Fine Structure of Spontaneous Atherosclerosis of the Aorta in the Squirrel Monkey

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SPONTANEOUS AND EXPERIMENTALLY INDUCED ATHEROSCLEROSIS have been investigated in several nonhuman primate species including rhesus (*Macaca mulatta*),[1-3] *Cebus*,[4,5] *Lagothrix*,[5] chimpanzee,[5,6] baboon,[7,8] and the squirrel monkey (*Saimiri sciureus*).[5,9,10] The aortic lesions in these monkeys were identified by gross examination or by Sudan IV staining of formaldehyde-fixed aortas. Such lesions have been studied extensively by light microscopy [1,3-7,9,10] and more recently by electron microscopy in the rhesus monkey [2] and baboon.[8]

In previous studies [5] in this department jungle-killed squirrel monkeys were found to have the greatest amount of aortic sudanophilia among New World monkeys. Furthermore, diet-induced aortic sudanophilia was greater in the squirrel monkey than either the *Lagothrix* or *Cebus* monkey fed similar purified diets.[11,12]

The purpose of this study was to investigate by light and electron microscopy the normal anatomy and spontaneous atherosclerosis in aortas of squirrel monkeys fed a basal semipurified diet for 1 year. This investigation forms the basis for ultrastructural studies of atherosclerosis in monkeys fed the basal diet with added fat and cholesterol.

Materials and Methods

Six young adult male squirrel monkeys (*Saimiri sciureus*), weighing from 650 to 920 gm., were obtained after being observed and having adjusted to the climate for 6 months (Trefflich's Pet Department Store, Inc., New York). Upon arrival in the laboratory they were caged in a separate room, fed Purina Monkey Chow, and observed for signs and symptoms of infectious disease for 30 days, then changed to a semipurified diet with the following percent composition: vitamin-free casein 25.0; corn oil 8.0; sucrose 62.4; salts IV [13] 4.0; choline 0.5; inositol 0.1. The following

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were added per kilogram of diet: thiamine 10 mg.; niacin 49 mg.; calcium pantothenate 30 mg.; folic acid 1 mg.; biotin 0.2 mg.; Vitamin A acetate 12,500 units; Vitamin D_3 2000 units.

After 1 year the animals were killed by femoral vein injection of a sublethal dose of sodium pentobarbital followed by exsanguination from the right ventricle after opening the thoracic cavity. The aorta was perfused gently by injecting 10 ml. of physiologic saline into the left ventricle after cutting the common iliac arteries transversely, to allow easy drainage and prevent backflow of blood. The aorta was perfused in the same manner with 10 ml. of 3% glutaraldehyde buffered at pH 7.3 by 0.1 M phosphate buffer, and opened longitudinally in situ starting from the left ventricle through the aortic valve to the bifurcation of the abdominal aorta. Each aorta was dissected free posteriorly, the heart removed by cutting the aorta just proximal to the aortic valve ring, and excess fat removed from the adventitial surface. Care was taken not to strip away the innermost layer of fat in order to preserve adventitial nerves and vasculature. The aorta was stretched gently and pinned flat on cardboard, adventitial surface down to the same length it occupied in situ, and immersed at room temperature in 3% buffered glutaraldehyde for 2 hr. It was divided into four segments as follows: (1) ascending thoracic aorta (from just distal to the aortic valve to the origin of the innominate artery); (2) descending thoracic aorta (from just distal to the insertion of the ligamentum arteriosum to the origin of the sixth intercostal arteries); (3) proximal abdominal aorta (from the origin of the ninth intercostal arteries to the origin of the celiac axis); (4) distal abdominal aorta (from the origin of the renal arteries to the bifurcation of the abdominal aorta). Eight small rectangular blocks of aorta, measuring 6 by 2 by 1 mm. each, were taken from the four regions—a total of 32 blocks per aorta. They were cut so that the short end of the rectangle (surface to be sectioned later) represented the transverse plane in four tissue blocks and the longitudinal plane in the other four tissue blocks. Care was taken not to touch the endothelial surface at any time during dissection or trimming. No blocks were taken near previously cut margins or the origin of a branching artery.

Tissue blocks were immersed in 0.1 M phosphate buffer at pH 7.3 for 1 hr., postfixed for 1 hr. at room temperature in 1% osmium tetroxide buffered with 0.1 M phosphate, rinsed in distilled water, dehydrated in ethanol (50%, 75%, 95%, and absolute for 15 min. each), immersed for 2–5 min. in propylene oxide, and left overnight uncapped in a well-ventilated hood in a mixture of 50% propylene oxide and 50% Epon 812. Blocks were embedded flat in aluminum weighing dishes containing Epon 812 and put in an incubator at 60° C. for 48 hr. to polymerize.

Sections were cut of every block using glass and diamond knives in Porter-Blum I or II ultramicrotomes. Thick sections of each block were mounted on glass slides, stained with 0.5% toluidine blue buffered at pH 11 with sodium borate, permanently mounted in balsam, and examined by light microscopy. Ultrathin sections were mounted on bare 200-mesh copper grids and stained with 5% uranyl acetate followed by lead citrate.[14] Sections were examined and micrographs taken in a Philips 200 electron microscope with a 60-kv acceleration voltage.

An additional 5 adult male squirrel monkeys between 2 and 3 years of age were killed by injection of an overdose of sodium pentobarbital into the femoral vein. The aortas were perfused in situ with 10 ml. of physiologic saline followed by 10 ml. of 10% formaldehyde. They were opened in situ and removed in the same manner as those used for electron microscopy. Each aorta was rolled, beginning at the cardiac end, into a tightly wound coil with the endothelium on the concave inner surface. They were pinned to avoid unwinding, fixed in 10% formaldehyde, dehyrated in step-graded ethanol, cleared overnight in cedar wood oil, and infiltrated with a mixture of melted paraffin and Tissuemat (Fisher Scientific Company) in a partial

vacuum at −25 to −30 mm. Hg before embedding in the same material. Sections were cut at 5 μ and stained with hematoxylin-eosin-alcian blue or Verhoeff-Van Gieson stains for study by light microscopy of the anatomic variations in the different regions of the aorta.

Observations

Atheromata were not seen by gross examination during dissection and trimming of tissue blocks. Pathologic intimal changes similar to those found in man were seen by light and electron microscopy.

The aorta varies in structure progressively along its length (Fig. 1). In the thoracic region (Fig. 2A) elastic tissue laminae were of uniform thickness and interspersed evenly with alternating bands of smooth-muscle cells. The internal elastic lamina was not distinct. In the abdominal region (Fig. 2B), where the aortic wall was not as thick, elastic laminae were spaced more widely, and the internal elastic lamina was more prominent and thicker than the other elastic laminae. In this region the adventitia was composed of a more dense or compact connective tissue than in the thoracic region.

The fraction of total surface area embedded per aorta (32 blocks) was estimated to be approximately 5%. The average number of lesions found per aorta was four. In Epon-embedded thick sections, atheromata in the thoracic region were recognized as intimal thickenings containing lipid. In the abdominal segment distal to the renal arteries only intimal thickening was noted by light microscopy. Approximately the same number of lesions were found in the ascending thoracic, descending thoracic, and distal abdominal aortas. Very few lesions were found in the proximal abdominal aorta (from the origin of the ninth intercostal arteries to the origin of the celiac axis). Ultrathin sections of areas having lesions and nearby uninvolved areas were studied by electron microscopy.

