ARTERIAL DISEASE

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SCIENTIFIC PUBLICATIONS
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FOREWORD

The subject of this book is an important one for three reasons. First, many people, particularly men, die in middle age after the abrupt onset of pain in the chest and with manifestations before and after death suggesting infarction of the heart. Second, the cause of this disease is not understood, and therefore no preventive measures can be undertaken. Finally, a vast number of man-hours of scientific research, and a corresponding amount of wealth is being consumed in trying to answer this riddle. Unfortunately, the elementary facts concerning the disease in man are by no means clearly established. The critical onlooker may therefore be forgiven if he is a little doubtful whether some of this effort will have great relevance to the human problem just outlined.

It was because of this important consideration that Mitchell and Schwartz undertook the work that is set out in the first part of this book. I myself am delighted with this work for several reasons. It represents the joint effort of a physician and a pathologist to illuminate what happens during life by what can be seen after death, an effort that is nowadays too rare. They have compared the changes found in the coronary arteries with those in other arteries of comparable size and in the aorta. They have used the best methods yet described. They have avoided unnecessary and unverified assumptions. They have used the same methods to investigate patients dying with clinical or post-mortem evidence of infarction of the heart, and an unselected sample of those dying in hospital in the same city. Thus although this is not the first study of this subject it is in many ways the most complete. The story seems to be this.

The immediate cause of infarction of the heart is in most cases, though possibly not in all, a thrombus occluding a branch of a coronary artery, particularly the left anterior descending. The thrombus has a characteristic structure—clumps of platelets fringed with leucocytes and interlaced with a fibrin meshwork which often contains red cells or the ghosts of red cells. As the thrombus ages the nuclei disappear, and it becomes more uniform in composition. Later it becomes organized and recanalized. The longer the interval between the clinical attack and death, the less likely is there to be a fresh thrombus and the more likely is there to be a characteristic plaque of intimal thickening producing coronary stenosis. A unitary view