A PATHOLOGICAL STUDY OF PRIMARY MYOCARDIAL AMYLOIDOSIS *

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There have been reported two distinct groups of cases in which amyloid infiltration of the heart has been observed.

Within the first, and by far the more prominent group numerically, is a series of cases such as those reported by Von Huebschmann,[1] in which amyloid has been demonstrated in the hearts of patients affected by a generalized amyloidosis. In eight such cases investigated postmortem by Von Huebschmann only on microscopic investigation was amyloid demonstrable within the myocardium, and in no case was its occurrence in this location associated with specific differential symptoms referable to the system involved. Microscopically, the myocardium was found to contain amyloid deposits within the connective tissue and vessel walls; rarely, it was demonstrable in the valves and endocardium. In no instance was amyloid degeneration of the muscle fibers observed. On the basis of his observations, this investigator concluded that the amyloid arises both by transformation of the connective tissue fibrils (into amyloid) as well as by an interpositional deposit.

Commenting on this series in its review, Beneke and Bönning[2] suggest that Von Huebschmann's observations are in agreement with all similar reported cases. But they advocate that in primary myocardial amyloidosis the peculiar nodular amyloid deposits in the heart are not only confined to the capillary walls, but are localized about the muscle fibers themselves. It is their opinion that the accumulation of amyloid in the ectoplasmic zone of individual tissue cells leads to complete cellular intubation by amyloid, and that cells in this manner deprived of their nutrition undergo inanition atrophy. This they believe to be indicative of a previously unrecognized causal relationship between the metabolic processes of the sarcolemma-free muscle fibers, and the local deposition of amyloid.

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The localization of amyloid confined to single foci is considered analogous to the development in scattered foci of degenerative changes in general, which, as Ribbert has advocated, are related to localized metabolic changes. Beneke and Bönning further suggest that the patchy distribution in the heart is no more remarkable than the generally known distribution of amyloid in other organs. With these cases of general amyloidosis the present study is not immediately concerned.

By far the less important group numerically, and, because of their uniqueness, perhaps unduly emphasized, are those cases in which the amyloid infiltrations are confined primarily to the heart, almost to the exclusion of those organs and tissues in which it is deposited in the usual cases of generalized or local amyloidosis. In this group there are, to our knowledge, only three reported cases at present.

In the case reported by Wild [3] there was found marked amyloid deposit in the myocardium associated with similar local deposits in the lungs, tongue, bladder and gastro-intestinal tract. The clinical report of this case is incomplete. The condition in the heart existed without true clinical signs referable to its existence. The patient, a 56 year-old woman who died of erysipelas had shown only the general clinical picture of heart failure. At autopsy the heart was small. Its walls were generally involved by nodular deposits of cartilaginous consistency, especially in the left auricle, and least pronounced in the left ventricle. Even the valve leaflets were infiltrated and thickened. There was an associated nodular infiltration of the peritoneum and intestinal serosa. According to Wild's observations the homogeneous masses which he found in the heart and intestine possessed a central zone which gave the amyloid reaction, and a non-specific hyaline peripheral zone.

The case of Steinhaus [4] presented changes remarkably similar to the findings of Wild. In a previously healthy 40 year-old man, the clinical syndrome consisted of vomiting, distention and intestinal hemorrhage, secondary anemia and clinical heart failure, the entire duration being but six months. Clinical observations on this case indicate a copious bleeding from the gastro-intestinal tract in a patient previously in good health, followed by marked anemia of the secondary type. The pulse was rapid. Albumin and traces of blood were found in the urine. In this case also there was apparent a stiff knot-like infiltration of the entire heart wall, stomach and

intestinal tract. The cut surface of the heart was hard, of a glassy homogeneity and appeared whitish gray as if cooked. In the markedly thickened stomach wall were many glassy nodules, and numerous erosions of the mucosa. In this case, too, the liver, spleen and kidneys were free of amyloid. The amyloid deposits adjoined extensive deposits of hyaline-like material in the connective tissue. Often, particularly when in the vessel walls, those nodules gave the amyloid reaction. The staining reactions have been questioned by Beneke and Bönning, although they admit that the striking similarity to the case reported by Wild favors the correctness of the dye tests.

The case of Beneke and Bönning was a 70 year-old man who entered the hospital moribund with a diagnosis of chronic bronchitis. The amyloid deposits were limited almost exclusively to the heart, the adjoining vena cava and the lungs. Because it has been reported fully and accurately this case will be referred to in the discussion of the following one which fell under our observation, a case which has points of similarity to all of the foregoing, but differs from each of them. Our case follows:

Report of Case

Clinical History: Feb. 8, 1926, a 65 year-old negro male, entered the hospital with a respiratory infection for which he was successfully treated. The essential findings at this time were: a generalized arteriosclerosis, normal heart, absent or sluggish reflexes, negative urine. Non-protein nitrogen 33.3, Wassermann and Kahn negative, sputum negative, stools negative, globulin cerebrospinal fluid, negative.