A fatty streak of the ascending aorta is seen in Fig. 3. Endothelium in thoracic lesions was bulged into the lumen by thickened intima composed superficially of cells containing clear lipid vacuoles and an occasional osmiophilic lipid droplet. Deeper in the intima were smooth-muscle cells containing lipid vacuoles, and a fragmented, discontinuous internal elastic lamina beneath which were smooth-muscle cells in their usual anatomic site, some containing smaller, osmiophilic lipid droplets. There were some lipid bodies in the extracellular space above and below the internal elastic lamina, but the lipid-laden cells and smooth-muscle cells were packed rather densely, filling most of the extracellular space. At higher magnification (Fig. 4) beneath the endothelium and seen occasionally between cells was amorphous, homogeneous material simi-

lar to that composing basement membrane. Toward the media there were large numbers of collagen fibers and clumps of elastin. Other areas were similar, but the fat-containing cells in the intima were less compact (Fig. 5). Occasional myelin forms were seen in the cytoplasm of these cells and were very numerous in the extracellular space. Nearby, normal areas of thoracic aorta were composed of flat endothelium, underlying elastic laminae that were not fragmented and deeper lying smooth-muscle cells that did not contain lipid droplets or vacuoles.

In less advanced fatty streaks of the thoracic aorta (Fig. 6), the endothelium was fairly flat, and lipid-laden cells were fewer in number (one to two cell layers thick). Smooth-muscle cells at the base of this type of lesion did not contain large lipid droplets or vacuoles. In these fatty streaks, smooth-muscle cells were present in the intima but were beneath lipid-laden cells. Smooth-muscle cells were seen on occasion in uninvolved areas away from fatty streaks on the luminal side of the internal elastic lamina and slightly bulged the endothelium (Fig. 7). These smooth-muscle cells were small, having large numbers of mitochondria, abundant rough endoplasmic reticulum, and a fairly prominent Golgi complex. They had numerous myofilaments, fusiform electron-dense bodies along the inner aspect of the cytoplasmic membrane, and did not contain lipid droplets or vacuoles.

Intimal lesions of the abdominal aorta distal to the renal arteries were different from those of the thoracic aorta. An area of uninvolved, normal abdominal aorta sectioned longitudinally is seen in Fig. 8. The endothelium was seen overlying the prominent internal elastic lamina. An area of intimal thickening of the abdominal aorta is seen in Fig. 9. These lesions bulged the endothelium slightly and were composed of smooth-muscle cells oriented perpendicular to the longitudinally running cells of the media, the long axes being at 90° to each other. Smooth-muscle cells were not closely packed, and the extracellular space was large, containing numerous collagen fibers and pale staining homogeneous material. At slightly higher magnification (Fig. 10), the extracellular space was seen to contain numerous osmiophilic lipid droplets. Similar lipid droplets were present in the cytoplasm of smooth-muscle cells. Lipid-laden cells seen superficially in the thoracic aorta (Fig. 3–6) were not found in lesions of the distal abdominal aorta. The internal elastic laminae had gaps here and there but were not as irregular and were not fragmented as they were in thoracic lesions. An occasional cell, similar to those seen in thoracic lesions (Fig. 5), was seen just beneath the endothelium of the abdominal aorta (Fig. 11). These cells contained irregular tubular

structures in the cytoplasm but were not identified in the typical lesion described in the abdominal aorta.

Discussion

The anatomic differences between thoracic and abdominal aorta described in this report may be of great significance in the study of atherogenesis at the ultrastructural level. Striking morphologic differences exist between intimal lesions in these areas.

Lesions of the thoracic aorta (Fig. 3–6) were composed of lipid-laden cells resembling blood monocytes lying superficially just beneath the endothelium. Deeper in the intima were smooth-muscle cells containing lipid droplets and vacuoles lying beneath the lipid-laden cells and adjacent to the slightly irregular, fragmented internal elastic lamina. When smooth-muscle cells were seen superficially just beneath endothelium in the thoracic aorta (Fig. 7), they were not associated with lesions and did not contain lipid droplets or vacuoles. Lipid-laden cells in thoracic lesions of the present study are similar morphologically to lipid-laden monocytes and could have originated as monocytes from the blood stream, although none were seen breaching the endothelium. Some lipid droplets in this study appear to have a limiting membrane at low magnification (Fig. 6). At higher magnification the trilaminar structure of typical lipoprotein membranes, according to Fawcett,[16] is lacking. The dark osmiophilic periphery of lipid droplets like these may be condensation of the phospholipid moiety at the margin as described by Schoefl.[17] How and where these cells phagocytize or synthesize their lipid needs further investigation. In the normocholesterolemic squirrel monkey, perhaps the lipid is phagocytized in the intima, and was not carried there by the monocytes.

Intimal lesions in the abdominal aorta (Fig. 9 and 10) were composed of widely spaced smooth-muscle cells containing lipid droplets. Lipid-laden cells seen superficially in thoracic aorta lesions were not found in the abdominal aorta. The large extracellular space contained definite osmiophilic lipid droplets (Fig. 10), which may have been derived from lipoproteins that entered the intima from the blood stream, by extrusion from smooth-muscle cells, or by smooth-muscle cell breakdown. The latter two possibilities are less likely, since no lipid droplets were seen leaving smooth-muscle cells, and degenerating smooth-muscle cells were not found. The origin and ultimate fate of these extracellular lipid droplets is of primary importance in understanding the mechanism of atherogenesis and certainly merits future investigation.

The cellular components of the lesions described in the present study

have been seen in experimentally induced lesions of the thoracic aorta in the rhesus monkey.[2] The intimal smooth-muscle cell was the predominant cell type identified in baboon aortic fatty streaks,[8] and ultrastructural studies of human atheromata [14,15,18,19] have resulted in the identification of similar cell types. Fatty streaks and atheromata described in these other studies were more advanced lesions than the spontaneous lesions described here in the squirrel monkey.

Orientation of tissue blocks in the transverse and longitudinal planes resulted in an interesting observation that may be of significance. Intimal smooth-muscle cells in abdominal aortic lesions were perpendicular to longitudinally running smooth-muscle cells of the underlying media, their long axes being at right angles to one another (Fig. 8–10). Andrus and Portman [5] thought this to be the case in their light microscopic studies of monkey atheromata. In the human fetus and newborn, smooth-muscle cells in the intima are considered to be normal during growth and development.[20] Perhaps the presence of circularly oriented smooth-muscle cells in the intima of adult humans and other primates may represent one of the earliest pathologic changes of atherosclerosis. Additional studies in young, immature primates subjected to diets containing various types of fat may help to answer this and other questions concerning the initial phases of atherogenesis.

Summary

Significant anatomic differences between the thick elastic thoracic aorta and the thinner abdominal aorta have been described in the squirrel monkey by light microscopy. The fine structure of spontaneous intimal lesions (one to five cell layers in thickness) varied considerably in the thoracic compared to the abdominal aorta, perhaps because of the anatomic differences.

