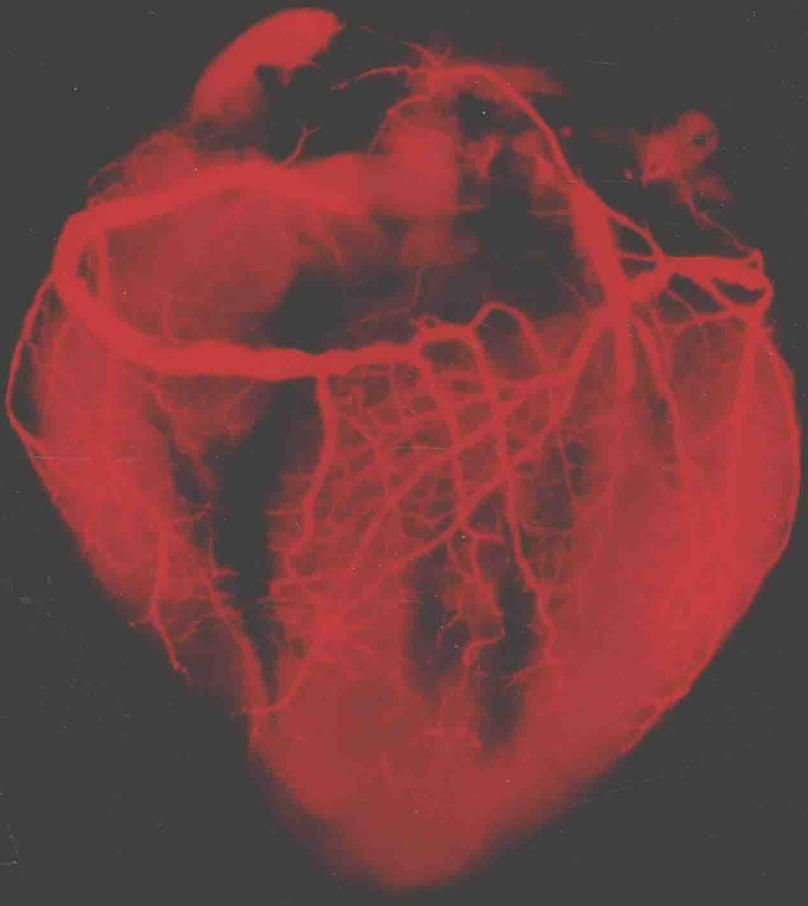
Pathology of Ischaemic Heart Disease

Theo Crawford



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POSTGRADUATE PATHOLOGY SERIES

Under the General Editorship of

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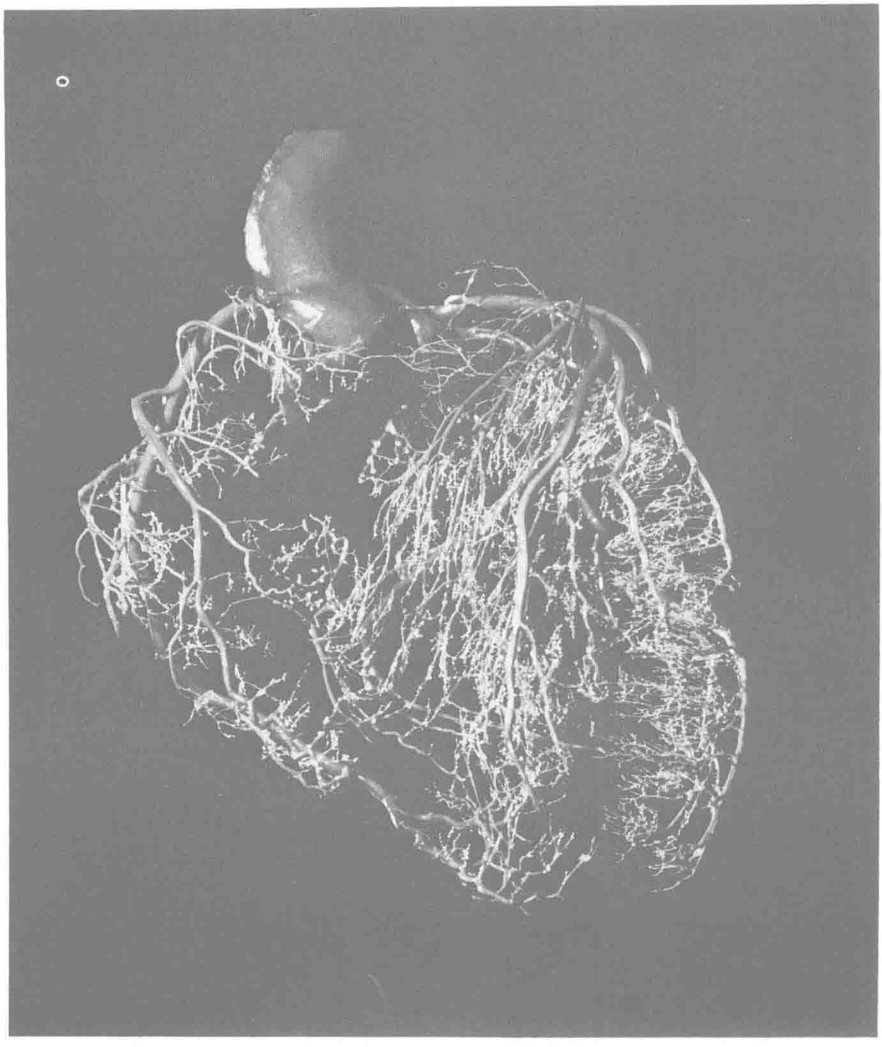

