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PULMONARY ARTERIAL BANDS AND WEBS: AN UNRECOGNIZED MANIFESTATION OF ORGANIZED PULMONARY EMBOLI

DAVID KORN, M.D.*; IRA GORE, M.D.; ANNE BLENKE, M.D., AND DANIEL P. COLLINS, M.D.

From the Department of Pathology, Harvard Medical School; the James Homer Wright Pathology Laboratories, Massachusetts General Hospital, Boston, Mass.; the Laboratory Service of the Veterans Administration Hospital, West Roxbury, Mass., and the Department of Pathology, Peter Bent Brigham Hospital, Boston, Mass.

It is well recognized that in the great majority of cases pulmonary embolism is clinically undetected. Documentation of its occurrence, therefore, often depends entirely upon meticulous necropsy examination of the pulmonary arterial tree by a prosector who is aware of the various morphologic transformations which pulmonary emboli may undergo during organization. This fact is, of course, equally valid in experimental studies of this disease.

In recent years there have been a number of attempts to elucidate the sequence of pathologic events which follow experimental pulmonary embolization.[1-10] Particular attention has been directed to the mechanisms which remove or organize the emboli and to the consequent structural alterations in the pulmonary vessels themselves. From these studies it has become recognized that, in the various animals employed, the major part of the pulmonary embolic material may be completely lysed; and that the organization of persisting clot often results in the formation of small, eccentric, fibro-elastic intimal plaques which may comprise the sole anatomic evidence of previous embolization.

A less common end result of organization of pulmonary thromboemboli is the formation within arterial lumens of delicate fibrous bands

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and elaborate fibrous webs. These structures have only rarely been described, and opinion concerning their cause has varied, some writers favoring a congenital origin,[11,12] but most suggesting that they are organized emboli.[13-20] In our experience these lesions are not rare, and it is probable that many of them are unrecognized at necropsy because pathologists are unaware of their existence.

From the individuals recently coming to necropsy in 3 Boston hospitals we have collected a series of 51 patients * who exhibited these lesions; 28 of these were personally examined by the authors. One additional case was referred to us.† It is the purpose of this paper to call attention to these lesions and to describe their morphologic features and the manner in which they are formed. Our findings corroborate the thesis that these structures represent healed thromboemboli.

Correlated Clinical and Pathologic Data

The pertinent clinical and pathologic features in the 52 cases have been summarized in Tables I and II. The cause of death in 8 patients was acute pulmonary embolism and in 5 others, chronic cor pulmonale due to recurrent pulmonary emboli. Thus, 25 per cent died of pulmonary embolic disease, an incidence considerably higher than that found in our necropsy populations in general. In 5 other patients cor pulmonale was

Table I

CLINICAL AND PATHOLOGIC OBSERVATIONS

Sex and age:	Males: 26	Age range: 35—90	Av. age: 68
	Females: 26	Age range: 38—87	Av. age: 71
Major pathologic features at necropsy			No. of cases
Carcinoma			
Pancreas			6
Colon			3
Stomach			2
Breast			2
Bladder			1
Esophagus			1
			Total: 15
Coronary heart disease			
Acute myocardial infarction			10
			Total: 11
Acute pulmonary embolism			8
Cor pulmonale			5
Cerebrovascular accident			5
Miscellaneous			8

* Four of the cases (all from the Massachusetts General Hospital) were reported individually previously.[17,21-23]

† We are indebted to Dr. R. H. Fennell, Jr., Pathologist, The Presbyterian Hospital, Pittsburgh, Pa., for permission to include his case.

present but was not considered to be the cause of death. Some degree of right-sided heart disease secondary to pulmonary vascular obstruction was thus present in 19 per cent of the group. The major associated pathologic features in the remaining patients were without special significance, except, perhaps, that in 6 of 15 patients with carcinoma, the neoplasms arose in the pancreas. This form of neoplasm has been re-

TABLE II

CARDIO-PULMONARY FEATURES

Cardio-pulmonary features of cases	No. of cases
History of pulmonary embolism	
Absent	38
Present:	
Old only	10
Recent only	4
Source of pulmonary emboli	
Not known	15
Known:	
Leg veins	25
Iliac veins (all of these also had thrombi in leg veins)	3
Inferior vena cava (all of these also had thrombi in leg veins)	4
Right heart: (1 also had thrombi in leg veins)	
Auricle (atrial fibrillation)	2
Ventricle (old transseptal infarct)	3
Other evidence of pulmonary embolism	
Old only	2
Recent only (including 8 cases of fatal acute pulmonary embolism)	15
Recent and old (including 8 cases of fatal acute pulmonary embolism)	18
Pulmonary infarcts	
Old only:	
Single	4
Multiple	5
Recent only:	
Single	7
Multiple	3
Number of bands and webs in each case	
Multiple:	
Bilateral	34
Unilateral	3
Not known	3
One or two	12
Pulmonary atherosclerosis	
Severe (all with cor pulmonale)	3
Moderate (2 with cor pulmonale)	6
Slight (none with cor pulmonale)	4
Cor pulmonale (including 5 in which cor pulmonale was cause of death)	10

ported to be associated with a particularly high incidence of intravascular thrombosis.[24,25]

Only 6 patients in this series had had surgical ligation of leg veins, and in 2 the procedure was carried out on the day prior to death. One patient had had bilateral saphenous vein ligations in the past and had no recent emboli at necropsy. Despite bilateral femoral vein ligations, 2 of the remaining 3 patients exhibited pulmonary emboli which had originated from thrombi proximal to the ligatures.

The data presented are in essential agreement with generally accepted conclusions concerning pulmonary embolic disease.[26-31] They emphasize: (a) the tendency of pulmonary embolic episodes to be multiple; (b) the striking frequency with which these episodes are clinically unrecognized, even when pulmonary infarcts occur; (c) the relative infrequency with which such infarcts do indeed ensue; (d) the prevalence of leg vein thrombi as the sources of pulmonary emboli; and (e) the rarity with which these emboli originate from the right heart.

