Noteworthy Decision Summary

Decision: WCAT-2003-01110-ad **Panel:** Randy Lane **Decision Date:** June 24, 2003

Carbon monoxide exposure and compensability for heart disease / heart attacks - Whether an automechanic's coronary heart disease and subsequent heart attacks were related to exposure to carbon monoxide in the course of his employment

The worker, who had worked for the same car dealership as a mechanic/trimmer 20 years until 1989, filed an application for compensation in 2000 alleging that his coronary artery disease and subsequent heart attacks were related to exposure of carbon monoxide in the course of his employment. His claim was denied and he appealed to WCAT.

The WCAT panel noted that three of the worker's nine brothers had heart problems. While blood tests done on the worker several days after exposure revealed carboxyhemoglobin determinations above the normal range, the tests did not establish toxic levels. Moreover, two physicians indicated that testing done several days after cessation of exposure did not reflect the levels measurable at the time of exposure and were of limited relevance. The panel also referred to a B.C. study of mortality rates among automechanics arising from arteriosclerotic heart disease which showed a proportional mortality ratio, or relative risk for automechanics, of 1.03. A relative risk of 2.0 is considered to be significant and often equated to a 50% likelihood that an exposed person's disease was caused by the agent. A relative risk of greater than 2.0 would permit an inference that an individual's disease was more likely than not caused by the implicated agent. A study done on tunnel workers reported a relative risk of 1.35, i.e. a 35% increase, in mortality rate from coronary heart disease, but their exposure was likely well above the levels to which the worker was exposed. These studies only deal with deaths and not disease.

The panel found that the evidence did not support a conclusion that the worker's individual work circumstances were such that he had such a significantly different level of exposure to carbon monoxide as compared to other automechanics with the result that his individual relative risk was 2.0 or more. Cardiovascular disease is very common in males of the worker's age, and a relevant family history could not be ignored. The panel preferred the opinions of two of the physicians over the others, in part because their comments are informed by the two studies and the worker's family history. The panel found that the worker's coronary artery disease and subsequent heart attacks were not related to exposure to carbon monoxide in the course of his employment.

Workers' Compensation Appeal Tribunal 150, 4600 Jacombs Road, Richmond, B.C. V6V 3B1
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Panel: Randy Lane, Vice Chair

Introduction

The worker filed an application for compensation on February 11, 2000 in which he linked his coronary artery problems to his employment as a mechanic/trimmer. He had worked for the same employer at the same building from 1969 until 1989. He had submitted a claim for carbon monoxide exposure in 1989 which had been disallowed. He returned to work after that claim, suffered a back injury, and stopped working.

In its August 9, 2002 findings a Review Board panel denied the worker's appeal from the March 29, 2000 decision of the Board that the worker's exposure to carbon monoxide in the garage where he worked was not of causative significance in the development of his coronary artery disease and subsequent heart attacks.

The worker has appealed the Review Board finding. In support of his appeal he provided an August 18, 2002 letter, a September 6, 2002 notice of appeal, a September 21, 2002 submission, and a December 16, 2002 submission. The worker's employer was notified of the appeal but it did not indicate that it wished to participate.

The notice of appeal indicates that an oral hearing is not required, and I agree.

Jurisdiction

This appeal was filed with the Appeal Division. On March 3, 2003, the Appeal Division and Review Board were replaced by the Workers' Compensation Appeal Tribunal (WCAT). As this appeal had not been considered by an Appeal Division panel before that date, it has been decided as a WCAT appeal. (See the *Workers Compensation Amendment Act (No. 2), 2002*, section 39).

Issue(s)

At issue is whether the worker's coronary artery disease and subsequent heart attacks are related to exposure to carbon monoxide in the course of his employment.

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Background and Evidence

The worker's written submissions to the Review Board state that he started as a trim man and general mechanic in 1957. From 1962 to 1969 he worked with C. Ltd. in the same capacity. In 1969 he moved to a different location with C. Ltd. and worked there from 1969 to 1989.

The worker filed a February 11, 2000 application for compensation.

