DIAGNOSIS AND TREATMENT OF
Cardiovascular Disease

VOLUME 2

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F. A. DAVIS COMPANY, *PUBLISHERS*
PHILADELPHIA
1957

Library of Congress
Catalog Card No. 57-6921

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CHAPTER PAGE

Cardiovascular Disease

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Coronary Disease Including Angina Pectoris

Introduction: The subject of coronary insufficiency, coronary disease, and angina pectoris should be a most interesting one, not only because the majority of patients seen by a physician will sooner or later suffer with such a pathological picture but also because a large percentage of physicians must expect to die with this condition. It therefore behooves us to learn as much as we can concerning its diagnosis, prevention, and treatment.

One of this chapter's main objects is to leave with the physician a more optimistic attitude towards the future of individuals suffering with coronary insufficiency, coronary disease, angina pectoris, or a healed coronary occlusion. The term coronary disease implies atherosclerotic changes of the coronaries with some diminution of the lumen. The term coronary insufficiency implies inability of the coronary vessels to deliver enough blood constituents to the myocardium to supply its needs with or without actual coronary disease. Coronary insufficiency may occur secondarily in patients suffering from pathology in the first part of the aorta, secondary anemia, aortic valvular insufficiency, and other conditions that impose an increased work load upon the normal myocardium.

At this point the facts should be stressed that usually the etiologic factor in coronary insufficiency, coronary disease, angina pectoris, or a coronary occlusion is one pathological picture, namely arteriosclerosis or atherosclerosis or both together with or without hypertension.

To urge optimism may seem strange when one realizes the enormous toll this disease is taking in our country. But from a selfish standpoint we should be optimistic. Such an attitude not only helps us develop a satisfactory philosophy towards life while suffering from coronary disease, but also prevents our patients from becoming unnecessarily discouraged. If so discouraged they may visit advertising quacks or those not adequately trained in medicine who may tell them there is nothing wrong with their hearts and receive credit for successful treatment, since often these patients can carry on useful active lives for many years. In this respect recent work by many investigators has given us a much clearer picture of this disease. Dr. Herman T. Blumgart has emphasized the race between the gradual occlusion of atherosclerotic arteries and the development of adequate collateral circulation. If the latter keeps abreast of the stenosing process,

R. Vieussens first correctly described the coronary vessels in 1715. In addition he noted the diagnostic features of pericardial effusion and gave the first description of aortic insufficiency (1695) and mitral stenosis (1705).

Edward Jenner, famous for discovery of vaccination against smallpox, was the first (with C. H. Parry) to associate coronary artery disease with angina (1788) proving his point when an autopsy revealed ossified and narrowed coronaries in the heart of his friend, John Hunter.

Heberden, in 1768, in a scholarly treatise, gave the first clear-cut clinical description of angina pectoris, establishing the condition as a disease entity.

Brunton, in 1871, noting a rise of the blood pressure in some patients suffering from angina, was the first to successfully employ nitrites in the treatment of the condition.

Herrick (1912) described coronary occlusion long regarded as an occasional necropsy finding and, separating it from angina, firmly established the features of sudden obstruction of the coronary arteries as a clinically recognizable syndrome.

FIGURE 1. Pioneers in the Study of the Coronary Circulation

symptoms may be minimal or never arise during life despite postmortem examination that may reveal severe major occlusions of the coronary arteries with minimal scarring of the myocardium. If, however, the collateral circulation lags or the occlusive process quickens, anginal symptoms may appear, only to vanish later as the collateral circulation regains ground. If the diseased artery occludes suddenly or an exceptional load is put upon the heart, an acute myocardial infarction may occur. Finally, pain that persists following an infarct may disappear eventually because of the opening up of new channels. With this in mind, we can and should give the patient a much more cheerful outlook than we could before such a concept was developed.

The head of one of the largest insurance organizations in this country has said, "It seems to me that most of the people you advise us to reject for life insurance because of their hearts act as pallbearers for the ones you tell us to accept." This is a challenge. It seems natural for an individual who knows he is not 100 per cent healthy from a cardiovascular standpoint to take such care of himself that he may live longer than the individual who considers himself sound. This was first brought home to the author while working with Sir James Mackenzie in 1920 at St. Andrews in Scotland. At that time the "beloved physician" was sixty-seven years old. Our attitude towards sudden death from coronary occlusion has definitely changed in the last few decades. Recent reports suggest that less than twenty-five per cent die suddenly in their first attack of myocardial infarction. Statistics compiled from cardiologists and life insurance companies indicate that the survival following an initial myocardial infarction is much higher than previously expected. The rate of survival without hypertension varies from fifty to eighty per cent at the end of five years and ten to fifty-seven per cent at the end of ten years with slightly lower figures for coexisting hypertension. In addition, most of the patients following their initial infarct are able to return to full-time work, the self-employed in over ninety per cent of instances, others in from fifty to eighty per cent. These studies include manual laborers as well as sedentary workers.

