# Carbon Monoxide in Indoor Ice Skating Rinks: Evaluation of Absorption by Adult Hockey Players

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**Abstract:** We evaluated alveolar carbon monoxide (CO) levels of 122 male, adult hockey players active in recreational leagues of the Quebec City region (Canada), before and after 10 weekly 90-minute games in 10 different rinks. We also determined exposure by quantifying the average CO level in the rink during the games. Other variables documented included age, pulmonary function, aerobic capacity, and smoking status. Environmental concentrations varied from 1.6 to 131.5 parts per million (ppm). We examined the absorption/exposure relationship using a simple linear regression model. In low CO exposure levels, physical exercise lowered the alveolar CO concentration. However, we noted that for each 10 ppm of CO in the ambient air, the players had adsorbed enough CO to raise their carboxyhemoglogin (COHb) levels by 1 percent. This relationship was true both for smokers and non-smokers. We suggest that an average environmental concentration of 20 ppm of CO for the duration of a hockey game (90 minutes) should be the reference limit not to be exceeded in indoor skating rinks. (*Am J Public Health* 1990; 80:594–598.)

## *Introduction*

Over the past few years numerous cases of carbon monoxide (CO) intoxication in indoor skating rinks have been described in medical literature.[1–9] Most often, the victims have been hockey players, and the source of the contamination has been the ice resurfacing machine.

When inhaled, CO quickly enters the bloodstream where once having displaced the oxygen, it becomes associated with hemoglobin to form carboxyhemoglobin (COHb). The percentage of COHb is the main indicator of the absorption of this toxic gas. It is generally equal to 1 percent among non-smokers[10] and may reach 5 to 10 percent among smokers.[11]

Following acute exposure to CO, there is a rapid increase in the COHb level. At the 3 to 4 percent level, the first signs of adaptation in healthy individuals take the form of an alteration of alertness[11] and arterial dilation in response to a drop in blood oxygenation.[12] These levels are sufficient to worsen coronary symptoms among persons with cardiac problems.[13–16] At 4 percent, careful driving skills are affected,[17] and, between 5 and 10 percent, visual acuity is decreased.[12] Levels between 10 and 20 percent cause headaches and difficult breathing.[12] At 20 percent, headaches worsen, nausea appears and manual dexterity is affected.[12] At 40 to 50 percent, all symptoms intensify, and between 50 and 60 percent, there is a possibility of coma.[12]

Gasoline or propane-driven ice resurfacers give off combustion by-products in their exhaust fumes. Depending on how well the unit operates, there will be a variable level of CO emitted. The problem is generally aggravated by poor ventilation in indoor rinks. The combination of these two factors often results in high levels. The CO reaches its maximum concentration when the ice resurfacer is in operation and gradually drops afterwards.[18]

Extremely high levels of CO have been documented in skating rinks with some levels exceeding 100 parts per million (ppm).[1,3,6,8,9,18–21]

CO absorption is up to three to four times higher during exercise than during rest.[22] We conducted a study among male adult hockey players of the Quebec City area to evaluate the CO absorbed by the players at the end of a 90-minute game. The data were collected between October 26, 1988 and January 28, 1989.

## *Methods*

We chose 10 of the 16 voluntary hockey teams, each playing in its own indoor rinks, guiding our choice on various indicators such as the age and size of the rink, the type of resurfacing machine, and whether or not there was a mechanical ventilation.

### Measuring Absorption

To measure absorption, subjects, standing up, took a deep breath which they held for 20 seconds. They then exhaled first into one polyvinyl chloride 900 mL bag and then into two other bags joined by plastic tubing (Tygon). The second sample filled with alveolar air was analyzed within 12 hours using a Bendix 8501-BA infra-red CO analyzer. The quantification technique was developed beforehand, and the correlation between the CO concentration in the alveolar air in ppm and the percentage of COHb was established using a sample of 24 volunteers (13 smokers and 11 non-smokers).[23] The square of the linear correlation coefficient ($r^2$) was .99, the intercept was −.003 ppm with a standard error of .005 ppm, the slope was 5.39 ppm (5.4 ppm of alv CO ≈ 1% COHb) and the standard error of the slope was .13 ppm.