April 16, 1928, the patient was seen in the Out-Patient department complaining of cough of one months duration associated with shortness of breath on exertion and epigastric tenderness. Digitalis therapy was instituted.

Nov. 19, 1928, the patient entered the hospital with a typical history of progressive left-sided heart failure.

Laboratory Findings: Electrocardiograms: Nov. 19, 1928, auricular fibrillation, left ventricular preopnderance. Dec. 13, 1928, auricular fibrillation, ventricular premature contraction. Dec. 14, 1928, auricular fibrillation, left ventricular preponderance. Dec. 15, 1928, auricular fibrillation, left ventricular preponderance, low voltage, inverted T in all leads.

He was discharged from the hospital Oct. 20, 1928, in a state of complete compensation. He had been diagnosed: general arteriosclerosis, arteriosclerosis of coronary arteries, cardiac arrhythmia-auricular fibrillation, cardiac insufficiency. He was sent out on digitalis 0.1 gm., and given instructions regarding diet, exercise and fluids. He returned to the Out-Patient department about two months later (Jan. 14, 1929) with slight dyspnoea and moderate edema. Digitalis was increased to 0.1 gm. b. i. d. and patient instructed as to conduct.

On March 19, 1929, the patient came into the hospital with a second bad break in compensation. With salyrgan and theocin he lost a huge amount of edema and became compensated. His mental befuddling was maintained, however. He was incontinent of urine and feces, and developed bed sores. On March 29th he developed a chill and high temperature. On March 30th he was in deep coma with stertorous breathing, neck rigid, and questionable Kernig. A spinal puncture gave a cloudy fluid with a fibrin coagulum and 2000 cells. Culture of this fluid and blood culture showed a hemolytic streptococcus. He died March 31, 1929, in coma.

From the autopsy the following diagnoses were made:

Septicemia, streptococcus hemolyticus, blood culture; meningitis, acute, diffuse; pericarditis, acute, purulent, with purulent effusion; cardiac dilation and hypertrophy; myocardial necrosis, acute focal with auricular thrombus, puriform; amyloid deposits, myocardial, pulmonary and renal; nephritis, chronic vascular; congestion, spleen, liver, adrenal and renal; decubitus ulcers.

Heart

The following description of the heart was given at autopsy:

The heart is a typical cor bovinum, weighing 700 gm. It is rubbery to compression, so that the depressed wall gives a sense of rebounding when the tension is released. The cut surface throughout is of a homogeneous reddish appearance as of stained liquid albumin, and gives one the impression that the light is reflected from the surface which is partially composed of a translucent substance. There is a puriform thrombus in the apex of the right auricle. The left auricle is clear to the limits of the appendage. The right auricular cavity is markedly dilated as is the left, and the walls of both are thickened to measure 0.5 cm. in diameter. The right ventricular cavity, it is estimated, is twice the normal size. The myocardium is 1 cm. thick at the base, and 3 mm. thick at the apex. The myocardium of the left ventricle is tremendously hypertrophied and the cavity increased to an estimated one-half greater than normal. At the base the wall measures 4 cm.; at the apex 2.75 cm. There are no grossly apparent necroses. The papillary muscles of both ventricles are markedly hypertrophied, some in the left ventricle measuring as much as 1.5 cm. in diameter at the base. The valves are free and flexible throughout. There are no vegetations. The mitral, aortic, tricuspid and pulmonic valves measure respectively: 11, 8.5, 8, and

15 cm. The endocardium throughout is clear and smooth with the exception of the right auricle which has already been described. The coronary arteries are moderately sclerotic. A small atheroma is present in the orifice of the left. It is recent. The coronary sinus is markedly dilated.

Because amyloid was suspected from the appearance of the heart, tissues were taken from every portion of this organ inclusive of pulmonary arteries, aorta, inferior vena cava, pulmonary veins, and pericardium. These tissues were rapidly stained in a Gram's iodine solution and transferred at once to sulphuric acid. The following tissues gave a positive reaction almost immediately.

Left Ventricle: A section of the muscle of the left ventricle with a specific amyloid stain, examined under the dissecting microscope, reveals an extensive bluish mottling of the tissue. These bluish areas are diffusely scattered throughout the entire section, running parallel to the line of fiber of the myocardium. In this manner the specimen is given a stippled or tigroid appearance with the stippling occurring in interrupted parallel bands. These bands are not complete in themselves, but have points of continuity with one another alternating with points of interruption. It is therefore evident that the deposit of amyloid is not uniform but diffuse throughout the myocardium. This observation has been recorded photographically by reflected light. The amyloid appears black in the reproductions. The appearance of the amyloid in the epicardium and beneath the endocardium of this ventricle is striking. Interlacing clusters of bluish material often related to definite vessels would by this stain indicate that, in the epicardium at least, the amyloid deposits have a definite relation to some portion of the vascular bed.

