INTENSIVE CORONARY CARE

A MANUAL FOR NURSES

Third Edition

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INTENSIVE CORONARY CARE

A Manual for Nurses

Preface

The system of intensive coronary care proposed in the original edition of this book (1965) is now a well established, standard method of hospital care throughout the world; indeed very few medical advances have been accepted as readily or with as much enthusiasm. It has been our premise from the outset that intensive coronary care is primarily and above all a system of specialized nursing care. The effectiveness of the plan in saving lives of patients with acute myocardial infarction depends finally on the ability of nurses to function as decision-making members of the coronary care team, capable of acting on their observations and judgment, particularly in emergency situations or whenever therapeutic decisions cannot be delayed. By demonstrating remarkable competence in this demanding role (and accepting the responsibilities that accompany it) coronary care nurses have broadened the horizons of clinical nursing, and have earned the sincere respect of their colleagues and patients.

We are extremely grateful for the extraordinary reception accorded the two previous editions of this book. *Intensive Coronary Care–A Manual for Nurses* has served as the standard textbook of coronary care nursing for the past 12 years; it has been translated into four languages, and more than 700,000 copies are in print.

In preparing this third edition we have attempted to increase the scope and depth of the presentation while preserving its simplicity and clarity. In effect, the style and format of the book remain unchanged but the text has been revised and expanded considerably. The revisions and additions are designed not only to present new concepts of coronary care that have evolved since the last edition but also equally important, to keep pace with the growing sophistication and quality of nursing education and nursing practice. To this end we have included detailed information about the pathophysiology of heart failure and cardiogenic shock, the pharmacology of antiarrhythmic drugs and diuretic agents, hemodynamic monitoring, coronary bypass surgery, and new concepts of the early management of acute myocardial infarction, among many other subjects involved in modern coronary care nursing.

We are indebted to Mrs. Elizabeth Meholick for her indispensable help in preparing this manuscript for publication.

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Philadelphia, Pennsylvania
September, 1977

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INTENSIVE CORONARY CARE

A Manual for Nurses

Contents

1

Coronary Heart Disease

When the incredible complexity of the human system is considered along with the vast number of possible sources of illness and death, it seems incongruous that the life of so many depends finally on the health of two small arteries; but the fact is undeniable. Disease of the coronary arteries has become the single greatest threat to life in industrialized countries throughout the world. In the United States, for example, more than 600,000 deaths a year—or one-third of *all* deaths—are directly attributable to this one disease.

As the sole blood supply to the heart musculature (myocardium), the coronary arteries assume extreme importance. Any significant interference with blood flow through these vessels can impair the entire function of the myocardium, with dire consequences including sudden death. Before describing the clinical aspects of coronary disease it is pertinent first to consider the coronary arteries and the basic disease process that affects them.

THE CORONARY ARTERIES

The two coronary arteries, the left and right, arise from the aorta just above the aortic valve. The left coronary artery then divides into two large branches: the left anterior descending artery and the left circumflex artery. The relationship of these three arteries is shown in Figure 1.1.

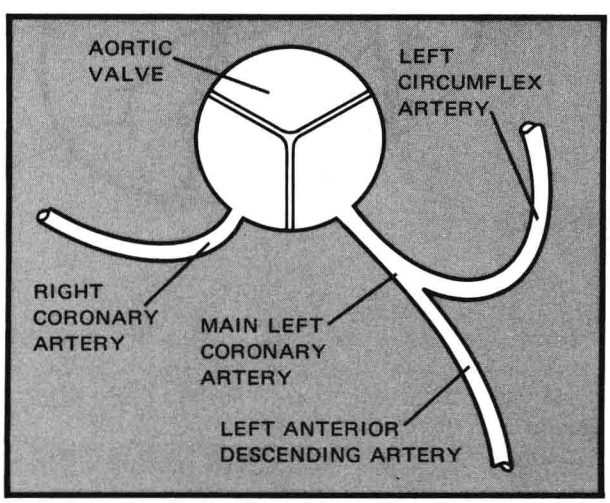

Figure 1.1. The coronary arteries as viewed from above (looking down at the aortic valve).