Marco-resin cast of the coronary arteries. The cast was prepared by D. H. Tompsett of the Department of Anatomy at the Royal College of Surgeons of England and photographed by Dr. Geoffrey Franglen of St. George's Hospital Medical School

Preface

The purpose of this volume, as with other volumes in this series on Postgraduate Pathology, is to present in handy form the amount of detailed knowledge of the subject that the author believes a trainee pathologist should acquire during his training period. This is not necessarily equated to the requirements for any particular examination but relates to what a young pathologist, who is not a super-specialist in the particular field, might be expected to be familiar with concerning ischaemic heart disease. He will, of course, extend his experience and no doubt develop his personal opinions concerning controversial aspects of the subject during his years as a practising pathologist.

There has been no attempt to make this an exhaustive reference book and only a selection of the more important and accessible references to the literature is provided at the end of each chapter. On controversial subjects the author's views are naturally given prominence but where they are grossly divergent from general opinion this will be made quite clear. An exception to the generalization concerning references will be found in the last chapter of the book, dealing with the still controversial subject of the relationship between hardness of water supplies and local death rates from ischaemic heart disease. This subject has been discussed and documented in more detail than have some other matters where the facts are more firmly and longer established. The author feels that this subject deserves special attention not only for the exciting possibilities that it opens up in terms of preventive medicine, but as an example of the way in which pathological surveys are required to lend substance and meaning to the data-collecting and statistical studies that are the primary activities of the epidemiologists. Furthermore, it seems probable that control of the modern epidemic of ischaemic heart disease is most likely to be achieved by a full documentation of aetiological factors and an understanding of how these factors exert their effects. It is in the development of this understanding that histopathology and experimental pathology are most likely to play a part.

T.C.

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It is a pleasure to acknowledge my indebtedness to Dr. M. J. Davies and Dr. J. P. O'Sullivan, of the Department of Pathology at St. George's Hospital Medical School, for providing some of the illustrations and helping in their preparation; and to my wife for her expert help in preparing and producing the typescript.

Theo Crawford

Contents

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Introduction

In the title of this monograph the term ischaemic heart disease has been preferred because it expresses precisely what is to be discussed: structural and functional abnormality of the heart as a consequence of an inadequate supply of blood to its tissues. Other terms commonly in use are less precise. 'Coronary thrombosis' emphasizes one pathological feature which, important as it is, is not a constant finding, and the same applies to 'myocardial infarction'; while the commonly used term 'coronary artery disease' describes something nearly everyone has though in only a minority of people does it cause damage to the myocardium and lead to heart disease.

