Chapter 11

Cardiovascular Diseases

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INTRODUCTION

It has been suggested repeatedly that smoking may have adverse effects on the cardiovascular system. Recently, studies of large groups of people have shown that cigarette smokers in particular are more prone to die early of certain cardiovascular disorders than non-smokers. Chief among these disorders is coronary artery disease, and the present chapter deals mostly with this subject. The chapter begins with a summary of information about the acute effects of smoking on the cardiovascular system. This is followed by a brief account of coronary disease, its frequency in different kinds of people, and the many factors known or thought to affect the likelihood of its develop ment. The aim here is not to review critically our knowledge of coronary disease but only to give background for what follows. Next is summarized the information currently available from study of large population groups on the association of cigarette smoking with an increased tendency to have coronary disease. There follows a brief discussion of smoking and non-coronary cardiovascular disease. Finally, there is a short review of evidence relating to the question of whether cigarette smokers may, as a group, differ from non-smokers in ways not caused by smoking itself. Mortality ratios showing the association between cigarette smoking and deaths from cardiovascular disease, especially coronary disease, do not indicate the magnitude of the burden. This can be better appreciated from consideration of the following facts: cardiovascular disease deaths now total more than 700,000 annually in the United States. Of these more than 660,000 were due to heart disease, with more than 500,000 due to arteriosclerotic heart disease including coronary disease. The remaining approximately 40,000 were ascribed to disease of other parts of the cardiovascular system. Deaths from lung cancer total approximately 39,000. A mortality ratio of 1.7 for coronary heart disease among cigarette smokers in the seven prospective studies represents from 32.9 percent to 51.7 percent of all excess deaths, whereas the much higher lung cancer mortality ratio of 10.8 from the same studies represents only 13.5 percent to 24.0 percent of total excess deaths (Chapter 8, Tables 19, 25).

PERTINENT PHARMACOLOGY

The acute cardiovascular effects of smoking in man and experimental animals are like those caused by nicotine alone. A smoker who inhales gets usually 1-2 mg of nicotine from a cigarette (56, 57).

Low concentrations of nicotine stimulate sympathetic ganglia, and high concentrations paralyze them. Parasympathetic ganglia respond in the same way but are less sensitive. Nicotine can also have a sympathomimetic effect

by causing the discharge of norepinephrine and epinephrine from chromaffin cells in various tissues, including heart, vessels, and skin (10, 11, 9). In addition, nicotine produces effects reflexly by stimulating the chemoreceptors of the carotid and aortic bodies. When nicotine is given intravenously in increasing doses to dogs or cats the first effects, at about 1 microgram/kg body weight, are increased breathing and sympathetic stimulation, with predominant vasoconstriction, cardiac acceleration, and rise in blood pressure, resulting from stimulation of the aortic and carotid bodies (17). Doses of 4 to 8 micrograms/kg can stimulate pulmonary and coronary chemoreflexes which produce opposite effects. If all these receptors are inactivated, much higher doses are needed to evoke the cardiovascular effects of sympathetic stimulation, presumably through action on sympathetic ganglia or chromaffin tissue. Intravenous administration of nicotine in the experimental animal causes a discharge of epinephrine from the adrenal medulla, and in man heavy cigarette smoking produces an increased urinary excretion of catecholamines (84, 99).

Smoking 1-2 cigarettes causes in most persons, both smokers and non-smokers, an increase in resting heart rate of 15-25 beats per minute, a rise in blood pressure of 10-20 mmHg systolic and 5-15 mmHg diastolic (76, 78, 85, 86). and an increase in cardiac output of about 0.5 l/min/sq.m (75). There is a decrease in digital blood flow and a consequent drop in finger and toe temperature (31, 78. 103). The decrease in peripheral blood flow which normally follows smoking does not occur in a sympathectomized limb, indicating that the effect is mediated primarily by the sympathetic nervous system rather than through the release of catecholamines from other sites or the direct effect of nicotine upon the smooth muscle of the blood vessels themselves (103). Intravenous nicotine, and probably cigarette smoking as well, can produce a slight transitory increase in the blood flow to resting calf muscle (79).

