THE AMERICAN JOURNAL OF PATHOLOGY

VOLUME XI SEPTEMBER, 1935 NUMBER 5

LESIONS OF THE LEFT AURICLE IN RHEUMATIC FEVER *

LOUIS GROSS, M.D.

(From the Laboratories of the Mount Sinai Hospital, New York City)

The influence of the nature and topography of tissues on the character of the resultant inflammatory process is better exemplified by rheumatic fever than by most other chronic diseases. Thus, for example, characteristic individual differences are found in the rheumatic lesions of the myocardium, valve rings, valve substance, pericardium, subcutaneous tissues, tendinous insertions and blood vessels, even though some of these may possess structural or cellular peculiarities in common. This relation between inflammatory process and tissue structure is strikingly shown in the left auricular endocardial lesions.

Rather vague and sketchy references to left auricular lesions were made by Corvisart,[1] Burns,[2] Baillie,[3] Wells,[4] Laennec,[5] Hope [6] and Bouillaud.[7] From these it is often difficult to determine whether the underlying cause was rheumatic fever, bacterial endocarditis or auricular thrombosis.

In 1898 Claude and Levaditi [8] gave a gross and microscopic description of what appears to have been an old rheumatic, left auricular endocardial lesion with calcification. They noted distended capillaries, inflammatory cells and obliterative vascular changes near the ulcerated (calcific ?) surface. Bacteriological cultures were negative. The case described by Cheadle and Lees [9] in the same year was undoubtedly one of bacterial endocarditis. This suggestion was made by Poynton, who performed the autopsy.

Huchard's [10] monograph (1903) contains two excellent drawings of

* Aided by grants from the Lucius N. Littauer and Walter W. Naumburg Funds.
Received for publication May 12, 1935.

typical rheumatic auricular lesions illustrating some of the gross and microscopic features of this process. The text description, however, is very vague. In 1914 Harper [11] reported 9 cases of rheumatic infection in childhood. In 1 case the "endocardium of the left auricle was found to be thickened and wavy. This was considered evidence of a former endocarditis." In 1920 Hertel [12] reported 10 cases of "parietal endocarditis." One of these was a "recurrent endocarditis of the mitral valve." Between the anterior and posterior cusps she observed large deposits, partly warty, partly polypoid, hanging in the heart cavity. These vegetations extended up the left auricular endocardium. An excellent histological description is given of these lesions which Hertel believed were a contiguity process from the mitral valve. However, although the description of this microscopic auricular lesion closely resembles in certain respects the true rheumatic process, the character of the gross lesions and some of the microscopic features suggest the possibility of at least a superimposed bacterial process, in spite of the reported negative bacteriological findings.

The study of this very interesting lesion took on new significance after the publication of the reports by MacCallum (1924–1925).[13] It is to the great credit of this author that he clearly recognized the essentially rheumatic nature of the left auricular process, gave an excellent description of its gross and microscopic features and, because of his extensive experience in the pathology of rheumatic fever, presented his findings on material which can hardly be subject to question. The most important points brought out by MacCallum were the infiltration of the endocardium and subendocardium with inflammatory cells, the banded appearance of the Aschoff bodies in the endocardium, the presence of necrotic bands of collagen, the edema, the distortion of the elastic lamellae and the extension of the process through the auricular myocardium to the pericardial mantle.

Soon after the publication of the first of these papers, Stewart and Branch [14] reported a case of rheumatic auricular endocarditis with calcification. The endocardium of the left auricle showed fibrous thickening with irregularity of the surface. The presence of adhesive pericarditis and myocardial Aschoff bodies, together with the clinical history, led the authors to conclude that the auricular lesion was of a rheumatic nature.

In 1926 VonGlahn [15] added materially to our knowledge of this lesion by a description of the auricular changes found in 9 out of 31 cases of rheumatic valvular disease. While no attempt was made to segregate the cases with respect to acuity or chronicity of the rheumatic process in the entire heart, a very careful and detailed description was given of the gross lesions and microscopic findings. In this paper VonGlahn was able to confirm MacCallum's findings and noted in addition the presence of large and small mononuclear cells, as well as polymorphonuclear leukocytes, oriented at right angles to the endocardial wall. He observed fibrin on the surface of some of these lesions and, at times, verrucous deposits. Considerable quantities of fibrin were present in the subendocardium. Capillaries were seen penetrating the endocardial lesion but never beyond the middle portion. Healing took place by means of cells resembling fibroblasts which arranged themselves perpendicularly to the surface in the superficial part of the endocardium, especially between the main connective tissue layer and the endothelium. Later on, elastic fringes and often calcium deposits were seen in these healing and healed areas. Further healing took place by the disappearance of the cellular accumulations.

These descriptions were subsequently amplified by Pappenheimer and VonGlahn,[16] and by VonGlahn and Pappenheimer.[17] In the latter report reference is made to the finding of 3 cases with lesions in the right auricle. Since the publication of these papers several new cases have been described by Shaw,[18] Perla and Deutch,[19] and by Klinge,[20] largely confirming the previously reported observations.

On reviewing the findings reported by the above mentioned authors, it appeared desirable to investigate these lesions on a much larger series of material, laying especial emphasis on the correlation between the gross and histological findings in the left auricle and the clinical course of the disease. This report will concern itself with a study of 87 rheumatic cases. Sixty-seven of these were active and showed Aschoff bodies in the myocardium, and 20 showed chronic valvular disease of the rheumatic variety but without evidence of activity either clinically or pathologically and with no demonstrable Aschoff bodies in the myocardium. The grouping as to activity and inactivity was based on the criteria outlined by Rothschild, Kugel and Gross.[21] Particular care was taken to avoid material which in any way indicated the possibility of a coexisting bacterial endocar-

ditis. A careful study of the clinical records and pathological specimens made it possible to divide the material into the following groups:

GROUP 1. Active cases where death took place in the first attack or where one preceding attack occurred within 1 year of the fatal outcome.

GROUP 2. Active cases in which one previous attack occurred at least 2 years previous to the fatal outcome.

GROUP 3. Active cases with repeated attacks, death occurring during an acute recurrence.

GROUP 4. Active cases where death was caused by decompensation without clinical evidence of a final recurrence. Some of these cases had no previous history of rheumatic fever.

GROUP 5. Inactive cases of chronic valvular disease of the typical rheumatic variety.

The technical methods used were essentially the same as those previously described by Gross and Ehrlich.[22] The findings here reported are based on a study of the routine left auricle sections (L.A.) and the auricular portions of the routine mitral valve posterior sections (M.V.P.) obtained in the standardized procedure suggested by Gross, Antopol and Sacks.[23]

Before describing the findings in this material it is advisable to present very briefly the histological characteristics of the normal left auricular endocardium, with special reference to the age period changes. It will be found that among the changes produced by rheumatic fever in the left auricular endocardium there occur alterations in the relations of the connective tissue, smooth muscle and elastic tissue components. Since alterations also occur as the result of age, it is necessary to have a clear-cut conception of these processes in order to differentiate the changes that are of normal evolutionary origin from those that may be due to the rheumatic process. This is particularly true for the findings in the inactive cases.

Descriptions of the histology of the left auricular endocardium have been reported by Königer,[24] Nagayo,[25] Miller and Perkins,[26] and by Perkins and Miller.[27] The descriptions by Königer and Nagayo appear to be unnecessarily complex and too rigid to fit the not inconsiderable variations found in the normal, human, left auricular endocardium. In the reports by Miller and Perkins, and Perkins and Miller, the age period changes in the left auricular endocardium

are described in a 2 day old infant, a 35 year old individual and an 85 year old individual.

In order to obtain a more plastic impression of the age period changes in a fairly representative number of cases, 50 normal hearts were chosen for study. These specimens were carefully selected to rule out such conditions as may be expected to affect the cardiovascular system. An examination of this material reveals the following histological and topographical features of the left auricular endocardium and their changes from birth to the eighth decade of life.