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Each artery supplies a different area of the heart. Briefly, the left anterior descending artery supplies most of the anterior wall of the left ventricle, the anterior portion of the interventricular septum, as well as the anterior wall of the right ventricle. The left circumflex artery supplies the lateral aspect of the left ventricle and the left atrium. The right coronary artery supplies the right atrium and the right ventricle along with the posterior portions of the left ventricle and interventricular septum. The arteries lie on the outer surface of the ventricles and give off numerous branches that penetrate all parts of the heart (Fig. 1.2). The terminal branches of the arteries have many interconnections, forming an extensive vascular network throughout the myocardium.

The function of the coronary arteries is to bring oxygen-carrying blood to the myocardium, oxygen being an essential ingredient in producing the energy the heart requires to contract. As a pump that works incessantly (contracting more than 100,000 times a day), the myocardium has very great oxygen needs. This constant demand can be met only by an adequate coronary blood flow. Indeed, 250 cc of blood per minute—or 360 liters per day—pass through the coronary arteries to oxygenate the myocardium.

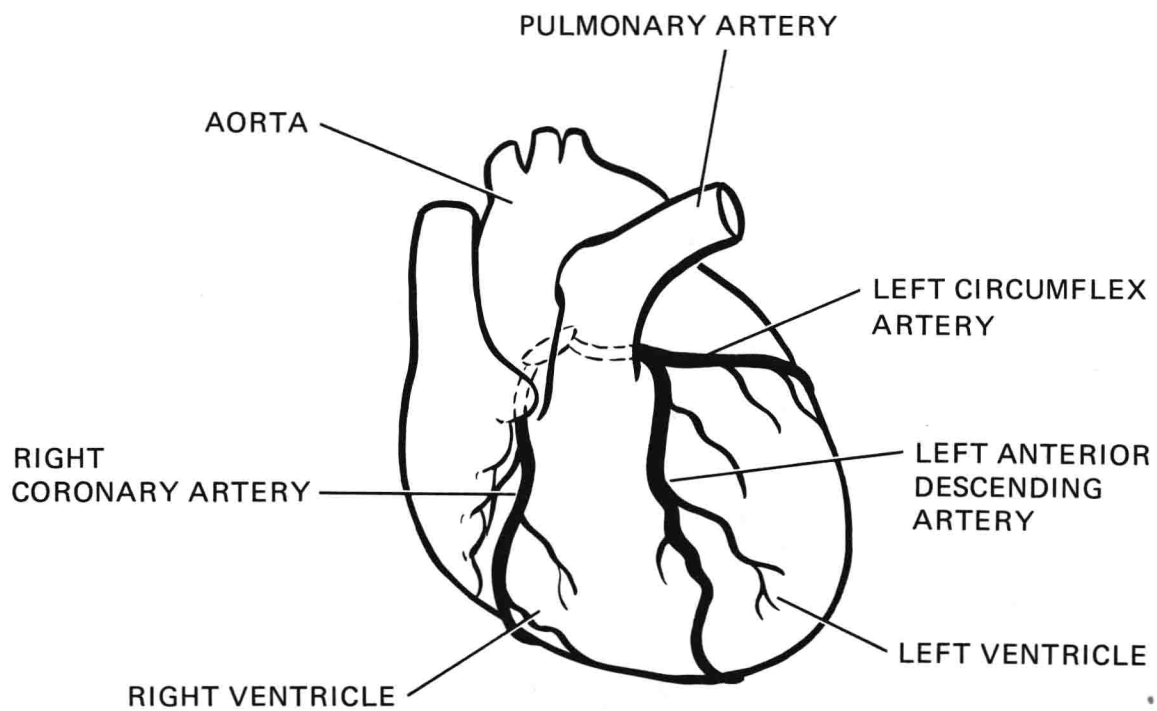

Figure 1.2. Coronary circulation.

