A PATHOLOGICAL STUDY OF SYPHILITIC AORTITIS AND ITS SEROLOGY.

By JOHN H. LARKIN, M.D., and I. J. LEVY, M.D.

(From the Strecker Memorial Laboratory of the City Hospital, New York.)

Plates 1 to 7.

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The Wassermann reaction as a diagnostic factor in syphilis holds a unique position in clinical medicine. There are those who believe it an infallible method for proving the presence of syphilis, but there are as many who are skeptical about accepting a serological diagnosis. Neither the serologist nor the clinician can hope definitely to ascertain the value of the complement fixation test in lues. As in all other problems in medicine, it is the work of the pathologist to demonstrate the probable focus of infection in an individual with a positive Wassermann reaction, in whom there is no clinical evidence of syphilis. With this in view it occurred to us to check the serological diagnosis on the autopsy table; that is, to find some definite pathological explanation for this type of reaction in so called latent syphilis. Since syphilis primarily attacks the blood vessels—in fact, it is regarded as a disease the manifold pathological changes of which have their origin in diseased blood vessels—we turned our attention first to the aorta.

Though the pathology of specific aortitis is fairly well understood and its various phases have been accurately described, and though there is a classical method of performing the Wassermann reaction, the problem presents many difficulties. Luetic aortitis, grossly and microscopically, presents many characteristic features, so our task is chiefly to point out what are the primary and early changes of a syphilitic process in the aorta. Since numerous modifications of the original Wassermann method are employed in doing the reaction, it is essential to study and to determine a certain routine serological

procedure, which, with the greatest degree of accuracy, will diagnose the presence or absence of specific changes in the aorta.

We obtained aortas from every case autopsied in the past four years in the Strecker Memorial Laboratory on which a serological examination of the blood had been made before death. Unfortunately, this includes only a small number of cases sectioned. With the aid of serology we have been able to point out definitely that the diagnostic lesion of specific aortitis is a histological one, and in the routine examination of a series of aortas a surprising number reveal this lesion in varying degree, without giving any gross evidence of syphilitic infection.

Gross Appearance.

In most instances in those who have died of aortitis the gross appearance of the aorta presents a typical picture. The lesion is confined to a definite portion of the aorta, usually the ascending and transverse parts, with a sharp line of demarcation between the lesion and the remainder of the aorta, which appears fairly normal (Fig. 3). The involved portion presents either a diffuse dilation or a saccular aneurysmal pouch, and the normal glistening appearance of the intima is lost, being replaced by confluent, pearly white, elevated scar areas, scattered through which are often seen yellowish patches of fatty degeneration (Figs. 1 and 2). To the touch it is hard, thickened, and fibrous. Though this gross appearance is characteristic, it is by no means diagnostic. In our series of seventeen cases eleven died of aortitis, and a little over one-half of these presented this typical picture; the others showed either a diffuse process, the dominating feature of which was an athero-degenerating sclerosis, superimposed upon a syphilitic process, or only slight changes in the intima were appreciable.

Microscopical Changes.

Chiari, in 1903, gave an excellent description of the microscopical changes. He calls attention primarily to the striking appearance of the media, in which one notes small areas of granulation and fibrous tissue, often with central necrotic zones and newly formed vascular elements, surrounded by infiltrating round cells. He also pointed out that in the adventitia the vasa vasorum were

distinctly thickened, and that a perivascular, round cell infiltration was associated with these changes in the adventitia. He regarded the changes in the intima as secondary, and presenting nothing characteristic of a luetic process, but showing various degrees of a simple atherosclerosis. Chiari's description, however, is only typical of an advanced case of luetic aortitis. It is the histological picture seen in the aortas taken from those who have died of the disease. We feel confident, from a study of the material at hand, that an earlier process of luetic aortitis presents quite a different picture. A productive inflammation in the aorta to our mind is pathognomonic of syphilis, and histologically it can easily be differentiated from the simple degenerating process in atherosclerosis. The earlier changes we have seen in aortas from individuals dying of tabes or syphilitic meningomyelitis, and, in several instances, from a non-syphilitic disease. The adventitia, undoubtedly, is the site of predilection, and here one sees the earliest evidence of a productive inflammation. Grouped around the slightly thickened vasa vasorum there is a distinct round cell infiltration. Associated with this perivascular inflammatory reaction the adventitia appears thickened; the fibers are coarser and heavier, and the thickening is directly proportionate to the degree of infiltration. It is important to note in this early stage that the inflammatory process is usually confined to the adventitia, and that the medial coat presents no evidence of a fibrous or lymphoid infiltration. In many instances, however, with a moderate perivascular infiltration in the adventitia the media and intima may be the seat of a marked degenerative process.

From a thorough study of a number of syphilitic aortas it is evident that a perivascular round cell infiltration is a constant feature of the diseased process. The typical cells closely resemble lymphocytes, small round cells staining intensely with a nuclear stain. With these one sees other types,—endothelial and plasma cells—but these are by no means invariably present (Fig. 7). Many have found giant cells in the infiltrations in the media, and a few have called attention to their presence in the adventitia, but in our series we were unable to observe any in our microscopical preparations. Gruber seldom found them and agrees with Thorel, that one may be unable to see giant cells, even in the examination of a large number of cases.

Perivascular Cellular Infiltration as a Diagnostic Feature of Luetic Aortitis.

