BRITISH MEDICAL JOURNAL

LONDON SATURDAY JUNE 16 1962

THROMBO-ENDARTERECTOMY AND ITS APPLICATION TO CORONARY ARTERY DISEASE*

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The problems presented by ischaemic heart disease cannot be considered in isolation. As Florey (1960) has emphasized in a comprehensive summary, they are associated with two general problems: those of atherosclerosis and of thrombosis. The ultimate solution to the combined clinical problem cannot be a surgical one, but it may be that, in the present period of ignorance and uncertainty, surgery may not only have something to offer to unfortunate sufferers, but, by a direct approach to the problem during life, it may contribute to the understanding of its pathology.

I propose, therefore, firstly to remind you rather sketchily of the background in so far as it influences or is influenced by surgical experience, emphasizing, with a surgical bias, the mechanical aspect of the disease processes. I then propose to discuss with you a surgeon's approach to arterial disease in general. Lastly, I hope to show how the techniques used for other vessels may be applied to the coronary arteries. Coronary thrombo-endarterectomy is a young subject. Its face is at present rather disfigured by failure, but it is well conceived and of good parentage: if you take to it it may develop handsomely.

The commonest cause of complete arterial occlusion is a thrombus superimposed upon an atheromatous plaque. It might be said that this is nearly all that is known for certain about vascular occlusive disease, for there is no agreement about the causes of either atherosclerosis or thrombosis or about the extent to which these two processes are dependent upon one another. There are those who believe that atheroma is essentially a disturbance of fat metabolism, and that cholesterol and phospholipids are deposited in excess in the arterial wall, and that the resulting tissue reaction leads to the formation of a fibrous plaque and to calcification. Those who take this view see a single, graded process from the fatty streak of relatively young arteries to the fibrous plaque, the calcified plaque, and the plaque that is complicated by thrombosis, ulceration, and haemorrhage. Others of a different opinion find no association between the fatty streak and the atheromatous lesion. The latter may be a result of disease of the arterial wall during which cholesterol is deposited. On the other hand, a thrombus, becoming adherent to the vessel wall from some unknown cause, may receive a covering of endothelium and become incorporated in the arterial wall to form atheroma.

Fortunately the surgeon is not expected to explain atheroma, but only to treat some of its complications. He is interested in the physical obstruction produced and also in the sites of the disease, particularly those at the bifurcation of the common carotid arteries, the origin of the renal arteries, the bifurcation of the aorta, the femoral artery, and the coronary arteries.

Mechanical Effect of Atherosclerosis

In considering any one diseased artery the surgeon is concerned about how far the plaques extend into the lumen to interfere with blood flow, and how far they extend into the media. There is usually a plane along which the diseased area of the arterial wall can be separated from the rest with ease. However, if calcification extends into the wall for varying depths over a long length of artery, at one point reaching the media and at another extending right through it almost to the adventitia, then stripping over the whole length may be very difficult, and at best leaves behind a rough and irregular surface to form the new lining of the artery.

The extent to which lesions project into the lumen and diminish its size is also of importance. If the diameter is decreased to one-half of the original the flow through the lumen is decreased sixteen times, for flow through rigid tubes varies as the fourth power of the diameter. When disease is being removed, however, the surgeon receives a corresponding interest for any slight improvement he may achieve, and this is the sort of investment that appeals to him. An abrupt but partial obstruction in an artery may affect flow in yet other ways. Laminar flow through rigid but smooth tubes is a relatively efficient process, but as soon as flow becomes turbulent, as in the wake of an obstruction, a greater amount of energy is needed to drive a given volume of fluid to the periphery, or, if the energy is not available, the flow decreases. Atheromatous projection into the lumen of an artery may therefore have the effect of producing turbulence as well as narrowing the lumen and so reduce blood flow in two ways.