Gross Description

Intra-arterial cords, bands and webs were found most frequently within primary, secondary and tertiary branches of the pulmonary artery and rarely in the main pulmonary trunks. ("Primary" branch refers to the major lobar artery and "secondary" and "tertiary" to succeeding orders of arterial branches.) There was no apparent predilection for either lung or for particular pulmonary lobes; in most cases the lesions were bilateral.

The lesions appeared in a great variety of forms. Most common was a delicate threadlike or ribbon-like structure of fibrous consistence (Fig. 1) covered on all surfaces by endothelium and anchored to the arterial intima at its two ends, with a free and unattached midportion. These structures, which we designate as "cords" or "bands," generally ranged from 0.3 to 2 cm. in length and from less than 0.1 to 0.3 cm. in width, and they lay within the arterial lumen, oriented in the direction of blood flow along the long axis of the vessel. Commonly they bridged the orifices of subsidiary channels, and less often they terminated at bifurcations. At attachment sites, particularly the proximal ones, there were frequently small, gray-yellow intimal plaques or ridges. Some of the shorter bands were attached to the vessel wall at multiple points along their lengths and could be related morphologically to short, ridgelike intimal thickenings which were generally astride a bifurcation at an angle to the axis of blood flow (Fig. 3).

In some instances the bands were themselves branched and formed networks of variable complexity ("webs") (Fig. 3). The webs ranged

from thin, fenestrated membranes to relatively thick, eccentrically situated, flattened, branching structures, which were the nodal points of attachment for a multiplicity of radially oriented cords and bands. These traversed the lumen to become attached at some other portion of the vessel wall. Often some of the fibrous trabeculae bridged the ostiums of branch vessels, while other strands actually extended for some distance within them.

Although the majority of the bands and webs in these patients were old and fibrous, a number of fresher lesions and structures of intermediate age were encountered, and in a few cases multiple lesions representing all stages of evolution were manifest in the same arterial tree. The morphologic sequence clearly demonstrated the development of the mature lesions from the organization and involution of emboli.

The earliest lesion (Fig. 4) which could be morphologically related to a fibrous band was a red-black, soft, cordlike thrombus, oriented in the direction of blood flow, and invariably attached at its proximal end to a somewhat older, adherent, red-gray, nonocclusive embolus which lay eccentrically within the arterial lumen. The latter embolus was occasionally laminated, presenting a gray, partially organized basal portion covered by one or more layers of more recent blood clot. The band-precursor was generally continuous with the more superficial, fresh thrombotic material. The band-precursor itself usually appeared homogeneous in color and consistence (and probably in age) along much of its length, but often its distal-most portion was softer and blacker than the proximal portion, suggesting that it had been more recently formed. At its proximal attachment the band-precursor was firmly adherent to the eccentric mass of embolus, whereas its distal attachment was often tenuous. Occasionally along the midportion of the fresh bandlike thrombus, there were one or more additional points of adherence to the vessel wall (Fig. 5). These attachments also varied in appearance, some being formed by delicate red strands of fresh thrombus, others by firm, gray, fibrous-like structures.

Somewhat older precursors varied from red-brown to gray, had increased consistence and smooth gray surfaces, and were more firmly anchored to the vessel wall at their ends. In these instances, at the proximal point of attachment, there was usually recognizable an eccentric plaque of organizing thrombus in process of incorporation into the vessel wall. Commonly, even at this stage of development, there was a clear gradation in color and consistence from the proximal to the distal portions of the band, indicating that the distal segments were less highly organized and probably more recently formed.

Entirely similar sequential changes were observed in the formation of

webs. Within these complex structures there was occasionally a striking disparity in age between strands which were gray and fibrous and branches of them which were red and soft; still other components were in intermediate stages of organization.

Occasionally, fresh emboli were found attached to bands or entrapped within the meshwork of a more complex web (Figs. 6 and 7). Somewhat older thrombi appeared as adherent, gray-red to yellow-gray nodules lying within the interstices of a web, or as verrucous excrescences on the surface of a band. With continued organization and shrinkage, the emboli were transformed into flat, knoblike swellings in the body of the original band. Such knoblike areas, single or multiple, were present in several bands, and produced in them a somewhat nodular contour.

In the 5 patients with fatal cor pulmonale there were literally hundreds of bands and webs involving the secondary, tertiary and quaternary arteries in both lungs. The gross characteristics of the individual lesions in this group of cases did not differ from those described above. A striking gross finding in all of these patients, however, was the presence of moderate to severe atherosclerosis in the main pulmonary arteries and marked mural thickening of the larger muscular arteries which gave them the appearance of systemic arteries. In each of these cases, the arterial thickening stopped abruptly at the proximal points of attachment of the fibrous bands and webs. The arterial wall distal to these points (including the arterial segments actually traversed by the bands and webs) was exceedingly delicate and almost translucent, and presented only small foci of thickening at the distal points of insertion of the bands and webs.

In most cases in which diseased leg veins were examined, they showed mural thickening and lumen narrowing or occlusion due to recent and old thrombi. In a few instances, however, the veins contained bands or webs (Fig. 2) identical in appearance to those in the pulmonary arterial tree.

Microscopic Description

The most recently formed structures which could be recognized as bands were elongated thrombi composed of a meshwork of fibrin and platelets within which there were entrapped red and white cells (Fig. 8). Even in these very early structures there was clear evidence of the axial orientation of the fibrin-platelet skeleton in the direction of blood flow, indicating the origin of the band from a propagated thrombus. At the proximal and distal points of attachment of the band, there was ingrowth of avascular granulation tissue from the arterial intima with organization of the thrombus. The entire band was enclosed by an endothelial lining continuous with the vascular endothelium at both attachment sites.

Just beneath the endothelium there was a thin, occasionally incomplete, layer of granulation tissue, also continuous with similar tissue at the sites of attachment; from this, organization was proceeding centrally along much of the length of the band. The result of these processes was that the central core of the band was generally the last zone to be organized, and even in somewhat older lesions there could often be found centrally situated foci of residual clot (Fig. 9).