That this histological lesion in an aorta, namely, perivascular cellular infiltration, has syphilis as its etiological factor, and further, that it is diagnostic of luetic aortitis, we base our opinion upon the following facts.

Eighteen of the forty-two aortas examined presented this picture, and seventeen of these gave a positive Wassermann during life. In other words, 94 per cent of the aortas showing a round cell infiltration gave a positive Wassermann reaction. On the other hand, twenty-four aortas presented no evidence of a perivascular infiltration, and twenty-two of these gave negative, while two gave positive reactions; that is, 91 per cent of these aortas gave negative complement fixations. Gruber working with postmortem serum in a series of 106 cases of syphilitic aortitis obtained identical results, 94.3 per cent positive fixations, and he states that any evidence of a productive inflammation in the aorta with a positive Wassermann gives us at least 0.9 per cent assurance that this type of inflammation is a result of syphilitic infection, even though the dominating feature is one of deforming atherosclerosis.

Such lesions as ruptured aneurysm, and aortitis with pure aortic lesions, undoubtedly syphilitic in origin, histologically present this in a marked degree. In other general metabolic and infectious processes, such as arteriosclerosis, interstitial nephritis, chronic ulcerative tuberculosis, carcinoma, pneumonia, vegetative endocarditis, and pernicious anemia, the aortas showed no such histological picture.

Most authorities, however, do not agree that a perivascular, round cell infiltration in the aorta is characteristic and pathognomonic of luetic infection. Stadler maintains that moderate infiltration around the vasa vasorum may be due to alcohol and to certain infections, and Mönckeberg describes it in a case of recurrent endocarditis with rheumatic joint lesions. Gruber admits that syphilis can cause only a productive inflammation with the typical gross changes in an aorta, but that an inflammatory infiltration may occasionally, though seldom, be caused by other processes, and while histologically he is unable to determine the etiological factor, the serological reaction gives him the diagnosis. Gruber agrees with Faber, that though the dominant feature of atherosclerosis is a degenerative process with calcification, ulceration, and deformity, undoubtedly a moderate inflammatory reaction is occasionally met with in this type of sclerosis. We will admit that fibrous connective tissue and thickened vasa vasorum surrounded with fibroblastic cells are occasionally seen in atherosclerosis, and that this is a chronic productive inflammation,

but that the typical round cell perivascular infiltration, already described, is a luetic process.

That infectious diseases, such as colon bacteremias or streptococcic endocarditis, are a possible cause of lymphoid infiltration in an aorta does not coincide with our experience. In two cases of vegetative endocarditis, and in one of pyonephrosis with colon infection, the aortas showed no inflammatory changes, and the serology was negative. In another, however, with focal necroses (a streptococcic infection) the aorta showed moderate perivascular infiltration, but the serology was positive, and the infection was secondary to an ulcerating gumma of the larynx. Gruber has noted two interesting cases of inflammatory infiltrations around the vasa vasorum in the adventitia of the aorta, secondary to an inflammatory process in the para-aortic tissue. In one case tubercular lymph glands in the mediastinum had ulcerated, producing a marked inflammatory reaction. In the other, perforation of a stenosis of the esophagus through surgical interference, from which the patient died several months later from sepsis, produced a typical productive inflammation in the aorta, which could not be differentiated from a luetic process. Both these cases gave negative Wassermann reactions, and Gruber concludes that they were non-specific. That a lymphoid infiltration and productive inflammation may result as an extension process is undoubtedly true, but that does not alter our opinion that this type of reaction produced in an aorta *per se* is pathognomonic of syphilis.

Treponema pallidum in Luetic Aortitis.

Though serology gives us definite proof that syphilis produces a typical aortitis, as further evidence in support of our view, we have attempted to find *Treponema pallidum* in our preparation. Wright and Richardson, with others, claim to have demonstrated the spirochæta in the aortas of acquired syphilis, but we have been unsuccessful in doing so. We have seen many artefacts in our preparations, which, by less skeptical men, could easily be interpreted as spirochætæ. Both Gruber and Fukushi have also failed to find the spirillum in their large series of cases.

Classification of Cases.

So, basing our diagnosis upon a round cell perivascular infiltration we have in our series eighteen cases showing evidence of syphilitic aortitis. We have divided this series into three classes: first, those showing this in a marked degree; second, those cases in which the infiltration is moderately present; and third, those in which there is only slight evidence of a cellular infiltration (Figs. 4, 5, and 6). In Class 1 there are eleven cases. Over one-half of the aortas presented the gross picture of specific aortitis; the rest showed a diffuse atheromatous process, with areas of ulceration. All these cases died of aortitis; three with ruptured aneurysm, the others with symptoms of cardiac decompensation. Five of the cases had marked aortic valve involvement, with normal mitral valves, or slightly thickened mitral cusps. One case had both valves involved. There was positive serology in all cases.

In Class 2, a series of five cases, none presented the gross typical picture. Three of the aortas were markedly atheromatosed, with areas of calcification and ulceration. The two others were fairly normal, with the exception of longitudinal striations and thickened, raised, whitish areas around the intercostal orifices. Histologically, however, the perivascular infiltration was distinctive and definite. In only one case was the aortic valve diseased. None of these cases died of their aortitis. One died with symptoms of cardiac decompensation, probably from localized fibrous infiltration of the wall of the left ventricle, involving the area of the bundle of His, with aneurysmal dilatation at this point. The others died of the following conditions: syphilitic meningomyelitis, carcinoma of the right bronchus, chronic ulcerative tuberculosis, and terminal bronchopneumonia with tabes. The serology was positive in all but one case.