In the thoracic aorta, lesions were three to five cell layers thick and were composed superficially of densely packed lipid-laden cells resembling blood monocytes; underlying smooth-muscle cells containing lipid; an irregular, fragmented internal elastic lamina; and very little extracellular lipid.

In the abdominal aorta, lesions were only one to two cell layers thick and were composed of loosely packed smooth-muscle cells containing lipid; a thicker, more distinct nonfragmented internal elastic lamina that appeared normal; and abundant extracellular lipid. Lipid-laden cells resembling blood monocytes were not found in these lesions. Of additional significance was the presence of definite round osmiophilic lipid

droplets of similar size and appearance in smooth-muscle cells and the adjacent extracellular space in abdominal aortic lesions.

Smooth-muscle cells found in the intima were oriented at right angles (90°) to the longitudinally running smooth-muscle cells of the underlying media. This significant finding was particularly evident in abdominal aortic atheromata.

Comparison of aortic intimal lesions in the squirrel monkey with the fine structure of more advanced atheromata in man and other primates has revealed numerous similarities. Accordingly, the squirrel monkey can be considered a satisfactory animal model for the study of aortic atherosclerosis in man.

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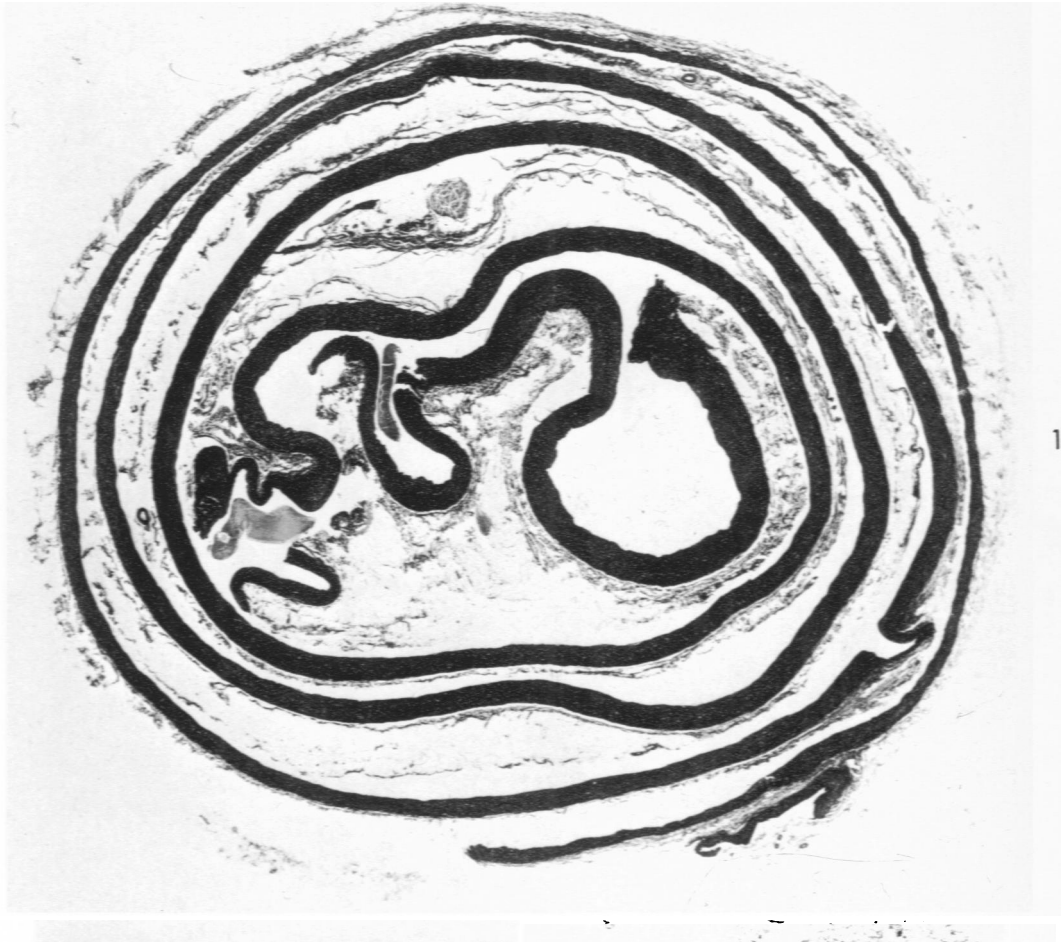
Legends for Figures

Fig. 1. Coiled squirrel monkey aorta varying in structure progressively throughout its length from the thick thoracic portion in the center of the section to the thin distal abdominal portion at the periphery. The endothelial surface is on the inner, concave surface of the aorta. Verhoeff-Van Gieson. × 15.

Fig. 2A. Thick ascending thoracic aorta (endothelial surface at top) is composed of dark, uniform, evenly spaced elastic laminae with interspersed light areas of smooth-muscle cells. The internal elastic lamina is similar to those of the rest of the aortic wall. Verhoeff-Van Gieson. × 100.

Fig. 2B. Thin distal abdominal aorta (endothelial surface at top) is composed of more widely spaced elastic laminae and light areas of smooth-muscle cells are relatively more prominent. Note the distinct internal elastic lamina, which is thicker than those beneath it, and the compact, dark adventitia along the lower margin. Verhoeff-Van Gieson. × 100.



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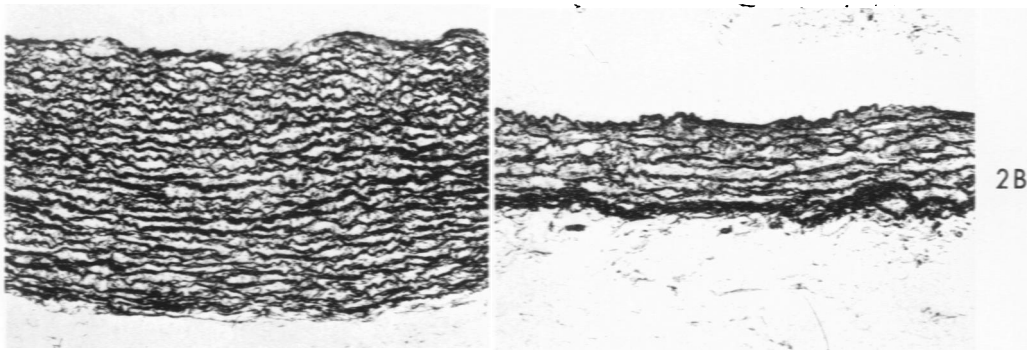