In his March 29, 2000 decision the claims adjudicator noted that the worker considered that his heart disease was due to exposure to carbon monoxide in the garage where he worked. The worker considered that there was a problem with an outlet pipe in the garage as it was constricted due to being dented during construction in 1969 and it was repaired in approximately 1989. The claims adjudicator noted that the worker felt that the station where he worked in the garage received even more carbon monoxide than other workstations, due to the layout of the shop, and that also contributed to his exposure.

The adjudicator noted the worker had two previous claims in 1989 for carbon monoxide exposure. (While Board records refer to two claims it appears the worker really had only one claim.) They were disallowed because when the worker was seen by physicians his levels were measured and they were considered very low and not sufficiently high enough to cause any disease or injury. The claims adjudicator noted that this was during the time when the vent was supposedly constricted.

The claims adjudicator referred to the opinion of a Board medical advisor and concluded that the worker's coronary artery disease and heart attacks were not caused by overexposure to carbon monoxide at work. He considered medical advice and evidence indicated it was more probable that the worker was suffering from intrinsic coronary artery disease which would be expected given his age and which was supported by the positive family history. He noted that the exposure levels which the worker suffered back in 1989 were fairly insignificant and would not lead credence to the worker's suggestion that he was overexposed during his employment.

In its August 9, 2002 findings the Review Board denied the worker's appeal. The panel reviewed the worker's evidence, the medical/scientific materials on file, and the worker's testimony. Those medical/scientific materials included test results, hospital records which contained consultation reports, a September 16, 1999 progress report and February 2, 2001 medical/legal report from Dr. J, the worker's family physician, the March 16, 2000 claim log entry by Dr. W, a Board medical advisor in the occupational health and safety section, opinions dated June 18, 2002 and August 6, 2002 from Dr. G, the Board's internal medicine consultant, and a photocopy of a book entitled *Carbon Monoxide Toxicity* edited by Dr. Penney, an American toxicologist. As well, the file contains materials associated with the worker's treatment for his heart problems.

The Review Board panel's analysis is found in the following two paragraphs from the section entitled "Reasons and Findings":

> The panel, in determining this appeal has reviewed the information supplied by the worker, his physician and the Board's Internal Medical Advisor. The worker relies upon the "Carbon Monoxide Toxicity" book to support his case. Unfortunately the panel can not, on the basis of the book, support the worker's belief that his coronary artery disease and subsequent heart attacks are a result of chronic low level exposure to carbon monoxide. There is no measurable evidence that the worker was exposed to low levels of carbon monoxide on a consistent basis. The air quality testing completed at the worksite fell within the acceptable range. Further there is no documented evidence to support that the worker's own medical problems are directly related to him being exposed to carbon monoxide over a period of time. As Dr. [G] pointed out, the lack of any identifiable risk factor in producing the worker's infarction is not an unusual occurrence. Atherosclerotic vascular disease is the leading cause of death in the worker's age group.
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> Accordingly, the panel finds that there is not enough evidence to support the worker's contention that he was continually exposed to carbon monoxide gases in the workplace and that it is of cause and significance in the development of his coronary artery disease and subsequent heart attacks.

Reasons and Findings

Carbon monoxide exposure

I do not doubt that the worker was exposed to carbon monoxide. He was a mechanic/trimmer at a car dealership and such exposure would be expected.

While the question of whether the worker's 1989 claim was properly denied is not before me, I consider that the information on that file is relevant to the issue of exposure. (I note in passing that while the worker indicated in his submission to the Review Board that he was appealing the May 5, 1989 decision to deny his 1989 claim, he would need to seek an extension of time in which to appeal that decision before WCAT could review it.)

According to his submissions to the Review Board, the worker did not go to work from February 20 to 24, 1989 owing to flu-like symptoms. Testing on February 24, 1989 revealed a carboxyhemoglobin figure of 0.03. He went back to work on February 27, 1989 without knowing the results of the tests and when the test came back on March 2, 1989 his physician told him to leave work. Some eight days after he stopped working,

on March 10, 1989, testing revealed a figure of 0.02. Testing on March 23, 1989 revealed a figure of 0.01.