In the third class, consisting of only two cases, characterized by a very moderate or slight infiltration around the vasa vasorum the aortas appeared practically normal, with the exception of patches of smooth, elevated areas around the intercostal orifices. One case died of carcinoma of the bronchus, the other of an ulcerating gumma of the larynx, with focal necroses of kidney and liver. Both cases had a positive serology.

Complications.

The most common complication of syphilitic aortitis is an insufficient aortic valve. In a study of forty-two cases with various types of aortas the aortic valve in fifteen showed evidence of disease. In seven of these fifteen valvular lesions the mitral valve was normal, and the aortas of these seven cases, in which only the aortic valves were involved, gave marked evidence of luetic aortitis, and the serology was positive, with the exception of one. In this case the aortic valve was practically destroyed with a vegetative ulceration; the serum was negative and the aorta normal. Of the remaining eight cases both valves were diseased; six consisting of fibrous changes, and two having acute vegetations. In only one aorta with thickened fibrous changes in both valves was there evidence of luetic aortitis, and in this case also the serum was positive. The seven other cases showed no productive inflammation in the aorta, and the complement fixation tests were negative. From this it seems evident that pure aortic insufficiency, with the exception of infectious endocarditis, is undoubtedly of syphilitic origin. With both valves involved the probability of a syphilitic infection of the aorta is small; an atheromatous or an infectious process should suggest the probable origin.

Luetic aortic insufficiency is, in most cases, probably a late process. In our seven cases of syphilitic involvement of the aortic valve, six were a complication of those specimens which showed marked histological changes in the aorta (Class 1) and all of these died of the vascular lesion. In other words, six out of ten specimens of late aortitis, or 60 per cent of the cases of advanced aortitis, had the aortic valve involved. On the other hand, of the seven cases of early luetic changes the aortic valve was part of the process only once, giving in our whole series the complication in about 40 per cent of the cases. This practically agrees with Stadler's findings, in which he states that while only one-third of his luetic aortas were complicated with a diseased valve, two-thirds of his cases dying of aortitis had insufficient aortic valves.

Ruptured aneurysm is a serious, though not so frequent a complication. Of course it is met with only in advanced cases. In our series of eleven dying with aortitis, three succumbed to a rupture of

an aneurysm. In two the rupture was in the thoracic cavity. In one the aneurysm was abdominal and had eroded the vertebra almost to the cord.

Cerebral luetic endarteritis is a complication of syphilitic aortitis as often as is aortitis. We can only consider cerebral endarteritis a complication of the aortitis when there are symptoms of a brain lesion, and evidences of brain softening in an individual dying of aortitis. Conversely, in individuals dying of their brain lesions the endarteritis is complicated with an aortitis, when there are either clinical or pathological evidences of aortic disease present. In our eleven cases of fatal aortitis two individuals had evidences of paralysis before death, and in these cases areas of brain softening were demonstrated. In seven cases of early aortitis only one died of cerebral softening.

Tabes complicates specific aortitis less often than it is complicated by the vascular lesion. In over ten fatal cases none gave during life any clinical evidence of tabes. It is of interest to note here, however, that the histological examination of the cord in one case revealed an early sclerosis of the posterior root fibers, the posterior tract of the cord not being affected. In two fatal cases of tabes diffuse atheroma with calcification and ulceration were present in both aortas. In one, however, there was a moderate perivascular infiltration, and the serum in this case was positive; the other gave no evidence of syphilis in the aorta or in the serum. Stadler found clinically in 248 cases of aortic disease that 6.2 per cent of these had tabes, and he further states that in his autopsy findings almost all cases of tabes showed syphilitic aortitis.

Aortitis is a frequent complication of general paresis. Gruber and Straub at autopsy found that 71 and 82 per cent, respectively, of their cases of general paresis showed evidence of luetic aortitis.

Stadler found narrowing of the lumen of the mouth and sclerosis of the coronaries in one-third of his autopsies on aortitis. This is undoubtedly true, but it is difficult to differentiate the dominating factor producing this change, whether an atherosclerosis or a productive inflammation. It is questionable, as the older writers have suggested, whether the earliest changes in syphilis of the aorta are seen around the orifice of its branches, producing a narrowed lumen.

Only occasionally have we noticed in advanced aortitis anything approaching a definite stenosis of these orifices.

In over two-thirds of our cases there was no cardiac enlargement (Figs. 1 and 2). This agrees with Grau's findings, but Gruber in a majority of his specimens reports a distinct hypertrophy of the heart. Some have stated that cardiac hypertrophy is associated with the aortic insufficiency, but in some of our most marked cases of aortic disease we have found surprisingly small hearts. It seems, however, that cardiac hypertrophy is not associated with luetic aortitis or any of its complications, but is a result probably of some type of nephritis.

Serology.

To discuss technically the serology of syphilitic aortitis is not the scope of this paper, but rather to point out that in our hands, at least, a definite serological procedure is preferable to either the original method, or several popular modifications, in diagnosing syphilis of the aorta, and that a high percentage of positive reactions in individuals who give no clinical evidence of syphilis of the vascular system can be explained by luetic aortitis.

