ON SUPRA-ARTERIAL EPICARDIAL FIBROID NODULES.

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Plates IX-XI.

The morbid condition to be described in this article is characterized by the presence of multiple, grayish-white, fibroid nodules situated upon the coronary arteries of the heart. Although similar nodules have doubtless been previously observed, I have been unable to find any satisfactory description of them in medical literature. I have, therefore, taken advantage of the opportunity to study histologically five well-marked examples which, during the last few months, have been observed among the autopsies at the Johns Hopkins Hospital.

These supra-arterial nodules differ from the ordinary tendinous or milky patches, often observed upon the epicardium, especially over the right ventricle, in their multiplicity, their smaller size, and their distribution over the coronary arteries to which they evidently bear some definite relationship. In gross appearance they resemble much more closely the nodular affection of the arteries first described by Kussmaul and Maier* in 1866 under the name “periarteritis nodosa.” In consequence of this resemblance, which, however, is only superficial and does not pertain to the histological structure, it is appropriate to direct attention to the latter affection.

Kussmaul and Maier observed nodular thickenings on all the smaller arteries of the body, except those of the central nervous system, in a man aged 27 who, after leading a vagabond’s life, entered the hospital at Freiburg complaining of weakness and muscular pains. He grew gradually worse, had an intermittent temperature, suffered from gastrointestinal disturbances and, late in his illness, with a progressive paralysis.

* Deutsches Arch. f. klin. Med., 1866, i, 484.
which showed from time to time short remissions. There were also symptoms of nephritis. He was ill four weeks.

At autopsy the most noticeable feature was the presence of countless opaque nodes on the smaller arteries of the body. The pericardium was smooth except where the coronary arteries were studded with these uniform grayish-white patches.

Microscopical examination showed usually no changes in the intima of the arteries, but there was a cellular infiltration and degeneration of the media, with infiltration and much hypertrophy of the adventitial coat, the lumen of the vessel being usually diminished, but occasionally dilated. Kussmaul and Maier expressed the opinion that the arterial changes were primary, and that the condition was a hitherto undescribed affection characterized by well-marked symptoms. They had no suggestion to make as to the cause of the arterial changes.

Chvostek and Weichselbaum* reported in 1877 the case of a soldier, aged 23, who became suddenly ill, complaining of headache, dizziness and nausea. There followed irregularity of the pupils and paralysis of the left ocular nerve, of the right side of the face and of the right extremities. He died after an illness of four months.

On section a ruptured aneurism of the left arteria profunda cerebri (posterior cerebral) was found, with hemorrhage into the cortex. Many of the medium-sized and small arteries of the body, but only at their bifurcation, were the seat of opaque gray swellings varying in size and shape. There were caseous areas in the lungs, pleurae, liver and kidneys.

Microscopical examination of some of the arterial tumefactions showed an increase in the cells of the intima with small-celled infiltration. These changes extended through the media and met a like proliferation of the adventitial coat, the elastic tissue offering the most resistance. There were aneurismal dilatations of some of the affected arteries. The condition was primarily, they consider, an endarteritis of luetic origin resembling that described by Heubner.† There were no other suspicious lesions nor was there a syphilitic history.

A somewhat similar case is described by P. Meyer.‡ A soldier, 24 years of age, after a life of dissipation, was admitted to a hospital complaining of great muscular pain, headache, sleeplessness, irregular fever and weakness. These symptoms became more marked and death took place in two months.

† Die leitische Erkrankung der Hirnarterien, etc., Leipzig, 1874.
‡ Virchow's Archiv, 1878, lxxxiv, 277.
The smaller arteries of the body were found at autopsy to be the seat of several yellowish-white dilatations from a mustard seed to a pea in size, most frequently situated at the branching of the artery involved. The vessels of the brain and cord were quite normal. The primary change Meyer conceives to consist in the rupture of the elastic coat of the artery leading to a dilatation of the other layers with thrombus formation and subsequent proliferation of the arterial walls.

He thinks that sudden changes in blood pressure from the ingestion of large quantities of fluid, from great exertion and from dissipation had weakened and broken through the strong elastic fibres with the above-mentioned secondary results.

This view is supported in part by Eppinger,* who brings the affection into line with aneurisms produced by increased blood pressure acting upon arteries whose elastic tissue is in places congenitally weak, and producing a bowing out of the wall with secondary hypertrophy of the intimal and adventitial layers.

The cases thus far have been those of young men, but that the process is not limited to this class is shown by the report of Fletcher.† A widow, aged 49, presented herself at the hospital complaining of muscular pain and weakness with occasional swelling of the legs. Later there were also intermittent fever, cough and albuminuria. The diagnosis rested between typhoid fever and miliary tuberculosis. Death occurred after two months.