Calcification of vessel walls may occur with very little narrowing, but it is the atheromatous plaque that impinges on the lumen of the vessel that may impair flow to the extent of producing symptoms. In a limb vessel this may cause only slight disability, but in the coronary vessels it may cause angina pectoris, in the renal vessels it may cause systemic hypertension, and in the cerebral vessels transient impairment of consciousness, strength, or sight.

*A lecture given to the Huddersfield Medical Society on November 8, 1961.

Arterial Thrombosis

Of more immediate interest to the clinician, and especially to the surgeon, is the complete occlusion that occurs when thrombosis takes place on an atheromatous plaque.

Thrombosis in a vessel must be distinguished from blood coagulation. Clotting does not occur in arteries when the blood is flowing, but sometimes stagnant blood between an obstruction and the next branch of the artery may clot. In one patient from whose carotid bifurcation an atheromatous plaque and thrombus were being removed, a propagated clot was drawn out which was in fact a cast of the internal carotid artery and its ophthalmic branch. A clot is a reticulum of fibrin with all the cellular elements of the blood entrapped within it. A thrombus, however, consists of masses of platelets surrounded by thin coverings of fibrin with only scanty red and white blood corpuscles. The distinction is an important one, as the two substances are formed in different ways and under different conditions. It seems likely that platelets, by virtue of their inherent stickiness, may tend to adhere to a roughened surface and so form the foundation of a thrombus. Unless anticoagulants have properties other than that of interfering with clotting, they would seem theoretically to have less to recommend them in the treatment of thrombosis than substances that would affect the stickiness of platelets.

There is less tendency for thrombus to form in a vessel if an even laminar type of flow is maintained. The risks are increased in the backwaters and eddies associated with turbulence. The mechanical factors concerned in such processes naturally appeal to the surgical mind because there is always the possibility of a mechanical solution. Cardiovascular surgery abounds with examples where mechanical causes of disturbed flow patterns are associated with thrombus formation or, as they are more frequently called in clinical practice, vegetations.

A persistent ductus arteriosus through which blood passes from the aorta into the pulmonary artery causes a turbulent, whirlpool-like churning of blood where the two streams meet, which is in that part of the pulmonary artery immediately on the cardiac side of the entry of the ductus. A finger placed on this area locates the millwheel murmur and thrill precisely. It is here that vegetations and infection are first found in the ductus complicated by endocarditis. Division of such a ductus was sometimes enough to eradicate the infection, for in this way the turbulence in the pulmonary artery was prevented and the vessel became "self-cleansing." A further example of such a process is in the dilatation of an artery beyond a stricture. The occurrence of such a dilatation can be explained by simple physical laws, and is comparable with the disturbed flow and erosion of the bank in a river below a narrow gorge. It may occur in man in association with a coarctation of the aorta, and the surgeon may find small non-infected vegetations on the wall of the aorta just below the stricture.

These effects were seen very dramatically in a patient with bilateral cervical rib whose subclavian arteries were compressed by the tendinous insertion of the scalenus anticus muscle. On each side a fusiform dilatation of the vessel occurred immediately distal to the stenosis. The patient complained of "poor circulation" in the hands, but in addition to this she had a number of episodes of pain, blanching, and numbness of the right

hand which were obviously due to peripheral emboli. At operation a large vegetation was found in the aneurysm (Fig. 1). The palmar arch, the radial and ulnar arteries, and the brachial artery nearly up to the axilla were packed with emboli of varying ages, that in the brachial artery being the most recent. As such effects of arterial obstruction multiply, vascular stenosis

FIG. 1.—Post-stenotic dilatation of the subclavian artery from a patient with cervical rib and vascular symptoms in the arm. The aneurysm is open to show a large vegetation which was the origin of repeated arterial emboli.

assumes a more and more sinister aspect. These mechanical considerations, however, important as they are, are overemphasized here because they present clinically and at operation. They occur in their proper perspective in relation to the whole problem of thrombosis in a recent review by Poole and French (1961).