He gives his own case history: "A doctor active in a country practice. In 1901, at age forty-eight, after running a short distance, heart became very irregular (auricular fibrillation). The attack lasted two hours and has not recurred up till now (1923). Since he was forty years of age, he has noticed extrasystoles. Beyond playing golf, has taken no violent exercise. In 1906, at age fifty-five, he had a severe attack of pain across the chest and into left arm. The attack lasted two hours when he fell asleep after 0.6 Gm. (10 grains) of veronal. Pain could be easily provoked at times under special circumstances as walking in the cold air or after meals. He found that walking rapidly for half a mile invariably produced this sensation. Yet he can play golf in cold and windy weather in comfort—the reason being that the effort is not continuous. Heart dulness extends just beyond the left nipple line. B.P. has varied during the past few years 140 to 170.

"At age seventy he still leads a fairly active life and, having noted the circumstances that provoke the pain, is able to go about in comfort. As soon as he stops walking it begins to pass off and in one or two minutes it is entirely gone and he can walk quietly in comfort. Occasionally has felt slight aching in left jaw and left side of tongue with an increased flow of saliva which precedes the pain in the chest."

FIGURE 2. Sir James Mackenzie. Approaching on the Royal and Ancient Golf Course at St. Andrews, Scotland, June, 1920, twelve years following his coronary thrombosis. His score was 82.

Sir James Mackenzie, at the age of sixty-eight, was still playing golf. In his youth he had been a scratch player from the Royal and Ancient Golf Club, and he played right up to 1922, fourteen years after his myocardial infarct. He had to stop then because of breathlessness. He died in 1925, seventeen years after his first coronary thrombosis.

Finally, a few words are in order about the importance of coronary disease to the population of the United States. Due to the recent preventive and therapeutic advances in medicine, the life span of our citizens is increasing tremendously. This increased duration of life brings with it mixed blessings; the mortality from cardiovascular renal diseases in 1955 accounted for three out of every four deaths reported among the Metropolitan Life Insurance policy holders. About thirty per cent of the deaths were directly attributable to atherosclerotic, arteriosclerotic, and degenerative heart disease. In other words, coronary artery disease at the present time is the leading killer.

ETIOLOGY

One of the biggest advances in the prevention, diagnosis, and treatment of cardiovascular disease in the last fifteen or twenty years has been a recognition of the etiological factors so that now no physician is justified in prognosing and treating a case unless he is as nearly positive as he possibly can be of just what the etiological factor is. These have now been narrowed to four main groups: (1) Congenital. (2) rheumatic, (3) syphilitic, and (4a) atherosclerotic, (4b) arteriosclerotic (with or without hypertension). Although coronary insufficiency can develop secondary to any of the first four factors, our primary concern is with the atherosclerotic group. These are the patients who develop signs of coronary insufficiency with angina pectoris and myocardial infarction with or without coronary occlusion.

FIGURE 3. This male patient experienced an anterior myocardial infarction in October 1938 at age forty-six. After six months he returned to his former occupation of building truck bodies. He needed nitroglycerin at times until 1941 when he had a posterior infarction. Five weeks after this second infarct, he returned to his job but needed nitroglycerin two or three times a week until January 1945. In December 1946 he was found to have diabetes with a blood sugar of 247. He required nitroglycerin frequently until 1951 but has needed none since then. During the summer of 1953 he had diarrhea and lost 9 Kg. (20 pounds). In November 1953 a combined abdominoperineal resection of an adenocarcinoma of the rectum was performed and the colostomy is functioning satisfactorily at the present time. Six months to the day following his operation he went fishing and caught a 5.4 Kg. (12 pound) striped bass.

Atherosclerosis is no longer dogmatically identified with aging. It is now clearly established that it is a disease. Although artherosclerosis may

strike any arterial vessel in the body, its most important manifestation is in the coronary arteries of men between the ages of thirty and sixty and next in frequency in the cerebral vessels and the aorta. It is rare to find symptoms or signs of coronary insufficiency in women until past the