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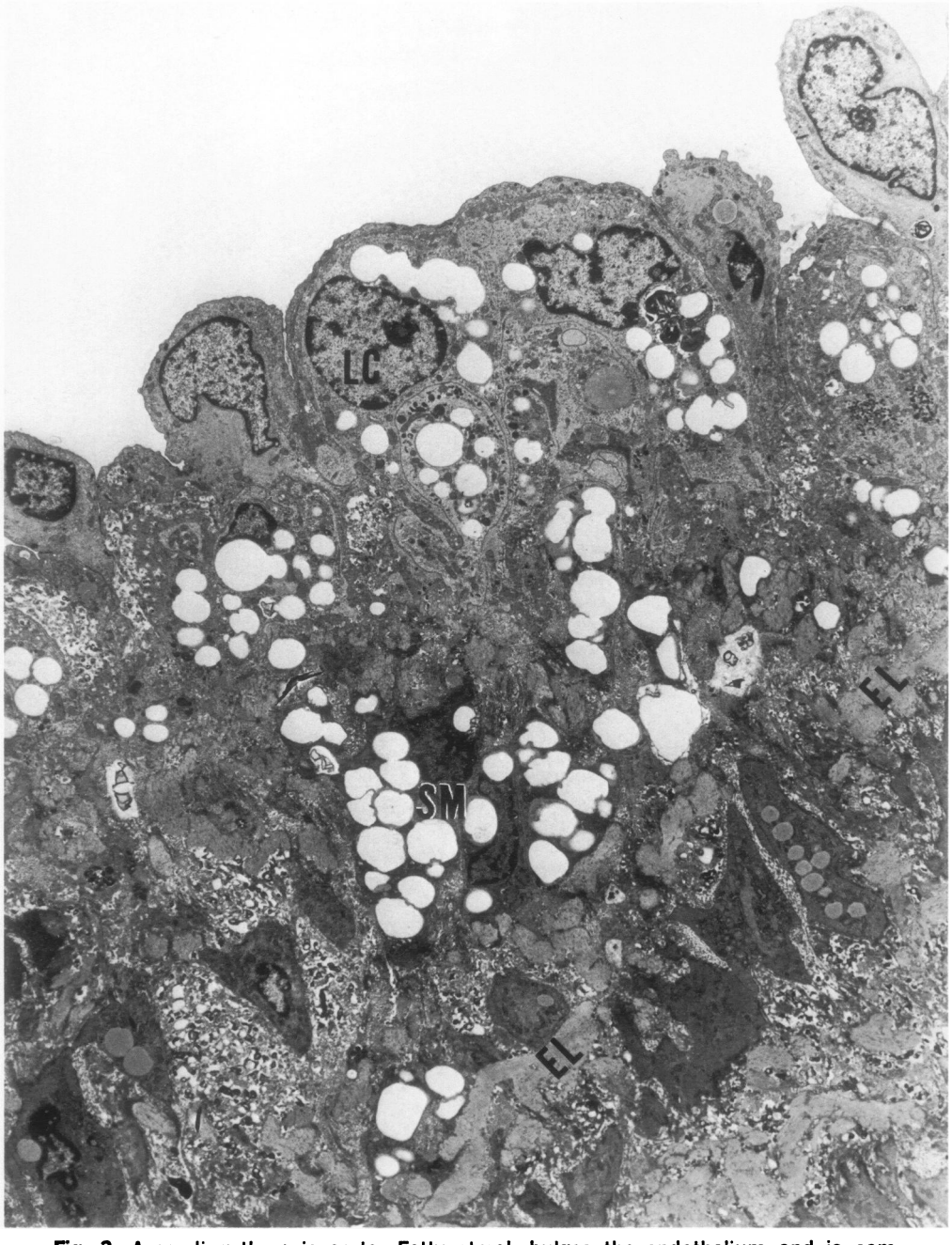

Fig. 3. Ascending thoracic aorta. Fatty streak bulges the endothelium and is composed superficially of densely packed lipid-laden cells (*LC*) containing clear lipid vacuoles and an occasional osmiophilic lipid droplet. Beneath these cells are smooth-muscle cells (*SM*) containing lipid vacuoles; an irregular, fragmented internal elastic lamina (*EL*); and deeper lying smooth-muscle cells containing osmiophilic lipid droplets. Uranyl acetate and lead citrate. × 3200.

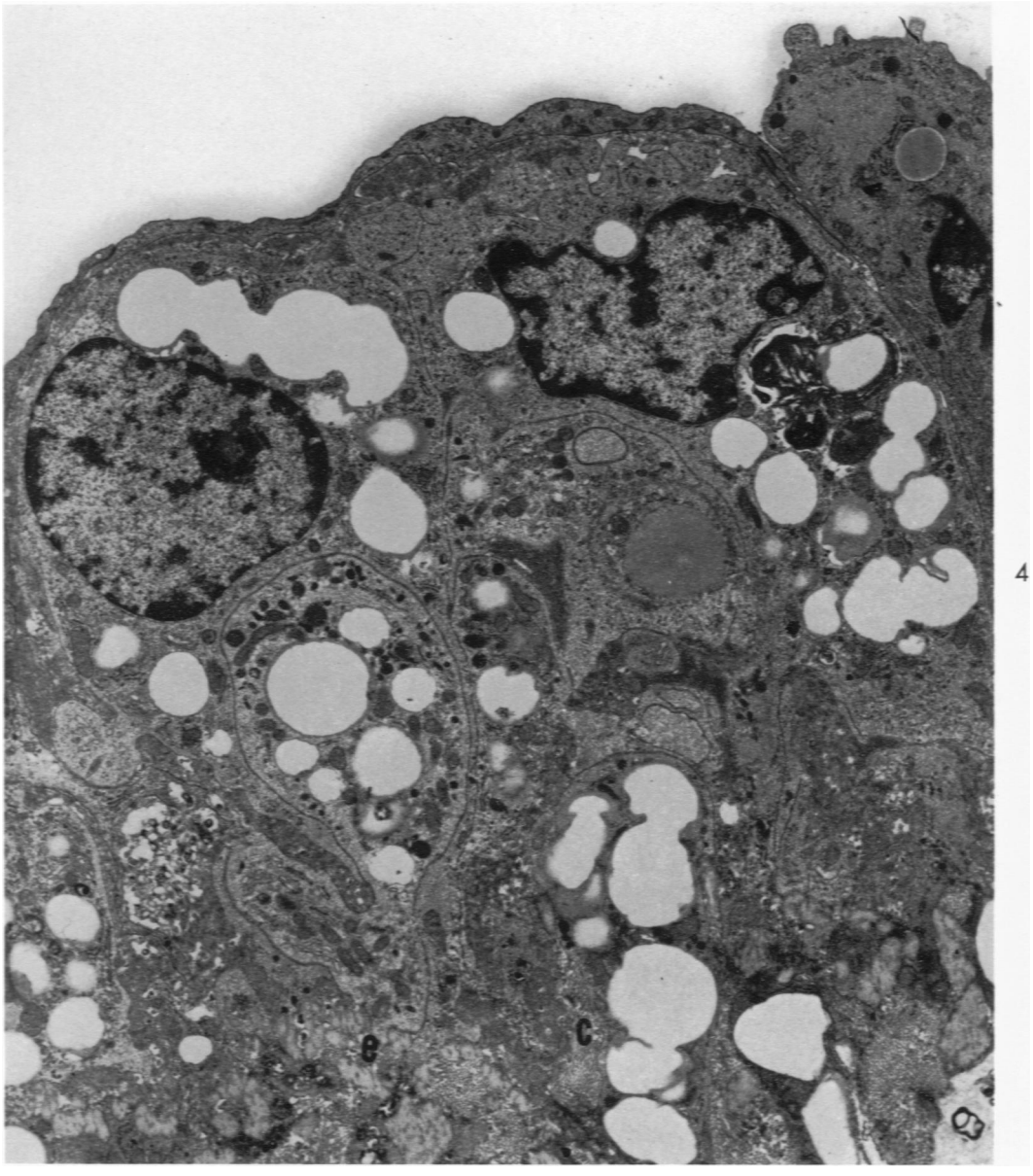

Fig. 4. Ascending thoracic aorta (higher magnification of Fig. 3). Note the thin, distended endothelium overlying the densely packed lipid-laden cells. Elastin (*e*) and collagen fibers (*c*) are seen in the small extracellular space. Uranyl acetate and lead citrate. × 9600.