Histology and Age Period Changes of the Left Auricular Endocardium

The fibro-musculo-elastic membrane lying internally to the left auricular myocardium may be divided into two layers, *i.e.* the endocardium proper and the subendocardium (Fig. 1). The former consists at birth of somewhat closely packed, anastomosing delicate sheets of elastic tissue separated by collagen fibrils. The elastic membranes lie parallel to one another and to the lumen of the auricle, running for the most part transversely to the axis of the blood stream flow. Dividing this endocardial layer arbitrarily into three equal zones, *viz.* inner third, middle third and outer third, it may be said that there are no conspicuous concentrations of the elastic tissue in any of the zones but that the outermost one occasionally contains a few smooth muscle fibers with their axis generally parallel to the elastic lamellae. Apart from these, the endocardium shows a large number of fibroblasts with fairly abundant cytoplasm and somewhat vesicular rounded nuclei. The cells are embedded in a mucinoid, more or less basophilic matrix. Within a few months after birth the cytoplasm disappears and the nuclei become denser and somewhat elongated. Scattered, large mononuclear cells, occasional lymphocytes and rare polymorphonuclear leukocytes may also be seen. These cells occur rather infrequently until about the fourth decade of life when they practically disappear from the normal endocardium. The number of fibroblastic nuclei appears to fall off sharply within the first year of life. They generally occupy the middle and outer thirds of the endocardium. From the third decade on they become very sparse. No blood vessels are seen in the normal endocardium.

The endocardium is covered by flat endothelial cells. Between these and the outermost elastic lamellae there are found a few scattered cells apparently derived from the primitive mesenchymal layer which, because of their multipotential properties under certain inflammatory conditions, will be referred to as the "mesenchymal layer." It is to be noted that this layer is most inconspicuous in the normal left auricle.

Immediately external to the endocardium proper, and intimately bound up to it, lies the subendocardial layer. This consists of larger and denser collagenous masses separated by interlacing elastic fibers which are thicker than those of the endocardium proper and generally run at right angles to the latter. The outermost zone of this subendocardial layer may at times take on a looser structure and merge with the collagenous framework of the adjacent auricular myocardium. A few scattered capillaries and very rare mononuclear cells are normally present in the subendocardium. Lymphocytes and polymorphonuclear leukocytes are practically never seen. The capillaries are often more conspicuous when fat tissue is present.

During the second year of life the regularity of the endocardial pattern may be somewhat disturbed. The elastic fibers may show focal concentrations or, in places, be missing. Thus, the beginnings of a mosaic pattern are already noted. This becomes more frequent during the second half of the first decade. About the end of the first decade an occasional specimen shows a somewhat greater concentration of smooth muscle fibers in the outermost endocardial zone. These run occasionally obliquely, but for the most part, transversely. At times the innermost endocardial layers may be scant in elastic fibers and present an appearance somewhat resembling the inflammatory structure which will be referred to as an "endocardial reduplication." These are, however, generally easily distinguishable from true reduplications during the first four decades of life, after which they occur with considerably greater frequency and can seldom be distinguished from the type of reduplication often found in chronic valvular disease of long duration.

From the second decade on, occasional specimens show a less distinct differentiation between the endocardium and subendocardium. During the third decade the subendocardium begins to show fat accumulations. As indicated before, these frequently contain a larger number of capillaries. The smooth muscle fibers, which gen-

erally occupy the outer zone of the endocardium proper and occur in more or less compact masses, become decidedly more conspicuous in the fourth decade and may produce irregularities in the distribution of the elastic lamellae, with the formation of elastic-muscular mosaics.

In the sixth decade of life a new pattern appears. The superficial layers of the elastic tissue may be either concentrated or quite defective. The inner zone of the endocardium generally shows atrophy of the elastic fibers with, at times, disappearance. The middle zone often shows dense concentrations of elastic fibers. The outer zone frequently contains conspicuous smooth muscle bundles, at times intermingled with dense elastic concentrations. The subendocardium frequently contains considerable fat tissue.

In the seventh decade the elastic fibers become extremely scant, being concentrated in irregular collections; the smooth muscle components are quite conspicuous and endocardial reduplications internal to the innermost elastic lamellae are frequently encountered. These reduplications are generally narrow, not elastified, of more or less even thickness and rest on a fairly heavy, elastic limiting membrane. The eighth decade frequently shows distinct atrophy of the endocardium with, generally, hypertrophy of the smooth muscle elements.

Macroscopic Appearance of Rheumatic Auricular Endocarditis

The macroscopic appearance of the rheumatic auricular lesions has been described in detail by VonGlahn.[15] The chief points brought out by this author are the predilection site of the posterior wall of the left auricle, at times spreading over almost the entire endocardial surface into the auricular appendage and up to the orifices of the pulmonary veins; the rare involvement of the foramen ovale; the appearance of irregular low ridges and hillocks, separated by furrows with no definite pattern; the smooth, glistening, at times slightly dull, irregular surface with, rarely, distinct projections resembling vegetations; the tawny gray color in the more acute lesions and grayer and translucent appearance of the older ones; and in the more extensive process, the flat or delicately ridged plaques of yellow color.

To the above résumé may be added the fact that not infrequently the only macroscopic evidences of endocardial lesions are the presence of very inconspicuous, flat, often rounded plateaus, sometimes measuring no more than 2 mm. in diameter, generally delicate pink but, at times, no different in color from the remainder of the left auricular endocardium. These delicate elevations merge imperceptibly with the adjacent tissues and are best brought out by sponging the inner surface of the auricle and allowing the light to fall on the lesion in such a way as to emphasize the shadows, much as is done in the photography of flat projections.

It is to be noted that the normal endocardium often displays a cross weaving of elastic fibers in somewhat sharply cut geometric pattern. These may appear as slightly raised, white or gray ridges which, however, are practically always in the form of straight lines. The flat rheumatic auricular lesions differ from these geometric elastic patterns of the normal auricular endocardium in their rounded contour, greater irregularity, broader base and, sometimes, in their pinkish color.

The only statistics available on the incidence of macroscopic left auricular rheumatic lesions are those by VonGlahn, who observed 9 instances among 31 cases (29 per cent), and Thayer,[28] who noted acute or chronic mural endocarditis of the left auricle in 10 out of 25 cases (40 per cent). Gross auricular lesions were observed in 80 per cent of the series of rheumatic hearts which form the basis of this report. The highest incidence was in Group 3 of the above mentioned clinical classification (repeated attacks) where they were found in every instance. The lowest incidence was in Group 5 (chronic valvular disease without Aschoff bodies) where they were found in approximately half the cases. Macroscopic lesions in the right auricle were very mild, not nearly as easily discernible as those of the left auricle and relatively infrequent. Their histological characteristics are similar to those of the left auricular lesions.

Microscopic Appearance of Rheumatic Auricular Lesions

As has been indicated by previous authors, the auricular lesions in rheumatic fever may present a variety of pictures depending on the acuity of the process and on the individual reactions of the tissues. These reactions in turn may possibly be influenced by the immuno-

logical state of the individual. An analysis of these histological changes indicates that they may be roughly classified into five types, each corresponding to one of the clinical groups mentioned above.

Group 1

Histology of the Left Auricular Lesion Found in Active Cases Where the Individual Died Either in the First Attack or Where One Preceding Attack Occurred Within 1 Year of the Fatal Outcome

There were 17 cases in this group, ranging in age from 17 months to 19 years. The endocardium proper in every specimen showed varying grades of infiltration with inflammatory cells (Fig. 2). These were generally polymorphonuclear leukocytes and lymphocytes, as well as occasional eosinophiles, plasma cells and large mononuclear cells. Many of the cells were oriented at right angles to the auricular lumen, appearing often as ameboid streamers. In approximately half the cases the infiltration was very marked. In almost every specimen there were noted palisades or banded arrangements of these cells on either side of swollen elastic and collagen fibers. There did not appear to be any site of predilection within the auricular endocardium for these palisades. Apart from these, however, the infiltration was generally diffuse with numerous, focal, intense accumulations scattered irregularly throughout the several zones of the endocardium. In approximately half the cases there were present stratified cellular accumulations with histological properties similar to the cellular components entering into the formation of Aschoff bodies. Thus, the cells possessed owl-eyed, fibrocytoid and pyknotic nuclei and were surrounded by rather abundant basophilic cytoplasm with ragged edges. These cells also appeared to be limited by the adjacent elastic and collagenous bands (Fig. 3) and sometimes arranged themselves around fragments or sheets of swollen eosinophilic collagen. These lesions will be referred to as endocardial Aschoff bodies.

Besides these cellular accumulations the endocardium, as indicated, generally showed conspicuous bands and foci of swollen eosinophilic collagen (fibrinoid change). The elastic fibers almost invariably showed a definite departure from the normal consistence and topography. Instead of the regular lamellated arrangement seen in normal controls, particularly in the younger age periods,

many of the endocardial lesions showed spreading apart of the elastic lamellae (Fig. 4) and deviation from their parallel arrangement so that arches were formed, often directed toward the subendocardium. Occasionally, the membranes showed rupture with irregular patches of thinning; in other areas they were thickened and concentrated (Fig. 5). For simplicity of description such irregularities in the distribution of the elastic membranes will be referred to as "elastica distortions." Approximately one-third of the cases showed marked edema of the endocardium. This generally occurred in the area showing greatest cellular accumulations.