The 1989 claim was denied primarily on the input of the Board medical advisor who commented that a figure of 0.03 was not significant. A smoker would present a reading of 0.08 to 0.10 and a reading of 0.15 to 0.2 would be required before a patient showed symptoms. The medical advisor also noted that the carbon monoxide levels would reach normal in 24 hours whatever the initial level.

A Board occupational hygiene officer took readings at the worker's place of employment in late March 1989. The four time-weighted averages of 9.3, 9.5, 10.7, and 13.9 ppm (parts per million) were well below permissible maximums which were then 50 ppm for eight hours and 400 ppm for 15 minutes and also below the later adopted permissible maximums of 25 ppm for eight hours and 100 ppm for 15 minutes). It was noted that the mechanics used the local exhaust ventilation systems. The worker arrived during the inspection and contended that the systems were not used. Inspection revealed that the system on the side of the shop where the worker worked was functioning as demonstrated by air current testing and air velocity measurements. The officer did write orders requiring the employer to attend to design deficiencies in the ventilation systems that permitted carbon monoxide to re-enter the shop. While orders were issued it is significant that the testing results were well below the maximum permissible figures.

I appreciate that as revealed by a December 17, 1999 memorandum documenting the worker's discussion with the claims adjudicator (more than 10 years after the inspection) the worker took exception to the method used to sample carbon monoxide at the garage. He advised the claims adjudicator that the testing was done at noon, there were no mechanics present, no cars were running, and the doors were open. I note that the March 29, 1989 memorandum of the occupational hygiene officer refers to mechanics using systems at the time of the inspection. I take that to refer to mechanics being active when the officer attended the worksite, contrary to the worker's assertion a decade later.

The December 17, 1999 memorandum makes reference to the pipe being creased halfway and the worker considering that it would only be exhausting the fumes 50 percent of the time. Thus I take the reference to a 15 percent restriction in the claims adjudicator's decision letter to be a typographical error.

Family history

That the worker has a relevant family history is established from information on file although there is some inconsistency in the information. An August 13, 1991 consultation report documented a positive family history with two brothers having undergone coronary bypass surgery between the ages of 55 and 60. The report

concludes with a reference to "his significant family history" and a later August 16, 1991 report by the same physician refers to "a very strong family history." I note in passing that in 1991 the worker sought medical attention when he woke with chest pain. It was considered that his ECG findings were highly suggestive of underlying ischemic heart disease.

In August 1997 the worker developed upper anterior aching chest discomfort. An August 27, 1997 consultation report documented that there was no family history of heart disease. He underwent a coronary angioplasty as there was significant narrowing owing to coronary disease.

Dr. J's September 16, 1999 progress report asserts that none of the worker's brothers had heart disease. Yet in his February 2, 2001 medical/legal report Dr. J indicated that two of the worker's 14 siblings had heart disease. The worker's materials submitted to the Review Board in May 2001 indicate that one brother had bypass surgery at age 67, another brother at the age of 80 was fitted with a pacemaker and underwent an angioplasty, and a third brother had bypass surgery at age 58. Exhibit #1, entitled WCB File Summary, prepared by the worker and tendered at the May 22, 2002 Review Board oral hearing, contains information regarding his family history. The information refers to three brothers who had cardiac problems. It refers to the brother who had bypass surgery at age 67, a brother who was over 80 before experiencing heart problems, and a third brother who had a bypass at age 58.

I consider the evidence supports a conclusion that three of the worker's nine brothers had heart problems.

Medical opinions

Dr. J considers that the worker was exposed to excess of levels of carbon monoxide which "undoubtedly raised his chance, if not solely produced of developing coronary heart disease." He commented the worker had a very good subjective and objective package of information which would indicate chronic carbon monoxide poisoning while working with his employer over the years. He likens the worker's case to that of a fire-fighter patient of his who was "given disability" for chronic carbon monoxide exposure because, at the time, protective measures were not taken. He comments that he does not know how one would explain carboxyhemoglobin levels like those demonstrated by the worker in 1989 if not for significant exposure while working. He notes that the measurements were done several days after the worker had been at work and that the half-life of carboxyhemoglobin is in the manner of several hours. He indicates he has read the book *Carbon Monoxide Toxicity* which he indicates seems to be the definitive treatise on carbon monoxide and parallels many of the symptoms the worker has suffered over the years.