In the dog, nicotine and cigarette smoke cause an increase in coronary flow as the blood pressure, cardiac output, and heart work increase (30, 53). These effects resemble those of epinephrine. Nicotine has been found to cause a transient decrease in cardiac oxygen utilization followed by a slight increase (53). Relatively little information is available about the effect of smoking on coronary blood flow in man. In normal subjects it is reported that cigarette smoking produces an early increase in coronary flow as heart work increases, but there is little change in oxygen utilization by the myocardium (2). With continued "steady state" smoking the coronary flow and cardiac oxygen utilization are maintained at the resting level in both normal subjects and persons with coronary heart disease, despite increased blood pressure, heart rate, and heart work (74). A larger experience must be gathered in this field before statements about the acute effects of smoking on the human coronary circulation can be made with assurance. The atherosclerotic rabbit heart, like the normal rabbit heart, shows an initial drop in coronary flow on administration of nicotine, but demonstrates less of a subsequent increase above the resting level than does the normal heart (97). These effects are said to be equivalent to those produced by norepinephrine in doses one-tenth as large as the nicotine dose.

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Little or no change in the electrocardiogram of most normal persons or cardiac patients, except for an increase in rate, is produced by smoking or by the intravenous injection of an equivalent dose of nicotine (82, 98). In some persons there is a slight depression of the S-T segment and a flattening of l-2 mm in the T wave of the limb leads. These changes are not like those associated with myocardial ischemia. Rarely in persons with true angina, an attack of pain is precipitated by smoking. An ill-defined syndrome consisting of chest pain, palpitation, and shortness of breath, known as "tobacco angina", has been described as occurring in smokers who do not have organic heart disease, but it is rarely diagnosed today (73, 82). Extrasystoles and other cardiac arrhythmias have been reported to be caused by smoking, but such cases appear to be unusual.

The ballistocardiogram obtained from a high-frequency table is sometimes changed by smoking a cigarette from a normal pattern to one said to be typical of coronary disease (78, 91). This phenomenon is rare in healthy persons below 50, becomes increasingly common with advancing years in apparently healthy persons. but is particularly prone to occur at any age in persons with actual coronary disease. The effect has been used as a "stress test" to help uncover coronary disease, but false positive and negative results are common. The ballistocardiographic changes on smoking have been variously interpreted as resulting from impaired myocardial contractility (78), from changes in the peripheral circulation (82), or from uncertain causes related to the physical properties of the high-frequency table as well as changes in the circulation.

Cigarette smoking causes an increase in the concentration of serum-free fatty acids in man (50), apparently mediated by stimulation of the sympathetic nervous system (51). Although continued administration of epinephrine to dogs over many hours can produce substantial increases in serum cholesterol, phospholipids, and triglycerides, such an effect has not yet been reported from nicotine or tobacco smoke (48, 92).

The clotting time of the blood can be decreased 50 percent or more in experimental animals by stimulation of the sympathetic nervous system or by administration of epinephrine (12, 13, 14), but attempts to demonstrate that cigarette smoking alters the clotting properties of the blood in man have been unsuccessful (5, 68). A decrease in platelet survival *in viva* has been found after smoking (68). Cigarette smokers have been reported to show substantial decreases in hematocrit, hemoglobin, and platelet counts after abstinence of l-2 weeks (25), but hemoglobin concentrations are alike in smokers and non-smokers of the same population group (4).

Attempts have been made to induce atherosclerosis in rats by the chronic administration of nicotine for periods up to a year without success (93).

Tobacco has antigenic properties (29, 43). Rats can he sensitized to tobacco extracts by intraperitoneal injection. Over a third of smokers demonstrate a positive "immediate" skin reaction to such extracts while only about 10% of non-smokers are said to give positive tests. The presence of serum reagins in persons with positive skin tests has been demonstrated by passive transfer techniques. Persons with thromboangiitis obliterans and smokers with occlusive vascular disease of other types are said to show a much higher incidence of positive skin tests than healthy smokers. The cardiovascular

diseases which have been related to smoking, however, do not in general resemble those usually ascribed to an immune mechanism.