As previously indicated, there is a tendency for the smooth muscle of the endocardium to increase with advancing age. This muscular stratum is generally but not invariably situated in the outermost zone of the endocardium and usually occurs in more or less compact form. In the typical auricular lesion falling in this clinical category, the smooth muscle elements may increase in number (Figs. 6 and 7). They are sometimes irregularly arranged; the bundles may be separated into smaller discrete islands and occupy various zones of the endocardium proper.

It has also been shown that the normal endocardium possesses no discernible capillaries (as opposed to the subendocardium, in which capillaries are invariably seen). In the auricular lesion under description there is seen in the majority of instances a penetration of capillaries from the subendocardium into the endocardium proper. These are frequently seen to lie between the smooth muscle bundles, often arranged at right angles to the endocardial surface (Fig. 7). In approximately half the cases they penetrate to the middle zone of the endocardium (Figs. 8 and 10) and in about one-third of the cases they penetrate as far as the inner zone (Fig. 9). This phenomenon will be referred to as "capillary penetration," although it is to be noted that in a few instances the penetrating vessels are of arteriolar structure.

Perhaps the most important feature of this lesion is the endocardial "reduplication." This is a term intended to designate the formation of a new layer of tissue situated between the innermost elastic lamella and the auricular endothelial lining. These reduplications occur in several different forms. In the clinical group under discussion the most frequent form is the embryonal uncovered type. This consists of a not inconspicuous layer of mucinoid-staining mate-

rial which may attain a width approximately equal to that of the endocardium proper (Fig. 6). Within the matrix of this material there are seen stellate and spindle cells often running at right angles to the elastic membranes and consisting of vesicular nuclei surrounded by a rather hazy, faintly staining cytoplasm. Inasmuch as these cells apparently possess the multipotentiality of mesenchymal tissue (transforming themselves usually into fibroblasts, smooth muscle and, rarely, bone) they are referred to here as embryonal mesenchymal cells. Occasionally in this group an elastic membrane is seen covering this reduplication (covered embryonal reduplication). It is apparently this lesion that VonGlahn considered one of the healing processes of the endocardial lesion.

In this clinical group more than half the cases show these reduplications as single strata. Occasionally, however, two strata are seen separated from one another by an elastic membrane. These are referred to as multiple reduplications (Figs. 6, 10 and 12). Another form of reduplication is the edematous type. This was seen only once and consisted of a rather gelatinous tissue infiltrated with small, round, inflammatory cells (Fig. 4). Although occurring infrequently in this group, the covered reduplication presents another variant. This consists of an endocardial reduplication, often with an embryonal matrix, lying inside the innermost auricular endocardial elastic membrane and showing various grades of elastification with more or less parallel rows of elastic lamellae (elastified reduplication) (Fig. 8). Another form of reduplication is the dense collagenous variety. As its name indicates, this consists of rather dense collagen bundles (Figs. 9, 10 and 11) which may or may not be penetrated with a varying amount of elastic tissue. This occurred only once in this series. Smooth muscle cells in the endocardial reduplications were seen only once in this group (Fig. 10).

In this clinical group the subendocardium invariably shows evidence of inflammatory change (Figs. 2, 3 and 4). This generally consists of a marked infiltration between the collagenous bundles. The latter may or may not show eosinophilic swelling. The cellular components are similar to those mentioned as forming the characteristic infiltration of the endocardium. The infiltration is generally diffuse but may be focal. Both the inflammatory cells as well as edema produce, at times, a marked increase in the width of the subendocardium (Figs. 3 and 4). In approximately one-third of the cases Aschoff

bodies were present in the subendocardium, these being of a somewhat modified mosaic pattern with the characteristic cells lying in the crypts between the collagenous bundles. The elastic fibers of the subendocardium also show considerable modification, such as thickening, swelling, rupture, concentration and, at times, disappearance.

The capillary component of the subendocardium generally shows a very definite increase in their number. Not infrequently they are larger, more conspicuous, often oriented toward the auricular lumen, sometimes with endothelial buds and swollen endothelial cells. One case in this group showed the characteristic, intimal, musculo-elastic hyperplastic changes similar to those described by Gross, Kugel and Epstein [29] as occurring in other coronary vessels in the heart in rheumatic fever (Fig. 11). Not infrequently delicate vascular channels are seen to be distended with lymphocytes (Fig. 1). This was pointed out by MacCallum.

Approximately half the cases showed considerable hypertrophy of the myocardium (Fig. 12). As Sacks [30] has indicated, such hypertrophy is not necessarily associated with valvular defects but appears to be a direct stimulating effect of the exciting agent. In the interstitium between the myocardial bundles several cases showed edema and early fibrosis. Practically every specimen showed some form of cellular infiltration, often with lymphocytes, sometimes with polymorphonuclear leukocytes, occasionally plasma cells, eosinophiles and large mononuclear cells (Fig. 1). These were arranged either diffusely or focally and very often showed a contiguity with the subendocardial infiltration on the one hand and with an inflammation of the pericardium on the other. Apart from these nonspecific cellular infiltrations, more than one-third of the specimens showed myocardial Aschoff bodies of various types.

As indicated in a previous report,[29] the vascular lesions occurring in the left auricular wall in acute rheumatic fever are not conspicuous or characteristic. Nevertheless, the majority of cases showed vessels with glassy medial hypertrophy, and with medial hypertrophy.* In some cases the capillaries were rather conspicuous. Rarely giant medial hypertrophy with metallaxis was encountered. In 2 cases intimal fibrosis was noted.

In every specimen some form of pericardial inflammation was

* For a description of these lesions see Gross, Kugel and Epstein.[29]

noted. In more than half the cases this consisted of a microscopic, mild or marked infiltration, generally with lymphocytes, occasionally with polymorphonuclear leukocytes, eosinophiles, plasma cells and large mononuclear cells. These infiltrations tended to occupy the outermost zones of the pericardium. In 2 cases pericardial Aschoff bodies were seen. Rarely, eosinophilic swelling of the collagen was noted. The pericardial capillaries were generally quite conspicuous. In 2 cases the vessels showed small thrombi, in 1 case there was seen polypoid endarteritis, giant medial hypertrophy with metallaxis and intimal musculo-elastic hypertrophy.

In one-third of the cases a distinct fibrinous pericarditis was present. It is to be noted, however, that this figure represents the incidence of this lesion as it was observed in the left auricular section studied. Macroscopic pericarditis, either fibrinous or adhesive, was observed on some portion of the entire heart in 80 per cent of the cases in this clinical group.

Group 2

Histology of the Left Auricular Lesion Found in Active Cases Where One Previous Attack Occurred at Least 2 Years Previous to the Fatal Outcome

There were 10 cases in this group, ranging in age from 7 to 34 years. The average endocardial lesion resembled very closely that described for the first group. For the sake of simplicity its chief histological features are listed seriatim as follows:

1. Palisades were observed in only 4 of the 10 cases, as compared to 15 of the 17 cases in Group 1. These palisades did not differ essentially in their histological characteristics from those previously described. The same may be said for the incidence of eosinophilic swelling of the collagenous tissue, which closely paralleled the incidence and extent of the cellular infiltration.
2. Endocardial Aschoff bodies were found of the same type as those previously described. The incidence, however, was appreciably lower (2 out of 10 cases).
3. Inflammatory cell infiltrations, elastica lesions, capillary penetration and increase in the smooth muscle component of the endocardium were similar to that described in Group 1.