For each player, the alveolar CO was measured within 15 minutes before and after the game. The difference between the two results represented the CO absorbed by the subject during the match in ppm (ΔalvCO).

### Measuring Exposure

Exposure was measured by collecting seven samples of air at 15 minutes intervals, from the players' arrival on the ice rink to their departure. The samples were taken from the penalty box located 1.2 meters above the ice using a Gilian HFS-113A pump and Calibrated Instrument 5 L aluminized bags. Pump flow was set at one liter per minute. These samples were analyzed within 12 hours on the Bendix spectrometer. The average of the seven samples was con-

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sidered the level of exposure by players during the game ([CO]).

**Other Variables**

Player's age and whether a smoker or non-smoker were obtained by questionnaire. The pulmonary function was quantified with a Roxon Compact vitalograph using the standardization criteria of the American Thoracic Society.[24] We determined the forced expiratory volume in one second ($FEV_1$), the forced vital capacity (FVC), the mean forced expiratory flow ($FEF_{25-75}$) and the $FVC/FEV_1$ ratio. The expected values were estimated using the Knudson method.[25] Finally, players' aerobic capacity ($\dot{V}O_2max$) was evaluated using a sub-maximum progressive effort test on an ergometric bicycle. The initial work load was set on the basis of the weight of the individual and increased progressively by 240 kiloponds meter (Kpm) for four two-minute levels. The $\dot{V}O_2max$ was estimated based on the equation suggested by the American College of Sports Medicine (26).

**Statistical Analysis**

The data were stratified according to smoking habits (smokers vs non-smokers). In addition to CO exposure, we examined the influence of age, $\dot{V}O_2max$ and the various spirometric indices on absorption, in a multiple regression model using a backward elimination procedure.[27] The preselected critical value of the F distribution was set for a significance level of .05. The resulting relationship between absorption and exposure was evaluated with a simple linear least squares regression model using individual values. When heteroscedastic, it was weighted by the inverse of the variance of the means to confirm the validity of the statistical tests.[27] To verify the possible parallelism between smokers and non-smokers, the coefficients were compared with the addition of a term of interaction.

## *Results*

We evaluated 122 players in 10 teams, 32 smokers and 90 non-smokers. Their age varied between 19 and 59 years old (mean = 34.04 years, standard deviation = 7.10 years).

The various spirometric parameters ($FEV_1$, FVC, $FEF_{25-75}$, $FVC/FEV_1$) as well as the age and the $\dot{V}O_2max$ were eliminated by the backward elimination procedure (data available on request to authors). It seems that these variables have very little effect on CO absorption, whatever the smoking habits. As a result, these variables were not considered factors likely to alter the exposure/absorption relationship.

Tables 1 and 2 describe by indoor rink, for non-smokers and smokers respectively, exposure concentrations in ppm, the number of players, the average quantity of alveolar CO (mean and variance) absorbed during the game and the estimated equivalent in percentage of COHb (5.4 ppm alv CO ≈ 1% COHb).

The exposure levels varied from 1.6 to 131.5 ppm. Seven rinks were below 20 ppm, whereas three exceeded 75 ppm.

Table 1 shows that with low exposures (rinks 1 and 3), non-smokers had, at the end of their game, lowered their COHb concentration. In seven other rinks there was absorption and it was proportional to the level of exposure. The concentration of 4 ppm noted in rink 4 represented a balance threshold where there was neither absorption nor elimination.

Table 2 shows an average drop in COHb among smokers at exposure levels as high as 15.1 and 16.2 ppm. This phenomenon is attributable to high basic levels among smokers.