In man and experimental animals smoking or the injection of nicotine causes increased secretion of antidiuretic hormone. The renal effects of this are easily demonstrable but the quantity of hormone secreted in response to smoking is probably too small to have significant vascular effects (17)

In summary, the acute cardiovascular effects of smoking and of nicotine closely resemble those of sympathetic stimulation, and to a considerable extent are mediated by excitation of the sympathetic nervous system. No additional or unique cardiovascular effects have been demonstrated which, in the light of our present understanding, seem likely to account for the observed association of cigarette smoking with an increased incidence of coronary disease.

GENERAL OBSERVATIONS ON CORONARY HEART DISEASE

Heart disease is the most common cause of death in our population, and coronary disease is the commonest variety of fatal heart disease (59). In 1961 there were 1,701,522 deaths from all causes in the United States. Heart disease deaths numbered 663,391 of which 502,351 were due to arterio-sclerotic heart disease.

The disorder consists of obstruction or narrowing of the coronary arteries, reducing the blood supply to the heart muscle. The underlying cause of the obstruction is coronary atherosclerosis, but an acute coronary artery occlusion is often caused by the formation of a blood clot in a diseased artery. The common manifestations of coronary disease are angina pectoris, recurrent brief attacks of chest pain caused by inadequate blood supply to the heart muscle; myocardial infarction, or necrosis of a portion of the heart muscle due to acute loss of blood supply; congestive heart failure, a chronic state caused by inability of the heart to pomp enough blood to satisfy the demands of the body; and sudden death resulting from cardiac standstill or ventricular fibrillation.

There are considerable differences in the prevalence of coronary heart disease in different countries, and often in different ethnic and socio-economic groups within a particular country (46, 62). The reported death rate of arteriosclerotic heart disease, which is primarily coronary disease, is higher in the United States than in other countries. It is also quite high in New Zealand, Australia, South Africa, Canada, and Finland, and moderately high in Great Britain. The death rate in Norway, Sweden, and Denmark is roughly half that in the high death rate countries (15). The death rate in Japan appears to be about one-sixth that in the United States, although persons of Japanese origin living in the United States are said to have a death rate similar to that of the general population of this country (52).

Because of changing diagnostic skills and revisions in nomenclature of disease, it is difficult to be certain of the change in incidence of coronary disease in the United States over the past few decades, but there is a general opinion that the incidence is increasing in this country and in England,

particularly in the younger male group (59, 62, 65, 83). In 1955 the mortality rate from arteriosclerotic heart disease was reported to be about 240 per 100,000. Although this is an increase of more than 50% over the rate in 1940, it has been estimated that less than 15% of the increase represented a real change in incidence of the disease, the remainder depending upon changes in diagnosis, in nomenclature and in the age of the population (59). Since 1955 the death rate from coronary disease (ISC 420) and from arteriosclerotic and degenerative heart disease (ISC-420 and 422) has continued to increase gradually. In 1960 the age-adjusted death rate from 420 and 422 was 330 per 100,000 for white males and 160 for white females (55).

Although the basic cause or causes of coronary heart disease are obscure, certain factors other than smoking are known or thought to predispose to the condition or to be associated with an increased incidence.

The incidence of coronary heart disease in men under 45 is about 5 times as great as that in women (Table 1) (15, 20, 59, 62). In both sexes the incidence increases with advancing years. After the menopause the incidence increases rapidly in women, and at age 80 the death rates from coronary disease are about the same for the two sexes. Coronary thrombosis plays a relatively more important role in precipitating myocardial infarction in young men than it does in old men (105). In studies of large population groups coronary disease has been associated with elevation of the serum cholesterol, hypertension, and marked overweight (19, 20, 24, 36, 46, 59, 62).