CORONARY ATHEROSCLEROSIS

The primary disease affecting the coronary arteries is atherosclerosis, a process in which fatty substances (particularly cholesterol) deposit as plaques along the inner lining of the vessels and narrow the passages. If the narrowing reaches a stage where the blood flow through the arteries is insufficient to meet the oxygen demands of the myocardium, then coronary heart disease (CHD) is said to exist.

Coronary atherosclerosis usually develops gradually over a period of years. However, the process begins at an early age so that by adulthood most men (and women, to a lesser degree) have some evidence of atherosclerosis in the coronary arteries. Autopsy studies have shown, for example, that among young American soldiers (average age of 22 years) killed in action during the Korean war nearly 80% had definite signs of coronary atherosclerosis. It is essential to realize, however, that the critical determinant of coronary heart disease is not the mere presence of atherosclerosis but rather the extent of arterial narrowing and the reduction in blood flow the lesions produce. Atherosclerosis can be categorized into four grades according to the degree of arterial obstruction. Grade 1 atherosclerosis indicates that the diameter (lumen) of the artery is reduced by no more than 25%; grade 2 represents a 50% reduction, grade 3 a 75% reduction, and grade 4 complete (100%) obstruction of the vessel (Fig. 1.3). It is believed that an obstruction of at least 75% is necessary to produce a significant reduction in coronary blood flow; lesser degrees of narrowing can usually be tolerated without affecting myocardial function. Obstruction may occur in any (or all) of the coronary arteries, but involvement of the left anterior descending artery is particularly dangerous. This vessel supplies a much larger portion of the total myocardial mass than the right coronary and left circumflex arteries and therefore has the greatest blood flow.

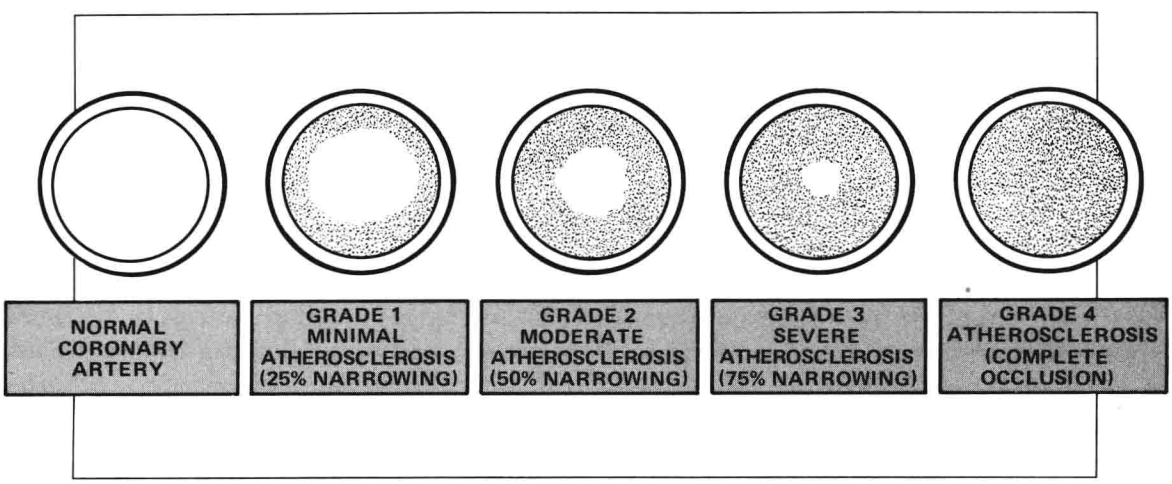

Figure 1.3. Criteria for the four grades of atherosclerosis.

Until recent years there was no way (except at autopsy) to determine the degree of arterial obstruction or the vessels involved. With the introduction of coronary arteriography, a technique which permits the arteries to be visualized by x rays, it is now possible to identify the site and extent of atherosclerotic lesions with reasonable accuracy. The procedure involves the insertion of a catheter into the aorta (by way of a peripheral artery) and the injection of a radiopaque dye through the openings (ostia) of the two main coronary arteries. As the dye is being injected a rapid series of x-ray films

are taken to outline the arterial tree; advanced lesions can be readily detected in this way. Figure 1.4 compares a normal coronary artery with one that has a 90% obstruction in one segment.