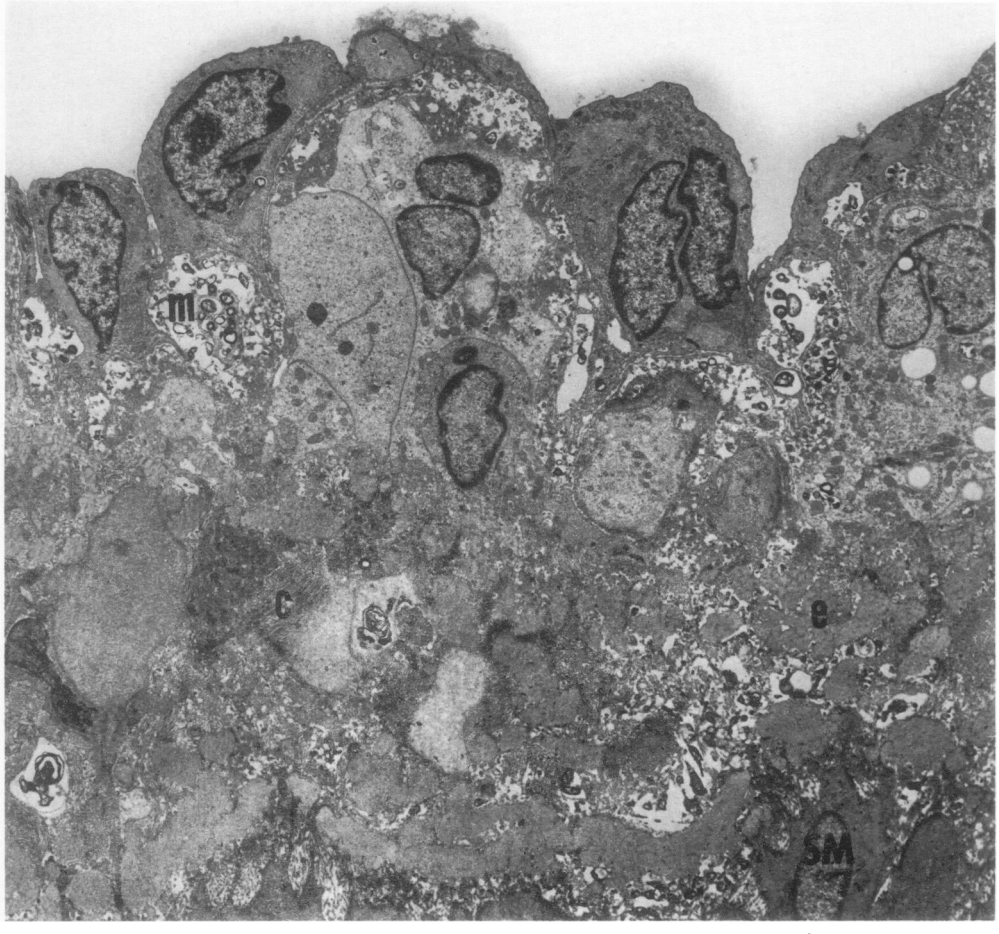

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Fig. 5. Ascending thoracic aorta. An intimal lesion is seen composed of cells similar to lipid-laden cells in Fig. 3 and 4. Very few lipid vacuoles are noted. Extracellular space is more prominent and contains numerous myelin forms (*m*), large amounts of elastin (*e*), and collagen (*c*). A smooth-muscle cell (*SM*) is seen breaching the internal elastic lamina at the lower right. Uranyl acetate and lead citrate. × 4400.

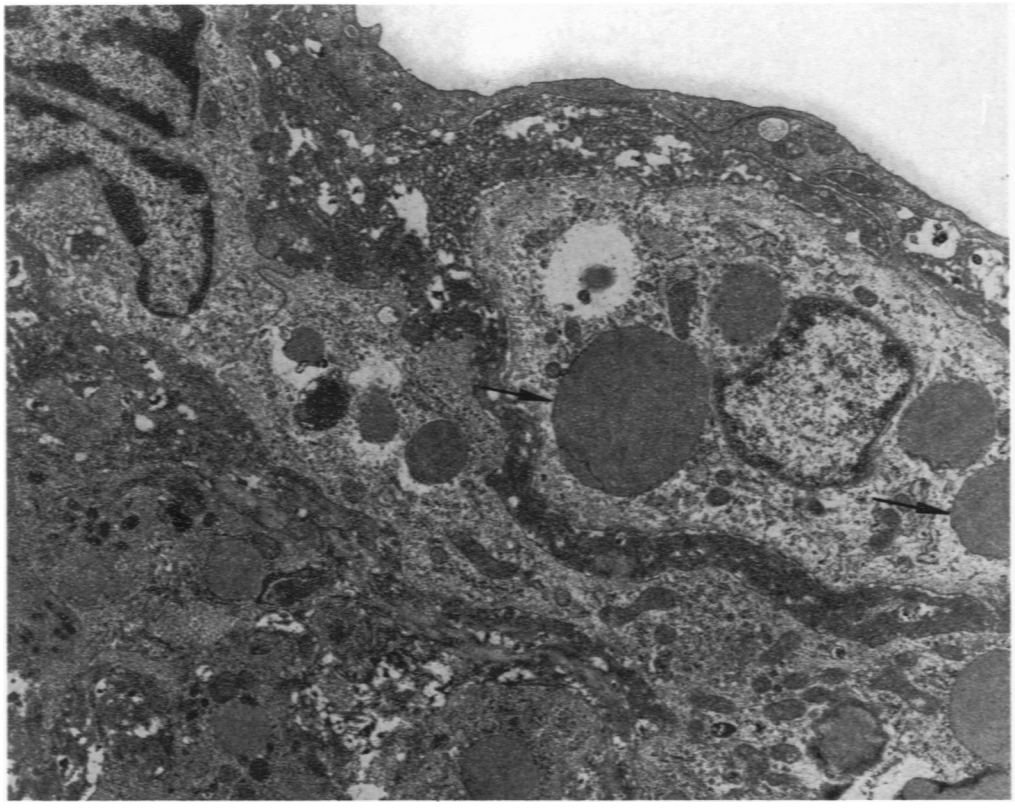

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Fig. 6. Descending thoracic aorta. A less advanced lesion bulges the endothelium only slightly and is composed of lipid-laden cells containing osmiophilic droplets that appear to have outer limiting membranes (*arrows*). This is probably condensation of the phospholipid moiety at their periphery. No lipid droplets are seen in the extracellular space. Uranyl acetate and lead citrate. × 11,600.