4. The most conspicuous difference from the first group lies in the nature and incidence of the reduplications. These were found in 7 out of the 10 cases. Multiple reduplications were seen in 1 case. All the reduplications were elastified. Some were covered. Two were of the dense collagenous variety and one showed a smooth muscle component.
5. As in Group 1, the width of the subendocardium was increased in approximately half the cases (Fig. 9).
6. Infiltration of the subendocardium with inflammatory cells occurred with about the same frequency as in Group 1, with perhaps a lower grade of intensity in approximately half the cases.
7. Subendocardial Aschoff bodies were found in only 1 case of the 10, as compared to 5 of the 17 in Group 1.
8. The increase in subendocardial vascularization was approximately the same as in Group 1, as was the nature of the vessels. One case showed arterioles and arteries with hypertrophied walls.
9. Eight out of the 10 cases showed myocardial hypertrophy. In the majority of these the hypertrophy was marked.
10. The interstitial myocardial infiltration, which was on the whole somewhat less marked than that in Group 1, occurred in 7 out of the 10 cases. Aschoff bodies were found in 4 cases.
11. Myocardial fibrosis was of about the same extent and incidence as in Group 1.
12. The myocardial vascular lesions were somewhat more varied. Thus, in 2 cases some of the blood vessels showed proliferation and desquamation of the endothelium; 1 case showed giant medial hypertrophy with metallaxis; 2, intimal fibrosis; 2, plugging of vessels with small thrombi; 1, intimal musculo-elastic hyperplastic changes, and 1, intimal elastification even in an early age period.
13. The incidence and type of microscopic pericarditis is similar to that described in Group 1. In 2 cases section of the left auricle showed adhesive pericarditis; 1 case showed a variety of rheumatic vascular lesions in the pericardium similar to those described in Group 1. (Macroscopic pericarditis, either fibrinous or adhesive, was observed on some portion of the entire heart in 7 of the 10 cases in this clinical group.)

Taken as a whole, the most conspicuous differences between the left auricular lesions in the first group and those in this clinical group lie in the lower incidence in the latter of palisade formations and of Aschoff bodies in the endocardium, subendocardium and myocardium; in the somewhat higher incidence and qualitative differences in the reduplications; and in the milder form of the interstitial myocarditis.

Group 3

Histology of the Left Auricular Lesion Found in Active Cases With Repeated Attacks, Death Occurring During an Acute Recurrence

There were 12 cases in this clinical group, ranging in age from 4½ to 36 years. The average endocardial lesion showed decidedly greater changes from the type described in Group 1 than did the previously described group. The following are the chief histological features:

1. While infiltration of the endocardium, as in the previous groups, was found in every case, this was marked in only 5 cases. The character of the cells and the incidence of eosinophilic swelling of the collagen was about the same. As in the previously described group, palisades were found less frequently than in Group 1. These were noted in 7 cases, in 5 of which the cells were of the large mononuclear variety.
2. Endocardial Aschoff bodies were found in 2 cases.
3. The elastica lesions were similar to those described in Group 1. They were found in every case.
4. Capillary penetration occurred in 11 of the cases. In 4 this was quite marked. In the rest of the cases penetration was very inconspicuous and affected only the outermost musculoelastic zone.
5. The smooth muscle increase was perhaps more definite in this group.
6. The most important difference from the previous groups lay in the incidence and type of reduplications. Thus, 6 cases of the 12 showed multiple reduplications, of which 3 possessed triple reduplications. Altogether, reduplications were found in 11 cases. The majority of them were elastified. A few of them were of the embryonal type and 2 showed smooth muscle components.

7. In practically every instance the subendocardium was somewhat increased in width. The infiltration, which was qualitatively of the same type as previously described, was marked in 7 cases.
8. In 3 cases subendocardial Aschoff bodies were present.
9. The increase in subendocardial vascularization, though still high in incidence (9 cases), was somewhat lower than previously described.
10. Eleven of the 12 cases showed marked myocardial hypertrophy.
11. Fibrosis between the myocardial bundles was found in 7 of the 12 cases, in 1 of which elastification was also present, even though in a relatively early age period (17 years).
12. In 4 cases inflammatory cell infiltration was marked. In 3 there was no infiltration noted. In the rest the infiltration was mild. Myocardial Aschoff bodies were found in 2 cases.
13. Vascular lesions of the myocardium occurred in even greater variety and frequency in this group. Thus, 3 cases showed intimal elastification; 2, intimal fibrosis; 4, thickening of the myocardial arteries or arterioles; and 1, intimal musculoelastic hyperplastic changes.
14. Eight cases showed organized or organizing pericarditis, a much higher incidence than was found in the previous groups. Typical rheumatic vascular lesions were found in 1 case. (Macroscopic pericarditis, either fibrinous or adhesive, was observed on some portion of the entire heart in 10 of the 12 cases in this clinical group.)

Considered as a whole, the features of this group are the tendency for the palisade formations to consist of large mononuclear cells, the higher incidence of multiple reduplications and adhesive pericarditis, and the lower incidence of interstitial myocarditis.

Group 4

Histology of the Left Auricular Lesion Found in Active Cases Where Death was Caused by Decompensation Without Clinical Evidence of a Final Recurrence. Some of these Cases Had No Previous History of Rheumatic Fever

There were 28 cases in this clinical group, ranging in age from 18 to 62 years. The average auricular lesion showed a decided decrease in active inflammatory phenomena. The following are the chief histological features:

1. Only 5 cases of the 28 in this group showed marked infiltrations of the endocardium; these were qualitatively similar to those described previously. Five cases showed no infiltration. In the remaining cases there was present only a mild grade of generally lymphocytic infiltration. Edema was also less marked and less frequent in this group.
2. Endocardial Aschoff bodies were found in only 4 of the 28 cases.
3. The incidence of palisade formations was decidedly lower, only 3 cases presenting these lesions. These were of the larger mononuclear cell variety. Eosinophilic swelling of the collagen occurred in only 3 cases, in 1 of which it was quite pronounced.
4. The smooth muscle components of the endocardium were very definitely increased, even after making allowance for the fact that the majority of the cases in this group belong to older age periods.
5. The elastica lesions were of the patchy variety and somewhat difficult to distinguish from age period changes (Fig. 11).
6. Nine cases showed multiple reduplications, of which 3 were triple reduplications. Altogether, 27 cases showed reduplications, generally of the elastified or dense collagenous variety. These were usually covered. In 1 case (Fig. 11), the reduplication was of the flat, collagenous, and somewhat elastified variety which, as will be noted later, is very characteristic of the cases belonging to the next clinical group, *i.e.* chronic valvular disease without activity. One

reduplication was of the embryonal type. Three of the reduplications showed smooth muscle components. One case showed calcific deposits on the superficial layers of the endocardium.

7. The width of the subendocardium was increased in the majority of instances.
8. Five of the 28 cases showed marked cellular infiltrations of the subendocardium; 17 cases showed mild infiltrations; 6 cases showed none.
9. Subendocardial Aschoff bodies were found in 7 of the 28 cases.
10. The vascularization of the subendocardium was increased in the majority of the cases. In 1 there was noted arteriolar hypertrophy; intimal musculo-elastic hyperplastic lesions were noted in 2.
11. In practically every case the myocardium was markedly hypertrophied.
12. In 18 cases there was no infiltration of the interstitial tissue of the myocardium. When it occurred it was generally mild and focal, consisting usually of lymphocytes and histiocytes. In 1 case myocardial Aschoff bodies were present.
13. The incidence of myocardial fibrosis was similar to that previously described. In addition, however, elastification occurred in 3 cases (in the somewhat later age periods).
14. The vascular lesions of the myocardium consisted of intimal elastification in about half of the cases. It is to be noted again that these occurred in the somewhat later age periods.
15. The pericardial lesions were on the whole mild. However, fibrinous pericarditis occurred in 1 case. Peculiar infoldings of the pericardial mantle with large swollen lining cells were found in 2 cases. Pericardial Aschoff bodies were found in 1 case. (Macroscopic pericarditis, either fibrinous or adhesive, was observed on some portion of the entire heart in 9 out of the 28 cases in this clinical group.)

Considered as a whole, the features of this group are the lower incidence of endocardial infiltration, palisade formation, Aschoff bodies and myocardial infiltration, and the higher incidence and peculiarity of the reduplications as well as the mildness of the pericardial lesions.

Group 5

Histology of the Left Auricular Lesion Found in Inactive Cases of Chronic Valvular Disease of the Typical Rheumatic Variety

There were 20 cases in this clinical group, ranging in age from 11 to 80 years. The average auricular lesion was marked by its extreme indolence. The following are the chief histological features:

1. Infiltrations of the endocardium occurred in practically every specimen. However, these were all very mild, consisting of scattered lymphocytes, occasional amebocytes and often large mononuclear cells. In many instances these infiltrations could not be distinguished from the occasional cellular infiltrations of the normal endocardium. However, inasmuch as the latter showed practically no infiltration after the fourth decade, its occurrence in many of the older cases of this clinical group was of some significance.
2. In 1 case there occurred a palisade formation of large mononuclear cells. This was associated with a moderate amount of eosinophilic swelling of the collagen. Edema was not found.
3. Aschoff bodies were not present in the endocardium.
4. A great variety of elastic changes was found in this group. None of them, however, showed the characteristic separation and stretching of the elastic fibers seen in the more active lesions. They consisted generally of exaggerations of the normally occurring age period changes of the elastic tissue, from which they were difficult and often impossible to distinguish. On the whole, it may be said that the elastic distribution in the endocardium in cases falling into this group is distinctly irregular, patchy, with, not infrequently, areas of cross-weaving of the elastic fibers and accumulation into compact bundles forming a mosaic with areas in which the elastic tissue was extremely sparse.
5. As in the previously described groups, the smooth muscle of the endocardium was increased. This, however, was frequently difficult to judge because of the older age periods in which most of these cases fell.
6. The endocardium itself was lightly infiltrated with calcium salts in 1 case.