Right Ventricle: Within the right ventricle deposits are essentially the same as those in the left, and practically as abundant.

Right Auricle: In a section taken through the right auricle to include the flap of the foramen ovale, the amyloid has practically the same anatomical distribution as in the ventricles. Wherever the muscle fibers have been cut longitudinally in this section the same tigroid appearance is observed as was apparent in the ventricular myocardium. Because of the line of muscle fiber in this section, however, many of the deposits appear as minute interlacing islands in the photograph. The focal areas of endocardial infiltration of the diffuse type are again apparent.

Left Auricle: In a tangential section of the left auricle the amyloid deposits have the characteristic tigroid appearance so apparent in the ventricles. In a cross-section they appear as minute, sometimes interlacing islets separated by the elements of the myocardium, endocardium, or epicardium.

Microscopic Examination

Myocardium: The muscle cells throughout the entire myocardium are hypertrophic, a condition which is especially marked within the wall of the left ventricle. An occasional minute scar is apparent, especially in sections from the posterior portion of the left ventricular apex. The adjacent muscle cells are markedly enlarged. An estimated 50 per cent of the volume of the myocardial sections is represented by amyloid, which, throughout the myocardium is present in the crevices between the muscle bands and their composite fibers, and intracellularly. It is homogeneous, and is present in the form of eccentric laminated bands which in many instances give to this foreign element a whorl-like appearance. There are areas in which there is so great an abundance of this material that the muscle elements are entirely wanting or occur only as vestiges of an original cell or cells in a field composed almost entirely of amyloid. Not every bundle or fiber is equally affected, for while amyloid is found in the interstitial space between practically every muscle fiber, thus giving it a diffuse distribution, its deposition is not at all uniform. At times it is present as a narrow band which forms a sheath at one side of an interfibrillar capillary, not encroaching on the adjacent muscle fibers. In other instances large areas composed only of amyloid are present. Marked indentation of the myocardial cells by amyloid is readily apparent, but in a few instances only is the integrity of the muscle cell wall interrupted by these apparent invaginations. In serial sections these invaginations can be readily traced from simple indentations of the cell wall to actual cellular invasion so that one finds in examination of a single fiber serially the transition from simple cellular indentations by amyloid to actual intracellular deposits of this substance. When the amyloid present is actually intracellular, having gained entrance to the cell in this manner, the amyloid substance is often surrounded by a clear zone of sarcoplasm, the nucleus displaced and the myo-

fibrillae concentrated in the peripheral portion of the cell. There is no evidence of degenerative change in these cells. It is of importance that in those instances in which this actual invasion of the cell has occurred, the pericellular tissues are often amyloid free except at that point at which the amyloid bud makes its invagination. It is further of importance that invaginating buds of amyloid are always traceable in serial sections to a point at which they become continuous with a pericapillary deposit of amyloid in the immediate vicinity.

There is no evidence of amyloid in the chordae tendineae, although it is present in abundance at the musculotendinous transition of the papillary muscles.

Endocardium: Focal collections of diffuse amyloid infiltration are here present as within the myocardium. The absence of large vessels in this tissue places the deposits, which here also are perivascular, about the capillaries and minute venules. Many of these deposits, because they appear primarily well myocardialward in this tissue are directly continuous or contiguous with the deposits of amyloid of the myocardium proper. In the right auricle alone the deposits have become sufficiently large, subendothelially to cause a protrusion of a large area of endothelium. The aortic valve is definitely infiltrated, especially in the proximal portion where the deposit of amyloid is continuous with that present in the myocardium. Peripherally in the valves the deposits consist of focal areas deep in the subendothelial tissues. Amyloid in similar distribution is found within the pulmonic, mitral and tricuspid valves. Never is it apparent from the surface. Its occurrence is always deep in the subendothelial stroma.

Pulmonary Artery: Distinct foci of amyloid are present in the outer media and adventitia of the pulmonary artery. In at least one instance vasa of the wall are demonstrable within the amyloid mass.

Aorta: Distinct foci of amyloid are apparent throughout the media. Many have a definite relationship to the vasa vasorum. A curious infiltration of the para-aortic adventitial veins is also evident. In this location there is a distinct localization of the amyloid within the media, though, at times the deposit has broken through and bulges the endothelium of the intima. There is no thrombus formation at the point of intimal protrusion.

Inferior Vena Cava: Distinct diffuse amyloid deposit is apparent,

especially medialward in this tissue. It has approximately the same relationship as that present in the para-aortic veins.

Lungs: Irregular intramural amyloid deposits are present generally throughout the alveolar walls of both lungs. The finer examination of these deposits shows that they are extensive, nearly diffuse about the alveolar capillaries where, at times it is so abundant as to cause the alveolar epithelium to bulge into the acinar space. The same transitions in the loss of the normal stromal connective tissue relationship between the capillaries and of the respiratory epithelium of the lungs is here observed as was so apparent in the interfiber deposits of the myocardium.