**TABLE 1—Variation in the Alveolar Absorption of Carbon Monoxide among Players during a 90-minute Hockey Game (ΔalvCO) and the Estimated Equivalent in Percentage of Carboxyhemoglobin (COHb) based on the Concentration of Carbon Monoxide ([CO]) in the Indoor Skating Rinks among Non-smokers**

| Arena | Players N | [CO] (ppm) | ΔalvCO Mean Variance (ppm) | ΔalvCO Mean Variance ($ppm^2$) | COHb % |
|---|---|---|---|---|---|
| 1 | 8 | 1.6 | −.46 | .10 | −.09 |
| 2 | 12 | 9.3 | 3.48 | .46 | .66 |
| 3 | 13 | 1.6 | −.24 | .10 | −.04 |
| 4 | 9 | 4.0 | −.04 | .44 | −.007 |
| 5 | 10 | 9.5 | 2.39 | .93 | .45 |
| 6 | 5 | 15.1 | 4.51 | 1.56 | .85 |
| 7 | 14 | 131.5 | 69.79 | 128.90 | 13.17 |
| 8 | 8 | 107.3 | 57.36 | 35.07 | 10.82 |
| 9 | 5 | 16.2 | 7.81 | .70 | 1.47 |
| 10 | 6 | 79.3 | 46.10 | 40.42 | 8.70 |

**TABLE 2—Variation in the Alveolar Absorption of Carbon Monoxide among Players during a 90-minute Hockey Game (ΔalvCO) and the Estimated Equivalent in Percentage of Carboxyhemoglobin (COHb) Based on the Concentration of Carbon Monoxide ([CO]) in the Indoor Skating Rinks among Smokers**

| Arena | Players N | [CO] (ppm) | ΔalvCO Mean Variance (ppm) | ΔalvCO Mean Variance ($ppm^2$) | COHb % |
|---|---|---|---|---|---|
| 1 | 3 | 1.6 | −6.38 | 46.32 | −1.20 |
| 2 | 2 | 9.3 | −27.72 | 20.58 | −5.23 |
| 3 | 3 | 1.6 | −10.68 | 11.30 | −2.00 |
| 4 | 1 | 4.0 | −18.13 | — | −3.42 |
| 5 | 1 | 9.5 | −13.79 | — | −2.60 |
| 6 | 4 | 15.1 | −2.38 | 31.42 | −0.45 |
| 7 | 6 | 131.5 | 51.50 | 299.45 | 9.72 |
| 8 | 6 | 107.3 | 45.66 | 98.87 | 8.61 |
| 9 | 4 | 16.2 | −9.93 | 1.81 | −1.87 |
| 10 | 4 | 79.3 | 34.80 | 357.27 | 6.57 |

Figures 1 and 2 show the relationship between exposure and absorption using a simple linear regression model.

Figures 1 illustrates this model among non-smokers. The $r^2$ was equal to 0.97 and the intercept to −1.72 ppm (standard error = .69 ppm). This small negative intercept reflected the CO elimination in low exposure concentrations for the non-smokers. The slope at 0.55 ppm (standard error = .01 ppm) indicated that for every 10 ppm of exposure, an adult hockey player (non-smoker) had, at the end of a 90-minute game, absorbed enough CO to raise his alveolar CO concentration by 5.5 ppm (≈1% of COHb). The F ratio was 2681.36 for a significance level below 0.0001. The test had to be interpreted with caution given the heteroscedasticity of the data. Indeed, there was an increase in the variability of absorption proportional to the level of exposure (see Figure 1 and Table 1). For exposures of 1.6 and 131.5 ppm, absorption varied respectively from −0.9 ppm to 0.1 ppm (range = 1 ppm, variance = 0.10 $ppm^2$) and from 51.1 ppm to 87.4 ppm (range = 36.3 ppm, variance = 128.90 $ppm^2$). To correct this situation, the results were weighted with the inverse of the variance of the means. The results of the regression and the test continued to be very significant ($F = 1454.70$, $p < 0.0001$).

