

* ASHD, arteriosclerotic heart disease; Obs, observed number of deaths; Exp, expected number of deaths; SMR, standardized mortality ratio (Obs/Exp); CI, confidence interval. Expected number of deaths are based on the death rates for New York City rounded to the nearest whole number.

† Significantly different from 1.00 ($p < 0.05$).

‡ Significantly different from 1.00 ($p < 0.01$).

employed for more than 10 years by the Triborough Bridge and Tunnel Authority (30 deaths observed compared with 16 expected).

The risk of arteriosclerotic heart disease mortality by age at death among tunnel officers and bridge officers is shown in table 4. Among tunnel officers, there were no deaths attributable to arteriosclerotic heart disease prior to age 40 years. After age 60, the relative risk of arteriosclerotic heart disease was found to be 1.0. However, during the working years, among tunnel officers, the relative risk of arteriosclerotic heart disease increased from 32 per cent over that expected for the age group 40–49 years to 74 per cent over that expected for the age group 50–59 years. No elevation in risk of mortality by age was observed among bridge officers, with the exception of those who had died prior to age 40. All tunnel officers who died from arteriosclerotic heart disease had started employment with the Triborough Bridge and Tunnel Authority prior to 1960 (mean year, 1951), and all were employed during the 1950s and 1960s, when exposures to vehicle exhaust, as represented by carbon monoxide, were considerably higher than in the 1970s (see Materials and Methods). It is acknowledged, however, that persons first employed after 1960 would have little risk of heart disease mortality by 1982 because of the young ages of this working group.

To help evaluate whether the excess risk from heart disease could have been related to smoking, we examined the other main smoking-related cause of death, lung cancer. Mortality from cancer of the lung was lower than expected among both bridge officers (18 deaths observed compared with 21 expected; SMR = 0.85) and tunnel officers (nine deaths observed compared with nine expected; SMR = 0.97) (table 3). The power of this study to detect a twofold increase in lung cancer for the cohort was at least 99 per cent, and among tunnel officers, it was 80 per cent, assuming an

TABLE 4

Mortality (1952–1982) from arteriosclerotic heart disease, by age at death, among male bridge and tunnel officers, Triborough Bridge and Tunnel Authority, New York City *

Age (years)	Bridge officers				Tunnel officers			
	Obs	Exp	SMR	90% CI	Obs	Exp	SMR	90% CI
<40	12	6	1.79†	1.15–3.24	0	2		
40–49	28	29	0.97	0.69–1.32	15	11	1.32	0.84–2.10
50–59	36	38	0.96	0.70–1.25	34	20	1.74†	1.25–2.26
60+	13	31	0.42	0.25–0.67	12	12	1.00	0.58–1.62
All ages	89	104	0.85	0.71–1.02	61	45	1.35†	1.09–1.68

* Obs, observed number of deaths; Exp, expected number of deaths; SMR, standardized mortality ratio (Obs/Exp); CI, confidence interval. Expected number of deaths are based on the death rates for New York City rounded to the nearest whole number.

† Significantly different from 1.00 ($p < 0.05$).

alpha level of 0.05 and a one-sided test. No substantial elevation in mortality from lung cancer was evident for either subgroup with increasing duration of employment or with increasing time since first employment. There was also a deficit of deaths from all other causes among both bridge officers and tunnel officers (table 3).

Proportional hazards model

Because of the significant excess risk from arteriosclerotic heart disease among tunnel officers observed in the standardized mortality ratio analysis, the Cox proportional hazards model was used to examine this finding in greater detail. Potential confounding and effect modification were examined by including duration of employment, age, and calendar time and their interactions in the models.