The anatomical findings were very similar to those of von Kahlden,‡ who, in 1894, reported two cases in which like alterations in the arteries were observed. Both patients were women beyond middle life, 49 and 52 years of age, the latter the mother of five normal children. In each case the symptoms were increasing weakness and fever to which in the older woman were added great pain and gastro-intestinal disturbance. The duration of the illness was 8 and 12 weeks respectively. On section, each of these three cases showed upon the small and medium-sized arteries circumscribed whitish nodules, most abundant in the heart, pericardium, mesentery, pleura, liver and kidneys, the arteries of the central nervous system being unaffected.

Fletcher and von Kahlden agree with Chvostek and Weichselbaum in considering that the process begins by a proliferation of the intima followed shortly afterwards by a like process with leucocytic infiltration in

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† Zeigler's Beiträge, 1891, xi, 323.
‡ Zeigler's Beiträge, 1894, xv, 587.
the adventitial layer. This cellular tissue, they assert, invades the media on both sides leading to its rupture and to the degeneration of both muscular and elastic tissues, with an accumulation of small round cells and fibrin; a thrombus filling the lumen in the later stages completes the picture. They do not accept the hypothesis that the affection is due to syphilis on account of the complete absence of syphilitic history and lesions, but they are disposed to consider the causal agent to be some other, bacterial or toxic, infection of the blood which attacks the internal coat of the vessels and produces those changes which they consider to be primary.

From this brief review there would appear to be several reported instances of an affection of the smaller arteries, characterized macroscopically by circumscribed whitish nodules distributed pretty generally through the body, except in the central nervous system, and microscopically by hypertrophy of both internal (except in the case of Kussmaul and Maier) and adventitial coats with weakening, and, in places, with rupture of the elastic coat. There was sometimes dilatation of the vessel, but usually the lumen was narrowed.

The lesions in these cases were accompanied by clinical symptoms having much in common. The onset was sudden and marked by muscular pain, weakness, intermittent fever and gastro-intestinal disturbances. There were occasionally also subsequent paralysis, anemia and nephritis. The course of the disease was progressively fatal, death resulting in all cases in from 7 to 12 weeks.

Three suggestions have been made as to the etiology. Chvostek and Weichselbaum reasoning from the close resemblance to arterial changes of known syphilitic origin, consider lues to be the cause of the lesions. Meyer and Eppinger think that the primary change is a weakening of the elastic membrane; while Fletcher and von Kahl- den are of the opinion that the proliferation of the intima is the first step, and that this is brought about by the direct action of bacteria or of toxins.

The gross appearance of the nodules on the surface of the hearts now under consideration agrees closely with that of the arterial thickenings scattered generally through the body in the cases of so-called periarteritis nodosa.
Description of gross specimens (Plate XI). In the specimens examined the extent of the process varied much, from tortuous, more or less uniform, elevations over the arteries, to whitish dots minute in size and few in number which almost escape attention. In most of the specimens the epicardium between the arteries was delicate, the only superficial lesion, other than an occasional “milky patch,” being the grayish-white nodules along the smaller arteries. These elevations may be separated from each other by wide intervals or they may be quite close, resembling beads strung along the vessel, or they may coalesce throughout the entire length of the artery (Case III).* They hold no demonstrable relationship to the points of bifurcation of the vessels, nor was any particular branch especially affected. In no case did the thickening tend to encircle the artery. In one case (Case V) they were noticed, not only over the ventricle, but also, although few in number, upon the auricle, and over the small vessels in the adventitia of the aorta in its ascending part, where a single small focus measuring 1x2 mm. in diameter existed. In the same case a few scattered nodules were present also over the large veins (Plate XI). There was no tendency to aneurismal formation.

Microscopical.—The appearances observed under the microscope differ somewhat in different cases, although the main lesion is, as I hope to show, constant. In Cases I, II, III and IV the following conditions were observed in cross-sections including the coronary arteries with the nodules upon their adventitial surfaces. The intima of the arteries in the majority of sections examined showed little alteration; there may be slight swelling and the inner margin may show a somewhat irregular surface, which is not excluded from being the result of irregular contractions of this coat in the hardening process. In several specimens there could be made out a marked increase in the intimal layer, either as a single mass bulging into the lumen, representing perhaps an organized mural thrombus or as several smaller, more diffuse protrusions consisting of fusiform and branched cells with some intercellular substance. In the last case examined (Case V) the intimal changes are much more pronounced, being most marked in situations corresponding to the nodes. The entire internal coat is thickened, but the increase is especially marked in the myocardial hemispherical segment of the artery. In this situation it is fully six times its normal thickness and the muscular tunic is here somewhat diminished and degenerated. The increase in the thickness of the intima on the epicardial side is relatively slight, although the muscle in this region is degenerated and attenuated to a greater degree than on the opposite side of the vessel.

* Protocols of the cases are appended to this article, pp. 257-260.
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The muscular coat (media) is in the main, except in the instance to be presently mentioned, unchanged, so far as can be determined, in the sections stained by haematoxylin and eosin. Occasionally there is an apparent degeneration of muscular fibres as shown in a reduction in number of muscle-cell nuclei and there is exceptionally an infiltration with small round cells. In Case V, as already mentioned, the media shows much more pronounced pathological changes, in that this tunic is reduced in thickness and areas of hyaline metamorphosis are apparent.