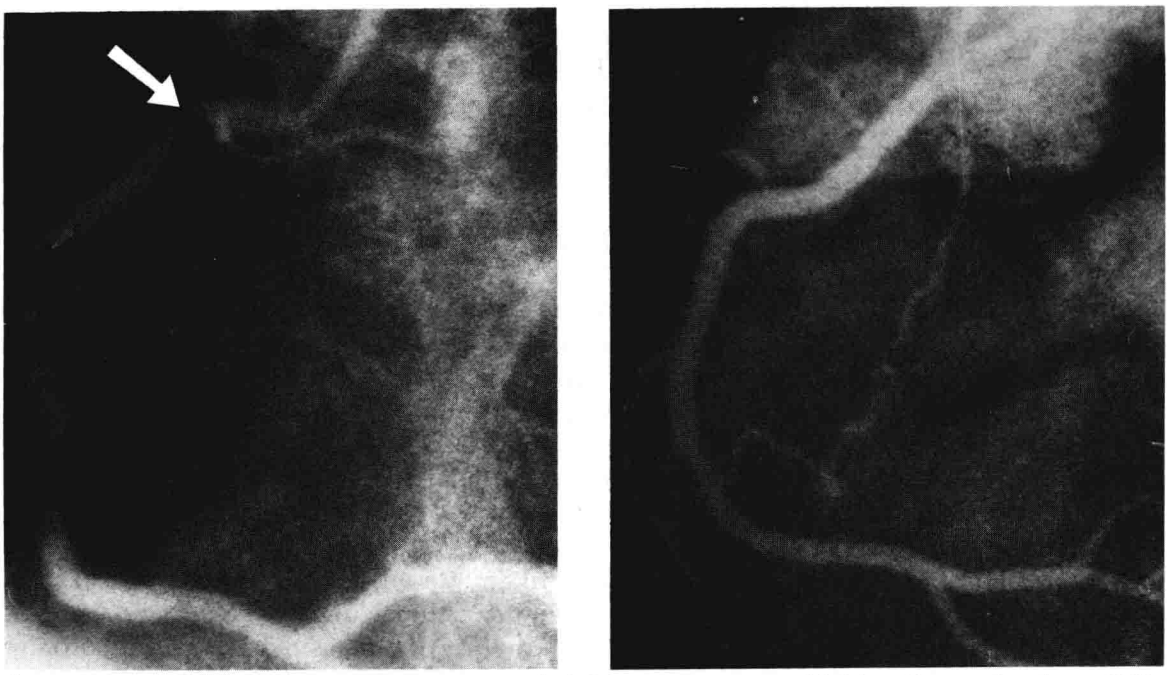

Figure 1.4. Arteriograms comparing a normal right coronary artery (right) with one that has a 90% obstruction (left).

CAUSES OF CORONARY HEART DISEASE

An unrelenting search has been in progress for more than 50 years attempting to ascertain why and how the coronary arteries are affected by atherosclerosis. The question has never been answered, and the cause of coronary atherosclerosis remains unknown. However, one fundamental fact has emerged: combination of several factors is undoubtedly involved in the development of CHD; no single mechanism can be held responsible in its own right. According to this concept, all of the following factors (called risk factors) may contribute to the formation and progression of coronary atherosclerosis.

Sex and Age

CHD is distinctly more prevalent in men than in women. Indeed, during the childbearing years women are seemingly protected from CHD unless they have many other risk factors (e.g., hypertension and diabetes). After the menopause, however, the incidence of CHD in females rises rapidly and equals the male rate thereafter. In contrast, symptomatic CHD may occur in men as young as 30 years (or even younger). This sex–age discrepancy suggests that hormonal influences may be important in the disease.

