ARTERIAL OCCLUSIONS PRODUCED BY EMBOLI FROM ERODED AORTIC ATHEROMATOUS PLAQUES *

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In a recent autopsy of a man with advanced arteriosclerosis, changes were observed in some of the small and medium-sized arteries of the kidney, spleen, pancreas, and thyroid. In the lumina of these vessels were spaces with the shape of cholesterol crystals and a few slit-like endothelium-lined channels. Foreign body giant cells partly surrounded many of the cholesterol crystal spaces. These occluding lesions were found in arteries having an external diameter of from 55 to 900 μ. Their appearance suggested that emboli, containing large cholesterol crystals, had lodged in these vessels and undergone organization. Since there was advanced erosion of the atheromatous plaques in the aorta of this man, it was believed that these eroded plaques were the source of the emboli.

It is known that the contents of an atheromatous plaque may serve as emboli if the surface of the plaque undergoes erosion,† [1, 2] and occlusion of coronary arteries by similar emboli has been described.[3]

Since there have been no recent studies on this subject, this investigation was made.

MATERIAL AND METHODS

Two hundred and sixty-seven autopsies were selected for review. Of these, 233 had been diagnosed as having "advanced arteriosclerosis" in the aorta. All of these had many atheromatous plaques in the intima of the aorta, and in some instances the atheromatous areas had eroded and were partially covered with mural thrombi. Thirty-four other cases were reviewed because the description of the aortas suggested that the degree of atherosclerosis was actually "advanced" although in the anatomic diagnoses it was called "moderate."

Of the 267 subjects, 191 were males ranging in age from 33 to 92 years, and 76 were females of from 35 to 86 years. One hundred and forty males and 57 females were over 60 years of age. The average age of all patients was 64.9 years. Sections of the kidney were ex-

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† The term "erosion" is used in this paper to describe the process of breaking down of the intimal surfaces of atheromatous plaques. This is seen commonly in the aortas, and occasionally in other arteries as well, of people with advanced arteriosclerosis. Since the flowing stream of blood is the force which removes the material of the plaque, the term "erosion" is appropriate in both geologic and pathologic senses. It is unfortunate that the term "ulceration" is used in some texts to describe this process, as the lesions produced are not inflammatory in origin and have no etiologic or histologic resemblance to ulcers.

amined in 244 cases, of the pancreas in 256 cases, of the spleen in 251 cases, of the adrenal in 109 cases, of the thyroid in 16 cases, and of the prostate in 20 cases. Often more than one section of an organ was examined. In selected cases serial sections were cut and the vascular changes studied in detail.

Vascular occlusions were found in 9 of these autopsies, yielding an incidence of 3.4 per cent among the 267 patients with advanced arteriosclerosis of the aorta.

Most of the sections were stained with hematoxylin and eosin; certain of them were stained also with Weigert's stain for elastic fibers. Frozen sections were cut of formalin-fixed tissues from case 1, and stained for fat with Sudan III.

The dimension of an artery was obtained by measuring the external diameter of the media with a calibrated ocular micrometer. This was done in the sections of fixed, paraffin-imbedded material.

The term cholesterol crystal spaces is used to describe the slit-like spaces which remain in tissues at the sites of cholesterol crystals dissolved during the preparation of the sections.

Report of Cases

Case 1 is given in detail. In Table I are listed the age, cause of death, blood pressure, and the degree of arteriosclerosis found at autopsy in each of the 9 cases. The distribution and frequency of the arterial occlusions are summarized in Table II.

Case 1

A white male, 61 years old, had a blood pressure of 180/100. He died with symptoms of coronary thrombosis.

At autopsy (autopsy no. 11016; by Dr. C. M. Flory) the entire aorta was markedly arteriosclerotic. In even the ascending portion and arch of the aorta some atheromatous plaques were superficially eroded and covered with mural thrombi. In the remainder of the aorta the intima was a mass of confluent atheroma, most of which was eroded, and covered with thrombi. Calcification was scanty. The coronary arteries contained many large atheromatous plaques and in places their lumina were stenosed. The heart weighed 710 gm.; its left ventricle was greatly enlarged. Healed infarcts were present in the myocardium. The iliac, splenic, celiac, right renal, and other medium-sized arteries were dilated and tortuous. The left kidney was a fluid-filled sac, and its ureter occluded by a scar. The right kidney weighed 155 gm.; its cortical surface was indented by deep wedge-shaped scars. In the area between the scars the cortex was of normal thickness. The small renal arteries were thick-walled. The spleen, pancreas, and thyroid gland appeared normal.

Histologic Observations

Great Vessels. In the intima of the aorta, 1 cm. above the aortic valve, were thick atheromatous plaques filled with cholesterol crystal spaces and lipid-filled macrophages. In the lower portions of the aorta the intima was 2.5 mm. thick and filled with structureless, gruel-like material containing many cholesterol crystal spaces. In some areas the intimal lining was eroded, and mural thrombi containing crystal-shaped spaces were attached to the atheromatous material (Fig. 1). About some of the cholesterol crystal spaces were foreign body giant cells.

Right Kidney. The cortex of the right kidney was slightly thinner than normal, and beneath the wedge-shaped depressed areas the glomeruli and tubules were atrophic or hyalinized. The large and small renal arteries were thick-walled. Their intimas were greatly thickened, and there was much reduplication of the internal elastic lamina. The arterioles were hyalinized.