Kidneys: The renal arteries of medium size present a definite thickening of their intima, and in some instances actual occlusion of the lumen is apparent. In association with these vascular changes there occur diffusely throughout the cortex numerous triangular scars containing atrophic renal structures with large numbers of hyalinized glomeruli. The scars are infiltrated with lymphocytes. Extensive deposits of amyloid occur in the pyramidal portions of the organ. They have a definite pericapillary arrangement.

Pararenal Ganglion: A definite deposit of amyloid is present in the interstitial fibrous tissue of the ganglion.

No deposits of amyloid were found in the spleen, liver, adrenals, pancreas, prostate, brain or any organ or tissue other than those mentioned.

Discussion

It is not the purpose of this paper to explain the clinical, physiological, or biochemical principles involved in amyloidosis. Clinically, it is sufficient to say that our case deals with an individual 65 years of age who in the last three years of life developed a progressive left-sided heart failure which was twice recompensated before death — a cardiopathy with hypertension without renal disease leading to complete decompensation.

It is rather our purpose to confine the discussion to the anatomical observations. The review of these findings in our case at once demonstrates definite and important variations from the observations made in those cases previously reported by Steinhaus, Wild, and by Beneke and Bönning. But one thing is unconditionally common to the foregoing and the case under our observation, *i. e.* the

primary localization of amyloid in the heart, especially the myocardium.

At autopsy, the heart, an organ of 700 gm., is dilated and hypertrophic throughout, and possessed of peculiar properties suggestive of amyloid deposition. In this lies the first variation of our case from all others in that the heart is markedly hypertrophic, whereas in all previously reported cases it was found to be atrophic. Unquestioned deposits of amyloid are not grossly recognized in the heart at autopsy, a feature contrary to the case of Beneke and Bönning in which the amyloid was readily detectable as large glassy nodules within the heart wall. Recognized tests for amyloid, however, reveal in our case a diffuse amyloid infiltration of the heart. In the premise the diffuse type of amyloid infiltration of the heart in this case bridges a gap in what has been previously known of primary myocardial amyloidosis, in that it demonstrates the possibility of diffuse as well as localized types of amyloid deposit in primary myocardial amyloid infiltration; a feature which places the primary myocardial amyloidosis in the same category as the generalized types of amyloidosis, in which both focal and diffuse types of amyloid deposit have been repeatedly observed, for example, in the spleen.

The microscopic findings place the only detectable amyloid deposits in this case in the heart and the neighboring great vessels, the lungs, the kidney, and a perirenal ganglion. In the heart the amyloid is deposited diffusely, though irregularly, throughout the tissues. In the epicardium it is present only in relation to veins some of which it occludes by pressure from without, and which form the centers from which it radiates into the adjacent tissue replacing all but the fat cells. In the endocardium it is present only in the deeper layers of the stroma and here again it is in definite relation to veins. In the less densely infiltrated areas of the myocardium, it is present primarily immediately adjacent to specific capillary endothelium whose connective tissue stroma it replaces; while other capillaries are entirely free of this deposit. This constitutes preeminently the primary site of amyloid deposit. Its occurrence in isolated areas in the interstitial tissue or about muscle cells is in every instance traceable in serial sections to a pericapillary relation. In the presence of slight traces of amyloid, therefore, it is located primarily about capillary endothelium. By examination of fields in

which the amyloid deposits are slightly more extensive it is apparent that the amyloid has extended from its pericapillary nidus into the interstitial tissue which it obliterates, ultimately replacing the stroma of the muscle cells which it surrounds, bridging the entire interval from capillary wall to muscle cell; but always one observes that the lamellae which stratify these deposits have their centers in a capillary. In the replacement of interstitial tissues it is evident that those capillaries about which the amyloid is primarily deposited are compressed to minute strands and occluded, ultimately completely disappearing. It is of equal importance, that other capillaries remain dilated and congested in many amyloid areas, and while this condition maintains there is little evidence of cellular atrophy; but once this group of capillaries is gone, the amyloid masses are seen to be mottled by hollow lacunae, the rests of preexistent muscle cells.