Adherent to the external surface of even recently formed bands there were frequently small fragments of fresh blood clot (Fig. 8). Similar fresh embolic material was also found commonly in association with old lesions (Fig. 16), and as this fresh material was organized it became incorporated into the substance of the band or web, often producing an eccentric bulge corresponding to the knobby thickenings observed grossly. The frequency with which these sequences were present indicated that these structures were continually undergoing alterations of size and form.

In some sections of early lesions there were histologic differences between the proximal and distal segments of individual bands (Figs. 8, 10 and 15). The distal portions were composed of more recently formed thrombus, either as yet unorganized or less highly organized than that in the proximal portions. These microscopic features corresponded to the gross observation that the distal segment of a band appeared to be composed of fresher thrombus than the proximal segment.

At the proximal site of attachment of the more recent bands, the arterial intima was thickened to form an eccentric plaque or ridge that lay primarily proximal to the origin of the band (Figs. 8 and 10). Within this zone of thickening there was usually evidence of organizing or organized embolus.

The older lesions exhibited diverse structural features. Upon completion of organization of the blood clot, the band was composed of cellular but avascular granulation tissue containing numerous macrophages laden with hemosiderin. With increasing age the fibrous tissue became less cellular and richer in collagen fibers which often ultimately underwent hyalinization. Even in such structures, however, few or many siderophages could often be demonstrated. Beginning in the early stages of organization, small amounts of smooth muscle and elastic fibers could be found within the bands. The proliferation of elastica and smooth muscle that occurs in the arterial intima at the site of an organizing mural thromboembolus is well known, and similar changes were present at the points of attachment of the bands. In some instances direct continuity of these intimal elastic and muscular elements with those in the band was apparent, but most often such a relationship could not be demon-

strated, suggesting that at least some of the elastic and muscle fibers in the bands developed *in situ*. In older lesions these fibers were often very abundant and closely packed (Figs. 11 and 12) and were usually oriented in parallel fashion to the endothelial surfaces of the band.

New vascular channels were formed within individual bands, but they were much more prominent in the webs (Figs. 13, 14 and 16). The recanalized lumens were lined by endothelium which rested upon a single elastic fiber, and they were often enclosed within a narrow sling of concentrically arranged smooth muscle fibers. Not uncommonly, the new vessels were themselves occluded by recent or organizing emboli.

In the complex, multipartite webs, the arrangement of collagen, elastic fibers, and smooth muscle produced a strikingly ordered histologic pattern (Figs. 13 and 14). In many of these structures, portions of different ages could be recognized by the content and the arrangement of the various fiber elements; in particular, eccentric knobs, representing various phases of organization of adherent thrombus could be clearly recognized. At times, histologic stratification of the lesions testified to their episodic growth in this manner (Fig. 14).

The walls of the arterial segments containing the bands and webs usually presented no abnormality (Figs. 9 to 11), except for the previously noted zones of intimal thickening at attachment sites. Occasionally, however, other parts of the wall did exhibit separate foci of intimal thickening, eccentric or concentric, which could not be distinguished from lesions representing old, organized emboli (Fig. 13); rarely, the arterial segment showed diffuse intimal thickening up to the distal attachment of the band (Fig. 15). Except in the 5 patients with fatal cor pulmonale, the pulmonary arterial tree proximal to the levels containing bands and webs was generally unremarkable. In these 5, a severe degree of pulmonary arteriosclerosis was evident both grossly and microscopically; the sections here also showed the striking tendency for arteriosclerotic changes to cease abruptly at the proximal attachment sites of the bands and webs (Fig. 16). The distal arterial wall remained relatively normal.

In those cases in which old pulmonary infarcts were identified, microscopic sections revealed the typical features of contracted, subpleural fibrous scars containing abundant fragmented elastic fibers in great disarray. In a very few instances, a pulmonary artery proximal to the infarct contained an old band or web, and the topographic relations were such that the lesions could possibly have been causally related. In several other cases, recent pulmonary infarcts were associated with recent emboli arrested in arteries at the sites of older bands or webs.

Discussion

The first recorded description of pulmonary arterial bands and webs was that of Zahn in 1889.[11] In a 52-year-old woman who died of massive pulmonary embolism, he found a fibrous web in the main artery to the left lower lobe and decided that it was of congenital origin, primarily because he had never previously encountered a similar lesion. A contrary opinion was offered by Møller in 1922 [13]; he described fibrous bands in 4 cases and postulated their origin from thromboemboli. He noted the tendency of the lesions to bridge the ostiums of branch vessels and their frequent content of stainable iron, and he observed structures of varying ages and stages of development. On the basis of his observations, Møller suggested that the lesions were formed by propagation of thrombi distal to the sites of lodgment of emboli.

The first notation of pulmonary bands and webs in English appeared in 1932 when Saphir[15] presented a detailed gross and microscopic description of multiple bands found in the pulmonary arteries of a 60-year-old man and proposed that they represented organized thrombi originating on foci of pulmonary atherosclerosis. Subsequently, Belt,[16] in 1939, described similar lesions in 3 patients, 2 of whom had severe cor pulmonale. He noted the tendency of the bands and webs to enlarge through the accretion of more recent thrombi, and he observed identical structures in the femoral veins in 2 of his cases. It was Belt's opinion that the pulmonary lesions clearly represented healed emboli; and he suggested that recurrent, clinically silent episodes of pulmonary embolism could lead to chronic, ultimately fatal cor pulmonale.

Brenner,[32] in his classic monograph on the pathology of the pulmonary vascular system, did not himself describe bands and webs but did refer to the earlier writings of Møller and Saphir. In more recent years, Castleman and Bland,[17] Bobek and Vaněk,[18] and Vaněk [19,20] have reported cases of fatal cor pulmonale associated with innumerable pulmonary arterial bands and webs interpreted as organized emboli. Irvin,[12] who reported a similar case in 1949, revived the thesis that the lesions were congenital.

Most of the studies concerned with experimental pulmonary embolism have stressed the remarkable fibrinolytic potential in the various animals employed and the tendency for nondissolved clot to become organized and reduced to small eccentric plaques of intimal fibro-elastic tissue. However, in several of these reports there are illustrations of fibrous bands and webs,[4,7,10] and in a few instances these structures have been specifically described.[4,10] Moreover, Dible,[33] in his detailed discussion of organization and canalization of arterial emboli, noted that the final

morphologic appearance of these lesions was very variable, depending on such factors as vessel size and local hemodynamics, and that often "tenuous bands . . . persist."