Some individual characteristics have been said to be associated with coronary disease. There is a significant familial tendency to develop it (36, 69, 81, 96). Persons with a mesomorphic constitution are said to be more vulnerable than endomorphs and ectomorphs (36, 62, 88). A coronary-prone personality has been described as the aggressive, competitive person who takes on too many jobs, fights deadlines, and is obsessed by the lack of adequate time for the performance of his work (33, 34, 35).

TABLE 1. --*Death rates per 100,000 from arteriosclerotic and degenerative heart disease* by sex and age, United States, 1958-60*

Age Group	Males	Females	Both Sexes
Under 35- - - - - - - - - - - - - - - - - - - -	3.3	1.2	2.2
35-44- -	90.2	18.3	53.3
45-54 -	353.7	79.3	213.5
55-64 -	928.5	314.5	610.2
65-74 -	2129.2	1082.0	1569.5
75 or over- - - - - - - - - - - - - - - - - -- - - -	4765.1	3738.4	4179.7

*Includes ISC numbers 420 and 422.

Source: WHO Epidemiological and Vital Statistics Report, Vol. 16, No. 2, 1963.

Certain occupations have been said particularly to favor the development of coronary disease, notably those which feature responsibility and stress (34, 81, 87). and which are sedentary in nature (7). Others (58, 72, 90) have not found that executives are more prone to coronary disease than non-executive personnel. Physicians have been said to have 3 or 4 times as much coronary disease as farmers or laborers (87), and general practitioners to

have 3 times as much as dermatologists (80). Occupations involving much physical activity are said to be protective (66, 67, 77). City life has been said to be more closely associated with coronary disease than suburban life, and men who drove more than 12,000 miles a year seemed, in one study, more prone to the disease than those who drove less (64).

It has been widely held, and occasionally denied, that a diet high in saturated fat predisposes to the development of coronary disease (46, 52, 69, 81). A correlation between the national incidence of coronary disease and the percentage of food calories available as saturated fat has been reported among those countries for which adequate data exist (46). The serum cholesterol tends to rise when saturated fat is added to the diet, and it falls significantly when unsaturated fat is substituted (46). It has also been suggested that general over-nutrition, rather than excess saturated fat predisposes to coronary disease, on the grounds that the correlation of coronary disease with total available calories or sugar consumption per capita is as good as that for percentage of calories in fat (106).

In general, it is apparent that multiple personal and environmental factors can markedly affect the incidence of coronary disease.

SMOKING AND CORONARY HEART DISEASE

Over the last two decades a considerable number of epidemiologic studies on different populations, employing different techniques, have shown with remarkable consistency a significant relationship between cigarette smoking and an increased death rate from coronary heart disease in males, particularly during middle life. There has been little dissenting evidence. The association of coronary disease with the use of tobacco in other forms has not been striking. The documentation for these statements is given in the following paragraphs. Particularly important is the information in Chapter 8, Mortality.

English et al. (26) found the incidence of coronary disease in male patients at the Mayo Clinic about 3 times greater in cigarette smokers than in non-smokers in the 40-59 year age range. but found little relation to smoking above 60. Russek (81) reported a similar relationship, but less striking, in young men with coronary disease. Mills (64) in a study of reported mortality in a Cincinnati population found that heavy smokers in the 30-59 year age range had twice as high a death rate from coronary disease as non-smokers. Male Seventh Day Adventists, who are non-smokers, were found by Wynder and Lemon (104) in a study based on hospital admissions to have significantly less coronary disease and to develop it later in life than the general male hospital population. Haag and Hanmer (37) reported that employees in the tobacco industry, who tend to smoke heavily, had a lower death rate for cardiovascular disease than the general population in their geographic region, but no report was made of mortality rates within the tobacco-worker group, divided by smoking habits. The study has been criticized on this and other grounds (16).