TABLE I

Clinical and Pathologic Data on Nine Cases with Emboli from Atheromatous Plaques

Case number*	Autopsy number	Age	Cause of death	Blood pressure	Arterio-sclerosis of aorta	Erosion of atherosclerotic plaques in aorta			Mural thrombi in aorta
						Thoracic aorta	Abdominal aorta	Site not stated	
1	11016	61	Coronary thrombosis	180/100	+++	++	+++		+++
2	8526	70	Cellulitis of neck	184/94	+++	0	++		0
3	9146	58	Coronary thrombosis	?	+++			+++	++
4	9855	69	Pyelo-nephritis	240/120	+++			++	++
5	10204	48	Carcinoma of stomach	140/80	+++	0	+++		++
6	10453	72	Coronary thrombosis	170/120	+++			+++	?
7	10633	67	Broncho-pneumonia	175/100	+++	+++	+++		0
8	10644	69	Peritonitis after lapa-rotomy	130/70	+++			+++	+
9	11226	50	Coronary thrombosis	160/108	+++	+++	+++		++

* All patients were males.
0 = No lesion.
\+ = Slight changes present.
++ = Moderate changes present.
+++ = Advanced changes present.
? = Information not given.

Cholesterol crystal spaces, partly surrounded by giant cells, and a few slit-like vascular spaces were seen in the lumina of the arteries in each of the 5 blocks of tissue examined. In 174 arteries over 50 μ in external diameter, 20, or 11 per cent of the total, contained these lesions. The arteries involved varied in diameter from 58 to 880 μ.

In a typical lesion the intima of the artery was hyperplastic and the entire lumen was filled with loose connective tissue surrounding choles-

TABLE II

Distribution and Frequency of Arterial Occlusions

Case number	Heart	Lung	Liver	Spleen	Pancreas	Kidney	Adrenal	Thyroid
1	o	o	o	++	+	++	o	+
2	o	o	o	+	+	+	—	o
3	o	o	o	+	+	+	—	—
4	o	o	o	o	+	o	o	—
5	o	o	o	o	o	+	—	—
6	o	o	o	+	+	o	—	o
7	o	o	o	o	+	o	—	—
8	o	o	o	+	o	o	—	—
9	o	ȯ	o	+	+	o	—	o
Totals	o	o	o	6	7	4	o	1

0 = Organ contained no occluded arteries.
+ = Organ contained one or few occluded arteries.
++ = Over 10% of arteries in the organ were occluded.
— = Organ not examined histologically.

terol crystal spaces and a few thin vascular channels. The crystal-shaped spaces were frequently bordered by foreign body giant cells.

A large lesion is shown in Figure 2. This was in an artery measuring 880 μ in diameter. In its lumen were four large cholesterol crystal spaces, about two of which were foreign body giant cells. Several small vascular channels ran through the hyperplastic intima which surrounded the crystal spaces. Many hemosiderin-filled macrophages were present, suggesting either that this was an organizing embolus or that there had been hemorrhage into this lesion. On following this lesion in serial sections, the pattern shifted rapidly, but the component parts—vascular channels, cholesterol crystal spaces, and giant cells—remained. This occluded vessel lay in the base of a large wedge-shaped area of cortical atrophy.

Another occluded vessel is shown in Figure 3. This measured 825 μ in diameter and was filled with small vascular channels and by large

cholesterol crystal spaces partly surrounded by giant cells. A few of these crystal-shaped spaces contained a homogeneous pink-staining, protein-like material, apparently the matrix of the crystals.

An earlier lesion is shown in Figure 4. A large branch of an artery measuring 920 μ across was plugged by cholesterol crystal spaces. These were partly surrounded by giant cells and loosely attached to the wall by hyperplastic intima. The mass projected into the lumen of the smaller vessel from the larger one.

Another type of lesion, consisting largely of vascular channels, is shown in Figure 5, A to E. This occlusion began at the bifurcation of an artery with diameter of about 600 μ. The first portion of the lesion consisted of a V-shaped group of vascular channels surrounding a few cholesterol slits (Figure 5-A). The bifurcation of this vessel is seen in Figure 5-B and its two channels in 5-C. In Figure 5-D the branches are separated and the lower branch is filled with cholesterol crystal spaces. These vessels lay in the base of a wedge-shaped area of cortical atrophy (Fig. 5-E). Many small branches of these arteries were filled similarly with crystal-shaped spaces. No hemosiderin was seen.

Many smaller vessels in the kidneys were also involved. These arteries varied from about 60 to 200 μ in diameter. In some the occlusions were similar to those in larger arteries. In others (see Figs. 8 and 9 of small splenic arteries) the cholesterol crystal spaces lay in the innermost portion of the thickened intima, and the vascular channel passed to one side. In some sections macrophages with vacuolated cytoplasm lay near the crystal spaces, which were partly surrounded by giant cells.

Frozen sections were cut from formalin-fixed blocks of the kidney tissue, stained for fat with Sudan III and counterstained with hematoxylin. Most of the cholesterol crystals did not remain *in situ* even in the frozen sections. In a few vessels, however, thin, rectangular crystals were seen. Most of the lipid which stained with Sudan III was in the outer portions of the intimas of the occluded vessels. The cholesterol crystal spaces were always found in the former lumina of the vessels, and often were surrounded by lipid-free tissue. In other vessels some lipid was present about the cholesterol crystal spaces, but the amount was never so great as in the outer layers of the intima.

