Smoking, Carbon Monoxide, and Coronary Heart Disease

Many studies have documented a significant association between cigarette smoking and the incidence of myocardial infarction and death from coronary heart disease, independent of other risk factors, especially in young and middle-aged men.1–5 Moreover, heavy cigarette smokers have a significantly higher incidence of myocardial infarction and mortality from coronary heart disease than light cigarette smokers. In addition, cigarette smokers who stop smoking have a significantly lower incidence of myocardial infarction and mortality from coronary heart disease than those who continue to smoke. Cigarette smoking has also been correlated with a shorter survival time in patients dying eight hours after the onset of a heart attack.9

Smoking cigarettes also causes patients with angina pectoris due to coronary artery disease to have a significant decrease in exercise performance before the onset of angina.7–9 Smoking high-nicotine cigarettes7 aggravates exercise-induced angina more than smoking low-nicotine8 or nonnicotine cigarettes.10 Smoking low-nicotine cigarettes8 aggravates exercise-induced angina more than smoking nonnicotine cigarettes.10

Smoking high-nicotine7, 11 or low-nicotine cigarettes8, 11 causes an increase in systolic blood pressure and in heart rate, consequently increasing the myocardial oxygen demand. This increase in systolic blood pressure and in heart rate does not occur after smoking nonnicotine cigarettes.9–11 However, smoking high-nicotine, low-nicotine, or nonnicotine cigarettes causes an elevated carboxyhemoglobin level,9, 11, 12 which decreases the amount of oxygen available to the myocardium. Therefore, anginal patients develop angina pectoris sooner after exercise following cigarette smoking for at least two reasons: increased myocardial oxygen demand in the presence of nicotine, and decreased oxygen delivery to the myocardium, whether or not nicotine is present.

Wald et al.13 have shown that carboxyhemoglobin levels in tobacco smokers correlate better with the presence of coronary heart disease than the smoking history. Cohen, Deane, and Goldsmith14 demonstrated an association between atmospheric carbon monoxide pollution in Los Angeles and case fatality rates for patients with acute myocardial infarction. Carbon monoxide exposure has also been suspected of playing a role in the pathogenesis of coronary atherosclerosis.15, 16 However, adequate prospective epidemiologic studies need to be done to determine the effect of atmospheric carbon monoxide pollution on the pathogenesis of coronary heart disease and on mortality from coronary heart disease.

Smokers who inhale develop high levels of carboxyhemoglobin. We observed that smokers who inhaled smoke from eight nonnicotine cigarettes, which they smoked at the rate of one cigarette every 30 minutes at their normal pace of activities, developed a mean increase in venous carboxyhemoglobin level from 1.58 to 7.79%.9 This increased carboxyhemoglobin level was associated with a decrease in exercise time until the onset of angina and with a reduction in the product of systolic blood pressure times heart rate at the onset of angina, with angina pectoris developing sooner, after less cardiac work.

Ayres et al.17 have shown that acute elevation of the venous carboxyhemoglobin level from a control level of 0.98% to 8.96% in patients with coronary heart disease and noncoronary heart disease caused a 20% average decrease in mixed venous oxygen tension. This greater reduction in mixed venous oxygen tension relative to the increase in venous carboxyhemoglobin level resulted from a leftward shift of the oxyhemoglobin dissociation curve, with tighter binding of oxygen to hemoglobin in the absence of carboxyhemoglobin. The increased carboxyhemoglobin level caused an increase in coronary blood flow in their patients with noncoronary heart disease but not in their patients with coronary heart disease. Myocardial oxygen extraction and extraction ratios also significantly decreased in their patients with coronary and with noncoronary heart disease, but the myocardial lactate extraction ratio significantly changed to lactate production only in their patients with coronary heart disease.

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Heavy atmospheric carbon monoxide pollution may also lead to increased carboxyhemoglobin levels. Carbon monoxide emission in motor vehicle exhaust is highest during idling and deceleration. Peak atmospheric carbon monoxide exposures have been reported to reach as high as 147 ppm in Los Angeles freeway traffic and 141 ppm in New York expressway traffic. The atmospheric carbon monoxide level also reached a peak level of 135 ppm at traffic intersections in Dayton, Ohio. We found that anginal patients who were driven for 90 minutes in Los Angeles County peak early-morning freeway traffic during the winter developed an increase in mean arterial carboxyhemoglobin level from 1.12% to 5.08%. Two hours after exposure to heavy freeway traffic, their mean arterial carboxyhemoglobin level was still 2.91%.