Large-scale prospective studies of mortality in British physicians (Doll and Hill, 21), United States males 50-69 recruited by volunteer workers

(Hammond and Horn, 38, 39, 40, 42) and V.A. Life Insurance policyholders (Dorn, 22) have confirmed the association of death from coronary disease with cigarette smoking. In the British study, a step-wise association was found between the amount of tobacco consumed (not entirely cigarettes) and the mortality from coronary disease. The association occurred in the 35-54 year age range, but not in older men. Hammond and Horn found a similar graded relationship between coronary deaths and cigarette smoking, the death rate being more than twice as great in men who smoked over a pack a day as in non-smokers. Men who had stopped smoking for more than a year at the start of the study had a coronary death rate lower than those who continued.

Studies on special groups of men, such as longshoremen (Buechley et al. 8) members of a fraternal order (Spain and Nathan, 89) and industrial employees (Paul et al. 71) which, in the latter two instances, incorporated clinical coronary disease, as well as coronary deaths, also have shown a relationship between coronary disease and smoking. The relationship was closer for men under 51 than for older men, and closer for myocardial infarcts and death than for angina pectoris (70, 89).

The long-term prospective studies of cardiovascular disease in Framingham (19) and in Albany (24) which have featured a painstaking search at regular intervals for clinical manifestations of disease, have, on pooling the data (Doyle et al. 23) shown a threefold increase in the incidence of myocardial infarction and coronary deaths in men who are heavy cigarette smokers as compared to non-smokers, pipe and cigar smokers, and former cigarette smokers. In the pooled data the incidence of angina pectoris did not show a significant association with cigarette smoking. The lack of this particular relationship had been suggested on the basis of clinical experience (White and Sharber, 102).

An apparent interplay of factors relating to smoking and occupation turned up in a short-term study of the development of coronary heart disease in a general North Dakota population (Zukel et al., 107). Farmers had about half the incidence of myocardial infarction experienced by others. In farmers, smoking had no appreciable effect on the incidence of infarction, but in others the incidence of infarction was twice as high among smokers as among the non-smokers. The farmers who smoked cigarettes smoked less heavily than males in other occupational groups.

In Chapter 8, Mortality, there is summarized the most recent information available from 7 large completed or current prospective smoking and death rate studies (Doll and Hill; Hammond and Horn; Dorn; Dunn, Linden and Breslow; Dunn, Buell and Breslow; Best, Josie, and Walker; and Hammond). The median mortality ratio for coronary disease of current cigarette smokers to non-smokers is 1.7 (range 1.5-2.0).

Table 2 presents data from some of the large prospective studies on the ratio of mortality rates due to coronary heart disease of male smokers to non-smokers, by age and amount smoked. The ratios tend in general to increase with amount smoked and to decrease with advancing age.

The data from the first 22 months of Hammond's (41) current study help to show the size of the coronary problem. For this purpose, actual numbers of deaths may be more informative than mortality ratios. Of nearly

TABLE 2.—*Ratios of mortality rates for coronary heart disease, male smokers to non-smokers, by age and amount smoked, in selected studies*

HAMMOND AND HORN—1958 (42)

Age Group	Cigarettes smoked per day		
	Less than 10	10-19	20 and over
50-54	1.4	2.0	2.5
55-59	1.4	2.0	2.5
60-64	1.2	1.9	1.9
65-69	1.3	1.6	1.6
Total (age adjusted)	1.29	1.89	(20-39) 2.15 (40+) 2.41

BUECHLEY, DRAKE, BRESLOW—1958 (8)*

35-44		2.0
45-54		2.8
55-64		1.8
65-74		0.9

FRAMINGHAM STUDY—1963 (47)

	(less than 20) 1.5	(20 and over) 3.2
30-62	1.5	3.2

DORN—1959 (22)

Total (age adjusted)	1.32	1.76	1.75

DOLL AND HILL—1956 (21)

Age Group	Grams of tobacco smoked per day		
	1-14 Grams	15-24 Grams	25 or more Grams
35-54	2.2	3.3	4.2
55-64	0.9	0.6	1.0
65-74	1.0	0.9	1.3
75+	1.3	1.5	1.6
Total (age adjusted)	1.1	1.1	1.4

*Persons smoking 1 pack per day or more compared with those smoking less than 1 pack per day (including non-smokers).