The emergence of ischaemic heart disease as a nosological entity constitutes one of the most fascinating and remarkable chapters in the whole of medical history. There is no doubt that the disease has been occurring for at least three hundred years – and probably much longer. William Harvey (1649) described the quite characteristic case of 'a noble baronet, Sir William Darcy', who suffered for many years from what can only have been angina pectoris, and ultimately died after an attack. Necropsy revealed rupture of the left ventricle, which was clearly the sequel to a myocardial infarct. Edward Jenner, of vaccination fame, in a letter to Heberden, probably written in 1778, described the finding of thickened coronary arteries with 'a kind of firm fleshy tube within the vessel' in the hearts from two sufferers from angina pectoris who had died suddenly. Jenner seems to have been the first to suggest that angina pectoris and the sudden deaths associated with it were due to the coronary arteries 'not being able to perform their functions'. It is remarkable that a country general practitioner should have made two such outstanding contributions to medical knowledge – vaccination against smallpox and the understanding of the mechanism of anginal heart attacks – using no more complicated instruments than observation and dissection. Unfortunately, Jenner's observation was forgotten or ignored by clinicians and pathologists alike and as late as the second decade of the twentieth century textbooks of pathology and of medicine were giving the sketchiest of descriptions of myocardial infarction or of its relationship to coronary artery disease.

Dr Evan Bedford, in his Harveian Oration to the Royal College of Physicians of London in 1968, confessed that as a student at the Middlesex Hospital, where he qualified in 1923, he had never heard of coronary thrombosis. Later at the London Hospital, working with Parkinson in 1926, he watched the epidemic spreading around the neighbourhood as local practitioners began to recognize coronary thrombosis, and by 1930 it had become a common and familiar illness. Today, with the decline of infective diseases and an increasing proportion of elderly people in the population, ischaemic heart disease is responsible for more deaths than any other cause in most parts of the world except the so-called under-developed countries. In England and Wales 153 250 deaths were certified to this cause in 1974, the nearest competitors being all neoplasms (123 022), all respiratory deaths (82 310), and cerebrovascular disease (78 833). There has, of course, been an enormous rise in certifications during the past 50 years, but the interpretation of the rise is not easy. Ischaemic heart disease affects principally the older half of the population, rates rising progressively with age, and it is commoner in males than in females; obviously, therefore, the rising proportions of the population in the upper age-groups, and the increasing male:female ratio will be associated with an increasing number of deaths from this cause. It is equally clear that awareness of the manifestations of ischaemic heart disease and its diagnosis which grew up in the 1930s took many years to spread widely among members of the medical profession, and it took even longer for the doctors to change their habits of diagnosis and death certification, so that only gradually have such vague terms as myocardial degeneration, arteriosclerosis and heart failure been replaced by more precise terms such as ischaemic heart disease, myocardial infarction and coronary thrombosis. It is impossible to determine, therefore, whether the present high level of ischaemic heart disease in the population of developed countries represents a modern response to changing ways of life or merely reflects a changed population structure together with improved medical awareness of the condition and its various manifestations. Most would probably support the view expressed by Morris (1964) that there is in fact a 'modern epidemic' of ischaemic heart disease, independent of changes in population structure and diagnostic fashions. Others, however, do not accept this and follow the conclusion of Robb-Smith (1967) that 'there is no known evidence for an increased age-specific incidence of coronary thrombosis or myocardial infarction from English or American post-mortem studies'. It is to be emphasized that Robb-Smith's is the minority view.

In studying ischaemic heart disease, then, we are studying a condition of high prevalence and probably increasing incidence in western civilized communities. It is a disease which kills more people in these communities than any other definable 'disease'. It will be the direct cause of death in about one-third of us who are medical men and a considerably higher proportion of us will suffer from its effects, whether we are aware of this or not. Ischaemic heart disease is itself, in all but a tiny proportion of quite exceptional examples, a local manifestation of the more widespread lesion or 'disease' – arterial atherosclerosis. This condition, which is virtually universal in mankind after the second decade of life, is the common pathological basis not only for ischaemic heart disease but in addition for much cerebrovascular disease and peripheral vascular disease as well as for abdominal aortic aneurysms and other less common manifestations. Because of its vast importance in this way the general pathology of atherosclerosis

forms the subject of a separate volume in this series (Woolf, in prep.) and here will be dealt with only in relation to its occurrence in the coronary arteries of the heart.

