TABLE I.—INDICATING THE NUMBER OF ANIMALS IN THE VARIOUS EXPERIMENTAL GROUPS WITH AND WITHOUT ATHEROSCLEROSIS AND THE DEGREE OF THE LESIONS IN THOSE SHOWING THEM

Experiment	*Total animals*	*With atherosclerosis*	*Without atherosclerosis*	*Average degree of atherosclerosis*
Scorbutogenic diet 42 days with ascorbic acid added from the beginning	12	0	12	0
Scorbutogenic diet for periods of from 21 to 30 days	27	11	16	2.5+
Scorbutogenic diet for 21 to 30 days, then ascorbic acid for 1 to 5 days	25	9	16	2.5+
Scorbutogenic diet for 21 to 30 days, then ascorbic acid for 7 to 27 days	25	7	18	2.5+

atherosclerosis in the various groups and the average degree of their lesions is set forth in Table I. Variations in the morphology of the lesions were as follows:

Atherosclerotic Lesions of Scurvy

The earliest lesions were characterized by a diffuse deposit of stainable lipid along the internal elastic membrane and in the immediately adjacent intima. This staining faded out gradually at the extremities of the plaques, blending into apparently intact internal elastic membrane. A heaping up of lipid in the middle portion of such early lesions eventually extending to involve the whole thickness of the intima was seen in the intermediate and advanced plaques (Fig. 1). Macrophages were noted in small numbers only. Each individual plaque appeared as a confluent mass of stainable lipid with no fat-free areas associated.

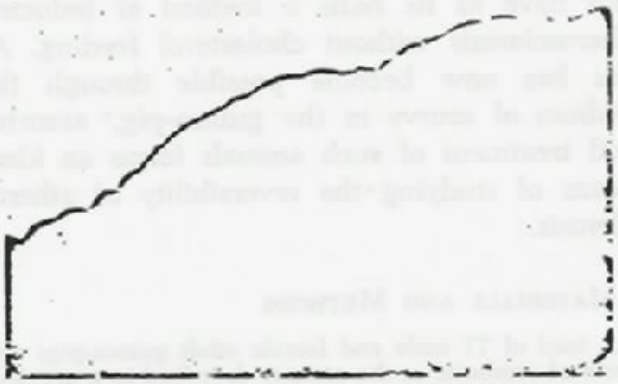

Fig. 1.—High power view of an early atherosclerotic lesion in a scorbutic guinea-pig. The dark internal elastic membrane and adjacent intima represents stained lipid (Scharlach R).

Atherosclerotic Lesions of Treated Scurvy

After as little as two days of ascorbic acid therapy, the early atherosclerotic plaques stained less intensely with Scharlach R and soon lost their diffuse lipid deposit completely. Numerous macrophages meanwhile became apparent, some on the intimal side of the internal elastic membrane and some on the medial side (Fig. 2). All these macrophages stained intensely for fat. Later stages of the process were characterized by a decrease in the bulk of lipid within macrophages. The end stages of lipid resorption were not detected, as this degree of atherosclerosis appeared to heal rapidly without permanent sequelæ.

Advanced lesions of atherosclerosis presented quite a different pattern, being considerably more resistant to resorption. After a period of about seven days, large plaques were found to be no longer a confluent mass of lipid filling the intima. Instead, the lipid was aggregated into separate islands with lipid-free areas intervening (Fig. 3). Sometimes such islands had a few macrophages around them but often none were noted. Sometimes there appeared to be diffusion of lipid into the inner layer of the arterial media. Even after a further period of nine days, very little further change could be noted in such plaques. Although animals killed as long as 27 days after the initiation of ascorbic acid therapy

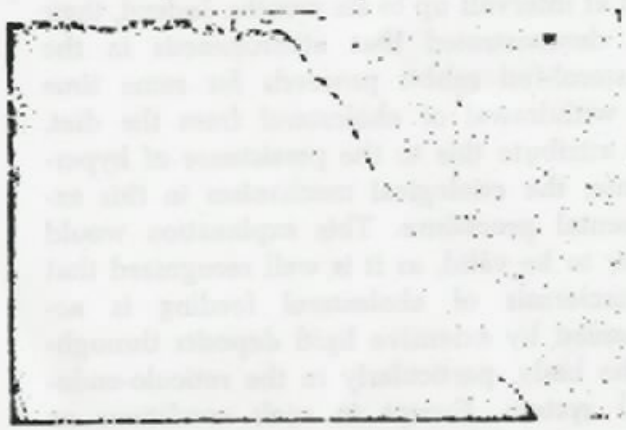

Fig. 2.—High power view of a plaque similar to that shown in Fig. 1. This plaque is undergoing resolution under the influence of two days of ascorbic acid therapy. Note that the extracellular stainable lipid is almost all gone while several lipid-laden macrophages are seen in apposition to the internal elastic membrane both on its intimal and medial surfaces (Scharlach R).