7. All the cases showed reduplications. In 2 cases these were multiple. In the great majority of cases the reduplications were of the flat, dense, elastified variety. Smooth muscle was found in one reduplication.
8. Many of the specimens showed a moderate widening of the subendocardium. This consisted frequently of fibrosis, to which there was often added a fat cell component.
9. Infiltration of the subendocardium was generally very mild and consisted of lymphocytes. In the somewhat younger cases the infiltration tended to be slightly more conspicuous. Two cases showed no infiltration. No subendocardial Aschoff bodies were present.
10. About half the cases showed increase in vascularization without distinctive features.
11. All the cases showed a very marked hypertrophy of the auricular myocardium.
12. Infiltration of the myocardial interstitium was even less marked than in the previous group. It occurred in approximately half the cases and was generally mild and focal.
13. Fibrosis of the myocardium occurred in about one-third of the cases.
14. The most conspicuous myocardial vascular lesion was congestion of the capillaries, which occurred in about one-third of the cases. For the rest, the arterioles appeared to be somewhat hypertrophied and intimal elastification was somewhat more frequently found. However, these corresponded to the age period changes to be expected in this group.
15. Pericardial lesions were found in only 12 cases and consisted of mild scattering of lymphocytes. (Macroscopic pericarditis, either fibrinous or adhesive, was observed on some portion of the entire heart in 4 out of the 20 cases in this clinical group.)

Considered as a whole, this group of cases is notable for the extreme mildness of infiltrative phenomena, for the absence of Aschoff bodies and for the possession of the characteristic, flat, elastified reduplications.

Discussion

Although the blocks of tissue on which this study was made represent single specimens from each case cut according to our routine procedure and generally without any special effort to include macroscopic lesions, every specimen showed some variety of histological lesion that can be reasonably attributed to rheumatic fever. It must not be inferred from this, however, that the lesions were always so distinctive as to permit a diagnosis of rheumatic fever solely on the auricular findings. Particularly in Groups 4 and 5 it is not infrequently difficult to distinguish the essential inflammatory lesions of rheumatic origin from the confusing concomitant age period changes. On the other hand, in the clinical groups which represented the more active cases (Groups 1, 2 and 3) the lesions were very varied, generally quite conspicuous and presented a sufficient number of individual pathological processes on which it is possible to make a diagnosis of rheumatic fever. These lesions consist of edema and marked infiltrations of the endocardium with inflammatory cells, the banded appearance of some of these cellular aggregations, the presence of eosinophilic swelling of the collagen, the presence of Aschoff bodies in the endocardium, subendocardium and myocardium, the distortion of the elastic tissue and the widening, hypercapillarization and marked infiltration of the subendocardium. To these, furthermore, may be added the two important features of capillary penetration into the endocardium and the presence of reduplications. A less important feature is the presence of scattered and increased smooth muscle elements in the endocardium, together with smooth muscle in the reduplications proper. In none of the material examined was there present a necrosis of the superficial layers of the endocardium of sufficient intensity to warrant the term "verrucous change." Together with these endocardial and subendocardial lesions these first three groups also presented a very high incidence of myocardial hypertrophy and interstitial inflammatory cell infiltration. In approximately half the cases microscopic section of the left auricle showed some pericardial lesion.

In the more chronic clinical groups (4 and 5) the inflammatory phenomena of the endocardium and subendocardium were present in almost every instance, but very much milder. Aschoff bodies occurred in extremely low incidence or not at all (Group 5), and the

subendocardial inflammatory phenomena were also less frequent and milder, as were those in the myocardium and pericardium. On the other hand, the almost invariable presence of reduplications, sometimes multiple, the penetration of capillaries into the endocardium, when present, the marked hypertrophy of the myocardium and the presence in the majority of cases of some form of pericardial lesion, together with the increase and irregularity of the endocardial smooth muscle, readily constituted criteria on which to entertain at least a suspicion of rheumatic fever.

Of great interest is a consideration of the differences in the microscopic lesions between each of the five clinical groups. Certain phenomena are observed throughout all the groups, even though usually in different proportions. On the other hand, each group presents certain characteristics of its own which are both of a quantitative as well as of a qualitative value. Thus, active inflammation of the endocardium and subendocardium, as well as of the myocardium, is generally noted in Groups 1, 2 and 3. These inflammations are less marked in Group 4 and extremely mild in Group 5. This is true for the cellular infiltrations, edema and eosinophilic collagen swelling. The incidence of small round cell inflammatory infiltration shows a rather abrupt decline in Groups 4 and 5 where large mononuclear cells are more frequently found. As previously noted, palisades were observed with considerable frequency in Groups 1, 2 and 3. They were infrequent in Group 4 and occurred only once in Group 5. The same may be said of the incidence of Aschoff bodies which was high in only the first group. Capillary penetration of the endocardium is seen in all the groups, being most marked, however, in the first four. As noted, inflammatory infiltrations of the subendocardium occur in all the groups, but are most marked in the first three. Increased vascularization of the subendocardium is found in all the groups, chiefly in Groups 2, 3 and 4. The incidence and type of pericarditis varies somewhat. It is somewhat similar in the first two groups, where it is generally of the lymphocytic or fibrinous variety. Organizing pericarditis, however, appears conspicuously in Group 3. The total incidence falls in Group 4 and especially in Group 5.

Besides the conspicuous difference in the incidence of Aschoff bodies, cellular infiltration, palisade formation and eosinophilic swelling of collagen, the incidence and types of endocardial redupli-

cations constitute the most characteristic difference between each of the five groups. Thus, in Group 1, 10 of the 17 cases showed reduplications usually single and of the embryonal, uncovered variety. In Group 2 the incidence is higher (7 out of 10), the reduplications are generally single but the type is changed to either the elastified or covered variety. In Group 3 multiple reduplications begin to make their appearance. The majority of lesions are elastified. Some are triple. In Group 4 the reduplications are multiple in approximately one-third of the cases; in the majority they are elastified or dense and covered. In Group 5 the great majority of the reduplications are of the flat, dense, elastified variety. It is seen, therefore, that the appearance of these reduplications constitutes one of the most significant differences in the lesions peculiar to the several clinical groups of rheumatic fever.

In spite of the different histological features of the several clinical groups of rheumatic fever pointed out in this report, it must be borne in mind that the observations were made on too limited a number of cases on which to place complete reliance on the statistics submitted, nor indeed is it considered justifiable as yet to attempt such fine distinctions in the appraisal of a given case. The observations are presented as a suggestive indication for further study and with the thought that other associated lesions in the heart and elsewhere in the body as a whole may reflect, at least to a certain extent, the differences in the reactions of the tissues to rheumatic fever as determined by the clinical course of the disease.

Summary and Conclusions

Gross and histological observations on the left auricle, based on an examination of 87 rheumatic hearts, are described. The material is classified into five clinical groups, depending on the course of the disease. It is shown that macroscopic lesions of the left auricle occur in 80 per cent of the cases and microscopic lesions in 100 per cent. In the acute cases the lesions are very significant and characteristic. In the chronic cases they are considerably milder and often difficult to differentiate from normally occurring histological changes. A description is also given of the age period changes of the normal left auricle, as observed in 50 hearts.