The incidence of CHD increases greatly with age in both sexes. For example, a man in his fifties has four times the risk of a heart attack as a man in his thirties. The fact that young persons may develop CHD makes it clear, however, that coronary atherosclerosis is not simply a disease of aging.

Diet and Cholesterol

Several epidemiologic studies have demonstrated that the incidence of premature CHD (i.e., coronary disease occurring before the age of 60) can be correlated with the different dietary patterns of various societies. Specifically, in affluent countries (as the United States), where animal fats constitute a large percentage of the total diet, the frequency of CHD is very high; and in poorer countries, where animal fat intake is much less, the incidence of the disease is low. The gross disparity in the amount of animal fat eaten (e.g., eggs, butter, cream, milk, and fatty meats) in different parts of the world is believed to account for the fact that "normal" serum cholesterol levels in the United States may be 200–240 mg%, whereas in those countries in which CHD is uncommon the comparable levels are only 100–120 mg%. Further evidence in support of the danger of high-fat diets is the reported decrease in the number of deaths from CHD during World War II in those countries where animal fats became scarce, followed by a prompt increase in the death rate after the war ended when the economy improved and fats again became available. From data of this type many researchers have concluded that overeating of animal fats (also called saturated fats) is a prime factor in the etiology of CHD.

More specific information about the danger of high serum cholesterol levels has been obtained from the Framingham Heart Study. In this study more than 5000 men and women in the town of Framingham, Massachusetts, have been examined at regular intervals for 25 years to determine which factors contribute to the development of CHD. The results indicate that the risk of a heart attack is at least three times greater in men with serum cholesterol levels of more than 240 mg% than it is in those with levels of less than 200 mg%.

Hypertension

High blood pressure is thought to predispose to CHD by accelerating the rate of atherosclerosis and by increasing the oxygen demands of the myocardium. In the Framingham Heart Study it was observed that blood pressures in excess of 160/95 were associated with a fivefold increase in the incidence of CHD compared with normal pressures. Thus from a statistical standpoint hypertension appears to be one of the most serious risk factors.

Heredity

A familial pattern of CHD has long been recognized, but the degree of risk is still uncertain (because family histories are unreliable in many instances). However, our own experience suggests that heredity ranks among the highest risk factors, particularly when CHD occurs during the fourth or fifth decade of life. In these latter cases it is commonly found that a man's father, grandfather, and brothers often developed CHD at about the same age. It has been postulated (but not proved) that the physical structure of the coronary arteries and the rate of atherosclerosis may be genetically determined.

Diabetes

CHD develops more frequently and at an earlier age among diabetic patients than among nondiabetics. Even when diabetes is mild or well controlled the risk of CHD remains substantially greater. These facts along with data indicating that other metabolic diseases (e.g., gout) are associated with a high incidence of CHD suggest that a biochemical disturbance may be central to the underlying disease process.

Cigarette Smoking

There is statistical evidence to indicate that heavy cigarette smokers have a higher incidence of CHD than nonsmokers. In the Framingham study the risk of a heart attack was nearly twice as great in cigarette smokers. However, the risk is associated primarily with middle-aged men and is much less impressive in older men and in women. Curiously, cigar and pipe smokers are at no greater risk than nonsmokers, presumably because they do not inhale. The manner in which cigarette smoking affects the coronary arteries is not understood. The suggestion that nicotine may cause sufficient constriction of the arteries to reduce coronary blood flow has not been confirmed. On the other hand, nicotine increases the work of the heart (by increasing the heart rate and blood pressure) and could produce a relative oxygen deficiency. Moreover, cigarette smoking is associated with elevated carbon monoxide levels in the blood, which may also interfere with myocardial oxygenation.

Sedentary Life

Lack of physical activity has been incriminated as a risk factor in CHD, but the evidence for this belief is still inconclusive. Several studies have revealed, for example, that CHD occurs more frequently in sedentary workers (e.g., postal clerks) than in those whose occupations demand substantial physical activity (e.g., mail carriers); yet many observers have questioned the significance of these findings, noting that there were so many other variables between the two groups that physical inactivity should not be singled out as a risk factor in its own right. Although there is good reason to believe that exercise may benefit the myocardium, it remains to be seen if physical activity (or inactivity) affects coronary arteries and influences atherosclerosis.