An important question that arises in vascular surgery is why the effects of thrombotic occlusion of an artery should be more serious than those of simple ligation. The rapid enlargement of collateral vessels makes it possible to ligate any large artery in the body that is not an "end-artery" and does not supply an organ whose demands are too exacting to await the development of anastomotic channels. Examples of arteries commanding respect as being end-arteries are the renal arteries and the hepatic artery close to the hilum of the liver. The vessels of the brain, spinal cord, and heart must be treated with great caution because these organs may perish before collateral channels have time to open. The iliac and femoral arteries, however, can be ligated with relatively little impairment of the function of the limb, whereas their thrombotic occlusion may cause ischaemic symptoms.

Factors in Thrombotic Occlusion

The local reaction to thrombosis is more intense than to ligation. Arterial spasm, while playing some immediate part, could hardly explain the prolonged effect. There would seem to be at least three other factors favouring the more serious effects of thrombotic occlusion. In the first place the vessels beyond the complete occlusion may themselves be diseased and contribute to the decrease in the flow of blood not only by their loss of elasticity but by atheromatous plaques diminishing the size of the orifices of the very branches that form the collateral channels. A second factor may be such a propagation of thrombus or clot beyond the initial site of obstruction as to occlude collateral channels. This may sometimes be seen in the leg, where the femoral artery may be full of thrombus from its origin to the bifurcation of the popliteal artery.

Lastly, there is the possibility that small peripheral vessels in the limb, such as the digital arteries, may have become obstructed by emboli formed at the site of atheromatous narrowing by the process that has already been suggested, and that the symptoms of ischaemia may be partly dependent on this contraction of the peripheral arterial bed.

The more centrally placed total occlusion, when it ultimately occurs, may, in such circumstances, give the *coup de grâce* to tissues that are already on the verge of dissolution. There is both clinical and pathological support for such a hypothesis. Patients with claudication and femoral artery disease may often complain of localized pain and tenderness over a small artery in the foot, but a more sensitive recorder is the brain, where stenosis of the internal carotid artery is associated with transient cerebral ischaemic attacks, and where the small emboli have been seen in the retinal arteries during life and in the cerebral vessels at post-mortem examination. On the experimental side, peripheral "white emboli" have been seen to be detached from a point of arterial injury by Wharton-Jones (1851) and by Florey (1925), and have been studied more recently by Honour and Ross Russell (1962).

Surgical Considerations

With such an incomplete background of hypothesis and experimental data about the aetiology and natural history of arterial occlusive disease, surgery might well be thought to have little to offer. However, good surgical treatment has often, in the past, preceded a full and accurate knowledge of disease. Furthermore, surgery is at its best when it is called to relieve a mechanical obstruction, and until the biochemist and the experimental pathologist find proper preventive measures for both atherosclerosis and thrombosis the surgeon has much comfort to offer those afflicted with ischaemia. Medical treatment has been beset by claim and counter-claim for this and that drug, but the very multiplicity of these, and the short-lived popularity of most, cast doubt on their efficacy.

Most clinicians, but by no means all, believe that anticoagulant treatment helps to prevent recurrence of cardiac infarction and death from infarction, but there is as yet no evidence that a drug that delays clotting can influence platelet deposition in thrombosis.

Arterial disease is the most common cause of death in this country and in the United States of America, and the two most important systems of vessels to be affected are the coronary and cerebral arteries. The Registrar-General's figures show that deaths from disease of the coronary arteries are increasing in these countries. I am grateful to Dr. R. A. Davies, the Medical Officer of Health for Huddersfield, for some local statistics which show that in 1958, 1959, and 1960 the commonest cause of death in this town was coronary heart disease. In 1960 deaths from this cause totalled 344, of which 206 were of men and 138 of women. As in all other records, the incidence in the young and middle-aged—that is, below 65 years—is much higher in men than in women, whereas after 65 the

series show an almost equal incidence. Records for the Radcliffe Infirmary at Oxford were admirably reviewed by Honey and Truelove (1957).

I have already said that if a thrombosed artery is incised longitudinally it is easy, with a blunt spatula, to find the plane in which a separation of the diseased intima from the rest of the arterial wall can be effected.