Wassermann Reaction as a Diagnostic Factor in Luetic Aortitis.

As stated, forty-two aortas have been examined and the sera of these cases tested serologically before death. Nineteen of these gave positive Wassermann tests; twenty-three were negative. Of the nineteen positive cases, seventeen aortas gave microscopical evidence of luetic aortitis; namely, 90 per cent of our positive reactions (those sera fixing complement according to the method above described) have, at least, a definite pathological explanation based on changes in the aorta which are pathognomonic of syphilis. It is interesting to note that fifteen of the nineteen cases died as a result of a syphilitic process (about 80 per cent), while eleven of the nineteen died from their luetic aortitis (about 60 per cent). The four cases dying from syphilis other than aortitis were one case of syphilitic cerebral endarteritis, one of tabes, one of meningomyelitis, and one ulcerating gumma of the larynx. Of the four cases with positive Wassermanns dying of non-syphilitic conditions but showing perivascular infiltra-

tion in the aorta, two died of carcinoma of the bronchus, one of chronic ulcerative tuberculosis, and one of interstitial nephritis.

Though the cases studied are too few to make any definite deduction, they are suggestive. It is highly probable that about 90 per cent of individuals dying with a positive Wassermann, if the infection is not of recent origin, have, at least from the histological point of view, luetic aortitis, that about 60 per cent of them die from their aortitis, giving clinical evidence of cardiac decompensation or rupture of an aneurysm, and that about 80 per cent of these individuals die of syphilis. Since only one case of perivascular cellular infiltration gave a negative Wassermann test in our series, it is fair to assume that 94 per cent of individuals suffering from luetic aortitis would give a positive reaction in their serum.

That our results correspond with the data in the literature is shown by the recent works of Stadler and Gruber. Gruber obtained identical serological reactions in his series of luetic aortitis; namely, 94.3 per cent positive Wassermanns on postmortem serum. Basing his results on the pathological findings, Stadler states that 82 per cent of 256 cases of syphilis had aortitis, and so considering a positive fixation as diagnostic of syphilis we find, as stated, that 90 per cent of syphilitics have evidence of aortitis. Our higher figure could easily be accounted for by considering (as we did) every aorta with perivascular infiltration as specific. Though only 46 per cent of Stadler's cases died of their vascular lesion, in our series we found that the aortic lesion accounted for deaths in 60 per cent.

The serology of aortic insufficiency is confusing. Serologists obtain positive reactions ranging anywhere from 50 to 80 per cent of the cases. The reason for this large discrepancy is due to the fact that the clinicians have failed to differentiate between the various types of this lesion. If one remembers that only pure aortic insufficiency, with the exception of an infectious endocarditis, is always syphilitic, and that the serology is positive in probably 100 per cent of the cases, and that the aortic lesion associated with a mitral incompetent valve is usually an atherosclerotic process, and only occasionally specific, one can readily understand the disagreement in the serological results.

Wassermann Reaction in Treated Cases.

The effect of treatment on the serology of luetic aortitis should receive passing notice. All the cases with varying amounts of treatment, including salvarsan and mercury, with the exception of four, remained positive throughout. The serology was modified in four cases, as follows: No. 292 was admitted with a positive Wassermann reaction; salvarsan was given, followed by several doses of mercury, and following this the Wassermann was negative. No. 678 was admitted with a weakly positive reaction; that is, 50 per cent hemolysis on the guinea pig antigen. Before admission the patient had received several doses of mercury. Without treatment at the end of a month the serology was strongly positive. Five salvarsan injections were given, and a month later the Wassermann test was still positive. A year later, however, just before death, the serology was reported doubtful; since, though there was complete inhibition of all antigens at the first reading, at the end of twelve hours there was complete hemolysis, even with cholesterinized antigen. No. 694 was admitted with serology noted as weakly positive, 50 per cent hemolysis on guinea pig heart antigen. Mercury had been given before admission. Eight months later (three months after the salvarsan and mercury treatment) the serum was reported positive. No. 691, which was admitted with a positive reaction after one salvarsan and several mercury injections, was reported as negative.

A careful review of these cases notes the importance in serologically diagnosing luetic aortitis of repeating the reaction on sera, especially if any form of antispecific treatment has been administered. Though in 75 per cent of the cases treatment did not alter the serological reaction in the blood, in 25 per cent it either weakened the reaction or produced a negative fixation.

Technique and Antigens.

In this series a positive reaction means complete inhibition of hemolysis when the controls have hemolyzed, and at the end of twelve hours. The antigen employed is an alcoholic extract of guinea pig heart. 0.1 cc. of complement and at least two units of amboceptor are used in the hemolytic system. Many of the sera were

done with other antigens; namely, alcoholic extract of syphilitic liver, acetone partition of calf's heart, cholesterinized alcoholic extract of human heart and guinea pig heart. But the findings with these various antigens were not as constant and as consistent as with the first named extract. We do not mean to infer that these extracts are not to be used in the Wassermann reaction, but rather that in the serological diagnosis of syphilitic aortitis, the alcoholic extract of guinea pig heart should receive first consideration.