Left Kidney. The glomeruli and tubules of the left kidney were atrophic and fibrosed, and the cortex and medulla were very thin. The arteries had thick walls but contained no lesions.

Spleen. The splenic pulp and malpighian bodies were normal. In the small arteries the intima was hyperplastic, and reduplication of the internal elastic lamina was prominent. The arterioles were hyalinized.

Two blocks of the spleen were available for study. In single sections

of both blocks were 63 arteries with a diameter of 50 μ or over, of which 12, or 19 per cent, had these lesions in their lumina. The occluded vessels varied from 58 to 540 μ in diameter. Three occluded splenic arteries (the largest measuring 540 μ) are shown in Figure 6. In branches of these arteries also the lumina were filled with cholesterol crystal spaces.

Figure 7 is a small branch of a larger occluded artery; the foreign body giant cells about the crystal spaces are clearly shown. Lesions of different histologic appearance than that seen in Figure 7 are shown in Figures 8 and 9. The vessel illustrated in Figure 8 measured 84 μ in diameter. Its muscularis and adventitia were vacuolated and contained scattered lymphocytes. Cholesterol crystal spaces, partly surrounded by giant cells, lay against the intimal surface of one side of the artery. In the second vessel (Fig. 9) the crystal-shaped spaces were attached to the surface of the intima and were surrounded by giant cells. A large vascular channel remained.

Pancreas. Histologic examination of the pancreas showed the parenchymal cells and islets to be normal. Fibrous tissue was excessive between the lobules of the gland. Intimal hyperplasia was marked in the medium-sized and small arteries. The arterioles were hyalinized.

Four arteries, varying in size from 77 to 270 μ in diameter, contained these lesions. The lumen of the largest was filled with intimal tissue in which were several slit-like vascular channels and two cholesterol crystal spaces surrounded by giant cells. The lesions in the other arteries were similar. One segment of a small artery contained a typical lesion; the next segment of the vessel was almost obliterated by intimal hyperplasia and by an accumulation of macrophages with lipid-filled cytoplasm.

Thyroid. The small arteries of the thyroid gland were thick-walled and their intimas hyperplastic. In the lumina of two small arteries were cholesterol crystal spaces and giant cells.

Other Organs. No occluding vascular lesions were found in the heart, lungs, liver, or adrenals.

Relation of Arterial Occlusions to Arteriosclerosis and Other Diseases

The 267 cases can be divided into three groups. In 63 cases erosion of atherosclerotic plaques in the aorta was not noted. No arterial occlusions containing cholesterol crystals were found in these cases. The second group consisted of 147 cases in which the erosion was of slight or moderate degree. Only two instances of these arterial occlusions (cases 2 and 4) were found in the group, an incidence of 1.3 per cent. In the third group the erosion of the plaques in the aorta was marked. Such advanced erosion was found in 57 cases, of which 7, or 12.3 per

cent, had arterial occlusions containing cholesterol crystals. The absence of this lesion in patients without advanced arteriosclerosis of the aorta has been substantiated by studying our routine autopsies. No arterial occlusions containing cholesterol crystals have been found in over 200 of these patients.

The occlusions have not been found among the very old, but in patients from 48 to 72 years of age, with an average age of 62.5 years. All were males. Of the 9 patients with this lesion, 7 had hypertension, 8 had narrowing or occlusion of coronary arteries, and 5 had myocardial infarcts. No patient, however, had diabetes, and only 2 had positive serological tests for syphilis.

Origin and Development of the Arterial Occlusions

The only early lesion found was a thrombus containing large cholesterol crystal spaces. This was attached to the wall of a medium-sized artery in the kidney of case 5 (Fig. 12). It seemed almost certain that these crystals were dislodged from eroded atheromatous plaques in the aorta and carried as emboli into this vessel.

The mass of cholesterol crystal slits projecting into the lumen of a medium-sized vessel of case 1 (Fig. 4) was an older, better organized lesion. There was a moderate degree of intimal proliferation about these crystal-shaped spaces. No remnant of the thrombus remained. Although many other medium-sized arteries were occluded in case 1, no crystal-shaped spaces were found within recent thrombi. The presence of many hemosiderin-filled macrophages in one occluded vessel (Fig. 2) suggested that a thrombus might have been present and organized.

Fully developed, well organized lesions were found in the medium-sized arteries in cases 1, 2, 3, and 9. The lumina of these vessels were filled with cholesterol crystal spaces surrounded by a few foreign body giant cells and intimal tissue. This type of occlusion is shown in Figures 2, 3, 5, and 6.

In every case except case 5 some small arteries measuring from 50 to 200 μ in diameter (Figs. 7 to 11) were partially or completely occluded by these lesions. No arterioles were affected. The histologic appearance of these occlusions in small vessels was more variable than that in the medium-sized vessels. In some of the small arteries the lumina were plugged by masses of cholesterol crystal spaces partly surrounded by giant cells. Figures 7 and 10 illustrate such lesions. Many of the small occluded arteries in case 1 and other cases were actually branches of larger obstructed vessels.

Emboli of cholesterol crystals from eroded aortic atheromata seemed to explain the origin of all of these lesions satisfactorily. In some small arteries, however, the cholesterol crystals lay against the hyperplastic

intimas of vessels in which the vascular channel was not occluded (Figs. 8, 9, and 11). It seemed possible that such cholesterol crystals might have been formed from the lipids in the thickened intima of the vessels. They probably, however, were examples of advanced recanalization of a vessel previously occluded by cholesterol crystal emboli.