Similar to the effect suggested by the standardized mortality ratio analysis, a direct comparison of job location (bridge vs. tunnel) showed significant elevation in risk from arteriosclerotic heart disease for tunnel officers when compared with bridge officers, adjusted for duration of employment, age, and calendar time (relative risk = 1.54, $p = 0.01$). To investigate the potential effect of chronic exposure to vehicular exhaust, we modeled trends in arteriosclerotic heart disease mortality with number of years employed. In contrast to the results of the standardized mortality ratio analysis, no significant trend was found with duration of employment in any of the models used. However, when the interaction of job location with age was examined, the result was found to be statistically significant ($p = 0.015$). Specifically, the risk of arteriosclerotic heart disease in tunnel officers increased approximately 5.5 per cent per year of age relative to bridge officers, starting at age 45 when the risk for the two groups was essentially the same.

Cognizant of the important finding of Rosenberg et al. (39) and others (16–23, 40) that the substantial excess risk from heart disease initially increases after cessation of cigarette smoking and then begins to decrease rather quickly thereafter, we chose to test a similar hypothesis in this study. Tunnel officers were directly compared with bridge officers with regard to their arteriosclerotic heart disease mortality experience after cessation of exposure, defined as that period of time following the date last employed. The time following cessation of employment was stratified into four intervals: zero to one month, two to 23 months, two to four years, and five or more years. The category zero to one month was chosen to distinguish the officers who died either on the job or shortly thereafter from individuals who died after cessation of employment and, hence, after exposure to the workplace environment. A direct comparison of tunnel officers with bridge officers in each time interval at each of three ages is presented in table 5. The risk of arterio-

TABLE 5

Relative risk estimates (90% confidence interval) of arteriosclerotic heart disease mortality, by time since last date of employment and by age at death, among male tunnel officers relative to male bridge officers, Triborough Bridge and Tunnel Authority, New York City*

Age (years)	Time since last date of employment			
	0–1 month	2–23 months	2–4 years	≥5 years
45	0.98	1.39	2.11	0.94
	(0.51–1.89)	(0.53–3.63)	(0.68–6.58)	(0.59–1.50)
55	1.59	2.25	3.41†	1.51
	(0.85–2.95)	(0.88–5.76)	(1.11–10.43)	(0.99–2.30)
65	2.57†	3.63†	5.53‡	2.45†
	(1.18–5.58)	(1.27–10.34)	(1.65–18.56)	(1.31–4.58)

* Estimated using the Cox proportional hazards model.

† Significantly different from 1.00 ($p < 0.05$).

‡ Significantly different from 1.00 ($p < 0.01$).

sclerotic heart disease among tunnel officers relative to bridge officers increases with age. However, after separation from employment for as little as five years, the elevated risk of arteriosclerotic heart disease mortality among tunnel officers declines and begins to approach that of bridge officers for each of the ages, albeit because the numbers in each strata are small, the confidence intervals are large and overlapping. These comparisons were intended to remove any unknown risk factors associated with time since separation from employment, including the possibility that cardiovascular morbidity may be a precipitant of termination. All results were adjusted for calendar time and duration of employment.

To investigate the effect of lowering of exposure which began after 1970 with the addition of fresh air ventilation in all tunnel booths along with an increase in tunnel ventilation, we modeled trends in arteriosclerotic heart disease mortality after that time. A significant decrease in relative risk of arteriosclerotic heart disease mortality was found ($p = 0.042$). Specifically, the risk of arteriosclerotic heart disease in tunnel officers was modeled as an exponential decline of 6.4 per cent per year after 1970 when compared with the less-exposed bridge officers. Figure 1 illustrates this decline in relative risk after 1970 for tunnel officers at 45, 55, and 65 years of age.

DISCUSSION

The present results suggest that exposure to carbon monoxide may be an important factor in arteriosclerotic heart disease mortality. In our study, tunnel officers employed by the Triborough Bridge and Tunnel Authority experienced a 35 per cent excess risk of arteriosclerotic heart disease mortality compared with the New York City population. The data suggest that two factors contributed to produce the elevated risk of heart disease: the higher levels of exposure to carbon monoxide experienced by the tunnel officers, and the movement into a critical higher age group. Both factors apparently act together to produce a significantly elevated risk of arteriosclerotic heart disease mortality.