The antigens made from syphilitic fetal livers were unreliable with us, while acetone partition antigens made according to Noguchi's method, though giving fairly good results, were influenced earlier by treatment and the completed extracts seldom were of uniform strength. These antigens have been discarded by us in reporting the routine Wassermann reaction in the laboratory. It is important to note, however, that Gruber working with fetal syphilitic liver antigens obtained results identical with ours. Our experience with the liver antigen (that it is so unreliable) is not surprising, since, though we attempted to obtain congenital luetic organs, in many instances we were not assured by spirochæta demonstration that our material was from a syphilitic infant. The highly sensitized cholesterin antigens we still employ with the assurance that complete complement fixation with this antigen probably does not always indicate the presence of syphilis. On the other hand, however, many other types of syphilis, such as tabes, give only positive reactions with this antigen.

Though 90 per cent of our positive reactions with guinea pig heart antigen showed evidence of syphilis in the aorta, only 77 per cent of the positive results with cholesterin could be explained by similar luetic changes.

Finally, we hope that these facts will be of assistance to the clinician in diagnosing syphilis of the aorta, and further, that they will emphasize the importance of a positive Wassermann reaction, and make it incumbent upon all physicians to institute treatment as a prophylactic measure in such cases.

Syphilis is an endemic infectious disease which ranks with tuberculosis and carcinoma as a menace to the community, and which, when untreated, probably kills 80 per cent of its victims and produces an aortitis in about 80 to 90 per cent, of whom 50 or 60 per cent die of the vascular lesion.

SUMMARY.

1. Syphilitic aortitis is a productive inflammatory process, the earliest and most constant feature of which is a perivascular round cell infiltration in the adventitia.

2. The typical gross picture of luetic aortitis is often obscured by a superimposed, diffuse atherosclerosis. In the early cases the aorta appears fairly normal, presenting only the characteristic histological changes.

3. A pure aortic insufficient valve, with the exception of an infectious endocarditis, is always luetic.

4. Cardiac hypertrophy is not a complication of luetic aortitis. When present it is usually associated with a nephritis.

5. The demonstration of *Spirochæta pallida*, even in advanced specimens of syphilitic aortas, is doubtful.

6. An antigen prepared from alcoholic extract of guinea pig heart with the original Wassermann technique should be preferred in diagnosing luetic aortitis.

7. Positive complement fixations in patients suffering with syphilis for a period of about fifteen years or longer suggest the probability, at least, of histological luetic changes in the aorta in 80 to 90 per cent of the cases. 60 per cent of these die from aortitis.

8. About 94 per cent of patients suffering with aortitis give positive Wassermann reactions.

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EXPLANATION OF PLATES.

PLATE 1.

FIG. 1. Note the small heart with concentric hypertrophy. The aortic valves are moderately thickened. The walls of the aorta are markedly thickened. There is a diffuse dilatation of the ascending and transverse portions of the aorta, and a marked fibrous appearance of its inner wall.

PLATE 2.

FIG. 2. The same as Fig. 1, but showing aneurysmal formation and involvement of the aortic valves. In both Fig. 1 and Fig. 2 the mitral valves were normal.

PLATE 3.

FIG. 3. Portion of a syphilitic aorta showing a definite line of demarcation between involved and normal parts at the junction of the lower third.

PLATE 4.

FIG. 4. Microscopical picture of a section of the aorta of the case shown in Fig. 1. Note the perivascular infiltration of the vasa vasorum, the thickened adventitia, the newly formed vascular elements in the media, with its round cell infiltration, also the thickened intima,—a secondary process.

PLATE 5.

FIG. 5. The same as Fig. 4, but not so advanced.

PLATE 6.

FIG. 6. An early process with a beginning atherosclerosis.

PLATE 7.

FIG. 7. A high power picture showing the type of cells. Note the great number of lymphocytic cells and several larger plasma cells.