The abundance of pathologic material available to us in this study has enabled us to observe pulmonary arterial bands and webs of all ages and in a variety of morphologic stages of development and has indicated conclusively that in every instance these structures originated as thromboemboli. Although the possibility that some of the fibrous bands and webs originated from autochthonous thrombi rather than emboli cannot be rigorously disproved, we believe that an embolic origin constitutes a more reasonable explanation. In none of our material was there evidence suggesting primary pulmonary thrombosis as the initiating event in band formation. Our interpretation is further strengthened by the previously noted experimental data and by the frequency with which identical structures, clearly of embolic origin, are observed in other, nonpulmonary sites,[34] such as in femoral veins, pulmonary veins (in cases of cyanotic congenital heart disease without pulmonary hypertension[35,36]), coronary artery (in a case of thrombotic thrombocytopenic purpura[37]), and (in one recent case) both renal veins.* Conversely, there is no evidence at all to support the thesis that these lesions are congenital.

The precise mechanism of formation of these structures may not be identical in all instances. One mechanism, most commonly indicated in our material, and also suggested by the clinical[13,16,18] and experimental[4,37] observations of others, is the propagation of a thrombus distally from the site of lodgment of an embolus. The reasons for the apparent readiness with which this sequence occurs in the pulmonary arterial tree can only be speculated upon, but two factors may be of particular importance. First circulating and pulmonary fibrinolysins operate to reduce the quantity of embolus which must be organized; second, organization tends to lead to incorporation of the clot into the vessel wall as an eccentric plaque of fibro-elastic tissue. Because of these factors, pulmonary emboli usually do not persist as totally occlusive lesions which are organized circumferentially, converted into large fibrous plugs, and ultimately recanalized; and blood flow past the site of impaction of the embolus is rapidly restored. That pulmonary bands and webs do not in fact often result precisely from such a process of recanalization of an occlusive thromboembolus is indicated by the usual finding that the intima of the arterial segment containing these structures is relatively normal except at attachment points. This is not the usual finding in recanalized thrombi, in which the newly formed channels are centrally situated in a matrix

* This case will be published in detail at a later date.

of fibrous tissue that still occupies a portion of the original arterial lumen. (In some instances, of course, such a recanalization sequence may lead to web formation.)

In none of our cases was there any evidence in support of the thesis [15] that the initiating event in band or web formation was autochthonous thrombosis due to pulmonary atherosclerosis.

It is apparent that the bands and webs in many, if not all, instances are not static structures but, rather, undergo frequent morphologic alterations through periodic accretion of more recent thrombotic and embolic deposits. Their appearance can thus vary from single, delicate, threadlike strands to webs of great complexity. The clinical significance of these lesions is largely dependent upon their number and distribution. In most instances the presence of single or scattered pulmonary arterial bands and webs is probably of no hemodynamic consequence. They do have potential significance, however, even when few in number, since an arterial lumen containing such a lesion may be totally occluded by a small entrapped embolus which ordinarily would have passed further distally. In such a circumstance a large pulmonary infarct (which we have observed), or even death, may result. When the lesions are present in great number bilaterally, they can so impede pulmonary blood flow that chronic, ultimately fatal cor pulmonale ensues.

As noted previously, the recognition at necropsy of pulmonary emboli, recent or healed, depends to a great extent on the vigilance and technique of the prosector. This is particularly apparent if the lesions are the delicate, easily destroyed or overlooked structures which comprise bands and webs. Although these structures represent a relatively uncommon end stage of organization of thromboemboli, their identification and proper interpretation are of importance pathologically in evaluating the life history of pulmonary emboli, the response of the pulmonary vascular bed to such emboli, and the relation of emboli to infarcts, to pulmonary vascular sclerosis, and to pulmonary hypertension. They are of importance, also, in the study of clinicopathologic correlations in pulmonary embolic disease and in the evaluation of various therapeutic procedures designed to prevent this disease.

Summary

The finding of intraluminal fibrous bands and webs in the pulmonary arterial tree is not uncommon, but these lesions are probably often overlooked or misinterpreted. We have presented a pathologic description of these structures based upon material in 52 cases and have adduced evidence in support of the thesis that these lesions represent organized thromboemboli. Three basic processes appear to be involved in their

evolution. These are initial arrest of an embolus, propagation of thrombus distal to this site, and frequent incorporation of more recently accumulated thrombus resulting in alterations in the size and form of the lesions. The recognition and proper interpretation of these structures are essential in pathologic and clinicopathologic studies of pulmonary embolic disease.

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Legends for Figures

Unless otherwise indicated, photomicrographs were prepared from sections stained with hematoxylin and eosin.

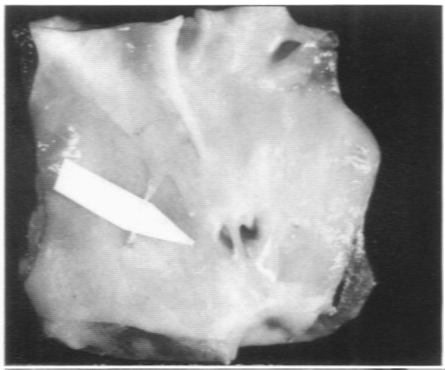
Fig. 1. A lobar pulmonary artery contains 3 lesions. The white paper arrow underlies a delicate, translucent cord. At one attachment (above) the arterial intima is thickened and forms a flat plaque. Above this lesion there is a linear zone of intimal thickening which represents a "ridge." To the right of the arrow, a thicker fibrous band bridges the ostium of a branch vessel.

Fig. 2. Femoral vein. The paper arrow lies beneath a thin fibrous band. A second, smaller band is visible below the base of the arrow.