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2

The Anatomy of the Coronary Arteries

The general arrangement of the coronary arteries follows a fairly regular pattern but the detailed distribution of the smaller branches is almost infinitely variable. Even the main trunks are far from constant. Much of this variability has been appreciated only quite recently by the use of radiographic methods (Fulton, 1965) and of injection-corrosion techniques (James, 1961; Baroldi and Scomazzoni, 1967). The practising pathologist should be thoroughly familiar with the usual layout of the main vessels and their larger branches, and with the commoner departures from this layout. The anatomical features of the main vessels are fully described in many anatomy texts and monographs and need be only briefly revised here.

THE MAIN LEFT CORONARY ARTERY

The main left coronary artery arises in the aortic sinus (of Valsalva) guarded by the left posterior cusp of the aortic valve, which is often conveniently referred to as the 'left coronary cusp'. Usually the ostium is just below the level of the cusp margin centrally placed in the sinus, and this is the safest place for it. Occasionally it is situated higher up, above the level of the cusp margin in the first part of the ascending aorta, and this is much more hazardous because in this position it may be caught up in the scarring processes that accompany various forms of aortitis, particularly syphilitic aortitis, or it may even be encroached upon by atherosclerotic plaques. Coronary embolism is also more liable to occur when the ostium is in the more distal situation. Emerging from the ostium the left coronary artery runs down and to the left across the root of the aorta to enter the anterior interventricular groove (*Figures 2.1, 2.2* and *2.3)* where it is usually easily visible on inspecting the heart from the front. The total length of this main left coronary trunk is rarely more than 2.5 cm and often as little as 2–5 mm (*Figure 2.3*), with a diameter of 3–6 mm in the absence of