The claims adjudicator relied on the opinion of Dr. W, a Board medical advisor, who holds a diploma in industrial hygiene and is a certificant of the Canadian Board of Occupational Medicine. Dr. W noted that coronary artery disease and heart attacks are comparatively common amongst males over the age of 50. Coronary artery disease, including heart attacks, is the most common cause of death amongst Canadians aged 55 to 70 according to a recent survey of cardiovascular disease published by the Canadian Medical Association. He noted that family history was one of the significant risk factors for coronary artery disease and heart attacks and this factor was present for the worker. He observed chronic exposure to carbon monoxide is rarely mentioned as a risk factor in the literature because the evidence is weak and often controversial.

Dr. W noted that a highly regarded study of heart disease amongst workers exposed to carbon monoxide compared workers in toll booths with minimal carbon monoxide exposure to workers in traffic tunnels with high carbon monoxide exposure (Stern et al. 1988. Heart disease mortality among bridge and tunnel officers exposed to carbon monoxide. *American Journal of Epidemiology* 128:1276-1288). He observed the concentrations of carbon monoxide to which the tunnel workers were exposed reached more than 50 ppm on average and there were peaks above 400 ppm. He observed that exposure was likely well above the levels to which the worker was exposed. He noted that the study reported an increase of 35 percent in the mortality rate from coronary artery disease. He commented that even if it was assumed the worker had similar exposure to carbon monoxide there was still a considerably less than a 50 per cent chance that his coronary artery disease and heart attack resulted from occupational exposure. He noted there would have to be a 100 percent increase in the disease before an occupational cause became a 50 percent probability.

Dr. W also reviewed the mortality statistics gathered by the Cancer Control Agency of British Columbia between 1950 and 1984. He noted this study isolated automechanics from other occupations and identified deaths arising from arteriosclerotic heart disease. The proportional mortality ratio of 103 was close enough to normal (100) to be not statistically significant. He observed that evidence in the literature that an acute exposure to carbon monoxide can cause an immediate attack of angina or heart attack should not be confused with long-term effects.

Dr. W observed that the worker's age, sex, and family history were adequate explanations for his heart problems rather than his occupational exposure. He contended the vast majority of evidence in the literature neither supported nor refuted the worker's carbon monoxide exposure being a significant aggravating or accelerating factor for his heart disease. He observed that the study of tunnel workers noted earlier suggested that excess mortality from heart disease was limited to the first few years after employment ceased. He commented that he could find no convincing medical evidence that allowed him to separate the worker from his age group which was recognized as having significant coronary artery disease and a high rate of heart attacks.

In response to the worker tendering material at the Review Board appeal including the book *Carbon Monoxide Toxicity*, the Review Board panel sought further medical input and received an opinion from Dr. G, who is a specialist in internal medicine. In its request the panel noted the worker's contention that there was a correlation between carbon monoxide inhalation and enlargement of his prostate gland. The worker claimed that one of the results of carbon monoxide toxicity was hearing loss and his hearing loss claim had been accepted by the Board. The worker contended that his breathing and speaking problems were one of the manifestations of carbon monoxide poisoning. The worker considered that his heart problems were due to his carbon monoxide exposure.

In response, Dr. G indicated that there was no legitimate medical data linking prostate enlargement to carbon monoxide exposure. He considered that while acute carbon monoxide poisoning produced tissue hypoxemia which affected all nerve function that did not mean that low level carbon monoxide exposure would have a similar effect. The European study noted in the book *Carbon Monoxide Toxicity* was not available in English and it was a survey only and did not include a control group. Dr. G did not consider that the results could be legitimately used in support of the idea that low dose carbon monoxide exposure produced hearing loss.