If it be a short obstruction, as, for example, in the aorta and common iliac arteries, the incision may be made as long as the obstructed segment, and "the core" removed and the arterial wall sutured (Fig. 2). For longer obstructions an incision is made at each end, the thickened intima is separated with the spatula under the incisions, and the part between is bored out by specially designed reamers. This operation of thrombo-endarterectomy leaves a tube of the patient's own tissues which quite rapidly becomes relined by endothelium.

FIG. 2.—Atherosclerotic intima and massive thrombus removed from the lower aorta and both common iliac arteries by thrombo-endarterectomy.

The amount of arterial wall left behind depends upon the depth to which the disease has penetrated; usually it is the adventitia with a variable amount of the media, but sometimes it may be little more than the adventitia. The operation has advantages over grafting, for not only is the new artery constructed from autologous tissue but the mouths of its branches are also opened. This last point is amply demonstrated by the immediate flow of blood into the lumen of the vessel from its side branches

FIG. 3.—Femoral arteries, the one filled with thrombus and the other six months after thrombo-endarterectomy showing the new lining and the amount of vessel wall left behind after operation.

after the thrombus has been removed and before the clamp at the top of the artery has been released. Fig. 3 compares a cross-section of a femoral artery completely occluded by thrombus with that of a slightly distorted section of a similar artery some months after thrombo-endarterectomy (from a patient who died of another disease). A large part of the media is still present and is lined by a smooth, re-formed intima. In contrast Fig. 4 shows a coronary vessel with marked thickening of the intima and another coronary vessel from a patient who succumbed after thrombo-endarterectomy. Both vessels are filled with injection material. The wall of the second vessel consists of little more than adventitia.

FIG. 4.—Coronary arteries both full of injection mass, the one showing extensive atheromatous occlusion, the other after thrombo-endarterectomy showing that the wall consists of little more than adventitia.

Application of Lessons Learned

To what extent can the lessons learned in vascular surgery be applied to the problems presented by the coronary arteries? There is at present no satisfactory answer to this, but it may be profitable to revise the position constantly as new developments occur. The advent of surgery for diseases of other organs has often stimulated the development of new techniques for more accurate diagnosis and assessment, and there are indications now that this is happening for the coronary vessels. It is enough for medical management to be able to say that there is clinical and electrocardiographic evidence of myocardial ischaemia or that infarction has occurred in a certain part of the ventricular walls. If, however, there is a possibility of a direct attack on the diseased vessel it is necessary to know which vessel has been occluded and what is the condition of the remaining vessels. To this end angiography of the coronary arteries has made very important advances in the last few years.

A catheter is passed through the brachial artery into the ascending aorta just above the valves. If the injection of radio-opaque dye is made during diastole when the heart is beating slowly, and particularly if a little positive pressure is applied to the airways through the endotracheal anaesthetic tube (Nordenström, 1960), rapidly changing pictures can be taken in two planes at right angles that will give fairly good definition of the coronary arteries. A finer technique of individual coronary artery catheterization, together with image intensification and cineradiography, has been developed by Sones *et al.* (1959), and it would appear at present that if the necessary apparatus is available and the practice obtained, this is the clearest way of demonstrating the vessels. As was the case in the early use

of radio-opaque medium for the outlining of the bronchial tree, much important information is now coming to light from this investigation that could not even be guessed at from clinical or electrocardiographic assessment. This is likely to have a profound effect on the diagnosis, prognosis, and treatment of ischaemic heart disease.