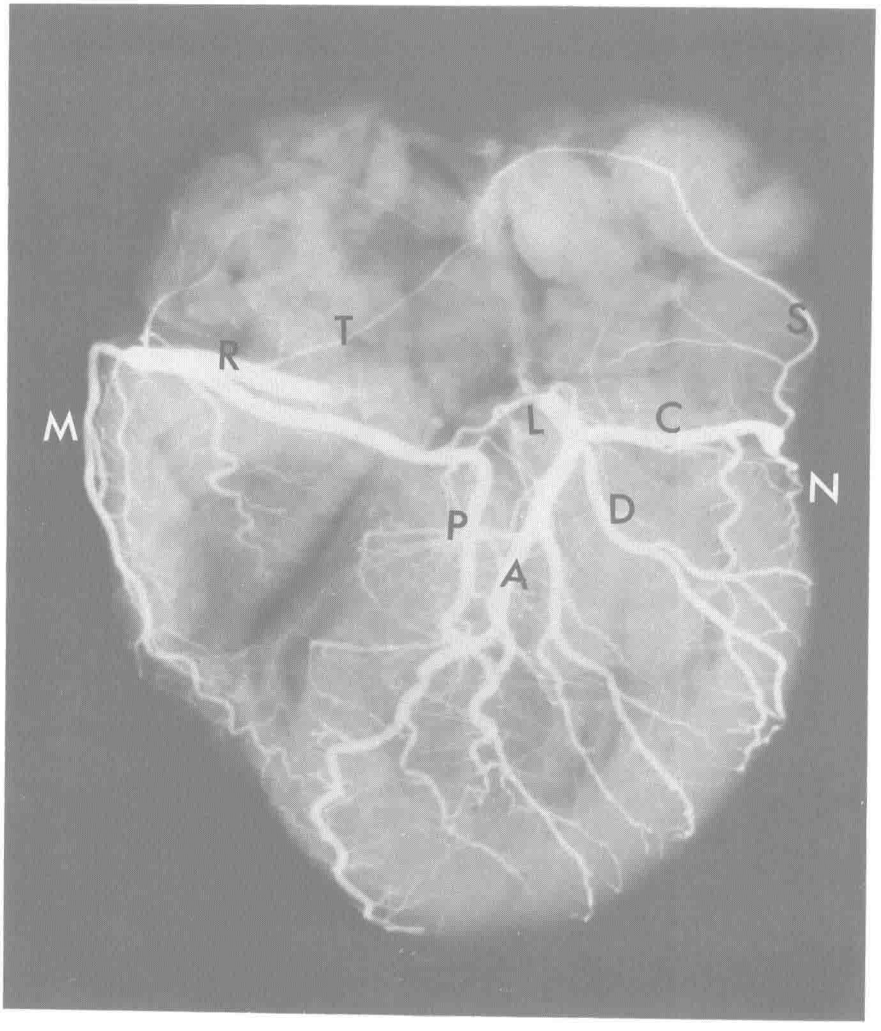

Figure 2.1 Post-mortem coronary arteriogram, showing normal arteries with right arterial dominance. L: Left main artery. R: Right artery. C: Left circumflex artery. A: Anterior descending artery. P: Posterior descending artery. D: Diagonal branch. M, N: Right and left marginal branches. S, T: Atrial branches of circumflex and right arteries, jointly supplying the sinus node

disease. The main vessel ends by dividing into the left anterior descending artery and the left circumflex artery; but not infrequently this termination is more complex with one or more additional branches taking intermediate positions between the descending and circumflex vessels (*Figures 2.1* and *2.3*). The left anterior and circumflex vessels are sometimes designated as branches of the left coronary artery rather than as named arteries in their own right; but with either system the meaning is clear and there should be no room for confusion.

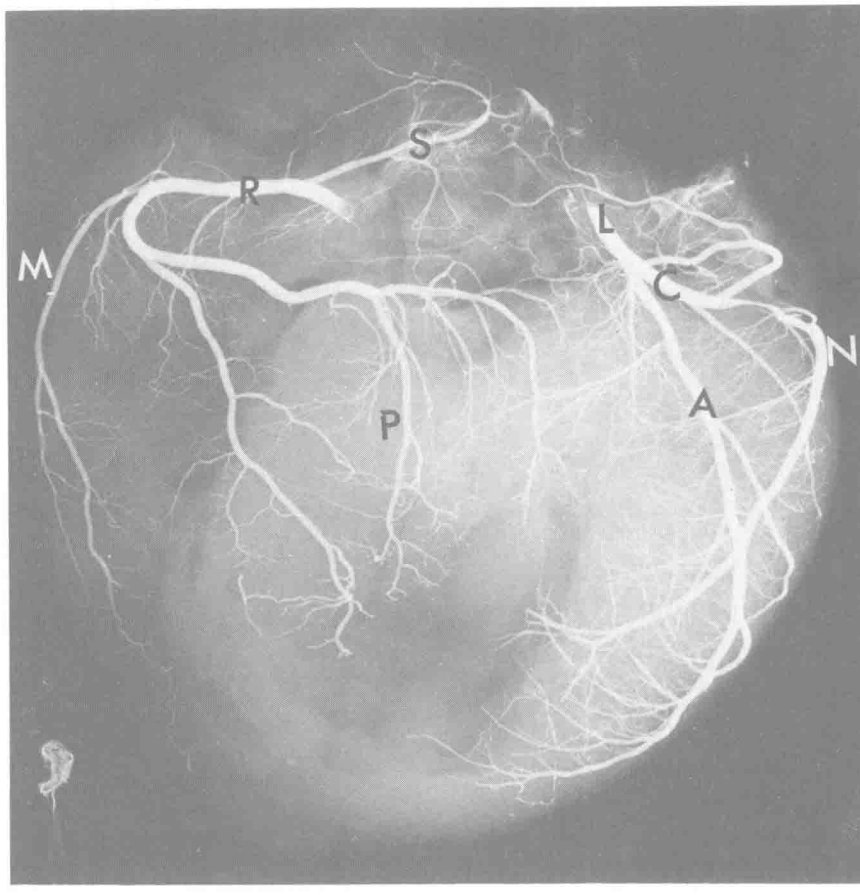

Figure 2.2 Post-mortem coronary arteriogram showing normal arteries with right arterial dominance. L: Left main artery. R: Right artery. C: Left circumflex artery. A: Anterior descending artery. P: Posterior descending artery. M, N: Right and left marginal branches. S: Sinus node artery arising from dominant right coronary

THE LEFT ANTERIOR DESCENDING ARTERY

The left anterior descending artery arising as described, by the terminal bifurcation of the main left coronary, descends in the anterior interventricular groove and terminates at or near the apex. Often it runs over the apex and ascends a few millimetres in the posterior interventricular groove (*Figure 2.2*). In its course it usually gives off one or two small twigs from its right side and four or five rather larger branches from its left, one of which – the left diagonal branch – may almost equal in size the descending artery itself (*Figures 2.1* and *2.3*). Small branches from the descending artery also pass posteriorly into the interventricular septum and usually form its principal supply, though joined in this function by similar branches from the posterior descending artery on the back of the heart.