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10,000 deaths of men aged 45-79, 46 percent were ascribed to coronary disease. 51.7 percent of the 2,630 "excess deaths" associated with cigarette smoking were caused by coronary disease. In approximate terms, nearly half of middle-aged and elderly males in the United States die of coronary disease. About half of these males smoke cigarettes. Cigarette smokers have been found in several studies to have 1.7 times as high a coronary death rate as non-smokers. If cigarettes actually caused the additional coronary deaths of smokers, they would account for many deaths of middle-aged and elderly males in this country, Like other studies (19, 21, 22, 23, 42) this one shows that the ratio of smokers' coronary death rates to those of non-smokers increases progressively with the daily cigarette consumption. In addition, at each level of consumption   ratio increases with the amount of inhalation reported by the smokers. Others (21, 23, 26, 89) have indicated

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that the risk of death from coronary disease in male cigarette smokers relative to that in non-smokers is greater in middle age than old age, and Hammond's current study supports this. The mortality ratio was 3.09 in the age range 40-49, and in successive decades was 2.20, 1.58, and 1.38.

Men who stop smoking have a lower death rate from coronary disease than those who continue (23, 42, 47). In the study of Hammond and Horn (42) the decrease in death appeared only after a year.

Angina pectoris is less closely related to cigarette smoking than myocardial infarction and sudden death. In the combined Albany-Framingham experience (23), angina pectoris showed no over-all relationship with smoking, and the association has not been strong in other studies (71, 89).

In summary, a significant association has been established between cigarette smoking and the incidence of myocardial infarction and sudden death in males, especially in middle life, in population groups whose members appear so far to be similar except for smoking habits, The question of whether they are, in fact, similar except for smoking is, of course, basic to the problem of whether cigarette smoking actually promotes the development of coronary disease or whether it is closely associated with some other factor or factors which promote the development of coronary disease. It has been pointed out that angina pectoris, which indicates advanced coronary atherosclerosis, is less closely associated with cigarette smoking than is myocardial infarction, and that this suggests that any etiologic role of smoking in myocardial infarction should relate more to acute occlusive mechanisms, such as intravascular thrombosis or coronary spasm, than to the development of chronic arterial disease.

SMOKING AND NON-CORONARY CARDIOVASCULAR DISEASE

In surveys of large groups cigarette smoking has not been found to be associated with an increased prevalence of hypertension (3, 4, 19, 47, 49). The study of Hammond and Horn (40, 42) did not show an increased death rate from hypertension in smokers. However, Dorn (22) found that the death rate of cigarette smokers from hypertension with heart disease was 1.53 times that of non-smokers, and from hypertension without heart disease, 1.41 times that of non-smokers. Hammond's current study shows similar figures (41). Smoking has not been found to be associated with an increased mortality rate from chronic rheumatic heart disease (22, 41, 42).

Hammond and Horn (42) found a moderate increase in the mortality rate from cerebral vascular disease in cigarette smokers as compared to non-smokers (ratio 1.30). Dorn (22) reported a ratio of 1.33, and Hammond (41) a ratio of 1.43. Although non-syphilitic aortic aneurysm is a relatively infrequent cause of death, the mortality ratio for smokers to non-smokers in this diagnostic category is large in relation to the ratios in other cardiovascular disorders. In the study of Hammond and Horn (42) it was 2.72, and in Hammond's current study (41) it is 3.10.

It has been reported (100) that diabetic males who smoke have a 50% greater incidence of clinically detectable arteriosclerosis obliterans in the legs than those who do not smoke. In general, however, there is little information about the relation of smoking to peripheral arteriosclerosis. Most experienced clinicians advise patients with obliterative peripheral arterial disease to stop smoking 145).

