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SERIAL ARTERIOGRAPHY IN ATHEROSCLEROSIS*

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THE PRESENT DAY concepts of the pathogenesis of atherosclerosis have been built upon autopsy studies in man and experimental animals. The drawback of this reliable approach to the problem is that the atherosclerotic process can be visualized only at one point in time in a given case. Nevertheless, by integrating all the facts from a large autopsy series, some idea has been gained as to the nature of early and late lesions. However, the fate of an individual plaque has never been followed during life, and there is thus little accurate knowledge as to the rate of progression of the disease, nor is it known definitely whether spontaneous regression ever occurs. This failure to visualize the atheromatous plaque from time to time during life has placed the assessment of aggravating or ameliorating factors largely upon subjective or, at best, non-specific grounds.

Perhaps the closest approach to a feasible periodic study of the vascular tree has been by ophthalmoscopy. This method has been extensively reviewed and although it permits direct visualization of the fundal vessels, certain objections have been raised.[1] The type of vessel observed in the retina is of arteriolar rather than arterial calibre, and only the external aspect is seen. Furthermore, in the great majority of cases of atherosclerosis there are no visible atherosclerotic plaques in the retinal vessels. Ophthalmoscopy is considerably more valuable in studying hypertensive vascular disease than it is in the assessment of atherosclerosis.

Other criteria used to assess atherosclerosis have in actual fact been only observations of secondary phenomena. Changes in symptoms, skin temperature, digital blood flow and electrocardiogram are all influenced by many factors other than atherosclerosis; they therefore fall short as accurate criteria of the disease. The relationship of biochemical studies to atherosclerosis is even more uncertain.

To overcome these difficulties it was decided to study the femoral and popliteal arteries by serial arteriography. In this way, using a standard x-ray technique, it is possible to observe the natural history of atherosclerotic plaques. The effect of influencing factors upon the disease can likewise be evaluated. In a pathological study of the femoral and popliteal arteries previously reported,[2] it was noted that atherosclerosis in these arteries was uniformly associated with atherosclerosis elsewhere. Thus of the 152 cases studied at autopsy, 27 had had a myocardial infarction at some time, and none of these 27 was free of atherosclerosis in the thigh vessels. Aortic atherosclerosis likewise tended to parallel the atherosclerosis of these lower limb vessels in degree. Dow[3] in his detailed examination of all the main arteries in the body, noted that the abdominal aorta, and common iliac, femoral and popliteal arteries were more extensively affected than any other vessel. A large combined x-ray and pathological study made by Lindbom[4] revealed that thrombosis of the lower limb vessels is considerably more common than coronary thrombosis in the older age group. On these grounds it may be taken that the degree of atherosclerosis in the femoral and popliteal arteries is a reliable indication of the degree of atherosclerosis likely to be present elsewhere.

MATERIAL AND METHODS

The patients studied by arteriography were selected from the Queen Mary Veterans' and St. Anne's Hospitals. All were men, varying in age from 55 to 77, with an average age of 64 years, and were those who had shown many of the clinical manifestations ordinarily considered to be associated with atherosclerosis. Frequently the cases had been clinically diagnosed as "generalized atherosclerosis."

Bilateral femoral arteriography was performed in all cases. Premedication included Seconal gr. 1½, atropine gr. 1/150 and morphine gr. 1/6 to ¼, given half to one hour before the procedure. No local anæsthesia was used, as this was found to make arterial puncture more difficult. In spite of this, there was only slight pain during the insertion of the needle. A preliminary test dose of 35% Diodrast was given to all patients, 0.5 c.c. being injected intravenously 15 to 20 minutes prior to the intraarterial injection. No instance of sensitivity to Diodrast was encountered. The

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