THE LEFT CIRCUMFLEX ARTERY

The left circumflex artery arises, as has been described, from the termination of the main left coronary artery, turning left and upwards at a wide angle to enter the atrioventricular sulcus. In the first few centimetres of its course it is obscured by the left atrial appendage which must be displaced for the vessel to be seen. The size and length of the circumflex artery are extremely variable. Often it terminates at a point within a centimetre on either side of the left border of the heart by turning down at a right angle to form the left marginal branch (*Figure 2.1*). This runs down along or close to the left border of the heart towards the apex. In some hearts the circumflex artery may be shorter, terminating after a centimetre or two by sloping down to the left border; or it may be much

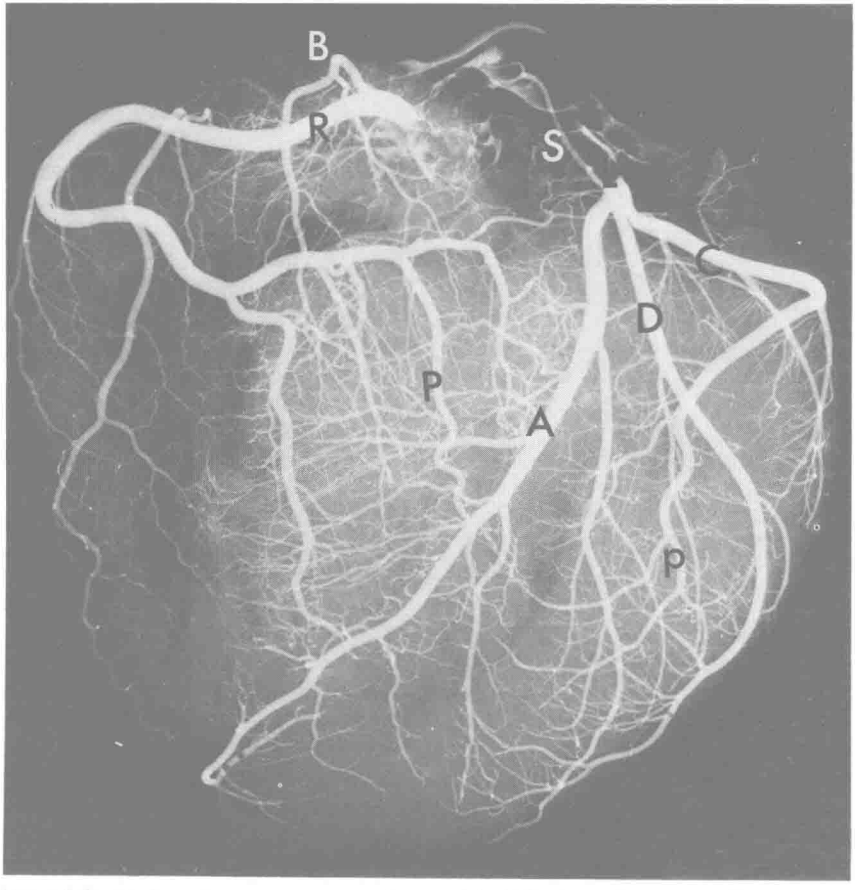

Figure 2.3 Post-mortem coronary arteriogram. The left circumflex artery (C) is large and continues onto the posterior surface of the right ventricle. The right artery (R) is also large so that there are in effect right and left posterior descending arteries (P and p). The nodal artery (S) is derived from the left circumflex artery. The anterior descending artery (A) is long, traversing the apex and ascending a few centimetres in the posterior interventricular sulcus. There is a large diagonal branch (D) arising from the trifurcation of the left trunk (L). A conspicuous conus artery (B) is seen as the first branch of the right coronary