Fig. 3. A lobar pulmonary artery contains a complex fibrous web. "A" indicates an area of intimal thickening which is the nodal zone of attachment of multiple fibrous bands. Many of the bands cross the arterial lumen, and a few bridge the ostiums of subsidiary vessels. "B" indicates a large intimal plaque, continuous with the fibrous core of the web. "C" indicates a short band with multiple attachments lying at a branch ostium. "D" marks a separate band which crosses the orifice of a branch vessel.

Fig. 4. A lobar pulmonary artery. "A" indicates an adherent, recent embolus. Extending distally from it are two fresh band-precursors with folded cardboard elevating their free midportions. The band-precursor marked by paper arrow "B" terminates at a bifurcation. Band-precursor "C" lies within a secondary branch and actually terminates in a tertiary branch vessel at still another bifurcation.

1



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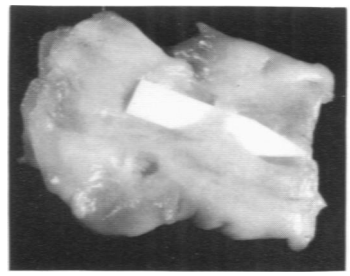

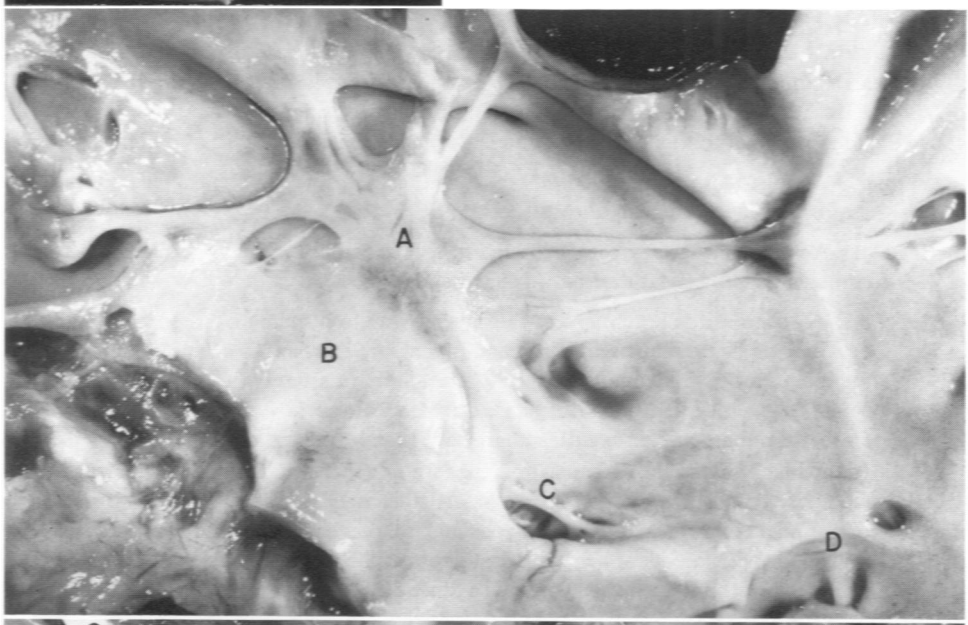

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FIG. 5. An adherent embolus lies at a bifurcation of a pulmonary artery and exhibits a free midportion (paper rectangle). Extending from it along a branch vessel is a fresh band-precursor with a point of attachment (paper arrow) at its midportion. Proximal to this point the band is gray-red, while distally it is red-black.

FIG. 6. A pulmonary artery contains a recent embolus (right) entrapped in the meshwork of a branching fibrous band. × 2.

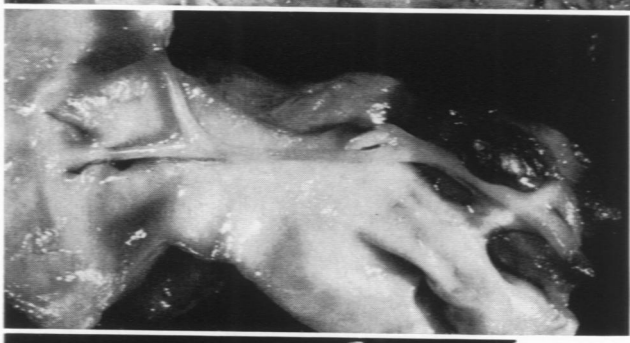
FIG. 7. A pulmonary artery contains a recent saddle embolus. The upper limb of the embolus passes beneath an old fibrous band (arrow).

FIG. 8A. A composite photomicrograph showing (top to bottom) distal, central, and proximal portions of an organizing band-precursor. The band is enclosed by endothelium and by an incomplete layer of granulation tissue, both continuous with similar tissue at the attachment sites. On the upper surface of the band there is fresh thrombus, and above this there is a small amount of postmortem clot (dark black material). Organization is more advanced at the proximal attachment site than at the distal. The proximal attachment lies at an arterial bifurcation, and the lower arterial limb proximal to the band shows intimal fibrous thickening with abundant hemosiderin (arrow). The rectangle encloses a segment which is illustrated at higher power in Figure 8B. × 14.

FIG. 8B. The midportion of the band-precursor contains 4 layers: (a) fibrous tissue; (b) organizing thrombus; (c) unorganized central core of band, composed of fibrin and platelets showing prominent axial orientation; (d) more recent, superimposed thrombus. Arterial intima is at left-hand edge of photograph. × 50.