Obesity

Insurance company statistics suggest that obesity predisposes to fatal CHD, but (as with physical inactivity) the issue is by no means settled. In fact in the Framingham study moderate obesity by itself was not associated with an increased incidence of CHD. However, overweight persons are especially prone to hypertension, diabetes, and elevated cholesterol levels, and it may be that the risk of obesity lies with these secondary effects. In any case obesity is classified as a risk factor even though its mechanism of action is uncertain.

Emotional Stress

Epidemiologic studies have consistently shown a markedly higher incidence of CHD in industrialized (civilized) countries than in primitive, less-demanding societies. Many believe that this gross disparity is a reflection or a direct result of emotional stress imposed by modern, fast-paced styles of life. For this reason CHD is considered by some to be a disease of "overcivilization." According to this theory civilized man has developed chronic anxiety in attempting to cope with rapidly changing socioeconomic and sociocultural forces, and this tension in some way promotes atherosclerosis. In principle, this is an attractive concept, since it has been demonstrated that anxiety is often accompanied by a distinct rise in serum cholesterol, which could favor the development of atherosclerotic plaques. Moreover, stress is known to accelerate blood coagulation, allowing small clots to form within the coronary arteries. Nevertheless, the relationship between emotional stress and CHD has been difficult to prove, particularly since there are no available methods to actually measure degrees of stress. Some re-

search studies in fact have cast doubt on the importance of stress as a risk factor. For example, one large investigation involving telephone company employees showed that the incidence of CHD was actually less common among high-level executives (who presumably function under great stress) than it was among workers who installed or repaired equipment. Further facts will be needed to determine the significance of emotional stress as a risk factor.

Behavioral Patterns

Attempts have been made to correlate CHD with certain personality traits and behavioral patterns. The coronary-prone person—called a type A personality—is said to be one who is aggressive, ambitious, highly competitive, and most of all possessed with a profound sense of the urgency of time. Those with this type behavioral pattern reportedly have significantly higher cholesterol levels and an increased incidence of CHD than their counterparts (type B personalities), in whom these particular characteristics are not as apparent. This interesting observation requires confirmation, but many now accept type A behavior as a distinct risk factor.

Summary of Risk Factors

It is essential to point out that there is no definite evidence that any of the risk factors just described actually *cause* CHD. All that can be said is that individuals with multiple risk factors are high-risk candidates for CHD; conversely, the absence of these factors predicts little likelihood of developing the disease. For example, a man with hypertension and high serum cholesterol levels who is a heavy cigarette smoker may have ten times the risk of sustaining a heart attack than a person with none of these factors. In other words, there is a statistical association between risk factors and CHD but, on the other hand, no proof that these risk factors in themselves are the direct cause of coronary atherosclerosis.

THE CLINICAL SPECTRUM OF CORONARY ATHEROSCLEROSIS

Asymptomatic Coronary Atherosclerosis

If the degree of arterial obstruction is moderate and does not significantly reduce the blood supply to the myocardium, the disease may never be suspected by the patient or his physician. Results of autopsy studies among persons dying of other causes indicate that this is a common situation. In fact, practically all men in the United States have evidence of coronary atherosclerosis by age 50; it is only the degree of involvement that varies.

Even if the coronary arteries are grossly narrowed by intimal plaques, it still does not follow that the disease will be clinically evident or produce symptoms. This paradox can be explained by the fact that as the coronary arteries gradually narrow small branches of these vessels may enlarge or new branches may form in order to bring more blood to the myocardium. This additional blood supply, called *collateral circulation*, is of great importance in determining the clinical effects of coronary atherosclerosis since this network of vessels is often substantial enough to maintain an adequate blood supply to portions of the myocardium despite the presence of advanced atherosclerosis in a major vessel. It is the *total* blood supply to the myocardium rather than the state of the main coronary arteries that determines whether the disease will be

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