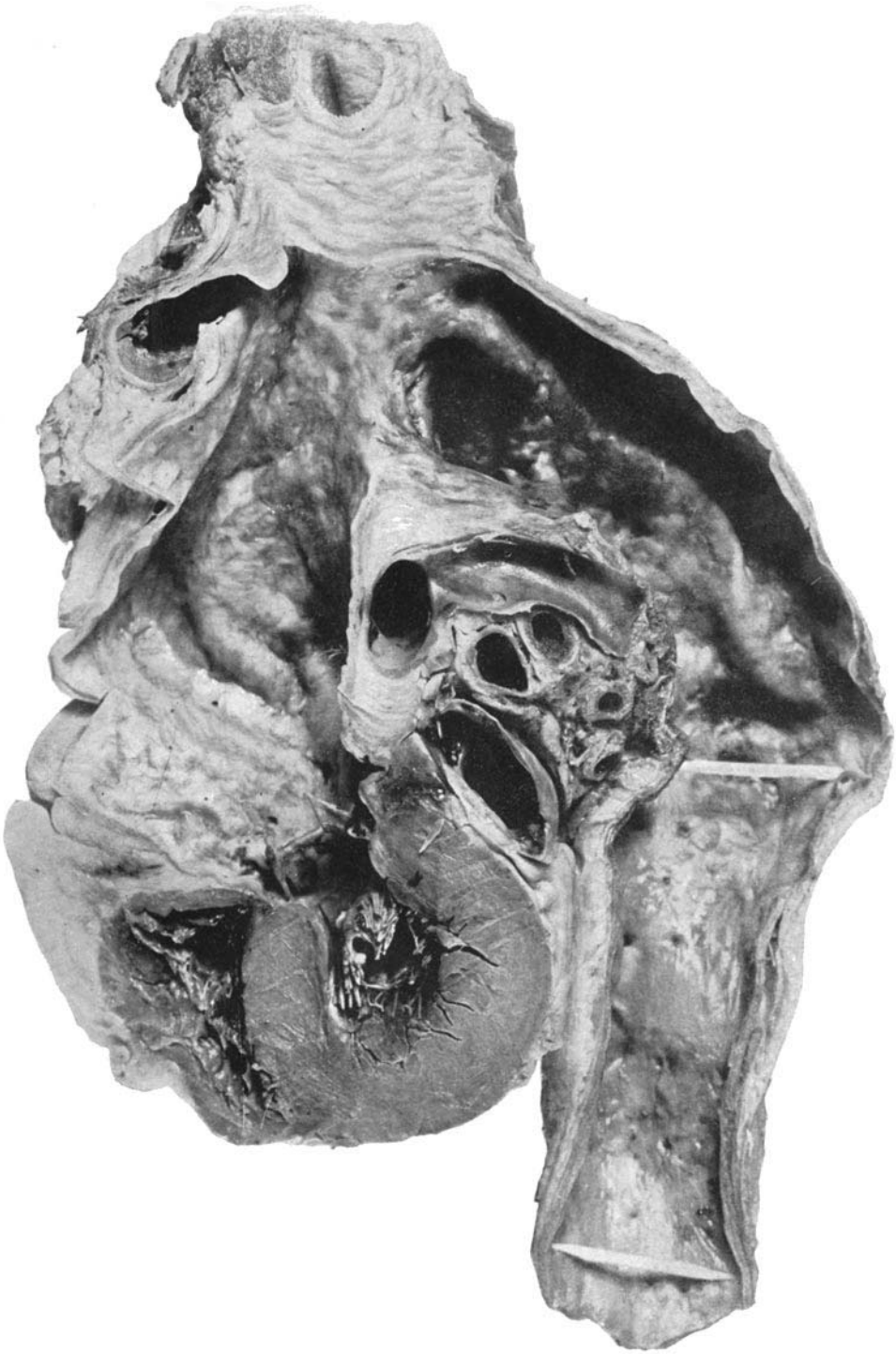

FIG. 1.

(Larkin and Levy: Syphilitic Aortitis.)

FIG. 2.

(Larkin and Levy: Syphilitic Aortitis.)

FIG. 3

(Larkin and Levy: Syphilitic Aortitis.)