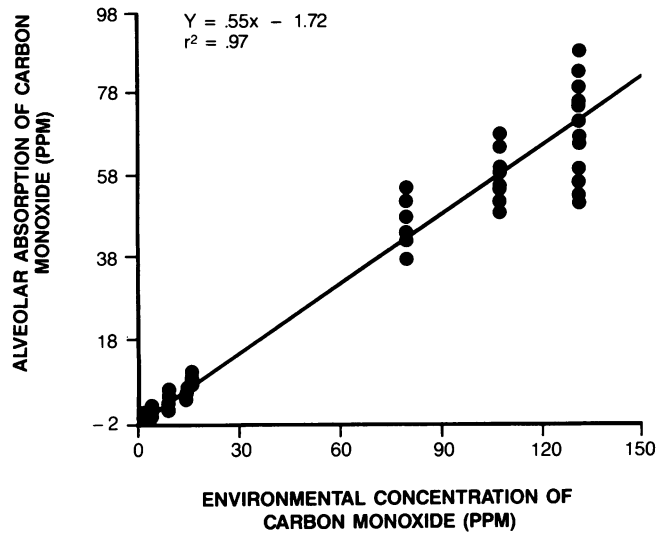


**FIGURE 1—Relationship between Alveolar Absorption of Carbon Monoxide among Players during a 90-Minute Hockey Game based on Carbon Monoxide Concentrations in Indoor Skating Rinks (non-smokers).**

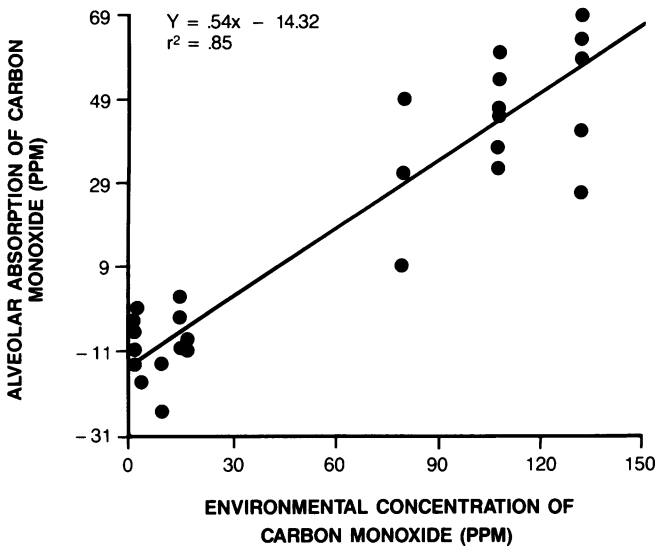


**FIGURE 2—Relationship between Alveolar Absorption of Carbon Monoxide among Players during a 90-Minute Hockey Game Based on Carbon Monoxide Concentrations in Indoor Skating Rinks (smokers).**

Among smokers (Figure 2), the $r^2$ was equal to 0.85 and the intercept to −14.32 ppm (standard error = 3.1 ppm) indicated the effect of exercise on CO elimination with elevated basic levels of alveolar CO. The F ratio was 169.50 ($p < 0.0001$). The slope at 0.54 ppm (standard error = .04 ppm) was not considered statistically different to that of non-smokers ($p = .6607$). As a result, after a 90-minute game of hockey, smokers and non-smokers alike increased their concentration of COHb by 1 percent for every 10 ppm of exposure to CO in the indoor rink.

*Discussion*

There are two types of standards for CO: standards for workers and standards for the public in general.

In an occupational environment the National Institute for Occupational Safety and Health (NIOSH) recommends a maximum time-weighted average (TWA) of 35 ppm for an eight-hour work day and a ceiling concentration of 200 ppm.[10] In the province of Quebec (Canada), the prevailing regulation stipulates 50 and 400 ppm for the same parameters.[28]

These values cannot apply to a hockey player playing in a recreational league. The latter, besides practicing his sport during his leisure time, is involved in an activity that required an energy expenditure greater than the workers. For example, the average energy expenditure of professional activities like lumberjack, mason, miner or carpenter is between 5.0 and 6.4 Mets (1 Met = 3.5 mL $O_2 \times Kg^{-1} \times min.^{-1}$).[29] For a hockey player, it is 12.9 Mets (min. = 9, max. = 15), a greater quantity than a soccer player (10.3 Mets, min. = 8, max. = 12) or a basketball player (11.1 Mets, min. = 9, max. = 13).[29] It is therefore necessary to look to criteria that apply to the general public.

