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Smoker's Heart

Increased incidence and severity of coronary artery disease, as manifested by myocardial infarction with an associated higher death rate in cigarette smokers, has been documented by a large number of epidemiologic studies.[1] Demonstration of underlying morphologic coronary atherosclerosis in cigarette smokers was first reported 11 years ago.[2] The study, based on information from autopsy protocols, showed that heavy cigarette smokers (two or more packs a day) had nearly three times more atherosclerotic changes than patients who never smoked regularly. A new prospective study, "Cigarette Smoking and Coronary Artery Disease," in *Chest* (70:697-705, 1976) covers 1,056 men who died from causes other than heart disease in a Veterans Administration hospital between 1965 and 1970. Patients with heart weights of more than 500 gm (to exclude hypertension) and diabetics were excluded from the study. The results show more than four times more atherosclerosis in two or more packs-a-day smokers vs the group that never smoked regularly. The increased incidence compared with the previous study is probably accounted for by the removal of the coronary arteries in the manner described by McGill and Strong,[3] and the opportunity for a more systematic study. The important new findings, however, were found in the microscopical slides. A total of 31,173 sections were prepared from the coronary arteries and arterioles. All the slides were read in a statistically designed fashion by one of us (O.A.). The major findings demonstrate a strong association between the amount of cigarette smoking and fibrous intimal thickening of the wall of the coronary arteries. Advanced fibrous thickening was found in 22.2% of two or more packs-a-day smokers and 0.8% in those who never smoked. The increase is greater for intramyocardial arteries than for subepicardial arteries. Atheroma was found only in the presence of fibrous thickening, but fibrous intimal thickening was found frequently in the absence of atheroma. The most substantial effects of cigarette smoking were found in the myocardial arterioles. Advanced hyaline thickening was found in 90.7% of two or more packs-a-day smokers, 48.4% of one to two packs-a-day smokers, and *not in any* of the group that never smoked regularly. An association of hyaline thickening with cigarette smoking has been demonstrated in other organs[4] and has been produced experimentally in dogs.[5] Hyaline thickening of an advanced degree in the myocardial arterioles with or without the associated fibrous intimal thickening in larger coronary arteries may properly be termed the "smoker's heart."

The mechanism through which the morphologic alterations are produced may be nicotine, carbon monoxide (acting either separately or together), or perhaps some other product of cigarette combustion. Favoring the nicotine hypothesis is the decreased coronary artery death rate in persons smoking low tar/nicotine cigarettes,[6] and a decreased amount of hyaline thickening in dogs smoking low tar/nicotine cigarettes, and the alteration of small myocardial arterioles in people with epinephrine-secreting tumors (pheochromocytoma).[7-9] It is well known that nicotine causes the release of epinephrine.[9,10]

We believe that these findings may explain some of the discrepancies between the clinical and autopsy findings, such as the presence of myocardial infarction without commensurate atherosclerosis of the larger coronary arteries.

OSCAR AUERBACH, MD
East Orange, NJ
HARRY W. CARTER, MD
Livingston, NJ
LAWRENCE GARFINKEL, MA
E. CUYLER HAMMOND, ScD
New York

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