At this stage, and without prejudice to surgical treatment, it might be profitable to look at the problem from the point of view of the general practitioner and the consulting physician. Having made the not too difficult diagnosis of myocardial ischaemia or coronary thrombosis, and having used all the medical measures

in which he believes, the question he is bound to be asked, and to which he has no authoritative answer, is "How long will the patient live?" An encouraging or an evasive answer may satisfy both patient and relatives, especially when the patient lives a long time, but it does not satisfy the physician who looks at large numbers of patients and sees how many have died that he hoped would live. If, among those patients with angina or a history of coronary occlusion, there were some way of sorting out those who had less than two or three years to live from those who would lead useful lives for ten to fifteen years, the physician would obviously be in a strong position to choose between medical and surgical treatment. It is possible that more extensive use of coronary angiography may ultimately contribute to his ability

to do this, though it still must be remembered that angiography can only indicate mechanical obstruction and that so far as is known at present this does not equate accurately with a patient's liability to thrombosis. A further difficulty is to weigh the risks of the disease against the risks of surgery, an ever-recurring one since the far-off day when arguments raged about the advisability of removing an acutely inflamed appendix.

Let us consider the present position and speculate on the future.

Speculation on the Future

The greatest contributor to the surgery of cardiac ischaemia has been Claud Beck, in Cleveland, U.S.A. His conclusions can be summarized as follows. Sudden death from coronary artery disease may be the result of single or recurrent severe infarction from which the myocardium cannot recover. It may, however, be the result of an electrical change in a heart which is otherwise capable of many years' further work. Under the title of "Hearts too good to die," Beck and Leighninger (1960) argue that electrical potentials are set up between well-oxygenated and poorly oxygenated parts of the heart which may send the ventricles into fibrillation. The recent development, at Johns Hopkins Hospital, of external cardiac massage followed by external defibrillation of the ventricles, lends great support to Beck's arguments, for there are people alive and working to-day who have "died" from an acute coronary attack and been resuscitated in this way. The aim of Beck's work is to increase the intercoronary anastomoses by causing a superficial inflammation on the surface of the heart and so to distribute oxygen to the heart muscle more evenly.

Attempts have been made to bring new blood-vessels to the heart from outside, notably by Beck (1929, 1935), by O'Shaughnessy (1936), and by Vineberg (1946). There is no doubt that such operations have been associated with some success and that injection studies have shown the new vessels in communication with the coronary branches, but experimental work by Sabiston *et al.* (1957) suggests that the blood-flow through them is small. They may be effective, as is Beck's operation, by reducing the irregularities of oxygenation.

The removal of obstruction in the coronary arteries by thrombo-endarterectomy has had an obvious appeal to those interested in vascular surgery elsewhere, but the sum total of experience is still very small. Sabiston (1960), at Johns Hopkins Hospital, has performed coronary endarterectomy on six patients suffering from severe angina pectoris with only one operative death. Cannon *et al.* (1959), in Los Angeles, have reported nine cases, and three of these patients died from operation. There are other reports of cases by May and Bailey (1958), Satinsky *et al.* (1959), Burke and Nardi (1961), and Senning (1961). In Oxford we have performed the operation six times, with three deaths on the table and two deaths two to three months after operation. All had extensive disease of all three main coronary arteries. The following brief summaries give an indication of the general physical state of the patients treated. In the arterial sense they are all seen to be "terminal."

Case Reports

Case 1.—A man aged 54, a known diabetic, had intermittent claudication for 16 years and threatened gangrene of both legs. Severe disease of both iliac and femoral arteries was demonstrated by angiography. Walking was finally limited to 20 yards (18 metres) by severe angina pectoris. There was a history of two previous cardiac infarctions. Coronary angiography was not done. At operation the heart was grossly enlarged with excessive epicardial fat. A calcified plaque in the anterior descending branch of the left coronary artery was excised. After operation the patient could walk three times round the hospital without pain in the chest or legs. He died three months later, and necropsy revealed severe atheroma of the aorta with narrowing of the aortic ostia. There was narrowing of the right coronary artery and of the left anterior descending branch except at the site of endarterectomy. Severe atheroma was present in both carotid arteries. The right external iliac artery was completely occluded, and there was severe stenosis of both left iliac arteries.