REFERENCES

1. Corvisart, J. N. Essai sur les maladies et les lésions organiques du coeur et des gros vaisseaux. Migneret, Paris, 1806.
2. Burns, Allan. Observations on Some of the Most Frequent and Important Diseases of the Heart. Bryce and Company, Edinburgh, 1809, (see Chapt. 9).
3. Baillie, M. The Morbid Anatomy of Some of the Most Important Parts of the Human Body. W. Bulmer & Co., London, 1818, Ed. 5.
4. Wells, Wm. Ch. On rheumatism of the heart. *Tr. Soc. Impr. Med. and Chir. Knowledge*, 1812, **3**, 373–424.
5. Laennec, R.-T.-H. De l'auscultation médiate. J.-A. Brosson & J.-S. Chaudé, Paris, 1819, **2**, 127.
6. Hope, J. A Treatise on the Diseases of the Heart and Great Vessels. William Kidd, London, 1832, (see page 149).
7. Bouillaud, J.-B. Traité clinique des maladies du coeur. J.-B. Baillière, Paris, 1835, **2**, (see page 28).
8. Claude and Levaditi. Endocardite chronique à forme ulcéreuse de la paroi auriculaire gauche avec infiltration calcaire consécutive. *Bull. de le Soc. Anat. de Paris*, 1898, **12**, Ser. 5, 641–642.
9. Cheadle, W. B., and Lees, D. B. Three cases of extensive venous thrombosis associated with severe rheumatic carditis; necropsies. *Lancet*, 1898, **2**, 206–209.
10. Huchard, Henri. Traité clinique des maladies du coeur et de l'aorte. O. Doin, Paris, 1903, **3**, 326–327.
11. Harper, W. W. Pathology of the heart in rheumatic infection in children. *Southern M. J.*, 1914, **7**, 261–267.
12. Hertel, M.-P. Das Verhalten des Endokards bei parietaler Endokarditis und bei allgemeiner Blutdrucksteigerung. *Frankfurt. Ztschr. f. Path.*, 1920, **24**, 1–57.
13. MacCallum, W. G. Rheumatic lesions of the left auricle of the heart. *Bull. Johns Hopkins Hosp.*, 1924, **35**, 329.
    MacCallum, W. G. Rheumatism. The Harrington Lecture. *J.A.M.A.*, 1925, **84**, 1545–1551.
14. Stewart, H. J., and Branch, A. Rheumatic carditis with predominant involvement and calcification of the left auricle. Report of a case. *Proc. N. Y. Path. Soc.*, 1924, **24**, N.S., 149–163.
15. VonGlahn, Wm. C. Auricular endocarditis of rheumatic origin. *Am. J. Path.*, 1926, **2**, 1–14.
16. Pappenheimer, A. M., and VonGlahn, Wm. C. Studies in the pathology of rheumatic fever. Two cases presenting unusual cardiovascular lesions. *Am. J. Path.*, 1927, **3**, 583–595.

17. VonGlahn, Wm. C., and Pappenheimer, A. M. Rheumatic disease of the auricle and blood vessels. *Tr. A. Am. Phys.*, 1928, **43**, 203-212.

18. Shaw, A. F. B. Topography and pathogenesis of lesions in rheumatic fever. *Arch. Dis. Childhood*, 1929, **4**, 155–164.

19. Perla, D., and Deutch, M. The intimal lesion of the aorta in rheumatic infections. *Am. J. Path.*. 1929, **5**, 45–56.

20. Klinge, F. Der Rheumatismus. *Ergebn. d. allg. Pathol. u. path. Anat.*, 1933, **27**, 1–354.

21. Rothschild, M. A., Kugel, M. A., and Gross, Louis. Incidence and significance of active infection in cases of rheumatic cardiovalvular disease during the various age periods. *Am. Heart J.*, 1934, **9**, 586–595.

22. Gross, Louis, and Ehrlich, J. C. Studies on the myocardial Aschoff body. I. Descriptive classification of lesions. *Am. J. Path.*, 1934, **10**, 467–487.

23. Gross, Louis, Antopol, W., and Sacks, B. A standardized procedure suggested for microscopic studies on the heart. *Arch. Path.*, 1930, **10**, 840–852.

24. Königer, H. Histologische Untersuchungen über Endokarditis. *Arb. a. d. path. Inst. zu Leipzig*, 1903, **1**, No. 2, 1–162.

25. Nagayo, M. Zur normalen und pathologischen Histologie des Endocardium parietale. *Beitr. z. path. Anat. u. z. allg. Pathol.*, 1909, **45**, 283–305.

26. Miller, A. M., and Perkins, O. C. Elastic tissue of the heart in advancing age. *Am. J. Anat.*, 1927, **39**, 205–218.

27. Perkins, O. C., and Miller, A. M. Dynamics of histogenesis in cardiac repair. The rôle played by connective tissue. *Arch. Path. & Lab. Med.*, 1927, **3**, 785–800.

28. Thayer, W. S. Notes on acute rheumatic disease of the heart. *Bull. Johns Hopkins Hosp.*, 1925, **36**, 99–104.

29. Gross, Louis, Kugel, M. A., and Epstein, E. Z. Lesions of the coronary arteries and their branches in rheumatic fever. *Am. J. Path.*, 1935, **11**, 253–280.

30. Sacks, B. The pathology of rheumatic fever. A critical review. *Am. Heart J.*, 1926, **1**, 750–772.

DESCRIPTION OF PLATES

PLATE 100

FIG. 1. Normal left auricle. Age 3 months. Low power. Weigert's elastic and Van Gieson's connective tissue stain.

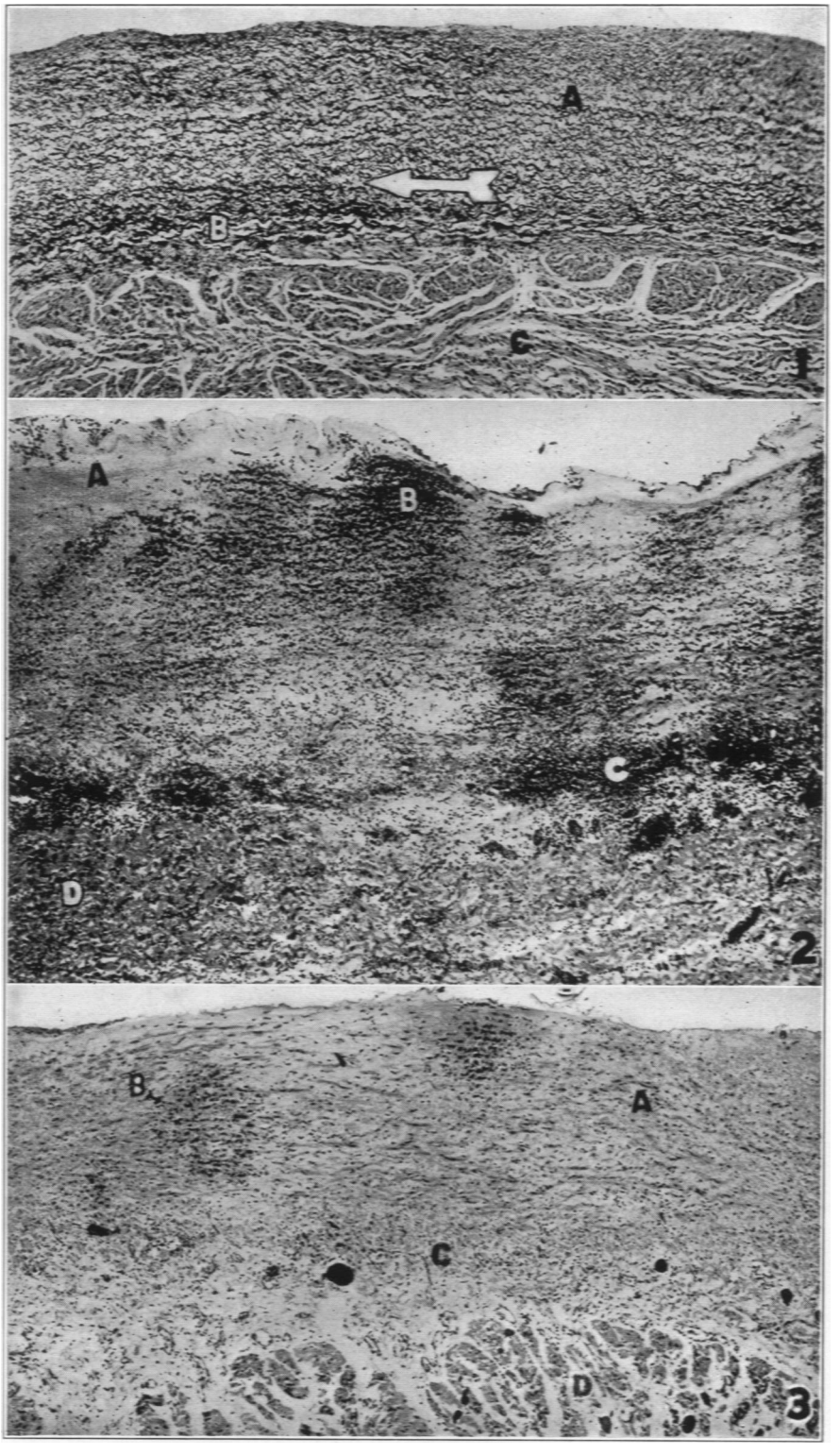
Arrow indicates division between A, endocardium and B, subendocardium; C = myocardium.