5

6



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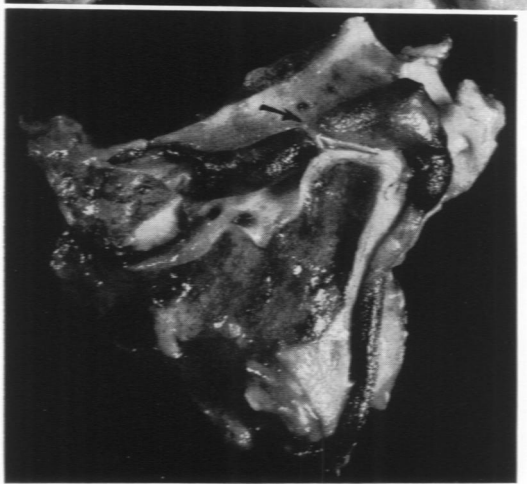
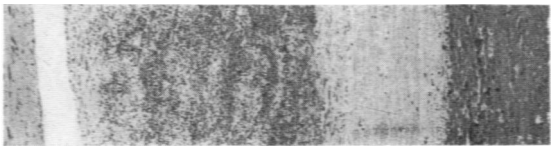
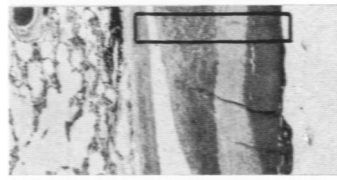


8B





8A

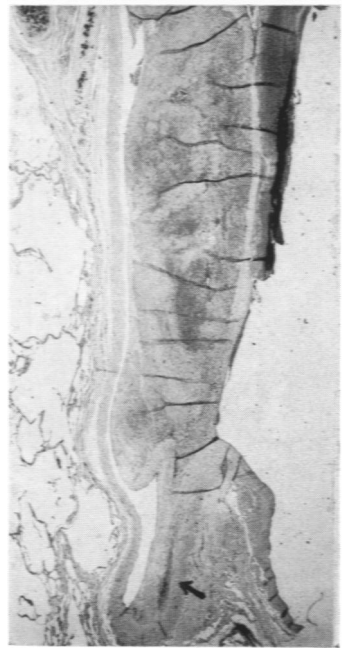
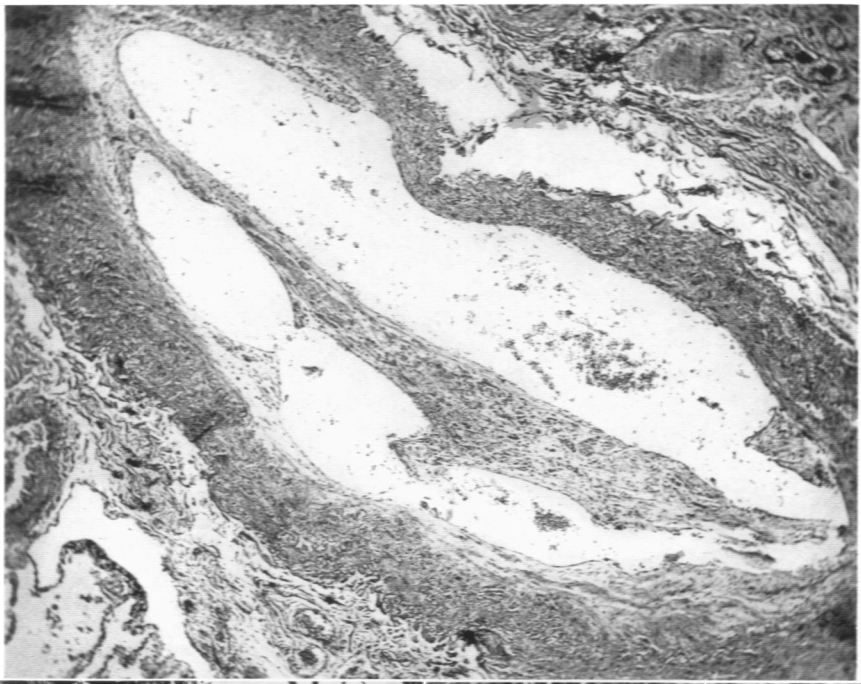


FIG. 9. A band of intermediate age with a residual central core of unorganized clot. The arterial intima is normal except at attachment points. × 50.

FIG. 10A. A band somewhat older than that in Figure 9 (proximal, above; distal, below). The rectangles enclose segments shown at higher magnification in Figures 10B and 10C. × 27.

FIG. 10B. An attachment site. The band is composed of loose, cellular fibrous tissue containing hemosiderin (dark granules). × 90.

FIG. 10C. The distal end of the band is composed of recent thrombus with very early organization. × 90.



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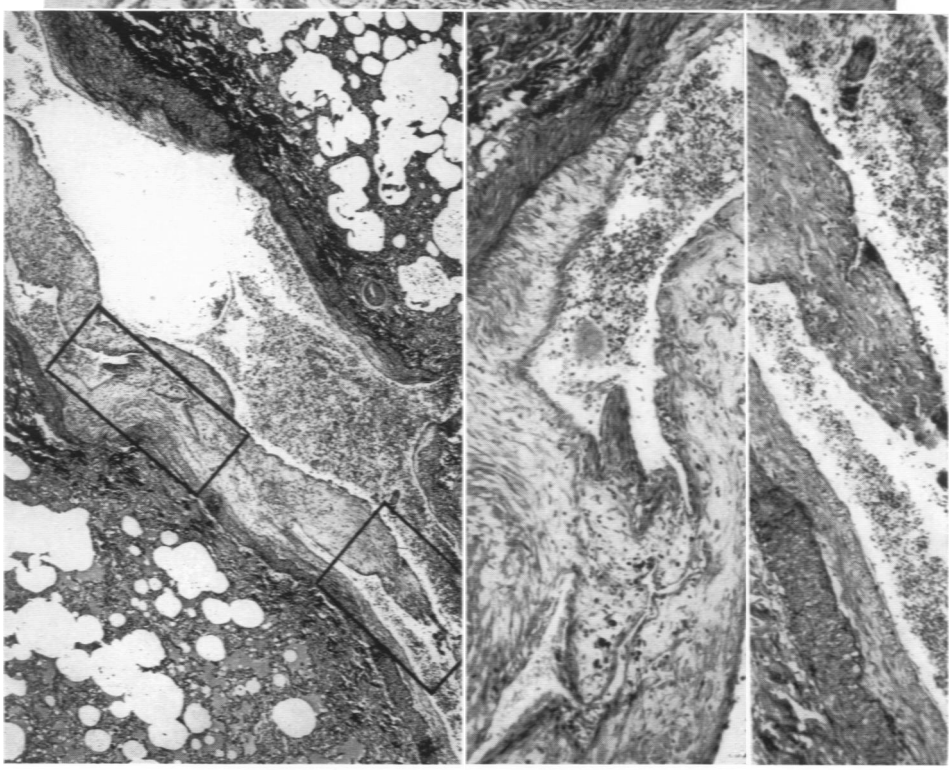