Case 2.—A man aged 51 complained of severe claudication in both legs at 20 yards (18 metres) for six months, and angina pectoris for six months. Eleven years previously he had suffered from coronary thrombosis and subsequently from duodenal ulcer and acute pancreatitis. Investigation showed irregularity and stenosis of the aortic bifurcation and both common iliac arteries, and stenosis of the right femoral artery and complete block of the right popliteal artery. Coronary angiography showed irregular atheromatous narrowing of the left coronary artery. The right was narrowed and irregular, and filled late. At operation a plaque was removed from the right coronary artery at a point about 5 cm. from its origin and a good flow through the segment was obtained. Since operation his exercise tolerance has increased, but the overall picture is obscured by domestic worries, depression, and persistent claudication in the legs.

Case 3.—A man aged 59 complained of angina pectoris of rapidly increasing severity for one year. Coronary angiography showed that the left artery was very irregular and narrow. The right was obstructed over 1–2 cm. at a point

5 cm. from its origin. Operation was performed under artificial circulation. The right coronary artery was extensively narrowed. The reamer was used and about 8 cm. of thrombus mobilized. The distal part was removed through the arterial incision, but the proximal part prolapsed into the aortic lumen, from which it had to be removed by an incision in the aortic wall. The left anterior descending artery was totally occluded and calcified over its visible length. Endarterectomy was performed but the left main artery was torn behind the pulmonary artery. Attempts to suture this led to stenosis and left ventricular fibrillation, which was fatal. At necropsy the treated arteries were seen to have a good lumen, but the stripping had included most of the media of the vessels. There was severe distal atheromatous stenosis of both arteries. Atheroma was present in the carotid arteries with stenosis of left common and left internal carotid arteries. There was also complicated atheroma of the aorta and iliac vessels.

Case 4.—A woman aged 57 had von Recklinghausen's generalized neurofibromatosis and also severe generalized arterial disease causing stenosis of all iliac and femoral and left carotid vessels with a long history of cold hands, fainting, and dizzy attacks. A successful right femoral thrombo-endarterectomy had been performed two years previously with relief from incipient gangrene. She was admitted to hospital with severe anginal attacks, at first when walking or with emotion but later at rest in bed. At operation the heart was huge, with a notable excess of epicardial fat. The right coronary artery was solid. During thrombo-endarterectomy the right coronary artery was torn at its origin from the aorta but was sutured. The patient collapsed and died as she was leaving the theatre.

Case 5.—A man aged 44 complained of angina of effort for eight years and for three years angina at rest, with as many as four attacks in the day and three at night. There was no relief from sympathectomy. Coronary angiography showed that the right coronary artery was obstructed at its origin, but that beyond this it had a good lumen filling from collaterals. The left anterior descending branch was blocked for 2 cm. at its origin and showed poor filling distally. Operation was performed under arrested circulation with partial by-pass between the left auricle and the left femoral artery. The patient was cooled to 25° C., at which temperature ventricular fibrillation occurred. Extensive calcification extended through the vessel walls to the adventitia. Attempted thrombo-endarterectomy resulted in multiple tears in the vessel wall. The adventitia was in parts so flimsy that closure was ineffective and a coronary circulation was never re-established. The patient died on the table.

Case 6.—A man aged 40 had familial hypercholesterolaemia. His father had died of coronary thrombosis at the age of 46. The patient had had intermittent claudication for three years and angina pectoris for 17 years. Coronary angiography showed that the right vessel was grossly narrowed and irregular, with small collaterals. The left anterior descending branch was irregularly narrowed and at a point 5 cm. down it was almost completely obstructed. The circumflex branch was tortuous and irregularly narrowed. At operation only the right coronary artery was treated: 6 cm. of thrombus and thickened intima was removed. Suture of the artery probably produced some narrowing. The patient made an uninterrupted recovery and returned home. Two months later he collapsed and died, presumably from coronary thrombosis, but no details are available.