FIG. 2. Left auricle from active case of rheumatic fever. Age 18 years. Low power. Hematoxylin and eosin stain.

A = edematous portion of endocardium; B = infiltration of endocardium with inflammatory cells in banded arrangement; C = markedly infiltrated subendocardium. Note hypercapillarization. D = marked infiltration of myocardial interstitium.

FIG. 3. Left auricle from active case of rheumatic fever (injected specimen). Age 12 years. Low power. Hematoxylin and eosin stain.

A = endocardium infiltrated with small round cells. Note palisade formations. B = palisade formation of the endocardial Aschoff body type. C = considerably widened subendocardium. Note infiltration with inflammatory cells and hypercapillarization with many capillaries oriented at right angles to endocardial surface. D = myocardium with distended capillaries, and shows mild interstitial infiltration.



Gross Lesions of Left Auricle in Rheumatic Fever

PLATE 101

FIG. 4. Left auricle from active case of rheumatic fever. Age 18 years. Low power. Weigert's elastic and Van Gieson's connective tissue stain.

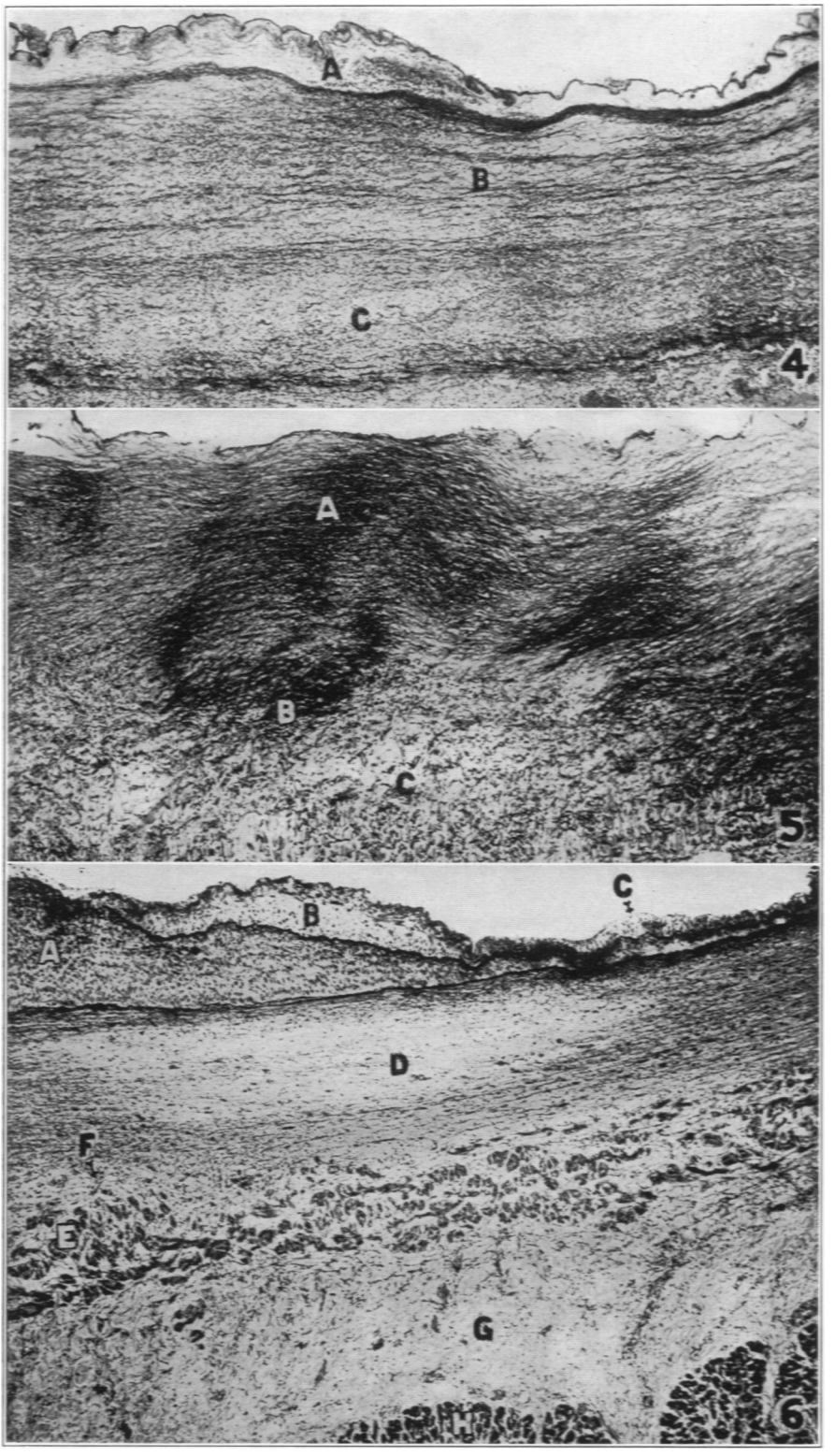
A = endocardial reduplication of edematous type. Note the small round cell inflammatory infiltration of this reduplication and the condensation of the elastic lamellae beneath it. B = endocardium markedly infiltrated with small round cells. Note edema, stretching and separation of elastic fibers. C = markedly inflamed, edematous and widened subendocardium.

FIG. 5. Left auricle from active case of rheumatic fever. Age 12 years. Low power. Weigert's elastic and Van Gieson's connective tissue stain.

A = endocardium showing distortion and condensation of elastic fibers; B = condensation of elastic fibers in subendocardium; C = edematous infiltrated subendocardium with hypercapillarization.

FIG. 6. Left auricle from active case of rheumatic fever showing multiple reduplications. Age 13 years. Low power. Weigert's elastic and Van Gieson's connective tissue stain.

A = older reduplication of embryonal covered type; B = more recent reduplication of partially covered embryonal type; C = uncovered portion of reduplication; D = endocardium proper showing marked edema, paucity of elastic fibers and mild small round cell infiltration; E = considerable increase in smooth muscle component of outer third of endocardium. Note irregularity of smooth muscle bundles, their separation by connective tissue and F, capillary penetration toward middle third of endocardium. G = markedly widened subendocardium with mild small round cell infiltration and marked hypercapillarization; H = myocardium.



Gross Lesions of Left Auricle in Rheumatic Fever

PLATE 102

FIG. 7. Smooth muscle zone in left auricle from active case of rheumatic fever. Age 13 years. High power. Masson's erythrosine-saffron stain. Note considerable increase in smooth muscle cells with irregularity in arrangement.