The increased arterial carboxyhemoglobin level caused by exposure to heavy freeway traffic caused a significant reduction in exercise time until the onset of angina pectoris, associated with a significant decrease in product of systolic blood pressure times heart rate at the onset of angina, immediately after breathing freeway air for 90 min and two hours after return from the freeway trip. Since our anginal patients could not adequately increase their coronary blood flow while exercising, and since their elevated carboxyhemoglobin level made less oxygen deliverable to the myocardium, their myocardial oxygen demand exceeded their myocardial oxygen supply, inducing angina pectoris sooner, after less myocardial work. That this significant decrease in exercise performance until the onset of angina pectoris, associated with a significant decrease in product of systolic blood pressure times heart rate at the onset of angina, was related to carbon monoxide exposure rather than to the stress of freeway travel was supported by the absence of these findings when compressed, purified air was supplied by mask to the same patients during an equivalent freeway trip.

We also observed that three of our ten anginal patients developed ischemic ST segment depression of at least 1 mm greater amplitude than in the control Holter electrocardiographic recordings while breathing freeway air during peak freeway traffic, whereas none of our ten anginal patients developed ischemic ST segment depression while breathing compressed, purified air during peak freeway traffic. The increased carboxyhemoglobin levels, plus exposure to other pollutants, and the stress of being driven during heavy freeway traffic may have precipitated the electrocardiographic abnormalities.

We found no significant difference in ischemic ST segment depression at the onset of exercise-induced angina after breathing freeway air in comparison with the control period or with the period after breathing compressed, purified air. However, ischemic ST segment depression at the onset of exercise-induced angina pectoris occurred earlier, after less exertion, and at a lower product of systolic blood pressure times heart rate in our anginal patients immediately and two hours after breathing freeway air when compared with the pretravel control periods or with the control period after breathing compressed, purified air.

Two very recent double-blind studies have also confirmed that carbon monoxide exposure in concentrations found during heavy atmospheric pollution aggravates exercise-induced angina pectoris. Anderson et al. demonstrated in a double-blind study that mean venous carboxyhemoglobin level in ten anginal patients who breathed 50 ppm of carbon monoxide for four hours increased from 1.3% to 2.9%. Their anginal patients had a significant decrease in exercise performance until the onset of angina pectoris after exposure to carbon monoxide compared to breathing compressed, purified air. Ischemic ST segment depression after exercise-induced angina pectoris in general appeared earlier and was deeper after breathing carbon monoxide compared to breathing compressed, purified air. However, these investigators did not quantify the amount of ischemic ST depression.

We observed in a double-blind study that ten anginal patients who breathed 50 ppm of carbon monoxide for two hours increased their mean venous carboxyhemoglobin level from 1.03% to 2.68%. After exposure to carbon monoxide, our anginal patients had a significant decrease from the control value in exercise time until the onset of angina pectoris, associated with a significant decrease in the product of systolic blood pressure times heart rate at the onset of angina, but the same phenomena were not observed after the patients breathed compressed, purified air. Ischemic ST segment depression was not observed in the Holter electrocardiographic recordings while the patients were breathing either carbon monoxide or compressed, purified air. We observed an increase in mean maximal ischemic ST segment depression from 1.30 mm to 1.45 mm after exercise-induced angina after breathing carbon monoxide compared to the control.
period. This increased ischemic ST segment depression after exercise-induced angina was not statistically significant. However, ischemic ST segment depression after exercise-induced angina occurred earlier, after less exertion, and at a lower product of systolic blood pressure times heart rate in our anginal patients after breathing carbon monoxide compared with the control periods or with the period after breathing compressed, purified air.

In summary, compelling evidence exists to indicate that cigarette smoking predisposes to coronary heart disease, causes an increased mortality from coronary heart disease, and aggravates angina pectoris. The medical profession should accept the responsibility to better educate the public against the hazards of smoking and to promote programs which will help people to stop smoking.

Heavy atmospheric carbon monoxide pollution also clearly aggravates angina pectoris due to coronary heart disease. Transient peaks of up to 90 ppm of carbon monoxide were measured passing across the face of a person who sat next to a subject who was smoking a cigarette for 10 minutes in an exposure chamber. Therefore, restrictions on smoking in public places may be necessary to protect patients with coronary heart disease. The medical profession should also assume leadership in educating the public on the need for measures such as mass transit systems (especially in congested urban areas with heavy atmospheric pollution), smaller and less polluting cars, and restricted use of the automobile in urban areas to protect the welfare of the significant segment of our urban population who have coronary heart disease.

Finally, the medical profession should assume responsibility for collaborative efforts with other members of the scientific community to obtain more comprehensive data to determine the safe levels of carbon monoxide and other pollutants in our environment for healthy people and for those with various coronary, pulmonary, and other disease states. Nonetheless, sufficient evidence already exists to indicate that we should act now.

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References


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