Dr. G noted that the normal range for the carboxyhemoglobin test was 0.05 to 0.015 and the worker tested at 0.03 after six days off the job. He commented that the major route of elimination of carbon monoxide is through the lungs and the half-time for elimination of carbon monoxide while tidal breathing at rest and at sea level is approximately four hours. He noted that carboxyhemoglobin determinations done several days after exposure would certainly not reflect the levels measurable at the time of exposure. He commented that systemic manifestations and neuropsychiatric sequelae of carbon monoxide poisoning noted in the *Carbon Monoxide Toxicity* are so broad and non-specific that they include most neuroses, many chronic neurologic abnormalities, and many common chronic disease processes. He commented that was not the kind of evidence one could use to narrow the differential diagnosis or make meaningful conclusions.

Dr. G observed that there was evidence which indicated that low carboxyhemoglobin concentrations cause adverse effects on the cardiovascular system. Incremental increases of as little as two percent had been shown to speed up the onset of anginal pain during exercise in patients with angina. He considered this fit with the understanding of the physiology of carbon monoxide poisoning in producing tissue hypoxemia. Tissue hypoxemia produced anginal pain. He noted the data presented by Dr. W which he considered to be the present state-of-the-art in terms of knowledge in the area. He observed mortality statistics did not indicate increased deaths from atherosclerotic heart disease in automechanics. He noted the lack of any identifiable risk factor in producing this worker's infarction is not an unusual occurrence as atherosclerotic vascular disease is the leading cause of death in his age group.

Dr G commented that he did not have a copy of the book *Carbon Monoxide Toxicity* but only had excerpts reported to support the relationship between the worker's symptoms and his exposure. He noted the website created by the editor of the book appeared to be mostly a marketing tool promoting its sale. He observed that using evidence available to him it appeared that the controversial thesis of chronic low-level carbon monoxide toxicity was supported by sparse data interpreted in what appeared to be a significantly biased manner. He did not consider there was enough evidence to support the claim that the worker's symptoms could be produced by the level of carbon monoxide exposure at his place of employment.

In response to a submission by the worker, the Review Board panel asked Dr. G to review all of *Carbon Monoxide Toxicity*. In his August 6, 2002 response Dr. G indicated he had reviewed the book. He commented that the chapter by Dr. Penny describing carbon monoxide poisoning confirmed his opinion that the thesis was supported by very sparse data interpreted in a significantly biased manner. He did not think the case for chronic carbon monoxide toxicity as presented in the book was supported by appropriately interpreted legitimate medical data or that it represented a valid support for the worker's claim.

Included in the package of materials submitted on the appeal is a July 1, 2001 letter from Dr. B, a cardiologist. He noted that carbon monoxide could lead to vascular problems but he felt that the worker's cardiovascular disease occurred independent of any carbon monoxide and might well be related to other risk factors for heart disease. I am aware from a copy of the worker's October 1, 2001 letter to a workers' adviser included in his appeal package that he considered that Dr. B's opinion was based on insufficient information about the worker's work situation and his family history. I note the worker does not indicate that he asked Dr. B to review the matter with additional information on those points.

I have considered the information from *Carbon Monoxide Toxicity* and the materials from Dr. Penney submitted by the worker in his appeal from the Review Board finding. The worker obtained an October 29, 2002 opinion from Dr. Penney who holds a PhD and is a professor of physiology at Wayne State University, an adjunct professor in occupational and environmental health, and the director of surgical research at a hospital. A review of the PubMed database establishes that he has authored or co-authored numerous articles concerning carbon monoxide exposure many of which concern rats.

In his opinion Dr. Penney notes that he reviewed several hundred pages of documents sent to him by the worker. He comments that the worker worked for several decades in an environment that often contained high concentrations of carbon monoxide, the ventilation system at the garage did not function as it should have, and public health officials were unaware of the serious health hazards posed by carbon monoxide in his work environment. He comments that the worker's blood tests are totally unusable and

irrelevant to a discussion of carbon monoxide poisoning as the blood was drawn more than 24 hours after he left the garage. He comments that the symptoms the worker relates as beginning or becoming especially problematic around 1989, headaches, nausea, sleep disturbance, etc. are symptoms consistent with carbon monoxide poisoning.