10A 10B 10C

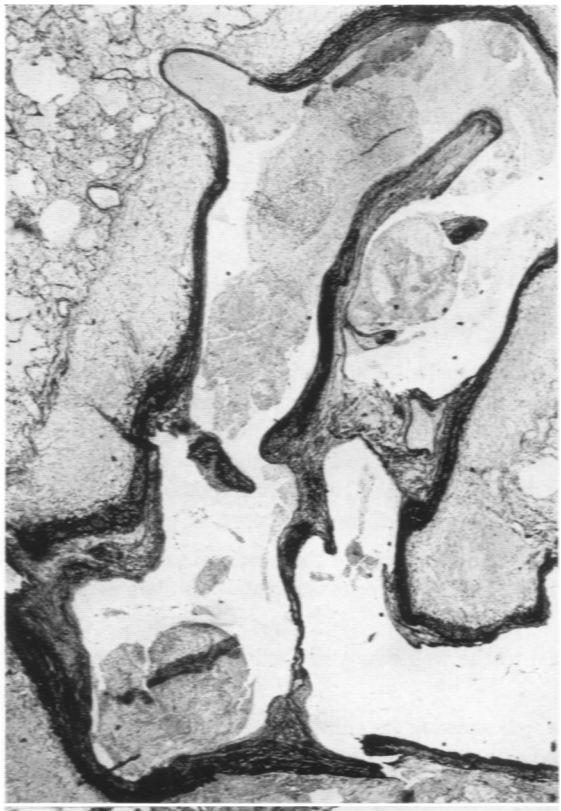
FIG. 11. A band containing numerous elastic fibers, most of which are oriented parallel to the surface. Note the tenuous portion of the band which attaches distally at a bifurcation. Near the band are several fibro-elastic structures that represent branches of the band disrupted in sectioning. The arterial wall is normal except for fibro-elastic intimal hillocks at attachment points. Verhoeff-van Gieson stain. × 22.

FIG. 12. A band containing abundant smooth muscle. The muscle bundles tend to be oriented parallel to the surfaces of the band. The arrows designate visible portions of the normal arterial wall. × 40.

FIG. 13. A web in cross section. The abundant elastic fibers are oriented parallel to the surfaces of the component trabeculae. There are several small plaque-like areas (arrows) containing fewer elastic fibers which may represent more recently organized thrombus. Around much of the circumference of the artery there is marked fibro-elastic intimal thickening. Verhoeff-van Gieson stain. × 20.

FIG. 14. The central portion of a complex web (no part of the arterial wall is shown). Note the prominent fibro-elastic stratification, suggesting episodic growth and remodeling due to incorporation of added thromboemboli. Verhoeff-van Gieson stain. × 24.

11



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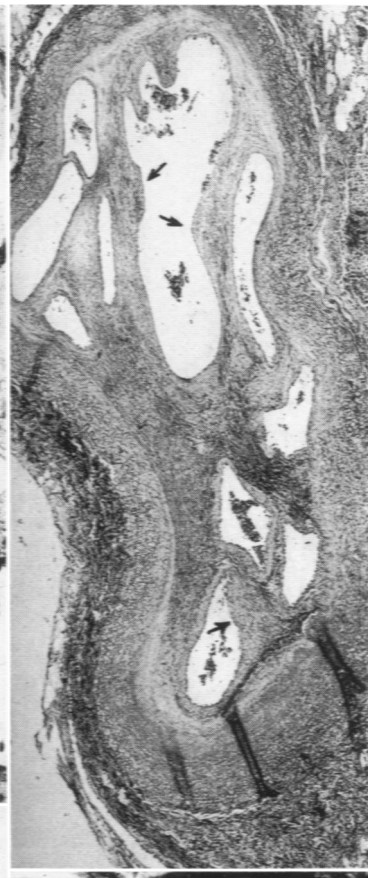

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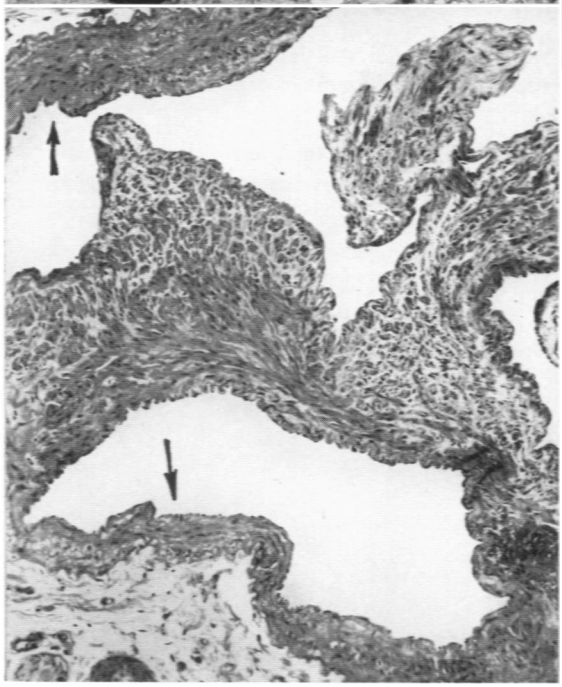

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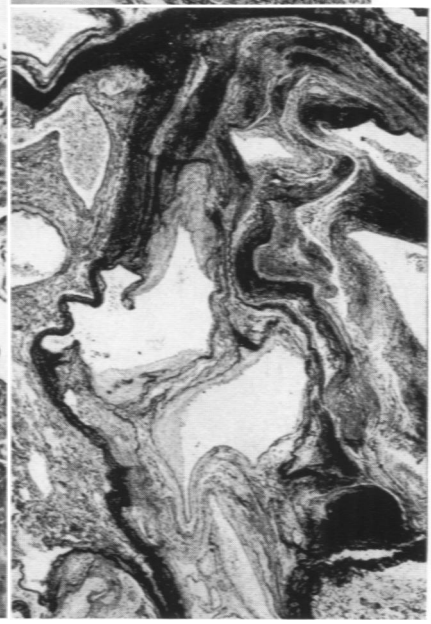


FIG. 15. A longitudinal section of an old, collagenized fibrous band, at the distal attachment site of which there was apparently marked impairment of blood flow. Note the intimal thickening to the left of the attachment site, compared with the normal arterial wall to the right. The loose fibrous meshwork extending distally (to the right) from the main part of the band is interpreted as representing more recently organized, superimposed thrombus. × 50.

