Current Practice

DISEASES OF THE HEART AND BLOOD-VESSELS

Recovery After Myocardial Infarction—1

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Coronary artery disease is a common clinical problem to every practitioner. It causes the loss of more years of working life than any other disease. It claims one-third of all deaths in middle age, and the mortality increases every year. Deaths from myocardial infarction in England and Wales climbed from 28,500 in 1949 to 106,000 in 1963, and the mortality from this disease now exceeds that of all forms of cancer.

The destruction of so many men in the prime of life has enlivened public interest, and advice is frequently sought by patients and relatives on the measures that can be taken to prevent further attacks. Yet despite the frequency of this disease neither the aetiology of the underlying atheroma nor the cause of the intravascular thrombosis is known. Rational treatment and prevention are therefore difficult, and authority, tradition, and prejudice flavour the therapeutic advice given to these patients. This review is primarily concerned with the management of the patient who has recovered from a myocardial infarction.

Prognosis

Many doctors regard the situation of a patient who has suffered a myocardial infarct as desperate. This is not so. Sixty-five per cent of survivors of an acute attack of myocardial infarction will be alive after five years with no treatment, and of those under 50 years of age 75% will survive five years. Ten years after the initial myocardial infarct the survival figures vary between 30% and 60%. An average figure taken from five large series of patients with a mean age of 57 years is 43% alive after ten years. While it is true that the chance of survival declines with increasing age, or if the acute episode is associated with congestive cardiac failure, arrhythmia, cardiac enlargement, previous infarction, or diabetes mellitus, it is equally true that the younger patient without these complications has brighter prospects. The mortality rate for patients with myocardial infarction under 60 years of age is half that of those over 60 years, and the chance of surviving five years is twice as great for those patients with no cardiac failure than for those in whom cardiac failure complicates the acute infarction. There is thus no place for gloom and despair.

Pathogenesis

The word arteriosclerosis means hardening of the arteries, and has been used for all diseases (apart from inflammatory) of all layers of all arteries. Atherosclerosis or atheroma was introduced as an alternative to arteriosclerosis to emphasize the fatty component of some arteriosclerotic lesions. It is a disorder affecting the intima of large arteries. Fatty deposits and fibrous nodules narrow the lumen of the artery and predispose to

thrombosis. Atheroma is not a specific disease, but a non-specific response of the arterial intima to a number of different assaults. Its aetiology is thus almost certainly multifactorial. The arteries particularly involved are the aorta, the coronaries, the internal carotid and vertebral arteries, the iliac, femoral, popliteal, and renal arteries. The coronary arteries differ from most other arteries in the body in that they have a clearly defined intimal layer, which contains longitudinal smooth muscle fibres. They also run part of their course within the substance of the myocardium, and compression of this portion during systole may lead to unnatural strain on the proximal epicardial segments.

Ischaemic heart disease or coronary failure results when the supply of oxygenated blood through the coronary arteries is inadequate to meet the demands of the cardiac muscle. Although theoretically such a discrepancy between supply and demand may be the result of excessive requirements of hypertrophied muscle or increased work of the heart due to hypertension or hyperthyroidism, it is in fact nearly always due to narrowing of the coronary arteries. Such occlusive disease of the coronary vessels is the result of atheroma (apart from rare cases due to syphilitic coronary ostial stenosis, coronary embolism, or polyarteritis). It must not be assumed that coronary occlusion is the inevitable end-result of atheroma. In the great majority of individuals with atheroma there are no ill effects, but in a minority thrombosis or severe narrowing precipitates coronary heart disease.

Atheroma and Infarction

There are four contributing factors in the aetiology of clinical coronary heart disease, and these are the factors that determine whether the atheroma, which is so universal in Western countries, will lead to clinical disease. These are: (1) The degree of atheroma and the site of the stenosing lesions. (2) The occurrence and extent of recent and old occlusions of the coronary arteries. (3) The collateral circulation. (4) Cardiac output.

Atheroma is the fundamental degenerative disorder of the vessel which provides the basis for the intravascular thrombosis that gives rise to coronary heart disease.

There is no precise correlation between the degree of atheroma on the one hand and the frequency of intravascular thrombosis and clinical coronary artery disease on the other. It is true that coronary thrombosis is never seen without disease of the vessel wall, but advanced atheroma often exists without thrombotic occlusion and clinical coronary artery disease, and the minor degrees of atheroma are often associated with intravascular thrombosis. If coronary insufficiency is sufficiently prolonged or severe, myocardial infarction results. Infarction depends on the severity and duration of ischaemia and not on the manner in which it is produced. Myocardial infarction is

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thus not synonymous with coronary thrombosis. Nevertheless it is true to say that the incidence of coronary thrombotic occlusion at necropsy in myocardial infarction is a direct function of the diligence and technique employed in the search for the arterial thrombosis.

Aetiology

The aetiology of atherosclerosis is not known. The two main theories ascribe the cause to (1) Infiltration of lipid into the arterial intima from the circulating plasma. (2) Intravascular thrombosis with subsequent organization of thrombus and incorporation into the vessel wall.