In the attempt to understand the etiology of arteriosclerotic heart disease in this cohort, another finding is of particular interest. The excess risk of arteriosclerotic heart disease mortality in tunnel officers when compared with the less-exposed bridge officers dropped in all age groups after cessation of exposure when individuals left employment in the tunnels. In addition, a similar reduction in risk was found after 1970, when substantially more ventilation was introduced, thereby reducing exposures. This effect suggests that, regardless of age, the adverse health effects experienced by the tunnel officers of the Triborough Bridge and Tunnel Authority are, in

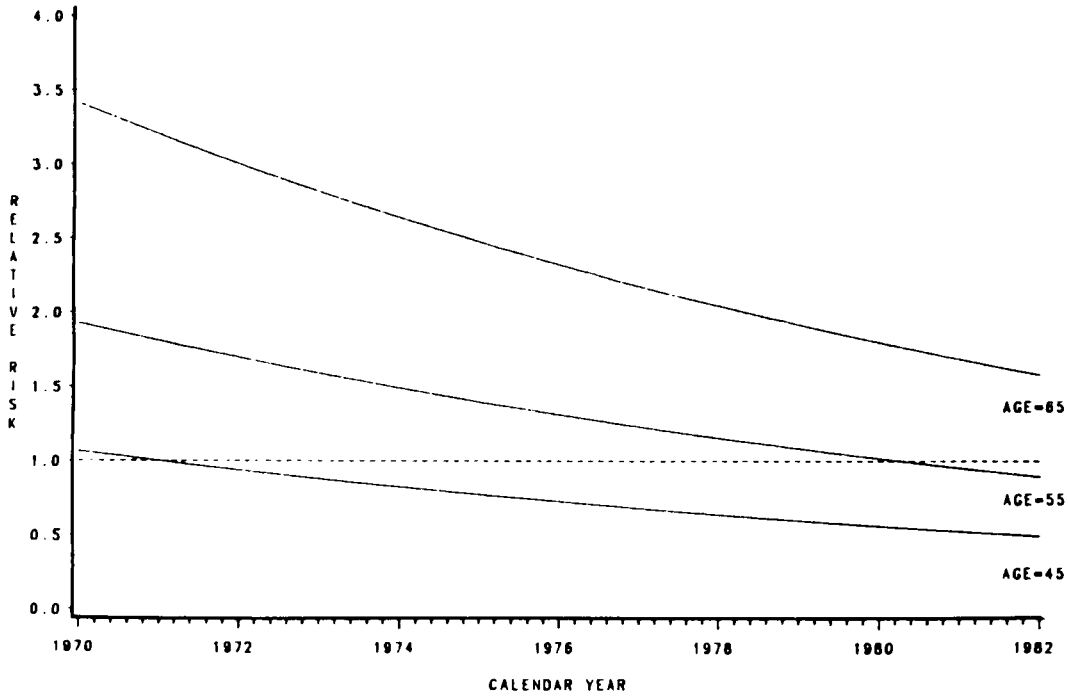

FIGURE 1. Risk of arteriosclerotic heart disease mortality in tunnel officers relative to bridge officers, as a function of age and calendar year, after introduction of ventilation in 1970.

large part, reversible. Similar results have been observed among cigarette smokers in whom the excess risk of heart disease morbidity and mortality begins to decrease after cessation of smoking, with much of the risk dissipating within two to 10 years (16–23, 39, 40).

Carbon monoxide may contribute to heart disease mortality either through the effects of current exposure or as the result of the accumulation of exposure over a sustained period. Length of exposure was not a risk factor for heart disease mortality in this study, suggesting that the acute effects of exposure to carbon monoxide are more important than the chronic effects. These results are consistent with studies of the effect of cigarette smoking on heart disease mortality which also demonstrate that it is the number of cigarettes smoked, not the length of years of cigarette smoking, that is associated with the excess risk of heart disease mortality (41, 42).