Dr. Penney contends that the worker and his workmates were chronically poisoned by carbon monoxide. The worker's loss of hearing is more likely than not due to exposure to carbon monoxide. Many of the worker's other health problems have some basis in his exposure to carbon monoxide. Dr. Penney considers that the worker's coronary artery disease is more likely than not due to exposure to carbon monoxide in his work environment. He notes that the 1988 study cited by Dr. W clearly shows a relationship between occupational exposure to carbon monoxide and development of coronary artery disease. He indicates other studies provide supporting proof and, indeed, possible mechanisms of such disease. Dr. Penney concludes that his opinions reflect a reasonable degree of scientific and medical certainty based on his professional experience in the field and his standing as a specialized toxicologist.

Discussion

As noted above, I accept that the worker was exposed to carbon monoxide. The comment by the Review Board that there was no measurable evidence that the worker was exposed to low levels of carbon monoxide seems to be based on a determination that the air quality testing established figures within the acceptable range. The fact that carbon monoxide was measured supports a conclusion that the worker was exposed. That figures may have fallen in the normal range would not change that.

While the worker may have been exposed to carbon monoxide, I do not consider that the evidence is sufficient to support a finding that the worker was exposed to excess amounts of carbon monoxide. The testing in 1989 established figures that were below the permissible maximum. As noted above the worker contends that the testing was flawed. I am unable to accept his contention that there were no vehicles running when the testing occurred. The contemporaneous 1989 report of the Board officer indicates otherwise. The worker considers that he was exposed to high levels of carbon monoxide on a daily basis; however, his opinion does not establish that is so. He notes that he has no quantitative evidence that establishes his contention.

The worker notes that Dr. G erred in indicating that the normal range on the blood test is .05 to 0.015. I accept that Dr. G erred as the reports clearly refer to a range of 0.005 to 0.015. Thus the worker’s readings of 0.03 and 0.02 on February 24, 1989 and March 10, 1989 were in excess of the normal range. That the figures were above the range does not mean that the tests establish “toxic” levels as asserted by the worker. He has tendered no persuasive evidence to that effect. I note the opinion of the Board medical advisor in 1989 that a figure of 0.15 would be needed before symptoms

resulted. While the worker seems to be arguing that figures of 0.02 and 0.03 after having stopped work must mean that his levels when working would have been much higher, I do not consider that the evidence supports a conclusion that they would have been at “toxic” levels when he was working. Both Dr. G and Dr. Penney indicate testing several days after cessation of exposure is of limited relevance. Further, the Board medical advisor commented in 1989 that readings would reach normal within 24 hours.
The worker comments that he does not consider that he had a significant family history of heart disease. As well, Dr J commented in his report that the worker developed cardiovascular disease "despite his rather good family history". I consider it significant that the 1991 consultation reports give a different impression of the history. That, of course, was at a time when the worker had two siblings with recognized heart disease. After 1991 a third sibling was found to have heart disease.

The Review Board's comment that there was no documented evidence that the worker's medical problems were related to him being exposed to carbon monoxide is problematic. The Review Board did have the benefit of an opinion from Dr. J which linked the worker's exposure to carbon monoxide to coronary heart disease. It may be that the Review Board meant there was no persuasive medical opinion.

It may be true that age and gender do not by themselves cause heart disease. The worker’s submission suggests that the Board considers heart disease is the norm for a man in his age group without any identifiable risk factors. However it cannot be ignored that cardiovascular disease is very common in males of the worker’s age. A relevant family history also cannot be ignored.

The worker seeks to buttress his case by pointing to the health problems of some of his co-workers. I do not consider that such information is persuasive as the worker has not provided medical reports regarding those workers or established that his co-workers’ problems are due to employment exposure as opposed to other factors.

I consider it significant that the mortality figures noted by Dr. W showed no significant difference for automechanics. The worker comments that a 3 percent difference means that 36 more mechanics died than expected; he considers that significant. I accept that there may have been some small excess but the fact is that the proportional mortality ratio or relative risk for automechanics is 1.03. A relative risk of 2.0 is considered significant. Relative risk is the ratio of the risk of disease or death among people exposed to an agent to the risk among the unexposed. A relative risk of 2.0 is often equated to a 50 per cent likelihood that an exposed person’s disease was caused by the agent, and a relative risk greater than 2.0 would permit an inference that an individual’s disease was more likely than not caused by the implicated agent.