Whether we believe that anticoagulant treatment is effective or not, and whatever we may believe about the prognosis of patients with angina pectoris or after a single infarction, we have to face the knowledge that, in this town alone in 1960, 120 patients under the age of 65 died from coronary occlusion and that more were probably lost in 1961. Those who survive stay with us and remind us of our success; those who succumb pass from our memory and so fail to influence our judgment

as much as they should. If it is right that when we give treatment and fail we should constantly review our methods and our skills, it is also true that when we avoid the responsibility of intervention we should be reminded of the results of our inactivity.

We can be sure that there will be deaths from surgical treatment but that these will diminish when patients are given a chance earlier, before all the large coronary arteries are extensively involved. Coronary angiography is likely to play an important part in the selection of such patients. If the history of anticoagulant therapy forms any sort of a parallel it will be a very long time before there is conclusive statistical support for surgical treatment. In the meantime it is important that patients should be considered for one or other form of treatment on the basis of the fullest possible investigation so that as numbers do become available they can be useful in statistical surveys.

Summary and Conclusion

Mechanical factors in arterial disease are discussed.

The part played by local arterial obstruction in the production of peripheral emboli is emphasized.

The application of thrombo-endarterectomy to the coronary vessels is described and case reports of six patients are given.

Coronary angiography has an important part to play in the detailed investigation of coronary artery disease. The early demonstration of stenosis or complete obstruction may well be of vital importance in assessing prognosis and in the selection of patients for surgical treatment. The most logical surgical treatment is removal of the arterial obstruction where this is possible.

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The Chartered Society of Physiotherapy has sponsored a film, "Posture and Movement in Childbirth," which deals, in two parts, with "Posture in Pregnancy" and "Post-natal Rehabilitation." The film is directed by Mrs. M. Williams, M.C.S.P., T.M.A.O.T., in collaboration with the Obstetric Unit of Edgware General and Bushey Maternity Hospitals. It is a 35 mm. colour filmstrip and details may be obtained from the producers, Camera Talks, 23 Denmark Place, London W.C.2.

ABO BLOOD GROUPS IN RELATION TO ISCHAEMIC HEART DISEASE

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The increasing incidence of ischaemic heart disease in certain countries has led many to the view that environmental influences are accelerating its development. Studies on groups have shown that Japanese who have come under the influence of the American mode of life in Hawaii and California have a greater susceptibility than Japanese in Japan (Keys *et al.*, 1958). There are similar group studies on recent immigrants into Israel (Toor *et al.*, 1957), and the differences in mortality dependent upon social class (Registrar-General, 1954) in England and Wales add to this view. Contrariwise, ischaemic heart disease has close associations with certain inherited disorders—for instance, essential xanthomatosis—and in clinical practice one is impressed by the high prevalence of the disease among members of the patient's family. Indeed, this impression is confirmed by the survey of Gertler and White (1954) on the frequency of the disease in parents and siblings of affected individuals. These authors, too, have emphasized the role of constitutional factors in the development of the disease by reporting the increased frequency of the disease in mesomorphic individuals as opposed to ectomorphs. Body build may be influenced by feeding in childhood, and likewise familial relationships could arise as readily from environmental causes. The differentiation of the role of genetic from environmental factors becomes an important issue, as environmental influences may be modified.

The increasing use of the blood-group distribution in the study of diseases has prompted us to use this technique in an attempt to assess the role of genetic factors in the development of ischaemic heart disease.

The Population of the Cape Peninsula

In a previous report (Bronte-Stewart *et al.*, 1955) a description was given of the three main racial groups resident in the Cape Peninsula—namely, Bantu, Cape Coloured, and White (European)—which was felt to be pertinent to their differing prevalence of ischaemic heart disease. In that report the Whites were treated as a homogeneous group, but for the purposes of the present study it became necessary to divide the Whites into English-speaking, Afrikaans-speaking, Jewish, and other groups and define the measure whereby this separation was achieved. There are distinct differences between these groups. Apart from home language, there are differences in outlook, occupation, temperament, dietary customs, and religious faith, but the environmental differences are not as marked nor are they dictated to