It is believed that the arterial occlusions containing cholesterol crystal spaces are the result of organization of emboli from eroded aortic atheromata. The mass of cholesterol crystals, mixed with lipid and thrombus material, is torn loose by the flow of blood and is carried into a medium-sized or small artery, where it lodges. About this embolus a thrombus forms and organizes. The blood clot is removed, but the cholesterol crystals remain and are encased by intimal tissue and foreign body giant cells. Recanalization of the thrombus takes place between or beside the crystals, forming slit-like vascular spaces. In a completely organized lesion the artery is occluded by cholesterol crystals, often surrounded by foreign body giant cells, slit-like vascular spaces, and hyperplastic intimal tissue.

The only anatomic changes associated with these arterial occlusions were found in the kidney where the renal parenchyma supplied by these obstructed vessels was atrophic, forming depressed, wedge-shaped cortical areas (Fig. 5-E). No changes attributable to these vascular lesions were observed in the pancreas, spleen, or thyroid.

Experimental Production of the Lesions

An unfixed human aorta was selected in which atherosclerosis was marked. Soft yellow material was scraped from several of the plaques and suspended in 5 cc. of physiologic saline solution. Microscopic examination of this fluid revealed many large, clear, thin rhomboidal crystals having the characteristic shape of cholesterol crystals. Fat droplets and red blood cells also were present.

Two and one-half cc. of this material was injected into the ear veins of 2 rabbits. One animal was killed after 24 hours. In its lungs many small arteries were occluded by masses of red blood cells, polymorphonuclear leukocytes, and large cholesterol crystals. The other animal was killed after 7 days. Many small pulmonary arteries were also occluded. In these vessels the cholesterol crystals were no longer surrounded by leukocytes but by foreign body giant cells and hyperplastic intimal tissue (Figure 13).

Discussion

The embolic theory of the origin of these arterial occlusions has been discussed. Another explanation is that the crystals formed *in situ* in the hyperplastic, lipid-rich intima of the arteriosclerotic vessels and

that the entire process is an unusual form of arteriosclerosis. If the interpretation of the histologic appearance of these lesions presented previously is correct, the arteriosclerotic hypothesis seems untenable.

Additional evidence against the theory of the formation of the crystals *in situ* in the arteries is the fact that in 63 patients with advanced arteriosclerosis of the aorta but with no erosion of aortic atheromatous plaques, crystal-containing arterial lesions were not found despite the fact that the splenic, pancreatic, and renal arteries were often very thick-walled and contained much lipid in their hyperplastic intimal tissues. In the 147 patients with slight or moderate erosion, the lesion was found in only 1.3 per cent. However, of the 57 cases with advanced erosion of atheromatous plaques in the aorta, 7, or 12.3 per cent, had these lesions. This suggests that embolism rather than arteriosclerosis is the cause. It may be argued, however, that the arteriosclerosis was actually more marked in the latter groups. This certainly was true in the aorta, but there was no histologic evidence that the arteriosclerosis in the smaller vessels was more severe in one group than in the others.

Another argument against the theory of formation of these crystals *in situ* is the location of the crystals in the arteries. If the lipid in the thick intimal tissue of arteriosclerotic arteries were to crystallize, one would expect to find vessels with crystals in their intimas as well as vessels in which the lumina were filled with crystals. Crystals have not been found in the intimas of medium-sized unoccluded arteries. In the similarly sized occluded vessels the crystals were always in the luminal portion of the vessel. This suggests that these large crystals entered the vessels as emboli.

In certain small arteries, however, crystals have been observed in the intimas of vessels in which large lumina were present. It would be dogmatic to say that these lesions were the result of partial recanalization of a vessel previously occluded by cholesterol crystal emboli, although this is a possible explanation. The thickened intimal tissues of these small arteries occasionally contained lipid-filled macrophages. In such vessels cholesterol might have crystallized from this lipid *in situ*.

The possible effects of these vascular occlusions should be emphasized. In case 1, where about 10 per cent of the renal arteries were occluded by these lesions, anatomic changes in the kidneys were produced. These consisted of many wedge-shaped areas of cortical atrophy. In the other cases where medium-sized renal arteries were occluded, similar wedge-shaped areas of cortical atrophy were also found distal to the occluded vessels. No infarcts related to these arterial occlusions have been observed.

It is possible, however, that cholesterol crystal emboli may produce

infarcts in the kidney or spleen, and that gangrene of a toe or some other portion of a lower extremity, occurring in an old person with advanced arteriosclerosis, may occasionally be caused by cholesterol crystal emboli.

Conclusions

Arterial occlusions produced by emboli from eroded aortic atheromatous plaques have been found in the small and medium-sized arteries of the spleen, pancreas, and kidney. In the nine cases in which such lesions were observed the frequency and the distribution of the lesions were variable. In one case the lesions were numerous, involving 19 per cent of the splenic and 11 per cent of the renal arteries; in other cases only a few vessels were involved.

In a typical lesion the lumen of the artery was filled with large cholesterol crystal spaces surrounded by hyperplastic intimal tissue and a few foreign body giant cells. In the kidney these occlusions caused wedge-shaped areas of cortical atrophy.

The intravenous injection of material containing cholesterol crystals, obtained from an atheromatous human aorta, has produced similar lesions in the arteries of the lungs of rabbits.