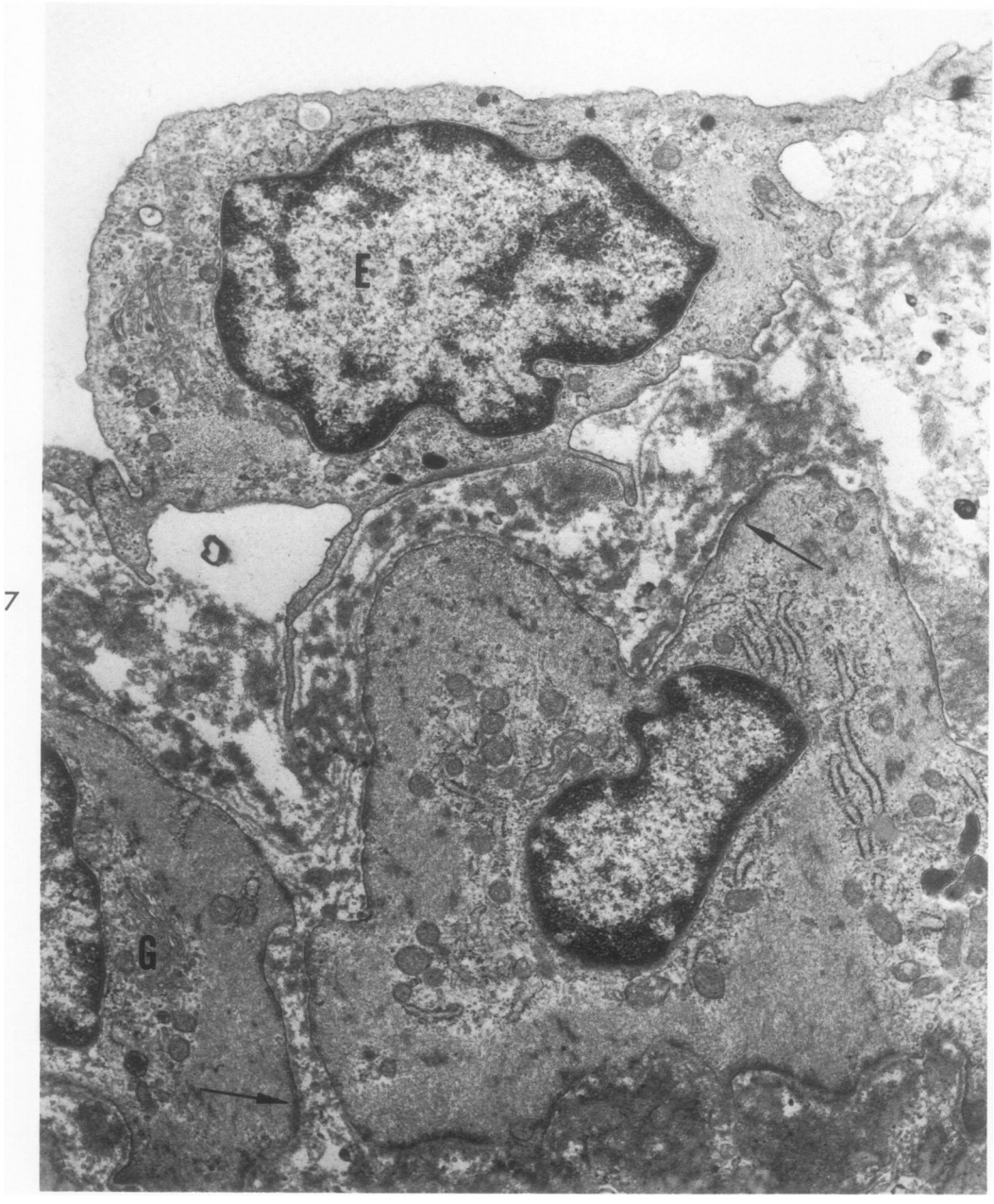

Fig. 7. Descending thoracic aorta. Beneath an endothelial cell (*E*) are two intimal smooth-muscle cells. Note the small nuclei, abundant rough endoplasmic reticulum, prominent Golgi complex (*G*), numerous cytoplasmic myofilaments, micropinocytotic vesicles, electron-dense bodies (*arrows*) along the cytoplasmic membrane, and basement membrane. Abundant collagen is present in the extracellular space but there are no lipid droplets or myelin forms. Uranyl acetate and lead citrate. × 18,200.