As noted by the worker, the Cancer Control Agency information only deals with deaths and not disease. As well the Stern et al. study noted above only concerned mortality. That study is significant for the fact that the standardized mortality ratio was 1.35. Thus

the relative risk for tunnel officers would not have been at all close to 2.0. The study notes that the elevated risk declined after cessation of exposure with much of the risk dissipating within as little as five years.

There are other articles which address carbon monoxide exposure, and I note the following recent publications:

- Koskela RS. 2000. Factors predictive of ischemic heart disease mortality in foundry workers exposed to carbon monoxide. *American Journal of Epidemiology*. 52(7):628-32

- Herbert, R. et al. 2000. Occupational coronary heart disease among bridge and tunnel officers. *Archives of Environmental Health*. 55(3):152-63. Erratum at 55(4):286.

The second article, like the 1988 Stern et al. article, involved a study of traffic control officers in New York. It is notable for the observation that perhaps some portion of the observed increase in coronary heart disease resulted from an historic, atherogenic effect of carbon monoxide; however, the authors considered that conclusion was "speculative". Unlike Stern et al. they found no statistically significant difference in risk between workers employed in tunnels versus bridges although there was a trend in that direction. They were unable to identify specific risk factors that led to the described increase.

While epidemiology informs decision-makers, it is not determinative of the cases of individual workers. It is still necessary to address individual circumstances. I do not consider that the evidence on file supports a conclusion that the worker's individual work circumstances were such that he had such a significantly different level of exposure to carbon monoxide as compared to other automechanics with the result that his individual relative risk was 2.0 or more. The worker's contentions which include submissions that work as a trimmer meant that he was breathing more deeply, that he worked shifts longer than eight hours, that his position in the garage exposed him to more carbon monoxide, and that he was exposed to higher levels of carbon monoxide from cars without catalytic converters do not provide persuasive evidence as there is no persuasive evidence that his experience differed significantly from other automechanics.

I am not persuaded by Dr. Penney's opinion. I do not consider that I need to request a fuller analysis from him as suggested by the worker who asks that Dr. Penney be contacted if it is found that there is insufficient evidence to support his claim. It is open to WCAT to seek out new medical information. There is even formal provision in the Act which sets out the procedure for WCAT to obtain the opinion of an independent health care professional. However I consider that I have enough information to make an informed decision on this claim. There is no obligation on a decision-maker to seek out

additional information when the record is sufficient to enable the decision-maker to render an informed decision.

I consider that the opinions of Drs. G and W are persuasive. They have qualifications that permit them to speak knowledgeably about the issue; their comments are informed by relevant BC data, the Stern et al. article, and the worker's family history. The worker places great weight on Dr. J's opinion owing to that physician's knowledge of his personal circumstances. With respect, I consider that Dr. J's knowledge of the worker's circumstances does not inform Dr. J as to the causative significance of the worker's occupational exposure to carbon monoxide.

I do not consider that the worker's heart problems are an occupational disease due to the nature of his employment.

Section 55

The worker's application in February 2000 was more than one year after his 1991 or 1997 heart problems. Neither the claims adjudicator nor the Review Board drew the worker's attention to the provisions of section 55 of the Act which require that an application must be made within one year of an injury or within one year of disablement from an occupational disease. A claim is barred unless there are special circumstances which precluded a worker from claiming within the one year period.

I have not considered the application of section 55 to this claim. Had I considered that there was a casual link between the worker's employment exposure and his heart problems, I would have given the worker notice of the section 55 issue and given him an opportunity to make submissions on the matter.

The worker has asked that he be reimbursed for various expenses which include the purchase of two copies of *Carbon Monoxide Toxicity* (one copy kept for reference and a second copy submitted to the Board), photocopying, and the cost of Dr. Penney's opinion. I have considered the request but decline to order reimbursement.

Conclusion

The worker's appeal is denied. I confirm the finding of the Review Board that the worker's coronary artery disease and subsequent heart attacks are not related to exposure to carbon monoxide in the course of his employment.

Randy Lane
Vice Chair

RL/dcl