It is further observed that amyloid occurs often in the myocardial venules, never in the arteries; that the extremely dense areas of amyloid in which all tissue structure has been obliterated often correspond roughly to a single arteriovenous capillary tree. In the endocardium and valves, amyloid deposit occurs only in the deeper tissues. Likewise in the aorta and pulmonary arteries the amyloid occurs primarily in the media, the site of the vasa vasorum; it presents in the intima only by continuity from medial deposits; and finally, it has been noted that in the papillary muscles of the ventricles, while amyloid is present abundantly in the muscle it is entirely wanting in the chordae tendineae, abruptly ending at the musculotendinous transition line. It is therefore apparent that amyloid deposit does not occur in those tissues which are avascular except by extension from deposits in vascular tissue; that it occurs in vascular tissue primarily in relation to venous capillary endothelium and vein walls, never in arteries except the pulmonic artery and aorta where it is present about the medial vessels and adventitial veins. Moreover the distribution and abundance of the deposits bear a specific relation to the area of the capillary bed. It is my opinion that the amyloid is deposited only in those tissues which are known to have a definite vascular bed, and here its deposition is proportional to the vascular bed; and its deposit is primary about the endothelium of specific venocapillaries and venules from which it expands to include adjacent structures. Ultimately it cuts off the

arterial supply to the part and this in turn leads to atrophy of the structures in the involved area with a persistence of amyloid. Amyloid apparently is not affected by the anemia but continues to accrue, leading ultimately to closure of the lacunae left by atrophic muscle cells. A sheet of amyloid is thus produced which is marked by whorls, whorls formed by concentration of contingent amyloid deposits which encroach from several directions on muscle cells and lead to concentration bands of amyloid about individual cells, prior to the time the vascular supply has been severed.

It has not, however, in any instance been my observation that amyloid occurs primarily in the ectoplasmic zone of the muscle cells from which it invades the cell at random as suggested by Beneke and Bönning. Indeed, as has already been pointed out, the primary deposits of amyloid occur about venocapillary endothelium, from which it extends to surround muscle cells which in densely infiltrated areas it encases. In these areas in which the muscle cells are entirely surrounded, many of the cells manifest no evidence of atrophy. Rather the myofibrillae and other intracellular components are simply concentrated, as by partial dehydration. In serial section these cells have been traced in every instance to a level at which they reach an amyloid-free field. Actual cell atrophy is apparent only when in serial cross-sections the amyloid is so abundant as to have led to complete vascular occlusion to the area and to the cell over its entire extent. Of those cells which show no evidence of atrophy, many are indented and invaginated by adjacent amyloid buds; more commonly they are symmetrically compressed by amyloid which forms a pressure concentration zone at their periphery. From these encasements many cells, their membranes intact, have retracted in fixation. There is microscopically no evidence that amyloid invades the cell from this periphery except by one method which will later be described. It is apparent that by far the greater amount of amyloid replacement occurs as follows: the cell first dies by loss of its blood supply, leaves its empty lacuna in a field of amyloid and, then only, amyloid expands to fill this interval. This feature explains the picture of multiple whorls so often seen in a field of solid amyloid.

Microscopically evidence is found that amyloid actually invades the cell by only one method, a method independent of the concentration of the pericellular amyloid, inasmuch as it occurs in

fields in which a pericapillary amyloid bud simply contracts with one side of a muscle cell as well as in fields in which the cell is at certain levels densely surrounded. There is evident from serial cross-sections an actual invasion of the cell by amyloid, with penetration of the cell wall by the amyloid which within the cell extends bipolarly leading to peripheral concentration of the myofibrillae and lateral nuclear displacement. This intracellular amyloid lies in the position of the sarcoplasm. The entire group of intracellular structures in cells invaded in this manner are intact and manifest no changes other than displacement. There is no evidence of vacuolization or other cell response usually seen in the presence of intracellular foreign bodies. This observation presents a feature thus far entirely foreign to our knowledge of cellular pathology. The actual penetration of a fixed cell wall by a foreign substance without cellular destruction or recognizable alteration other than displacement is unique. The fraying out of the cells at the portal of entry of these invading buds offers many possibilities as to the nature of the muscle cell wall, which hitherto have been unsuspected.

The serial cross-sections, therefore, establish the fact that the apparent indiscriminate invasion of the muscle cells from the peripheral concentration zones is purely a sectional and distortional artifact. Cellular invasion occurs only by the method which we have just described, and by that method in comparatively few instances.

The deposits in the lungs and kidneys are likewise pericapillary in distribution. We therefore find no substantiation for the opinion advanced by Beneke and Bönning to the effect that amyloid is deposited in the ectoplasmic zone of the tissue cells as a result of disturbed metabolic equilibrium, which ultimately by encasement of the cell by amyloid leads to inanition atrophy. In fact we find no evidence that the amyloid bears relation to disturbed metabolism in any specific cell. It is our opinion that amyloid is deposited from the tissue lymph as a result of changes in the permeability in the venous endothelium. Its presence may be simply the result of abnormalities in the capillaries which are non-receptive to a substance to which normally they may be permeable.

Summary

1. The distribution of amyloid within the myocardium in primary myocardial amyloidosis may be diffuse as well as focal.

2. Its deposition within the heart may occur in the presence of hypertrophy as well as atrophy.

3. It is deposited only in those tissues which have a known vascular bed. Its presence in avascular tissue is only accomplished by continuity with deposits in vascular tissue.