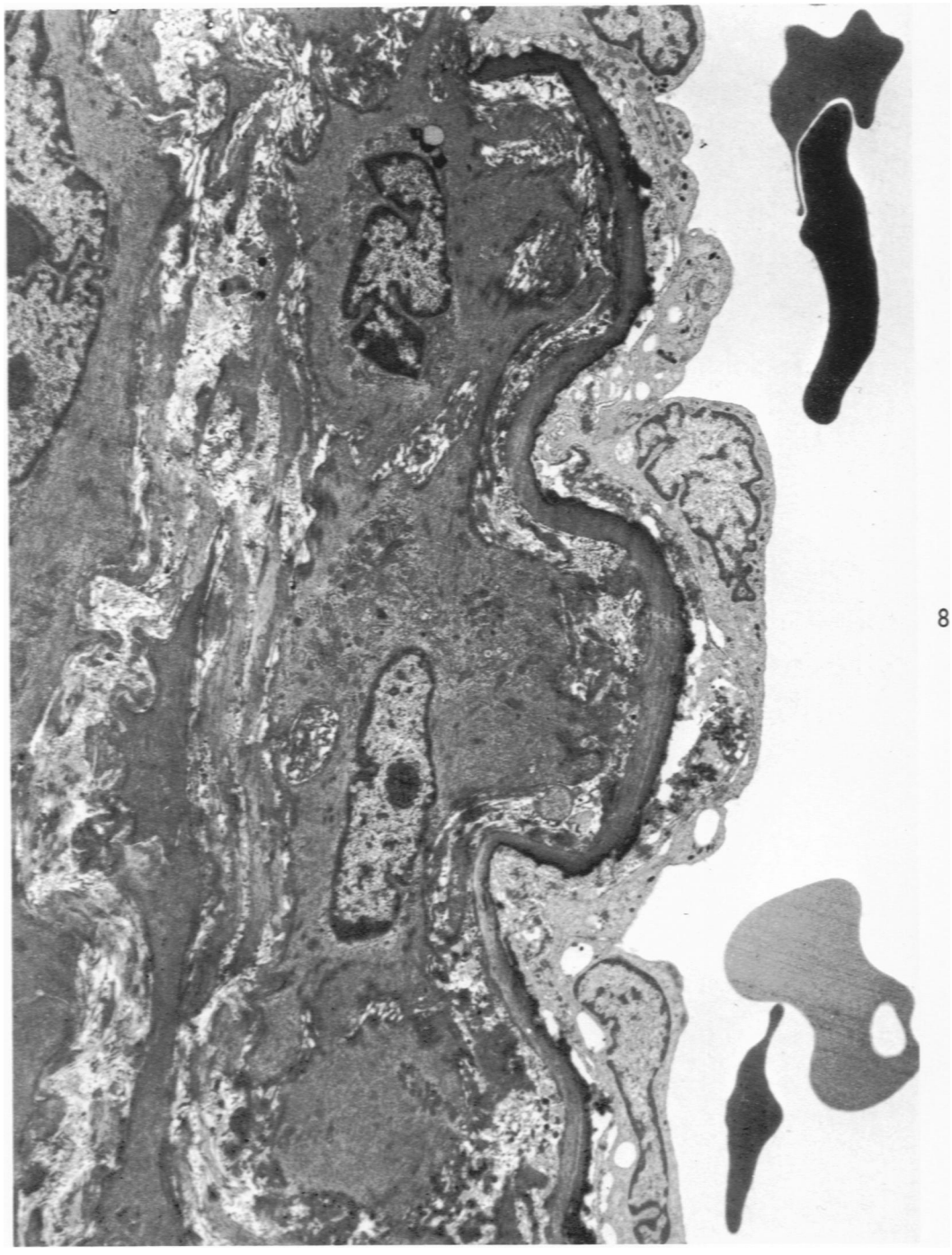

Fig. 8. Normal distal abdominal aorta. Endothelial cells are closely adherent to the prominent internal elastic lamina. Underlying smooth-muscle cells are uniform and oriented longitudinally. Phosphotungstic acid, uranyl acetate, and lead citrate. × 5800.

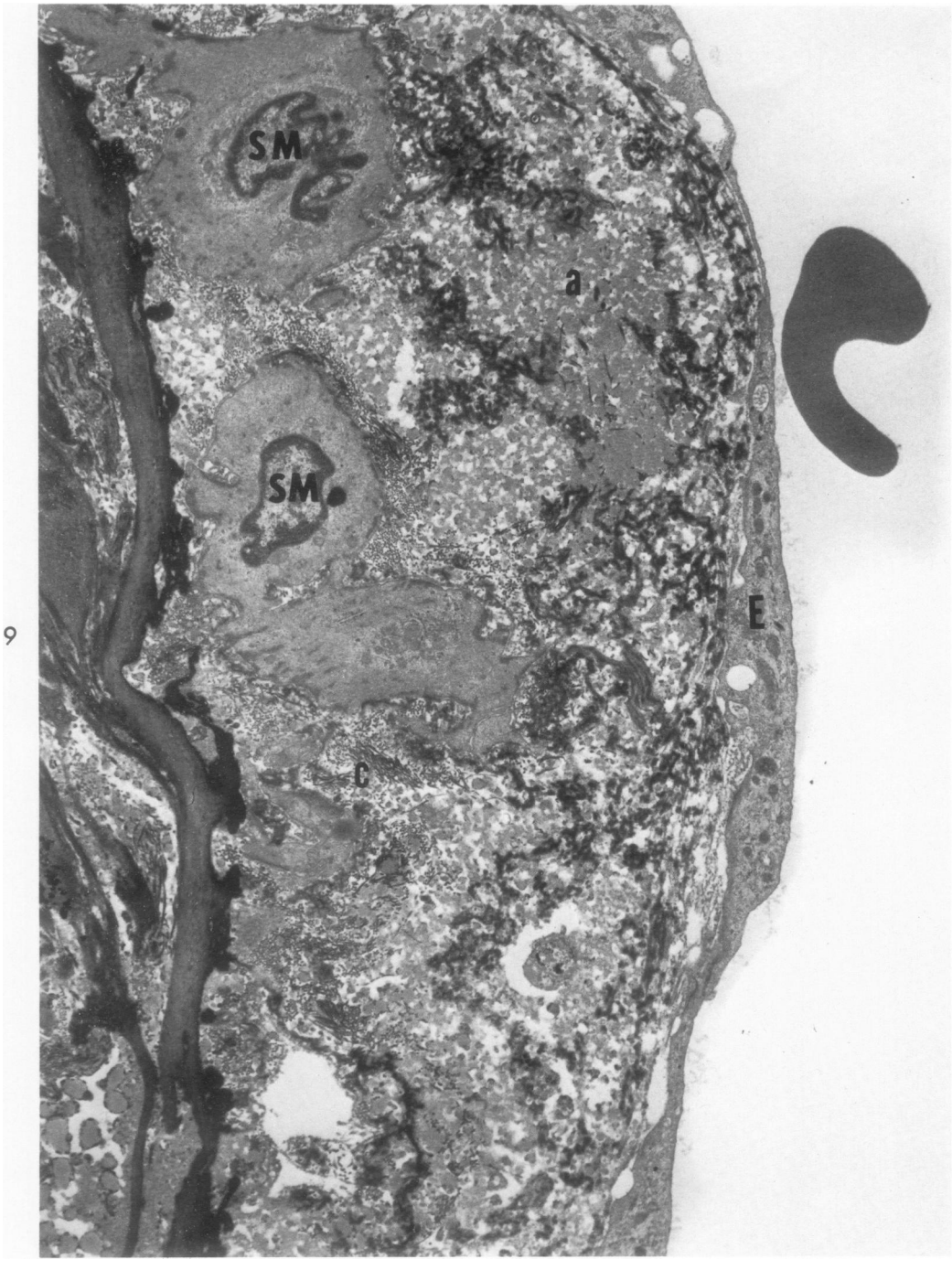

Fig. 9. Distal abdominal aorta. Endothelium (*E*) is thin and distended. Note the two intimal smooth-muscle cells (*SM*) that appear to extend at right angles (90°) to medial smooth-muscle cells (Fig. 8). A large extracellular space is seen containing numerous collagen fibers (*c*) and large amount of pale amorphous material (*a*). Phosphotungstic acid, uranyl acetate, and lead citrate. × 4620.