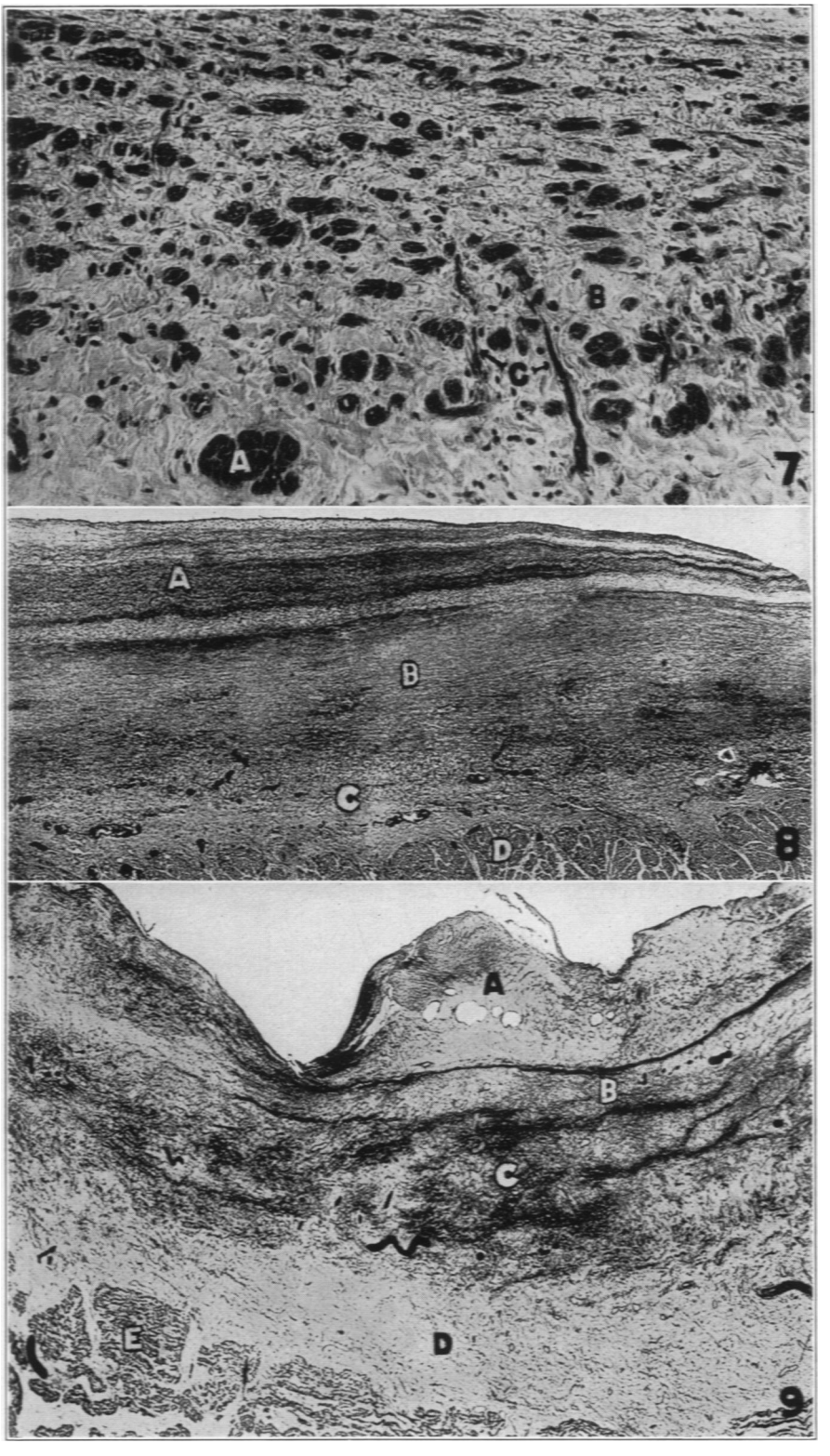
A = smooth muscle bundle; B = large amount of collagenous tissue between dispersed smooth muscle bundles; C = penetrating capillaries oriented at right angles to endocardium.

FIG. 8. Left auricle from active case of rheumatic fever. Age 15 years. Low power. Weigert's elastic and Van Gieson's connective tissue stain.

A = elastified reduplication with suggestion of origin from multiple reduplications; B = inflamed endocardium with capillary penetration to middle third; C = considerably widened, inflamed and hypercapillarized subendocardium; D = myocardium.

FIG. 9. Left auricle from active case of rheumatic fever (injected specimen). Age 7 years. Low power. Weigert's elastic and Van Gieson's connective tissue stain.

A = irregular collagenous reduplication with large vascular channels and some small round cell infiltration; B = capillary penetration of endocardium as far as the limiting membrane overlying the inner third; C = middle zone of endocardium showing marked elastica distortion and capillary penetration; D = widened subendocardium with hypercapillarization; E = myocardium.



Gross Lesions of Left Auricle in Rheumatic Fever

PLATE 103

FIG. 10. Left auricle from active case of rheumatic fever showing multiple reduplications. Age 14 years. Low power. Weigert's elastic and Van Gieson's connective tissue stain.

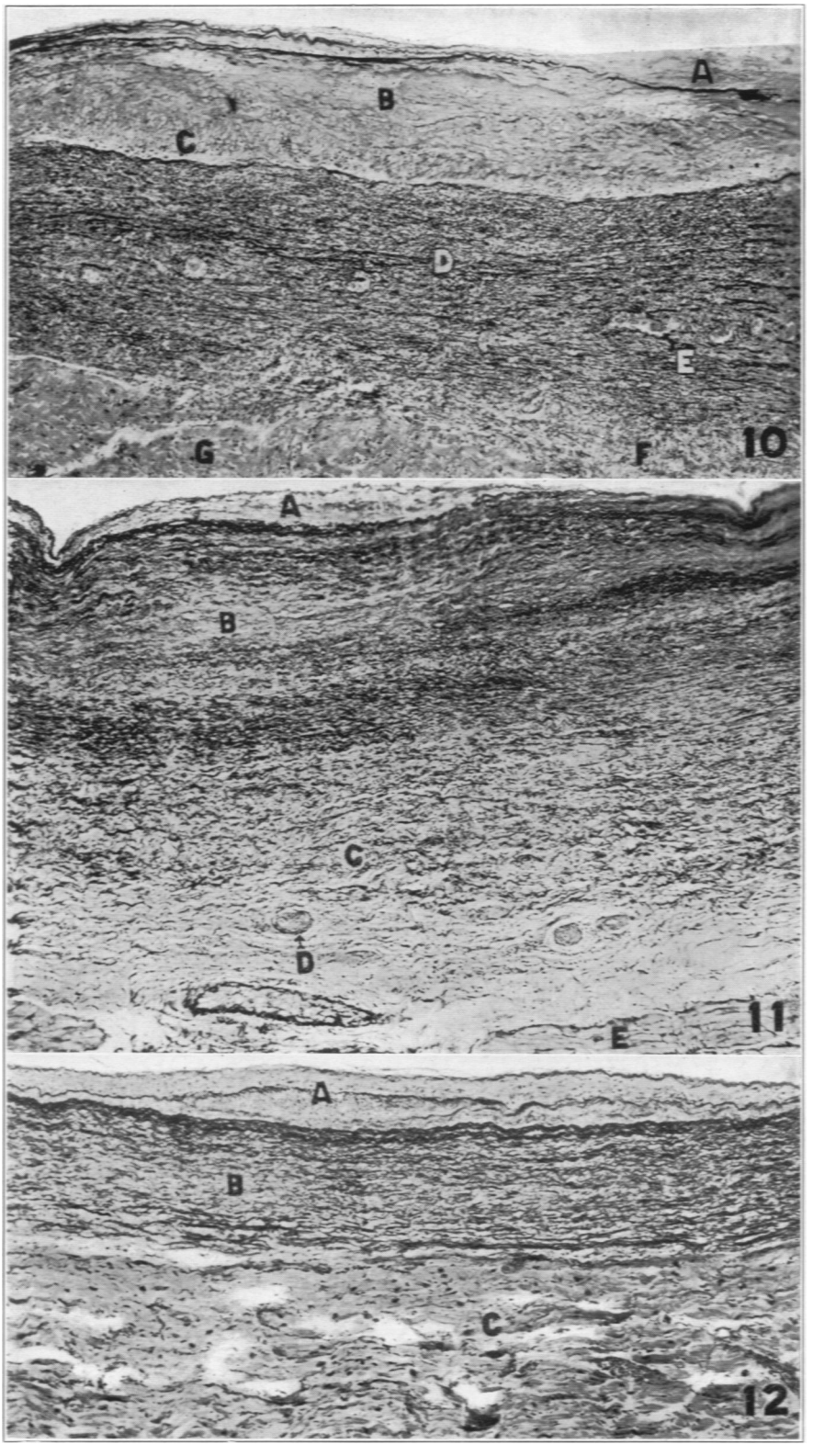
A = most recent reduplication, uncovered; B = older collagenous reduplication; C = smooth muscle component in reduplication; D = endocardium showing marked small round cell infiltration with stretching and separation of elastic fibers; E = penetrating vessels of arteriolar type, in endocardium; F = somewhat widened and infiltrated subendocardium; G = myocardium with mild interstitial infiltration.

FIG. 11. Left auricle from active case of rheumatic fever. Age 39 years. Low power. Weigert's elastic and Van Gieson's connective tissue stain.

A = collagenous covered reduplication; B = endocardium showing patchy arrangement of elastic fibers; C = markedly widened subendocardium with moderate small round cell infiltration; D = new formation of subendocardial vessels of the intimal musculo-elastic hyperplastic type; E = myocardium.

FIG. 12. Left auricle from active case of rheumatic fever showing multiple reduplications. Age 49 years. Low power. Weigert's elastic and Van Gieson's connective tissue stain.

A = multiple reduplications of endocardium. The reduplication resting on the endocardial limiting membrane is of the covered collagenous type. The innermost reduplication is of the covered embryonal type; B = endocardium; C = myocardium showing marked hypertrophy and some vascular engorgement.



Gross Lesions of Left Auricle in Rheumatic Fever