Table 3 gives a summary of Canadian[30] and American[31] standards and of the recommendations of the World Health Organization (WHO).[32] These levels have been deduced using empirical equations based on the controlled exposure of volunteers. Apart from the desirable Canadian standard, they aim at maintaining the COHb concentration for non-smokers at 2 percent, thus protecting the most vulnerable segment of the population from the harmful effects of CO. The desirable Canadian standard aims at allowing an average COHb content below 1 percent, namely the equivalent of the endogenic production of a non-smoker.[30]

In indoor rinks, the British Columbia Department of Labor (Canada) has proposed a reference limit of 25 ppm[8] and, following a review of the literature on the subject, Luckurst and French suggested CO concentrations of 25 and 12 ppm for one hour and eight hours, respectively.[33]

The study that we conducted allowed us to ascertain extremely high CO concentrations in three indoor rinks out of 10. These levels even exceeded the standards set for workers.

Beyond environmental measurements, the best way to describe the risk involves using biological measurements. Only one author has shown interest in this aspect. Spengler evaluated alveolar breath samples among 12 Harvard University players before and after 90 minutes of exercise. An average concentration of 22.5 ppm of CO in the air was sufficient to raise the average COHb level by 1.1 to 3.2 percent.[18]

We succeeded in determining the absorption curve of adult hockey players who practice their sport on a recreational basis, and we noted that absorption was directly related to exposure.

There was great individual variability of absorption at the same CO exposure. For the non-smokers, at low level of CO, the intrateam variability was very low (variance = .316 $ppm^2$ for teams 1 and 3). At higher exposures, the observed

**TABLE 3—Recommended Carbon Monoxide Levels for Ambient Air**

| | Average per 1 hour (ppm) | Average per 8 hours (ppm) |
|---|---|---|
| WHO* (1979) | 25 | 13 |
| United States (1985) | 35 | 9 |
| Canada (1987) | | |
| Desirable | 13 | 5 |
| Acceptable | 30 | 11 |

*World Health Organization

variability may be explained in part by the analytical error on the measure of alveolar CO itself.[23] Also, other variables undocumented here can probably affect the individual CO absorption at these levels. The ratio of the tidal air to the dead space of the lung and the diffusion constant of the lung can explain some divergencies in the CO uptake.[22] Moreover, factors which affect the players' activity may be important. For example, Green showed differences in the time-motion characteristics (actual playing time, number of playing shifts, recovery time between shifts, etc.) between players depending on the position played (center, wing, defense).[34] In addition for the smokers, the variability of the pre-exposure alveolar CO level is certainly an important factor.

In the more exposed teams (#10-8-7), the average increase of COHb were respectively 8.70 percent, 10.82 percent, and 13.17 percent. Some players had an increase up to 17 percent. Levels like this are certainly sufficient to cause headache, breathing difficulties and, for some people, nausea.[12] They can also alter the choice reaction time,[11] the driving skills,[17] and the visual acuity.[12] All these toxic actions can contribute to accidents on the skating rinks and on the road at the end of the game.

At the present time there are no reliable data on cardiac pathology among hockey players. However, sudden increases in COHb as noted here may have a significant impact on the triggering of an irreversible ischemia. Particularly, if they are within the framework of intense physical exercise. Montgomery estimated for 12 adult recreational players that the intensity of hockey play assessed by the Karvonen method was 85 percent.[35] This represents a strenuous activity and, by itself, it can strain the cardiovascular system.[35] All these considerations make CO a significant threat for the population under study.