4. The deposition of amyloid occurs primarily about venocapillary endothelium from which it extends to surround the normal tissues, ultimately cutting off the vascular supply to the part. Then only the tissues atrophy and are replaced by amyloid. This constitutes the primary mode of amyloid infiltration.

5. Amyloid gains entrance to occasional cardiac muscle cells by a process of invagination and ultimate penetration of the cell wall. This is a direct method by which myocardial cells may be replaced by amyloid.

6. There is no evidence that amyloid deposit is dependent on localized metabolic changes, nor is there evidence of primary pericellular deposition of amyloid, from which it freely invades living cell substance.

7. The deposit of amyloid apparently is dependent upon changes in endothelium, especially of venocapillaries, which may possibly become impermeable to some substance in the tissue lymph which may normally be present in tissue lymph and capable of permeating venocapillary endothelium.

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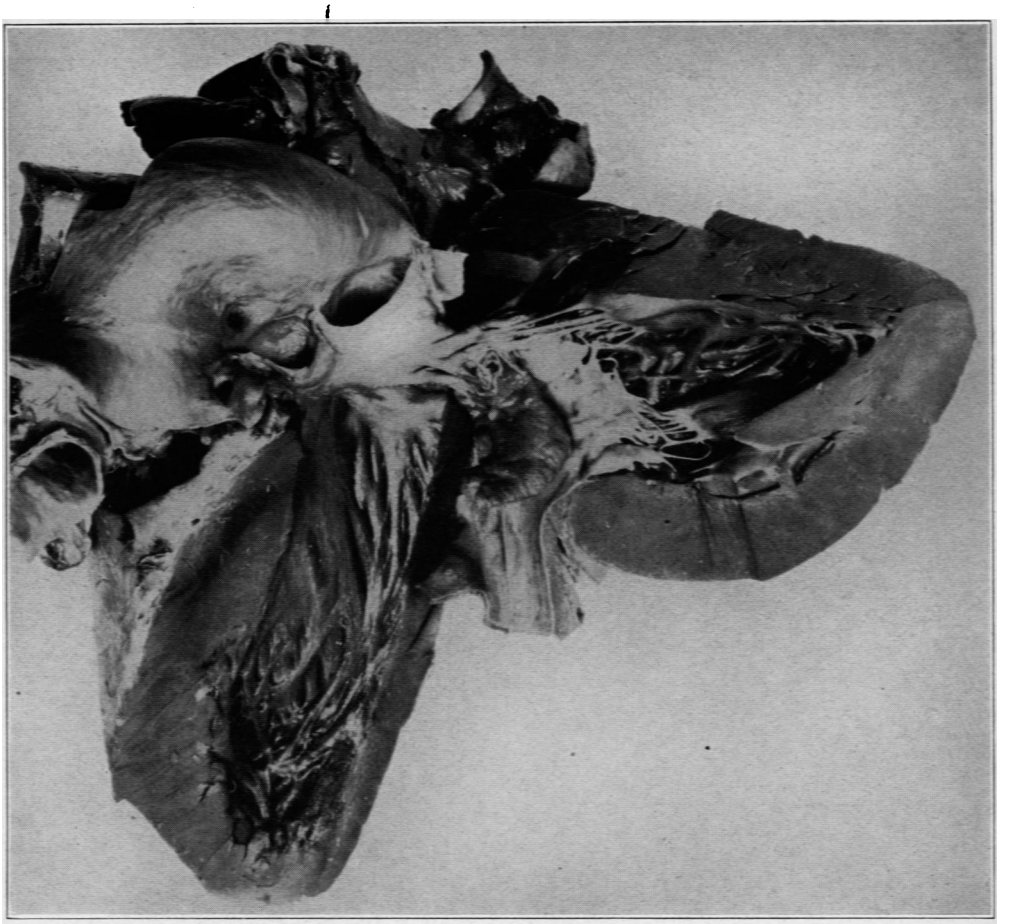
DESCRIPTION OF PLATES

PLATE 32

FIG. 1. Gross photograph of heart.

FIG. 2. Right auricle. Iodin-sulphuric acid test. Reflected light. × 13.