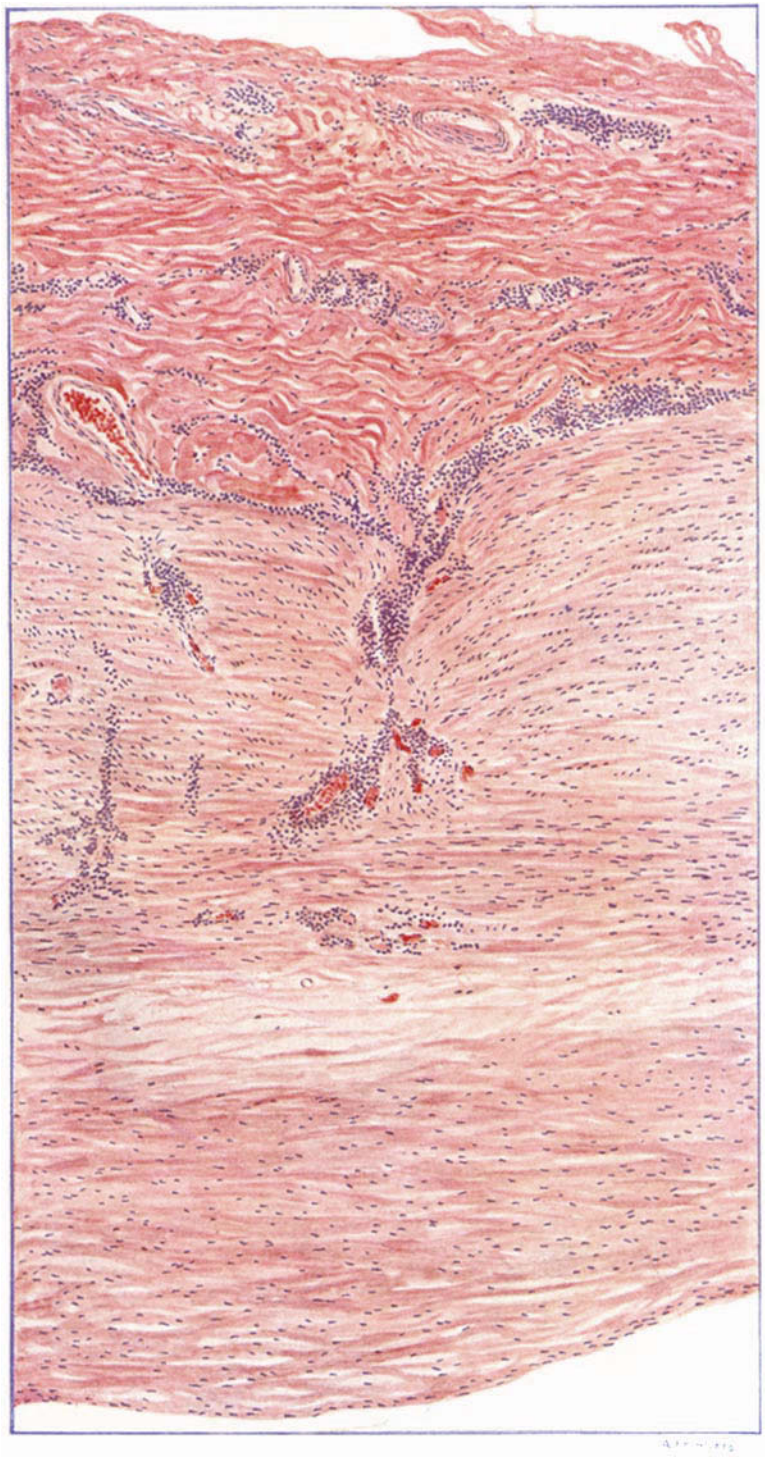

FIG. 4.

(Larkin and Levy: Syphilitic Aortitis.)

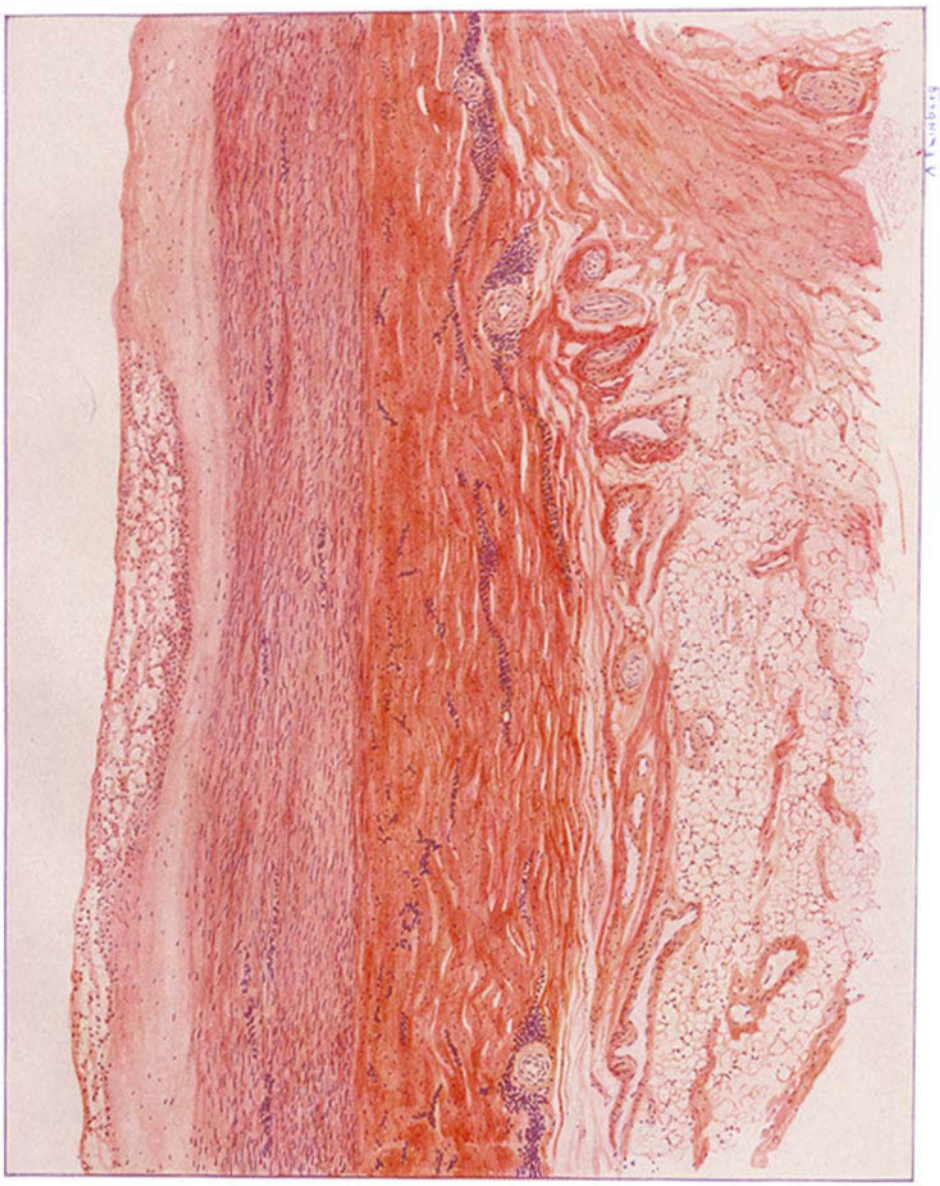

FIG. 5.

(Larkin and Levy: Syphilitic Aortitis.)