Prior to 1970, it had been documented that carbon monoxide levels in the tunnels averaged over 50 ppm, the current Occupational Safety and Health Administration standard, with excursions frequently exceeding 400 ppm. These excursions were due to the congestion of cars within the tunnels caused by motor vehicle accidents and rush-hour traffic and to the limitations of the ventilation systems. This level of exposure to carbon monoxide for tunnel officers would result in a range of carboxyhemoglobin that is close to the carboxyhemoglobin level observed in cigarette smokers (2, 24–26), who are also exposed to an average carbon monoxide level estimated at 50 ppm (43), with excursions up to 475 ppm (44), and who are also known to experience a greater risk of heart disease (16–23).

Mortality from arteriosclerotic heart disease has a complex multifactor etiology. The presence of other risk factors in addition to occupational carbon monoxide exposures, including cigarette smoking, hy-

pertension, hyperlipidemia, family history of heart disease, marked obesity, socioeconomic status, and sedentary living, increases the risk of developing arteriosclerotic heart disease (45–47). Since this study was conducted using employment records, information concerning many of these risk factors for the bridge and tunnel officers was limited. However, inferences about some of these risk factors can be made from the results of this study and from other information.

Cigarette smoking, uncontrolled in the analysis, could explain some of the excess risk for arteriosclerotic heart disease if the officers' smoking habits were substantially different from those of the comparison population (48). Although the tobacco smoking habits of the officers had not been recorded at the time of employment, information on smoking status of many active members of this cohort had been collected in two separate studies (32, 33). Among bridge officers, the percentages of current smokers, ex-smokers, and nonsmokers were 43.1, 28.6, and 28.3, respectively. Among tunnel officers, the percentages were almost identical—43.7, 28.6, and 27.7, respectively. These percentages resemble those among white males in the general population during the 1970s (49)—41.4 per cent, 29.9 per cent, and 28.7 per cent, respectively (50). Apparently, if this cross-sectional view of the smoking habits obtained is reasonably representative of the cohort as a whole, then the smoking habits among the bridge officers and tunnel officers were not substantially different from each other or from the general population. Thus, smoking probably cannot explain the substantially increased risk of arteriosclerotic heart disease mortality seen among tunnel officers of the Triborough Bridge and Tunnel Authority. Furthermore, if smoking in this cohort had been higher than that in the general population, a significant excess of lung cancer mortality among bridge officers and tunnel workers would also have been expected, since smoking is nearly seven times more strongly associated with lung cancer than with arteriosclerotic heart disease (17, 22). No increase in lung cancer mortality, however, was observed among either subcohort. Also, no increase in deaths from emphysema was found. There were no deaths attributable to hypertensive heart disease among tunnel officers (compared with 1.6 deaths expected), and the mortality risk from stroke was lower than that expected on the basis of rates in the New York City population (SMR = 0.92). Since hypertension and cigarette smoking are the major risk factors for stroke (51), the absence of elevated mortality from this disease suggests that neither hypertension nor cigarette smoking substantially contributed to the excess mortality due to arteriosclerotic heart disease.

Selection bias is also unlikely to explain these results. Self-selection into jobs by individuals with increased risk from arteriosclerotic heart disease was observed in studies of London bus drivers and fare collectors (52). However, the Triborough Bridge and Tunnel Authority employment practices were such that a recruit would not know his job location until after finishing the preemployment physical examination and training. There was no difference in the qualifications for bridge officers and tunnel officers. Thus, the requirements of the Triborough Bridge and Tunnel Authority rather than the officers' preferences determined their worksite selection. Those requirements would have been determined by the turnover of employees, which was similar in both bridge and tunnel officers. However, to limit the possibility of selective migration between bridges and tunnels for officers with an increased risk of arteriosclerotic heart disease, we eliminated from the study those officers whose personnel records indicated transfer between job locations. In addition, the duties of the bridge officers, mainly toll collection, are more sedentary than the duties of the tunnel officers, who must also man the observation booths within the tunnels. Thus, if prefer-

ence of the officer was a major factor in initial or future worksite selection, those less fit because of obesity or other factors would most likely be attracted to assignments on the bridges rather than in the tunnels.