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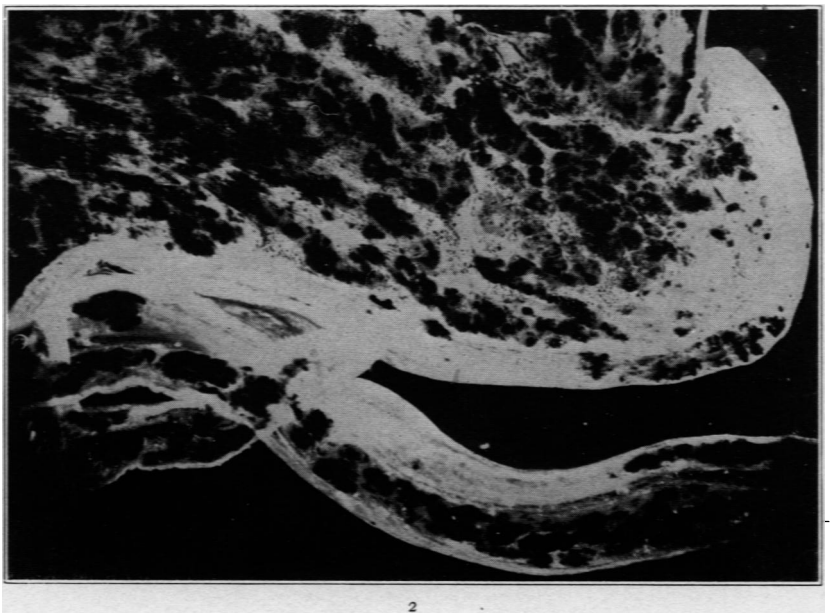

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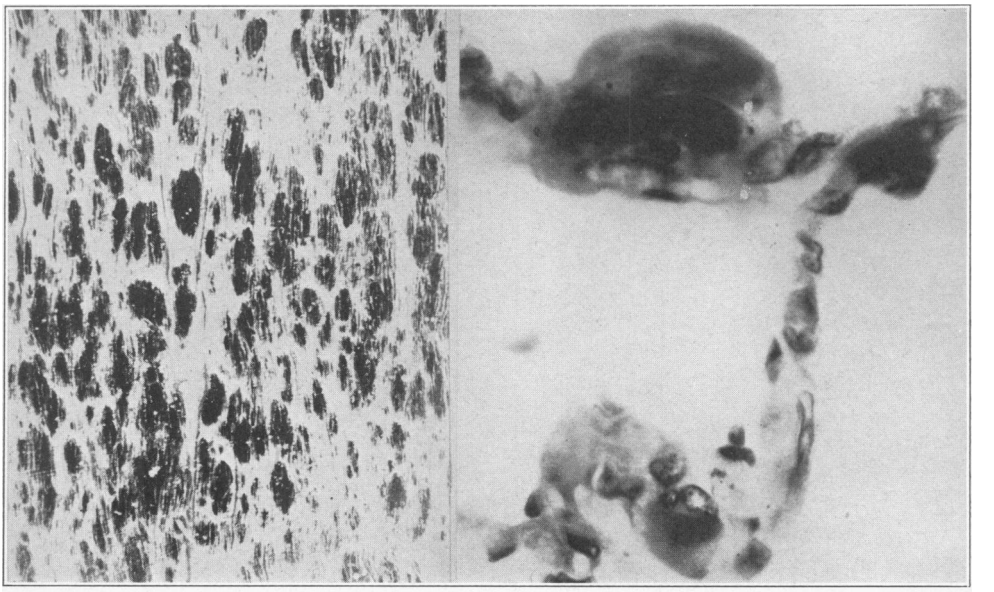
PLATE 33

FIG. 3. Left ventricular myocardium. Iodin-sulphuric acid test. × 13.

FIG. 4. Lung. Pericapillary amyloid in alveolar wall. × 114.

FIG. 5. Left ventricular myocardium. Diffuse amyloid deposit. × 540.



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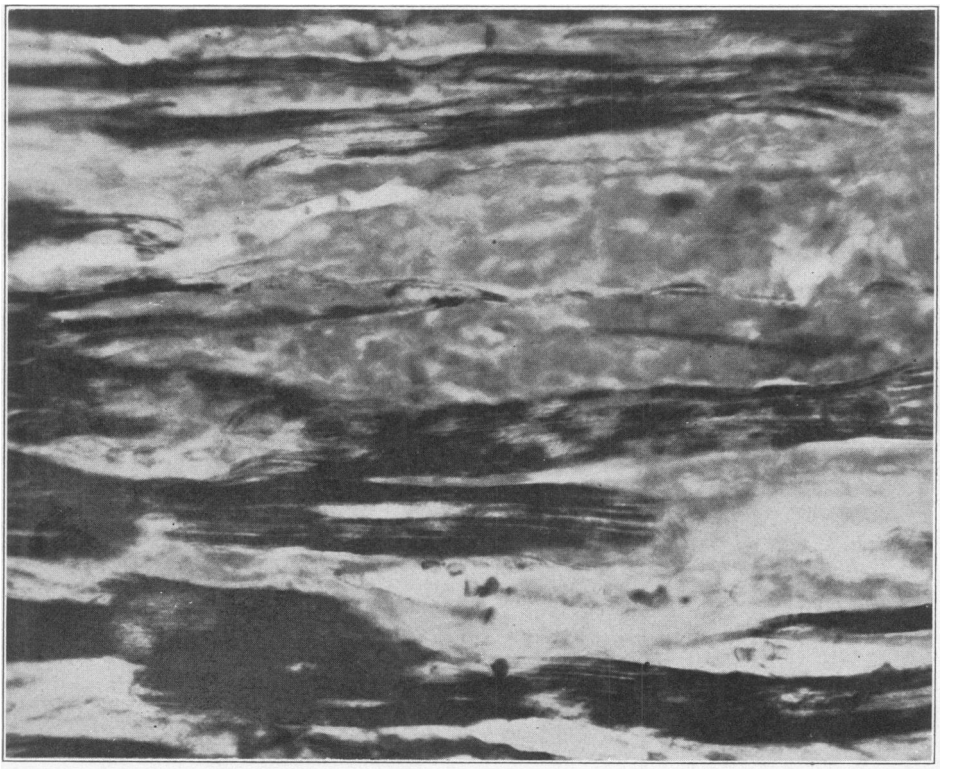

