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The serialographic films taken over a 7½ second period demonstrated successful filling of the same vessels on repeat examinations. The degree of rotation in subsequent arteriograms matched well with the originals.

Table I shows the results in the control group while the results of ascorbic acid therapy are demonstrated in Table II.

Fig. 2 illustrates the arteriographic evidence of progression of an atheromatous plaque in case 12, while Fig. 3 shows the improvement in case 11.

Cases 1 and 5 were followed up for a time as controls and a second arteriographic study was made. They were then placed on treatment as cases 9 and 8 respectively and a third x-ray study was made after an interval of treatment. Case 15 was always in the treated group but was reviewed by arteriography on three occasions.

It will be seen from the tables that intimal plaques may enlarge or become smaller, change being restricted to only some of the plaques visualized in a given case. Regression and progression of plaques were never found co-existent in the same case. Without treatment none of the 6 cases improved; 3 cases deteriorated while 3 were unchanged. In the treated group, 6 out of 10 cases improved while 3 became worse and 1 was stationary. The development of post-stenotic dilatation distal to a plaque was observed only once (Case 1).

Although old occlusions were observed radiologically several times, recanalization was not a striking feature. The channel was never observed to increase in calibre between examinations. Collateral vessels formed a much more prominent source of blood supply following occlusion. No entirely new plaques were seen to develop during the period of observation.

There was a correlation between the arteriographic changes and the signs and symptoms in only some of the cases. The plasma cholesterol levels failed to fluctuate in relation to the changes in the plaques.

DISCUSSION

Except for a single case reported by Lindbom,[4] this is the first time that the evolution of atheromatous plaques has been observed over a period of time. Not only have the plaques been demonstrated, but they have been followed up and any changes noted in them. It is true that, in the projection employed, the plaques situated along the medial and lateral walls of the arteries were best visualized and those along the anterior and posterior walls were indistinct. However, by standardizing the method, on all subsequent examinations the same plaques were demonstrated. The period of observation so far has been short, but it is believed that it has been possible to visualize the same atheromatous plaques in the same patient on different occasions even though slight rotation of the limb was sometimes unavoidable and the relationship of the vessel to the femoral shaft changed a little. The fact remains that on some of the patients it has been possible to take three different arteriograms at various time intervals, and some of the plaques have remained perfectly constant in outline while other plaques along the same vessel showed a change.

From these serial arteriograms it may be appreciated that atherosclerosis is not always a slow and inevitably progressive disease. Plaques may enlarge or become smaller with surprising rapidity. The fact that only some of the plaques in a given case are seen to progress in the interval of observation is evidence in favour of the importance of local rather than systemic etiological factors. This local factor is most likely partly mechanical in nature, as outlined in a previous paper.[5] Intimal hæmorrhage is very common[6] and plays an important part in enlarging the plaque in which it occurs.[4] Although there is no proof, it would seem likely that in the present series some of the instances of enlargement of plaques are attributable to intimal hæmorrhage.

Of major importance is the observation of regression of plaques. Regression of plaques in cholesterol-fed herbivorous animals is said to be very slow when cholesterol feeding is discontinued.[7] This is probably due to the prolonged persistence of the etiological factor, namely hypercholesterolæmia. In the dog, on the other hand, hypercholesterolæmia passes off in less than a week from the cessation of cholesterol feeding and regression of lesions is rapid.[8] The report of regression of atherosclerotic lesions in human subjects dying of wasting diseases[9] is speculative, as the arteries were seen only at one point in time. Furthermore advanced atherosclerotic plaques are a striking feature of the arteries of victims of malnutrition.[10]