Generally, these players drop out of hockey several years and then return to improve their physical fitness or for the friendship obtained.[35] They are often ill-prepared for such intense physical exercise and, of all the groups frequenting indoor rinks, they are probably the most susceptible to the harmful effects of CO given the high level of absorption conferred by the intensity of their activity. If a standard is to be issued, we believe that they should serve as a reference.

We find absurd the idea that a hockey player must adapt to a toxic gas. A 3 percent COHb threshold, which is sufficient to induce physiological compensation mechanisms, must be the reference index. Considering that non-smokers have COHb levels of 1 percent and less, an increase of more than 2 percent cannot be accepted during the hockey game. This corresponds, for the duration of the hockey game (90 minutes), to an average concentration of 20 ppm in the air of the indoor rink, and as a result, it is the limit we propose.

If we rely on the data of our study, this norm is realistic since seven indoor rinks in 10 fell within it. When an ice resurfacing machine with a non-polluting motor (electrical, hydrogen) is not available, there are three means of respecting this norm: good maintenance of the ice resurfacing machine, sufficient mechanical ventilation used wisely, and routine monitoring of CO levels in indoor skating rinks.[7,8,20,36]

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### REFERENCES

1. Anderson DE: Problems created for ice arenas by engine exhaust. Am Ind Hyg Assoc J 1971; 32:790–801.
2. Cleary WM: Indoor ice rinks: A public health problem. Mich Occup Health 1973; 19:1–3.
3. Johnson CJ, Moran JC, Paine SC, Anderson HW, Breysse PA: Abatement of toxic levels of carbon monoxide in Seattle ice-skating rinks. Am J Public Health 1975; 65:1087–1090.
4. Coueffin K: Carbon monoxide: A problem in enclosed ice arenas. Environ Health Rev 1981; 25:64–66.
5. Carbon monoxide intoxication associated with use of a gasoline-powered resurfacing machine at an ice skating rink. MMWR 1984; 33:49–51.
6. Carbon monoxide exposures at an ice skating rink. MMWR 1986; 35:435–441.
7. André D, Kosatsky T, Bonnier J-G: Intoxication au monoxyde de carbone dans les arénas: Problématique et moyens d'intervention. Can J Public Health 1988; 79:124–129.
8. Carbon monoxide in ice arenas—British Columbia. Canada Dis Weekly Rep 1984; 10–14:53–55.
9. Miller RK, Ryan MC, Bilowus P: Carbon monoxide poisoning in indoor ice skating arenas. Va Med 1989; 116:74–76.
10. Criteria for a recommended standard: Occupational exposure to carbon monoxide. Cincinnati: National Institute for Occupational Safety and Health, Pub. no. 73-11000, 1973.
11. Shephard RJ, Wilber CG (eds): Carbon monoxide: The Silent Killer. Springfield: Charles C. Thomas, 1983.
12. International Labor Office, Parmeggiani L (ed): Encylopedia of Occupational Health and Safety. 3rd Ed. Vol. 1. Geneva: ILO, 1983.
13. Anderson EW, Andelman RJ, Strauch JM, Fortuin NJ, Knelson JH: Effect of low-level carbon monoxide exposure on onset and duration of angina pectoris. Ann Intern Med 1973; 79:46–50.
14. Aronow WS, Harris CN, Isbell MW, Rokaw JN, Imparato B: Effect of freeway travel on angina pectoris. Ann Intern Med 1972; 77:669–676.
15. Aronow WS, Isbell MW: Carbon monoxide effect on exercise-induced angina pectoris. Ann Intern Med 1973; 79:392–395.
16. Aronow WS: Aggravation of angina pectoris by two percent carboxyhemoglobin. Am Heart J 1981; 101:154–157.
17. Wright G, Randell P, Shephard RJ: Carbon monoxide and driving skills. Arch Environ Health 1973; 27:349–354.
18. Spengler JD, Stone KR, Lilley FW: High carbon monoxide levels measured in enclosed skating rinks. J Air Pollution Control Assoc 1978; 28:776–779.
19. Sorensen AJ: The importance of monitoring carbon monoxide levels in indoor ice skating rinks. J Am Coll Health 1986; 34:185–186.
20. Kwok PW: Evaluation and control of carbon monoxide exposure in indoor skating arenas. Can J Public Health 1983; 74:261–265.
21. Ouellet J-Y: Le monoxyde de carbone dans les arénas. Sherbrooke: Université de Sherbrooke, 1975.
22. Forbes WH, Sargent F, Roughton FJW: The rate of carbon monoxide uptake by normal men. Am J Physiol 1945; 143:594–608.
23. Lavoie R, Lévesque B, Dewailly E: Evaluation of an alveolar carbon monoxide analysis method—A reliable measurement to monitor environmental and occupational exposure to carbon monoxide. Clin Chem 1990; 36:160.
24. American Thoracic Society: Standardization of spirometry—1987 update. Am Rev Respir Dis 1987; 136:1285–1298.
25. Knudson RJ, Slatin RC, Lebowitz MD, Burrows B: The maximum expiratory flow volume curve: Normal standards, variability, and effects of age. Am Rev Respir Dis 1976; 113:587–600.
26. American College of Sports Medicine: Guideline for exercises testing and prescription. Philadelphia: Lea and Febiger, 1986.
27. Kleinbaum DG, Jupper LL, Muller KE: Applied regression analysis and other multivariable methods. Boston: PWS-KENT, 1987.
28. Gouvernement du Québec: Règlement sur la qualité du milieu de travail. Québec: Éditeur officiel du Québec, 1986.
29. Groupe d'étude de Kino-Québec sur le système de quantification de la dépense énergétique (GSQ). Rapport final. Québec: Gouvernement du Québec, Ministère du Loisir de la Chasse et de la Pêche, DE 175-79, 1979.
30. Révision des objectifs nationaux en matière de qualité de l'air ambiant pour le monoxyde de carbone: Teneurs souhaitables et acceptables. Ottawa: Comité consultatif fédéral-provincial sur la qualité de l'air, Ministère des Approvisionnements et Services Canada, 1987.
31. Review of the national ambient air quality standards for carbon monoxide. EPA. Final Rule, 40 CFR Part 50. Federal Register Sept. 13, 1985; 50:178–37484.
32. Environmental health criteria 13—Carbon monoxide. Geneva: World Health Organization, 1979.
33. Luckurst DG, French W: Carbon monoxide in indoor skating arenas. Can