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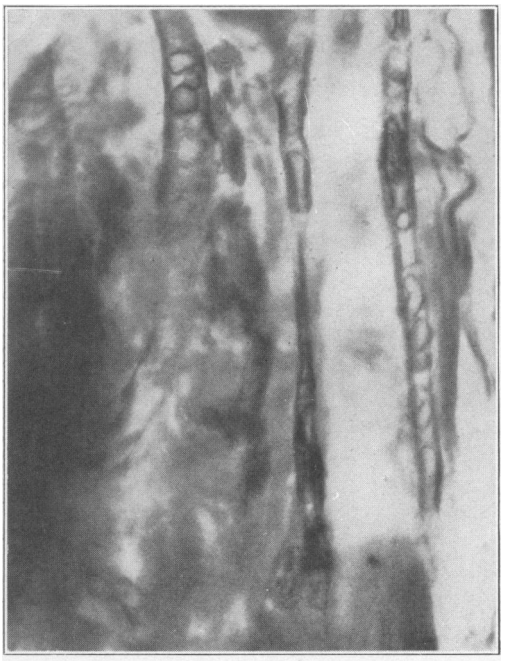
PLATE 34

FIG. 6. Myocardium. Pericapillary deposits of amyloid with partial capillary occlusion in left field. × 1000.

FIG. 7. Myocardium. Abrupt termination of muscle fibers at a point at which capillaries pass deep into tissue.

FIG. 8. Myocardial cell invaginated by amyloid. × 2000.

FIG. 9. Same cell as Fig. 8, in serial section showing penetration of amyloid into sarcoplasm. The mass of amyloid is continuous with that in Fig. 8. × 2000.



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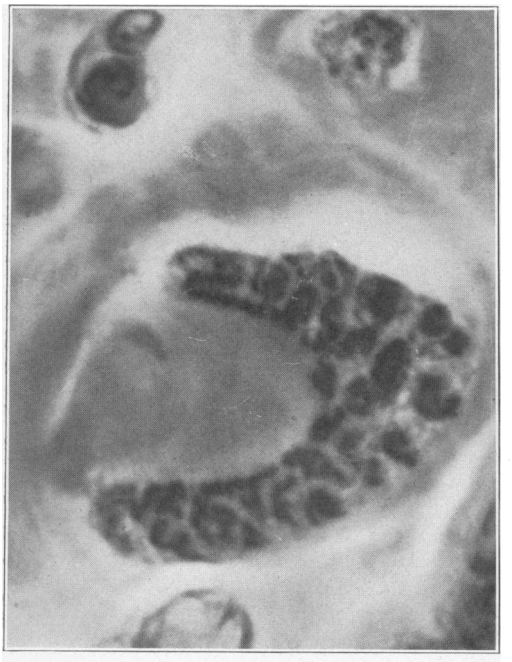

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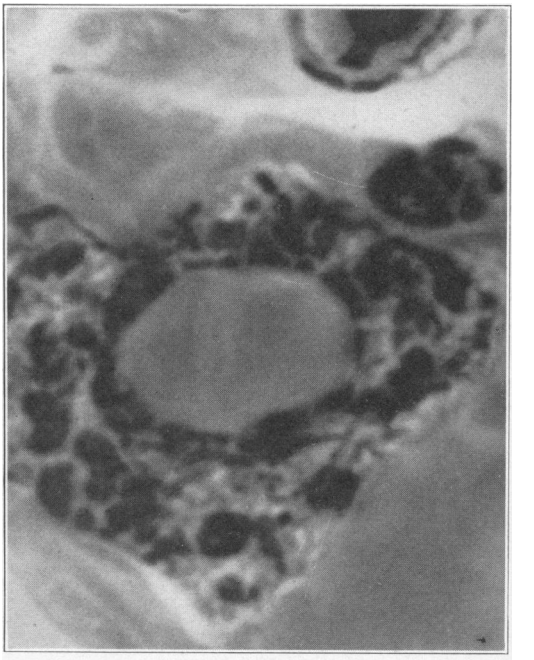

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