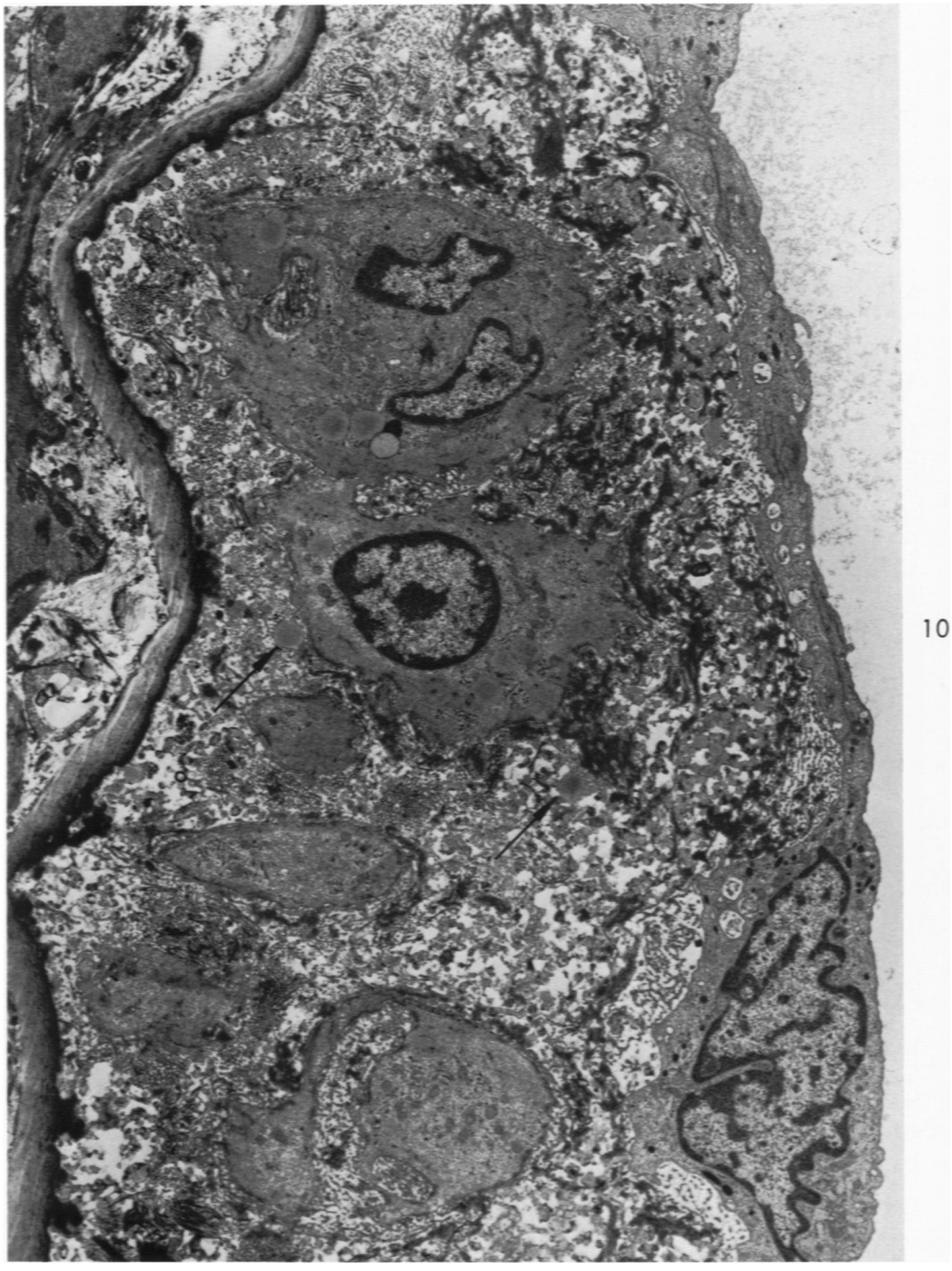

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Fig. 10. Distal abdominal aorta. Another intimal lesion is seen at slightly higher magnification than Fig. 9. Note the round, osmiophilic lipid droplets in smooth-muscle cells and similar droplets (*arrows*) in the adjacent extracellular space. Phosphotungstic acid, uranyl acetate, and lead citrate. × 6900.

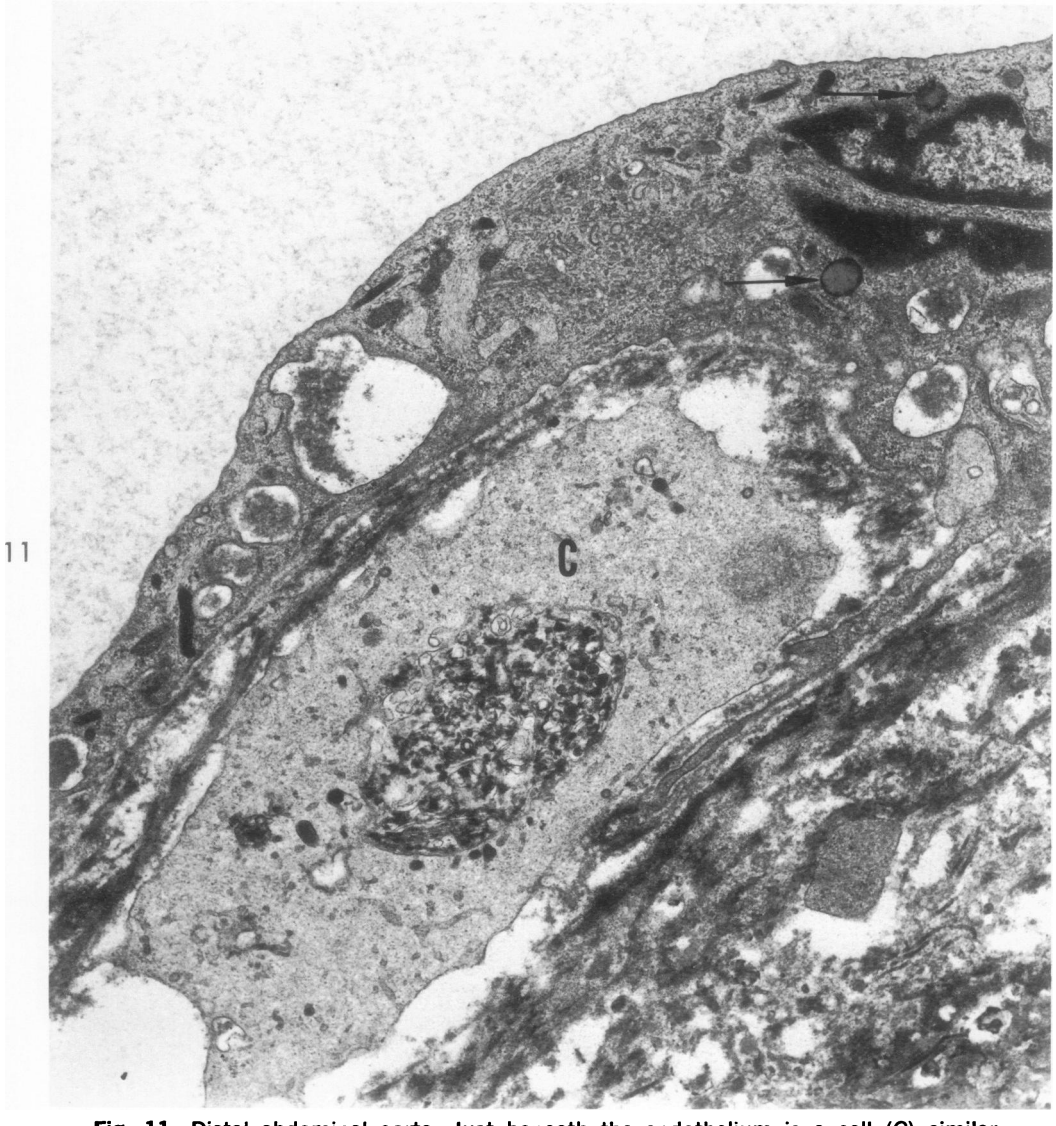

Fig. 11. Distal abdominal aorta. Just beneath the endothelium is a cell (*C*) similar to lipid-laden cells seen in thoracic atheromata. It contains numerous electron-dense bodies and structures that appear to be dilated smooth endoplasmic reticulum. Lipid droplets (*arrows*) are seen in the endothelium but not in the extracellular space. Uranyl acetate and lead citrate. × 11,600.