Lipid hypothesis. This theory is based on the observations that intimal plaques contain lipid, that patients with myocardial infarction show abnormal serum lipid patterns, and that atheroma can be produced in animals by feeding cholesterol. The assumption is that lipid material, having filtered through the endothelium, excites an inflammatory reaction in the intima, which leads to cellular infiltration, fibrous tissue reaction, and the formation of a nodular plaque. As the arterial intima derives its nourishment from the circulating blood, a secondary blood supply from the adventitia and media develops when the intima is thickened by disease. This leads to secondary complications such as haemorrhage into the plaque. Atheroma is thus regarded as a reaction of the arterial wall to invading lipids. It is certainly true that damage to the arterial wall from a variety of agents predisposes the area to infiltration by lipids, but whether the infiltration of lipid is a primary aetiological factor remains uncertain.

Thrombotic hypothesis. The laminated structure of many atheromatous coronary arteries suggested to morbid anatomists that previous thrombosis had occurred in most patients with coronary artery disease. Thrombi formed in circulating blood differ from the blood clot that forms after haemorrhage; they do not start as fibrin clots. Clumping of platelets on the internal surface of the artery initiates the process of intravascular thrombosis. Leucocytes are attracted and fibrin is formed. Such mural thrombi may vary from microscopic layers of platelets and fibrin up to masses of clot large enough to occlude the lumen. Under the force of the blood flow these deposits become moulded into the shape of the vessel wall. The blood-stream washes away much of its cellular content, leaving a laminated fibrous layer. In time not only does the thrombus become organized but it becomes covered by endothelium, so that it is incorporated into the vessel wall and any further change (such as softening or fatty infiltration) that occurs in it appears as if it was in the vessel wall rather than on the surface. Cholesterol, like calcium, tends to accumulate in necrotic material and the cholesterol content is likely to be enhanced

in hypercholesterolaemic states without the disorder being primarily a result of abnormal lipid metabolism. At first the thrombi do not completely occlude the vessel, or, if they do, collateral vessels are adequate to nourish the myocardium. When, however, intravascular thrombosis occurs in vessels whose collaterals are narrowed or thrombosed by previous disease then clinical manifestations of coronary heart disease appear.

Dietary Fat

Neither theory entirely explains all events. Many atheromatous lesions show little fat, and yet there is no doubt that a high dietary fat or hypercholesterolaemia will, under certain circumstances, contribute to lipid deposition in the intima. In the animal kingdom birds have the highest serum cholesterol and the most atheroma and reptiles the lowest and the least. There is also little doubt that mural thrombi are not infrequently deposited on the internal surface of an artery and that these become covered by endothelium and incorporated into the vessel wall. Patients with coronary artery disease have more atheroma of the aorta and iliac and carotid arteries than unselected patients of comparable age and sex and also have a higher incidence of thrombotic occlusion of the carotid and iliac arteries. This suggests the possibility of a generalized thrombotic tendency in patients with coronary heart disease. Furthermore, it is usually a thrombotic episode that precipitates clinical coronary artery disease.

However, intravascular thrombosis is unlikely to be the sole factor in the aetiology of atheroma, as the degree of coronary atheroma has not increased over the last 60 years and yet the incidence of intravascular thrombosis has done so enormously. Coronary atheroma is a feature of many races—such as the Bantu and Jamaican—and yet thrombosis of the coronary vessels and also thrombo-embolic disease remain rare. Moreover, in Britain and Scandinavia during the war there was a fall in mortality from coronary artery disease and a similar fall in thrombo-embolic diseases, suggesting that during this period there was a reduction in intravascular thrombosis, presumably due to dietary factors. Races, such as the Japanese, which have a low incidence of coronary artery disease also have a low incidence of thrombo-embolic disorders. Several studies have now shown a link between lipids and platelet aggregation—a high fat diet enhancing platelet aggregation. There is evidence that the type of free fatty acid with which the platelet comes into contact affects its stickiness, and this depends on the nature of the dietary fat.

(This article will be concluded next week with a discussion of the epidemiological studies of coronary thrombosis and advice on after-care)

ANY QUESTIONS?

We publish below a selection of questions and answers of general interest.

Pyloroplasty and Vagotomy for Duodenal Ulcer

Q.—*What are the indications for pyloroplasty with total vagotomy or pyloroplasty with selective vagotomy in the treatment of duodenal ulcer? Has pyloroplasty without vagotomy a place in treatment?*

A.—Vagotomy is being increasingly employed in the surgical treatment of chronic duodenal ulcer because of the widely held belief that there is usually, if not always,

an excessive amount of vagal stimulation to the stomach in these cases. Acid secretion in patients with duodenal ulcer is generally larger than usual, and is attributed to overaction of the vagi. A prime aim of vagal section is to reduce the hypersecretion.

However, since vagal section, in addition to its effect on acid secretion, impairs gastric emptying, some form of gastric drainage procedure is required to prevent symptoms due to slow emptying of the stomach. Originally most surgeons practised gastrojejunostomy with total vagotomy, and the

results were generally satisfactory. More recently pyloroplasty has been preferred by many surgeons, mainly on the ground that, if affording satisfactory drainage, it would seem more physiological to allow the food leaving the stomach to enter the duodenum than to be diverted to the jejunum. In support of this consideration it has in fact sometimes been found that post-operative sequelae such as "dumping," bilious vomiting, or bowel upset have been attributable to a gastrojejunostomy and cured by conversion of this form of drainage procedure to a pyloroplasty. However, pyloroplasty is occasionally followed by similar symptoms, and it is not known as yet whether it is significantly superior to gastrojejunostomy as a drainage procedure. There are currently