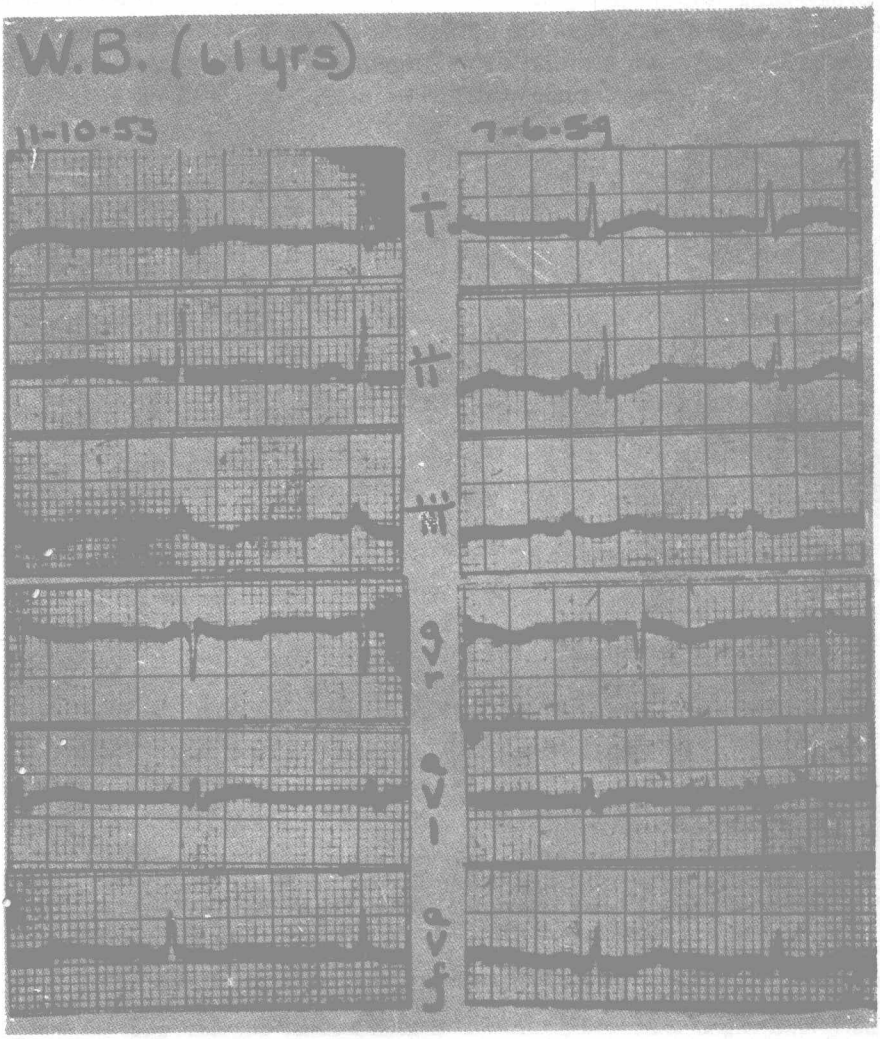

FIGURE 4. Electrocardiograms of W. F. B. It is interesting that the above electrocardiograms remained abnormal after 1941. The tracings on the left above were taken just before his abdominal operation. Eight months later they had returned to normal! He died a metastatic death on March 24, 1956.

menopause unless they are suffering from hypertension, diabetes mellitus, or some chronic valvular heart disease. Studies in the last twenty-five years have clearly established that atherosclerosis is a disease following upon alterations in cholestrol—lipid-lipoprotein metabolism. Population groups and experimental animals ingesting a diet rich in cholesterol-lipid over the life span manifest a tendency for plasma cholesterol to rise postnatally, remain at an elevated level in the early decades, and rise further

in the later decades. Such population groups invariably experience an extensive morbidity and mortality due to atherosclerosis. Reduction in such diets as caused by experimental withdrawal or the European experience during World War II reverses this trend of atherosclerosis and the concomitant morbidity and mortality. It should be noted in this regard that the present day "normal" American diet—rich in cholesterol-lipid (derived in large measure from dairy and poultry products) is a relatively recent innovation in nutrition.

Thus, we see that there are at least two and possibly more factors involved in the production of atherosclerotic changes in human arteries. The present concept lists the following:

1. An inherited factor and/or factors.
2. A high fat diet rich in cholesterol-lipid.
3. Sparing effect of estrogens.
4. Diabetes mellitus.
5. Inhalation of tobacco products (questionable).
6. With hypertension, inheritance of the "spasmogenic aptitude" (?).

The well known evidence of a familiar tendency suggests the first factor. The second factor has already been discussed and is available in the extensive literature. The estrogen sparing effect has been demonstrated experimentally as well as circumstantially in human morbidity and mortality rates. The role that diabetes mellitus plays in the production of atherosclerosis is well known, even under rigid dietary control and well adjusted glucose blood levels. There is some question whether the disease itself produces the changes or whether the administration of insulin to diabetics over the life-span exerts a significant artherogenesis-intensifying influence. Further studies are needed. The role of tobacco in the production of atherosclerosis, particularly of the coronary arteries, is a highly controversial one at the present time. Again, further well controlled studies of a more objective and clinical nature are necessary. The role of heredity or inheritance of emotional approach to life is a hard one to rule in or out. Any physician with an extensive practice cannot have failed to observe certain families in which from generation to generation there is an alarmingly high death rate among the male members. In some families all the males in successive generations die before the age of sixty from coronary artery disease. Whether this is due to an actual inherited anatomical weakness of the coronary vessels and/or the "spasmogenic aptitude" approach to life is a highly controversial question.

There is no doubt that frequent emotional upsets, long hours of nervous tension and mental concentration, inadequate vacations, excessive or prolonged physical effort indulged in sporadically by those past a certain age, and excess of stimulants, may play some role in the production of angina pectoris and ultimate myocardial infarction with or without coronary occlusion or hypertension alone or accompanied by atherosclerosis. However, it is highly unlikely that the psyche can ever produce coronary atherosclerosis as it has never been conclusively demonstrated that there is such an entity as spasm of the coronary or cerebral arteries. It is much