I wish to thank Mr. Julius Mesiar, who made the photographs which illustrate this paper, and Miss Helen Hirschbein for their technical assistance.

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DESCRIPTION OF PLATES

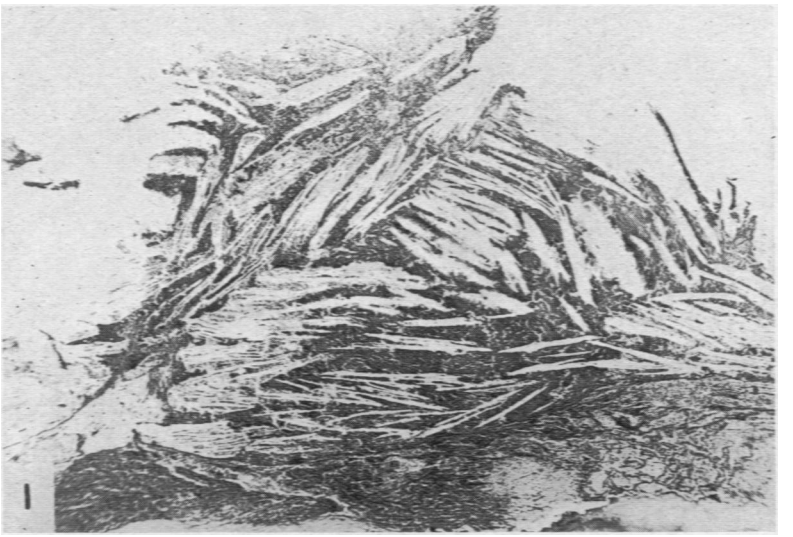
Plate 92

Fig. 1. Case 1. Surface of eroded atheromatous plaque in aorta. The mass of cholesterol crystal spaces is mixed with red blood cells; the surface of this lesion is very rough. × 50.

Figs. 2-A and 2-B. Case 1. Occlusion of a medium-sized renal artery. In *Figure 2-A* the entire artery is seen; this measured 880 μ in diameter. The large slit-like spaces in the former lumen of the vessel are cholesterol crystal spaces. × 50. In *Figure 2-B* giant cells can be seen at the ends of some of the crystal-shaped spaces. The dark masses of cells in the left side of the figure are hemosiderin-filled macrophages. × 160.

Fig. 3. Case 1. Occluded medium-sized renal artery. In this artery, which measured 825 μ in diameter, the large spaces are cholesterol crystal spaces, the small slits, vascular channels. × 50.