Buerger's disease, or thromhoangiitis obliterans, has been traditionally associated with smoking. and the literature contains numerous clinical reports describing the arrest of Buerger's disease when smoking is stopped and its reactivation on resumption of smoking. The existence of Buerger's disease as an entity separate from arteriosclerosis obliterans has been recently challenged (101,) but well defended (61).

It is apparent that much more work will have to be done to determine what relationship may exist between non-coronary occlusive vascular disease, aneurysmal disease. and smoking.

CHARACTERISTICS OF CIGARETTE SMOKERS

If it could be shown that cigarette smokers and non-smokers had significant constitutional differences apart from any differences that might be caused by smoking itself, then a possibility would exist that some predisposition of smokers to a particular disease might also be of constitutional origin and not caused by smoking. Cigarette smokers have, in fact, been found to differ as a group from non-smokers, but the differences, such as serum cholesterol concentration and resting heart rate, could have resulted from the smoking habit itself, so far as present knowledge indicates.

The concentration of serum cholesterol has been found to be slightly higher in smokers than in non-smokers by a number of investigators (6, 18, 49, 63, 95), but others have found no relationship (1, 54). Dawber (19) found not only that serum cholesterol was higher in smokers than in non-smokers but also that it remained higher in those who stopped smoking.

Smokers tend to be leaner than non-smokers, but to gain when they stop smoking (3, 18, 49).

A few personality differences have been reported between cigarette smokers and non-smokers. Friedman's type A men (the coronary type) tended to be heavy smokers (33.) Smokers are said to be more easily angered and to eat more when under stress (94). They have been reported to marry oftener, to change jobs more frequently, to be more often hospitalized, and to participate more actively in sports than non-smokers (60).

Thomas (94, 95) has reported that the parents of medical students who smoke have a significantly higher incidence of arteriosclerotic and hypertensive cardiovascular disease than parents of non-smokers. Clearly, this finding is open to more than one interpretation.

Smokers tend to have a higher heart rate than non-smokers (3, 94).

The matter of constitutional predisposition to smoking has been investigated in twins. It has been found (27, 28, 32) that the smoking habits of monozygotic twins are significantly more alike than those of dizygotic twins. even when members of a twin pair are brought up separately.

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In spite of some bits of suggestive evidence the existence of basic constitutional differences between smokers and non-smokers is not presently established. The constitutional hypothesis, which links smoking and predisposition to disease, is discussed in detail in Chapter 9, Cancer.

PSYCHO-SOCIAL FACTORS OF SMOKING IN RELATION TO CARDIOVASCULAR DISEASE

Even less conclusive information is available on the role of psycho-social factors of smoking in relation to cardiovascular disease. Studies which have focussed on this are limited in number according to Heinzelmann (44). Even fewer, he found, are those which have specifically examined the relative weight of these variables or their interaction. Reviewing those available, he observes that the evidence is highly fragmentary and uncertain. The findings suggest that the relationship between smoking behavior and coronary heart disease may reflect the influence of stress factors and/or personality mechanisms. However, they permit no definitive statements with respect to the relative role of pyscho-social factors and smoking in relation to etiology of the disease.

SUMMARY

Smoking and nicotine administration cause acute cardiovascular effects similar to those induced by stimulation of the autonomic nervous system, but these effects do not account well for the observed association between cigarette smoking and coronary disease. It is established that male cigarette smokers have a higher death rate from coronary disease than non-smoking males. The association of smoking with other cardiovascular disorders is less well established. If cigarette smoking actually caused the higher death rate from coronary disease, it would on this acconnt be responsible for many deaths of middle-aged and elderly males in the United States. Other factors such as high blood pressure, high serum cholesterol, and excessive obesity are also known to be associated with an unusually high death rate from coronary disease., The causative role of these other factors in coronary disease, though not proven, is suspected strongly enough to be a major reason for taking countermeasures against them. It is also more prudent to assume that the established association between cigarette smoking and coronary disease has causative meaning than to suspend judgment until no uncertainty remains.

CONCLUSION

Male cigarette smokers have a higher death date from coronary artery disease than non-smoking males, but it is not clear that the association has causal significance.

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