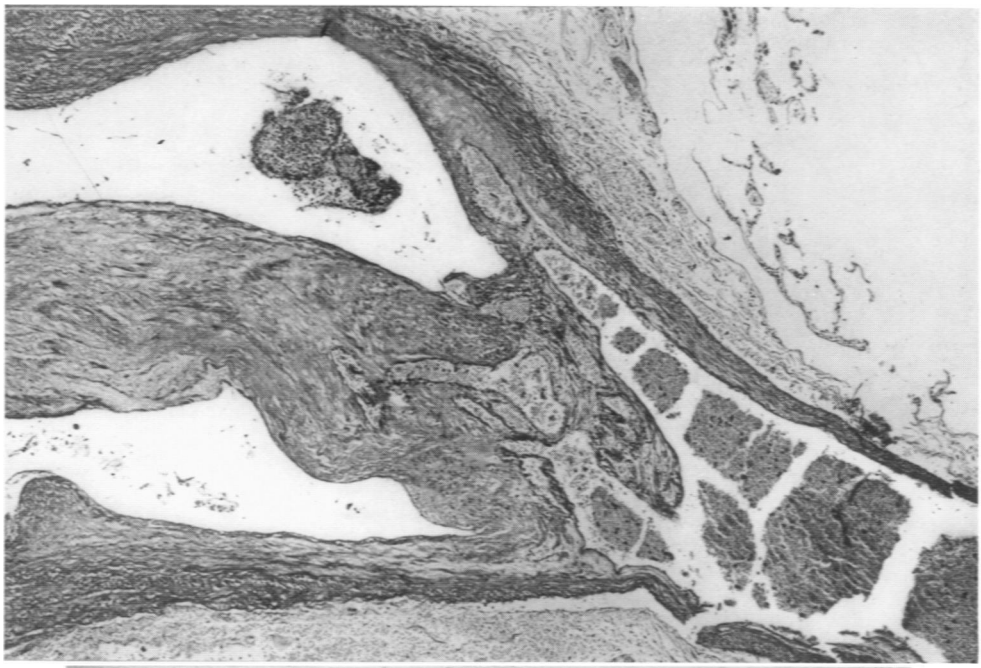
FIG. 16A. A complex web showing marked thickening of the proximal arterial wall. The brackets enclose segments illustrated at higher magnification in Figures 16B to 16E. × 8.5.

FIG. 16B. The arterial wall proximal to the web shows marked intimal thickening. × 20.

FIG. 16C. Proximal portion of the web showing its continuity with the thickened arterial intima (at the left). Beneath the body of the web the arterial wall is only slightly thickened. The arrows indicate small masses of organizing thrombus. × 20.

FIG. 16D. The midportion of the web. The arterial wall is much thinner than in Figure 16B. The arrow indicates another polypoid mass of organizing thrombus. × 20.

FIG. 16E. The arterial wall distal to the web of normal thickness except for small hillocks at the attachments of fibrous bands which have extended to this region from the main body of the web. × 20.



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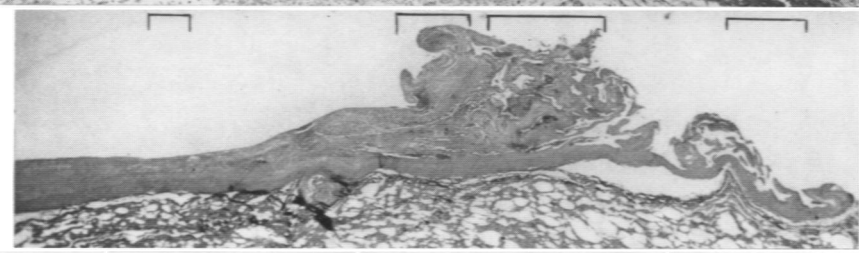
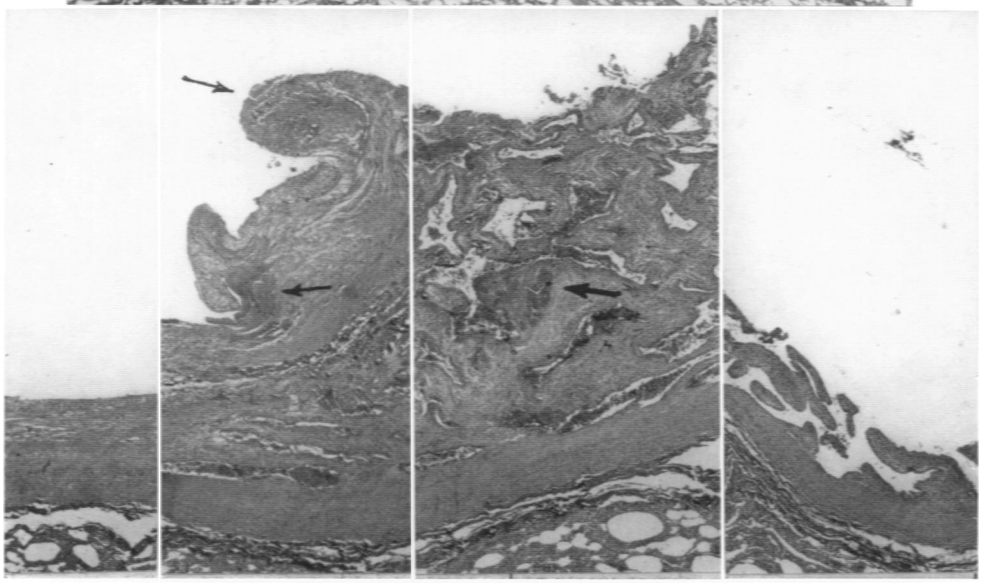


16A



16B 16C 16D 16E