Med Assoc J 1979; 121:1053–1056.
34. Green H, Bishop P, Houston M, McKillop R, Norman R, Stothart P: Time-motion and physiological assessments of ice hockey performance. J Appl Physiol 1976; 40:159–163.
35. Montgomery DL: Intensity of old-timer hockey play. Phys Sports Med 1982; 10:61–66.
36. Davis BP, Drenchen A: Carbon monoxide of concern in ice arenas. J Environ Health 1979; 42:120–122.

## AADS to Offer Oral Health Education for Elderly

The American Association of Dental Schools (AADS) is preparing an education packet to help older persons take better care of their oral health. Funding for the program is being provided by a $49,479 grant from the W. K. Kellogg Foundation.

Through the AADS project, copies of the oral health education packets will be distributed to 55 dental schools and 200 dental hygiene education programs nationwide. Beginning in the Fall 1990, the dental and dental hygiene students will give one-hour presentations at senior centers, clubs, churches, and volunteer organizations, with a special emphasis placed on reaching minority older persons.

The educational materials will be prepared by a team of dentists, dental hygienists, a social worker, and dental educators. By conducting the training, the students will gain first hand experience about oral health and its relation to the aging process, as well as learn to communicate effectively with elderly persons and to network with other health professionals.

For more information, contact the American Association of Dental Schools, 1625 Massachusetts Avenue, Suite 502, Washington, DC 20036; (202) 667-9433.