Fig. 4. Case 1. Intimal proliferation about a mass of cholesterol crystal slits in a medium-sized renal artery. In subsequent sections these slit-like spaces fill the lumen of a large branch of this vessel. × 50.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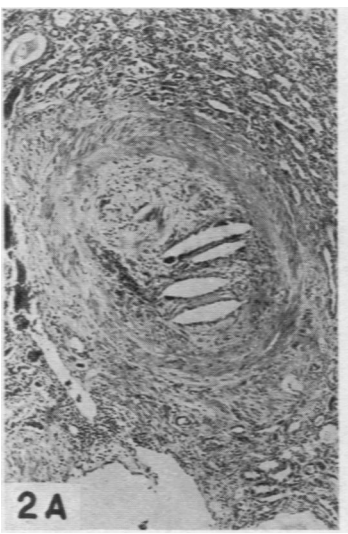
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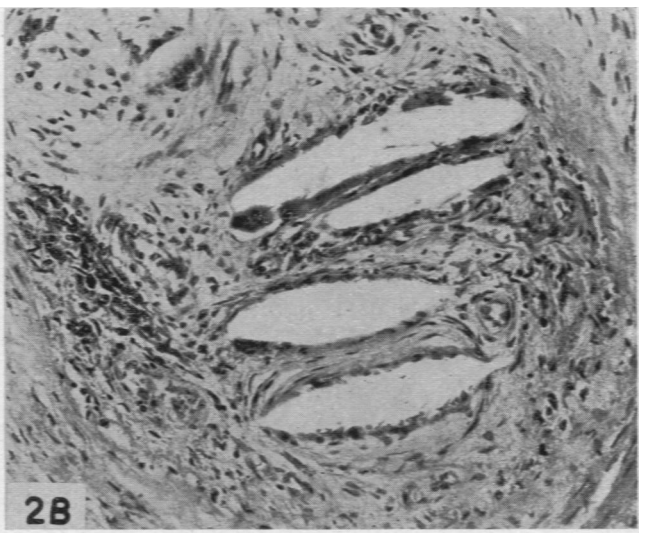
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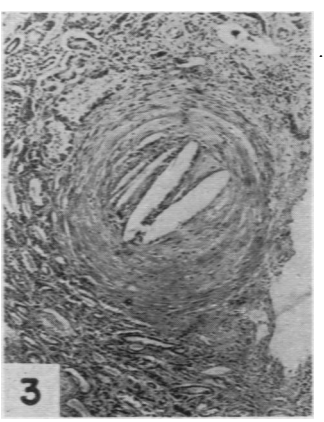
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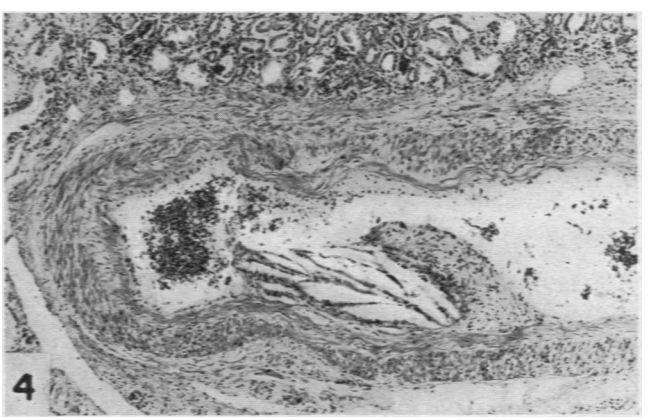
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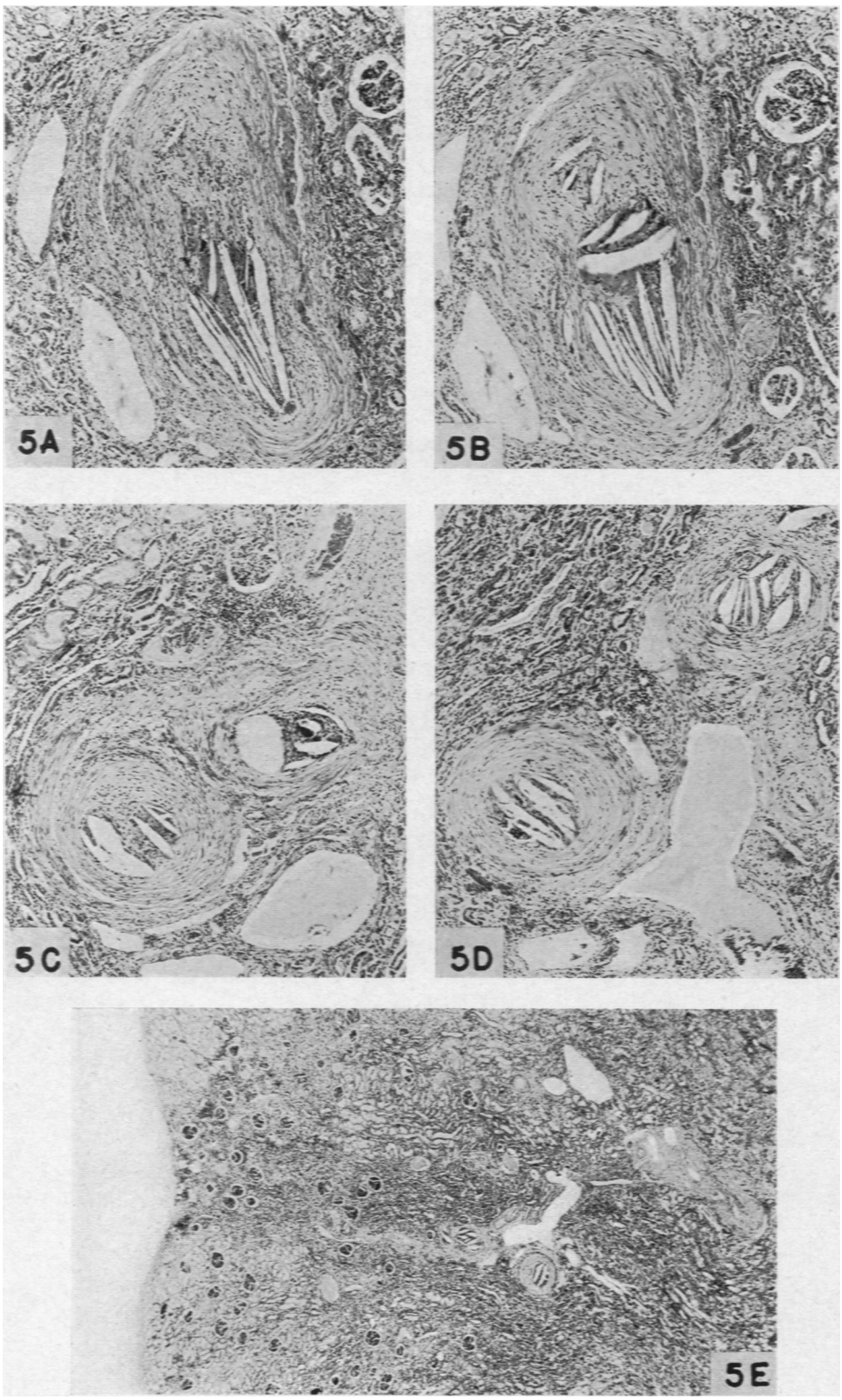
PLATE 93

FIG. 5. Case 1. Variability of the pattern of the occlusion in a medium-sized renal artery. In *Figure 5-A* the beginning of the bifurcation of the occluded vessel is shown. Many of the slit-like spaces are cholesterol crystal spaces, others are thin vascular spaces. *Figure 5-B* is the bifurcation of the vessel. In *Figure 5-C* the bifurcation is complete. The branches are partly filled with cholesterol crystal spaces. In *Figure 5-D* the vessels are farther apart. In the upper artery are many small cholesterol crystal spaces; in the lower vessel several large crystal spaces surrounded by giant cells. × 50. In *Figure 5-E* the area of cortical atrophy lying above the vessels shown in Figure 5-D is seen. × 20.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5C
5D
5E