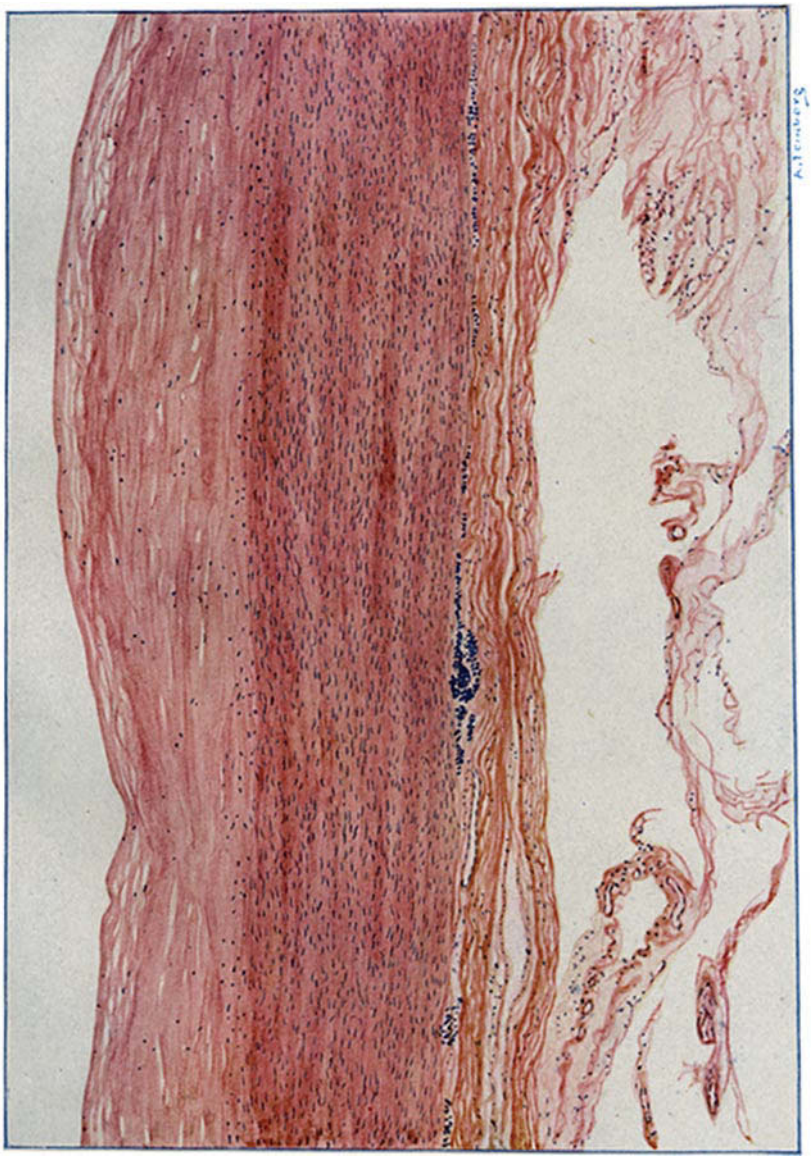

FIG. 6.

(Larkin and Levy: Syphilitic Aortitis.)

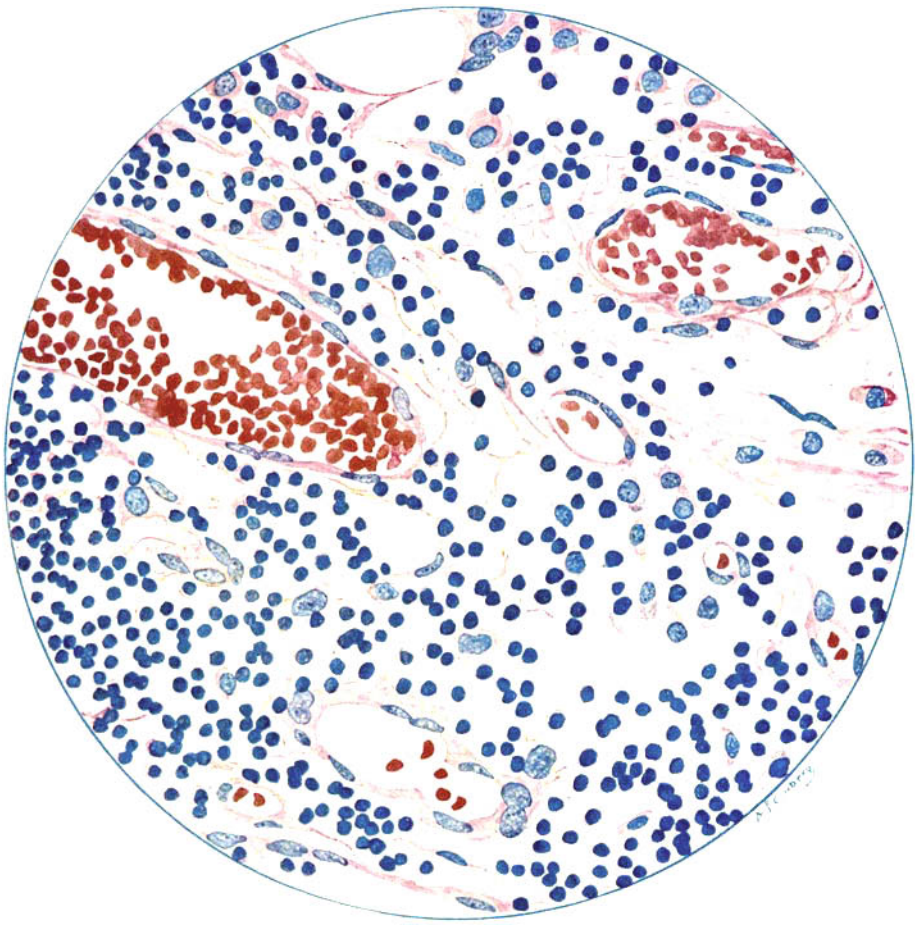

FIG. 7.

(Larkin and Levy: Syphilitic Aortitis.)