Finally, bridge officers and tunnel officers are drawn from the same applicant pool and hence the same socioeconomic class, which makes it less likely that there would be great differences between the two groups with regard to factors such as race, dietary habits, alcohol consumption, and physical activity during leisure time.

In the absence of data to implicate other causes for the increased mortality risk from arteriosclerotic heart disease among tunnel officers, the available evidence suggests that the factor was directly associated with employment. The most likely factor would be exposure to carbon monoxide. However, the possibility should be considered that one of the other constituents of vehicular exhaust (e.g., nitric oxides, hydrocarbons, particulates, lead, sulfur, or aldehydes) or some other characteristic of the worker environment (e.g., noise or stress) could have played a role in aggravating cardiovascular disease, although the implication of these other factors and their effect upon the heart is highly speculative (53–55).

This study had some potential strengths and limitations that should be considered. First, the choice of the comparison population used may have underestimated the true risk of mortality in this study. The New York City population, while more appropriate than the US population as a basis for calculating expected deaths, includes, like most generalized populations, numbers of chronically ill or otherwise unemployable persons. Since most populations of workers are healthier than the general population, comparison of the mortality experience of bridge officers and tunnel officers to that of a general population may produce an apparent deficit in mortality, as seen among bridge officers—the so-called "healthy worker effect" (56). However, the bridge officers were considered to be ideal for use as an internal comparison group in the proportional hazards model. Bridge officers are drawn from the same applicant pool as tunnel officers, undergo the same preemployment selection process, have comparable duties, and belong to the same socioeconomic class.

Second, in lieu of detailed monitoring data of past environmental carbon monoxide exposures for many years of the study, we had to define exposure by classifying officers according to the duration of their employment and their assignment to either bridge or tunnel work, which are crude surrogate measures of the actual exposure. Actual measurements throughout the years of the study would have been preferable to define more precisely the excess risk from arteriosclerotic heart disease mortality.

Third, ascertainment of vital status among our cohort was 97 per cent complete, and while this percentage of follow-up is well within the range generally considered acceptable for cohort mortality studies, the 3 per cent deficit had the effect of inflating person-years at risk and thus lowering the standardized mortality ratios. Furthermore, death certificates were located and cause of death was recorded for 460 (97 per cent) of the 474 known deaths. This had the effect of lowering the cause-specific standardized mortality ratios by an additional 3 per cent, on the average. These last two factors could have contributed to an underestimation of the magnitude of the arteriosclerotic heart disease mortality risk from employment with the Triborough Bridge and Tunnel Authority.

Finally, since this is a study of mortality as ascertained from death certificates, we are unable to assess the accuracy of the stated diagnoses or to define the date of myocardial infarction in contrast to the date of death.

In summary, this study indicates that occupational exposures to carbon monoxide contribute to arteriosclerotic heart disease

mortality. The excess risk of cardiovascular disease mortality that we observed may have been caused by long-term continuous lower exposure to carbon monoxide, short-term repeated peak exposures, or a combination of both. However, the absence of any significant relation between duration of employment and arteriosclerotic heart disease mortality tends to discredit the contribution of long-term exposure. The significant effect of age on arteriosclerotic heart disease mortality implies that age is an essential contributing factor in the excess risk of arteriosclerotic heart disease. The adverse effects of carbon monoxide poisoning on the risk of arteriosclerotic heart disease mortality seem to be reversible upon cessation of exposure.

We believe that our results have three important implications. First, given the magnitude of the effect that we have observed for a very prevalent cause of death, exposure to carbon monoxide in combination with underlying heart disease or other cardiovascular risk factors could be responsible for a very large number of preventable deaths. Second, parallel to the effect of cessation of cigarette smoking on the risk of cardiovascular disease, cessation of occupational exposure resulted in a decline in risk for heart disease mortality. Third, the parallel findings of this study of occupational exposure to carbon monoxide and those studies of the relation of cigarette smoking to cardiovascular mortality suggest that carbon monoxide may play an important role in the pathophysiology of cardiovascular mortality associated with cigarette smoking.

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