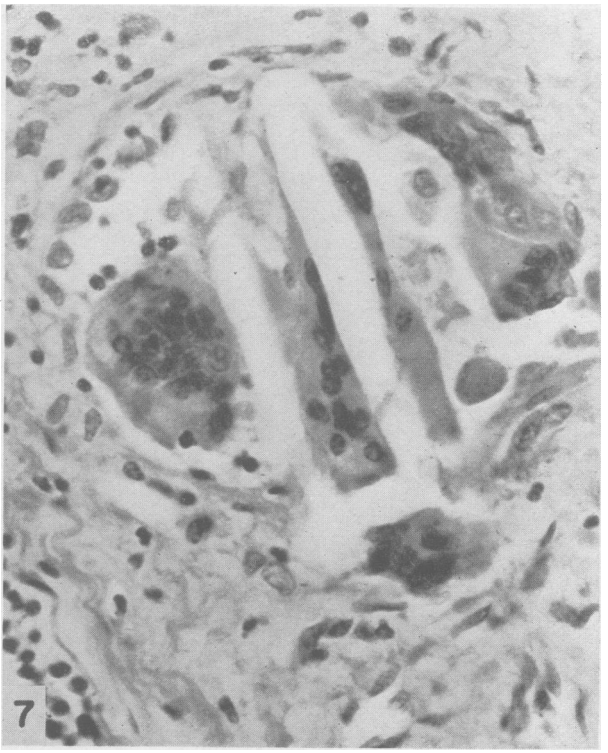
PLATE 94

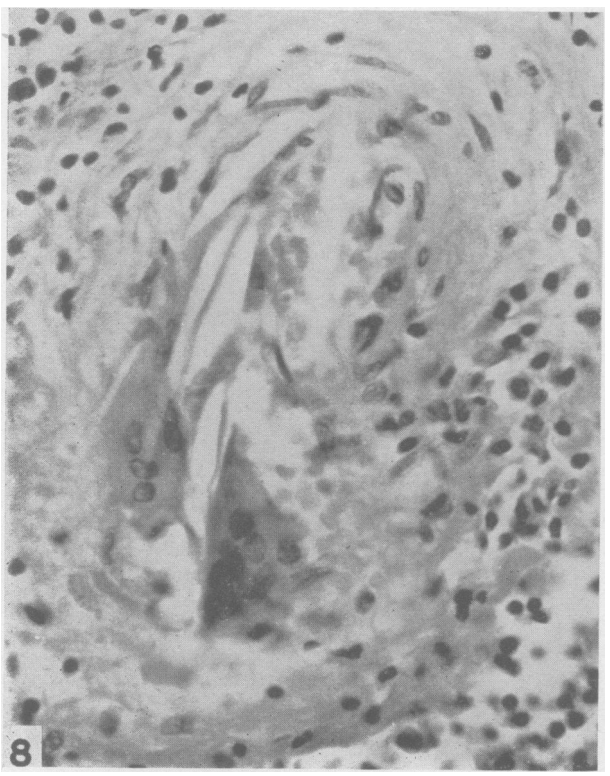
FIG. 6. Case 1. Three occluded splenic arteries. These arteries, the largest of which measured 540 μ in diameter, are probably all branches of a single larger vessel. They are filled with cholesterol crystal spaces. $\times$ 50.

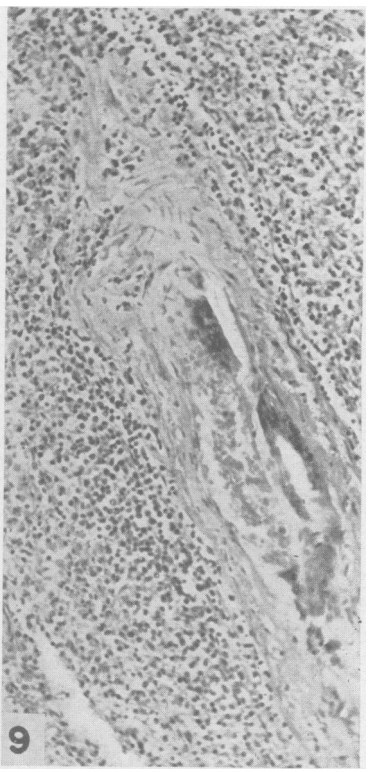
FIG. 7. Case 1. An occluded small splenic artery. This artery is a branch of one of the vessels shown in Figure 6. It measures 94 μ in diameter and is filled with cholesterol crystal spaces surrounded by large foreign body giant cells.

FIG. 8. Case 1. A partially occluded small splenic artery. In this artery, which measured 84 μ in diameter, the cholesterol crystal spaces lie against the intimal surface of one side of the artery and are partly encased by giant cells. Several endothelium-lined vascular channels remain. $\times$ 640.

FIG. 9. Case 1. Partially occluded small splenic artery. The crystal spaces are attached to the surface of the intima of the artery and are almost encased by giant cells. This vessel measures 96 μ in diameter. $\times$ 160.







Flory

Emboli from Atheromatous Plaques

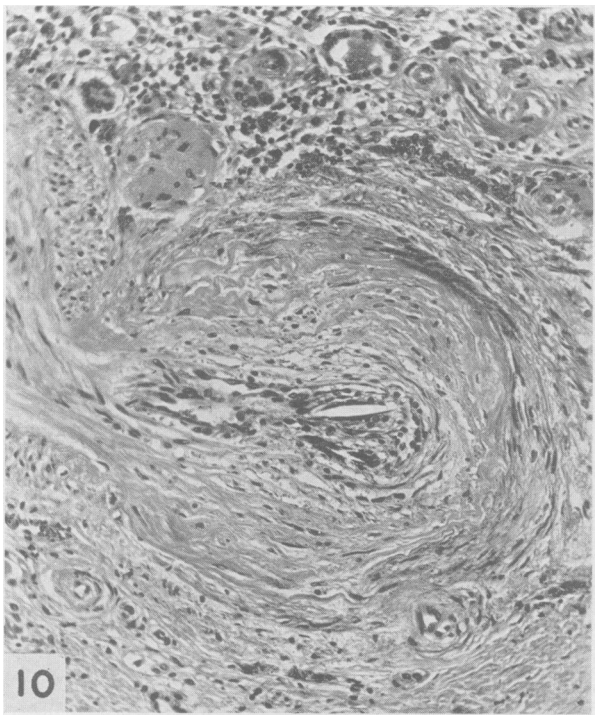
PLATE 95

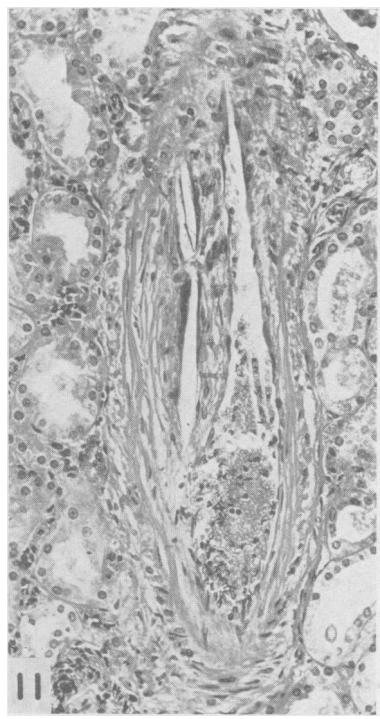
FIG. 10. Case 2. Occluded renal artery. In the center of this vessel are two small cholesterol crystal spaces partly encased by giant cells. Several small vascular channels are present in the intimal tissue. × 160.

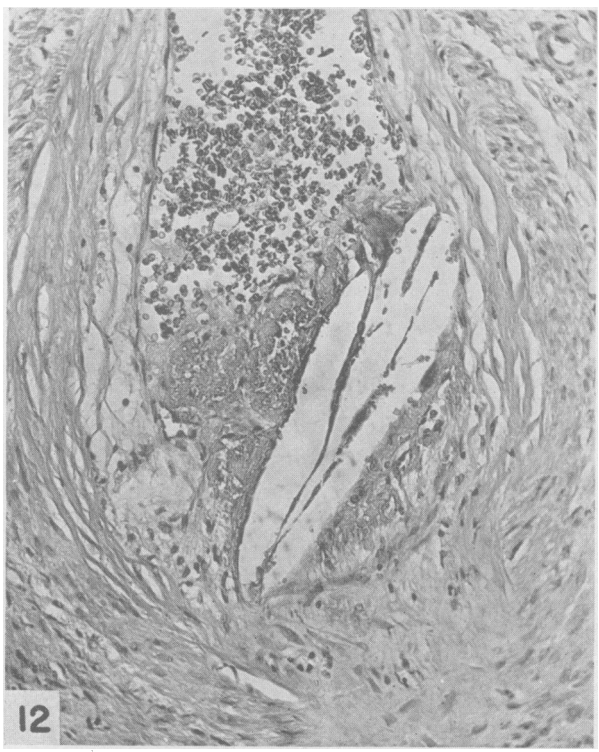
FIG. 11. Case 3. Partially occluded renal artery. Several large cholesterol crystal spaces are embedded in the hyperplastic intimal tissue of one wall of the vessel. A large lumen remains in the artery. This is probably an old lesion, and the vessel is almost completely recanalized. × 160.

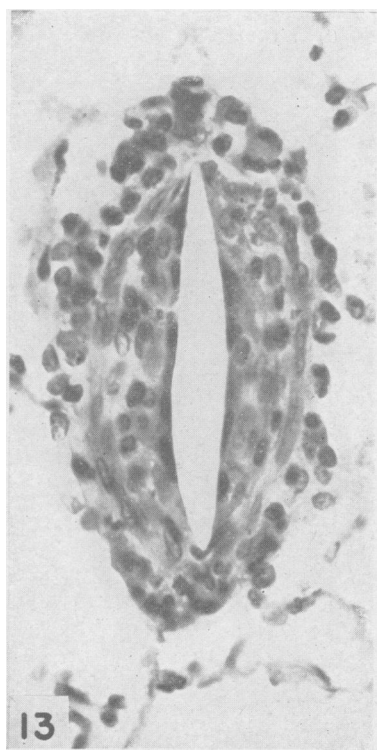
FIG. 12. Case 5. Recent thrombus containing cholesterol crystal spaces in a medium-sized renal artery. The lumen of this longitudinally cut artery is occluded or partially occluded by a recent, partially organized thrombus in which are several large cholesterol crystal spaces. × 50.

FIG. 13. Cholesterol crystal space in artery of lung of rabbit. One week before death, this animal was injected intravenously with a suspension of cholesterol crystals from an atheroma of a human aorta. The large crystal space is surrounded by giant cells and intimal tissue. × 640.









Flory

Emboli from Atheromatous Plaques