The changes in the adventitia proper are slight and inconstant. Only occasionally is there any hypertrophy.

The nodular formations lie upon the vessels within the epicardium, being seated primarily in the layer of connective tissue between the endothelial covering and the delicate layer of elastic fibres which rests upon the main layer of loose vascular connective tissue containing the epicardial fat. Their situation corresponds, therefore, to that of milky patches as determined by Ribbert.* In their immediate neighborhood are found the usual loose adipose and connective tissues, vessels and nerves. But the nodules differ from the normal connective tissue, being at once distinguished from this through their dense and fibrous, often sclerotic, appearance.

The appearance on cross-section is as though a compact mass of firm connective tissue, convex on its inner surface, were set upon the artery in the loose epicardial tissue. The lateral edges of the thickening slant gradually upward to the surface and are continuous there with the epicardium (Plates IX and X). Scattered through the firm mass are a few fusiform connective-tissue cells. At the base of the area and at the sides are often groups of lymphocytes and sometimes a small number of polymorphonuclear leucocytes (Plate IX, Fig. 1 and Plate X, Fig. 1). The firm tissue pushes up beyond the level of the rest of the epicardium and forms the opaque nodule seen in the gross specimen. The endothelium and occasionally some subjacent tissue cover the prominence and are continuous with the serosa over the rest of the heart. Beneath the nodules the layer of epicardial elastic tissue can usually be demonstrated, but the subjacent loose connective and adipose tissues are more or less atrophied.

The size of the fibrous thickening varies within wide limits, both in depth and in lateral extension. It may form a comparatively narrow band which is not raised above the surface of the epicardium; or, again, it may appear as a high, irregular, almost pediculated projection rising from a compact base above the vessel (Plate X, Fig. 2).

* Virchow's Archiv, 1897, exlivii, 211.
The nodule may not only cover the outer surface of the vessel but may extend a considerable distance in the loose cellular tissue on each side (Plate X); or, on the other hand, there may be an oval patch over only a portion of an artery, the remaining peri-adventitial covering being quite normal (Plate IX, Fig. 1).

The early stages of the process leading to the supra-arterial nodules presented a tissue richer in cells, both fibroblasts and lymphoid cells, situated superficially to the vessel and on its epicardial side. As the nodule becomes older, the more homogeneous and less cellular becomes its structure; the nuclei are fewer and the focus is more sharply differentiated from the surrounding tissue. In no instance was any tendency to a similar fibrous formation noticed on the side of the affected vessel next to the heart muscle, nor were any similar alterations seen about the arteries in the substance of the myocardium.

We have, then, in these cases, nodules on the surface of the heart macroscopically resembling those described by Kussmaul and Maier but totally dissimilar in their minute structure and relations; for the specimens stained in hematoxylin and eosin failed completely to show any constant degeneration or proliferation of the arterial walls, such as is present in periarteritis nodosa. The uniform relationship to arteries, however, suggested that there was probably some alteration in the vessel which was at least associated with the nodule. Hence a representative number of the sections were stained for elastic tissue by the fuchsin method of Manchot * and by the method of Weigert †.

Results of stain for elastic tissue. There were of course variations in the sections, but the general results were sufficiently uniform to be quite suggestive. The stains brought out the yellow elastic fibres in dark-red or violet, in contrast to the pale, partly decolorized tissues. In a few of the sections there were distinct breaks in the inner elastic coat just opposite the nodule.

The most noticeable alteration, however, and one present in two-thirds of the specimens was a diminution in the strength of the outer elastic coat between the muscle and adventitial layers. This membrane was well represented in the inner (myocardial) side of the artery, often by a heavy dark band, but toward the outer (epicardial) half it became

* Virchow's Archiv, 1890, cxxi, 111.
† Centralbl. f. allg. Path., 1898, ix, 389.
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thinned, the fibres appeared looser and separated and more or less interlaced by the coarser bands of the adventitial coat until, beneath the thickening over the vessel, perhaps the last trace of the elastic membrane disappeared (Plate IX, Fig. 2).

In no instance was this change accompanied by an increase in the thickness of the inner elastic layer; in fact, at times the latter also appeared reduced.

The extent of this reduction or disappearance of the outer elastic coat differed widely. The thinning of the elastica was often present not only beneath the fibroid nodule, but extended a considerable distance in each direction, affecting perhaps half the circumference; or, again, it was more localized and perhaps somewhat to one side of the thickest part of the node (Plate X, Fig. 2).

The normal relative strength of the inner and outer elastic layers varies considerably; usually the inner coat is the stronger and the fibres seem more compact, but in other specimens it appears to be the weaker of the two and the main elasticity of the vessel wall is evidently supplied by an outer membrane which is thick and firm. In a rupture of this strong band there is usually not a simple break in the continuity of its fibres, but they split up into a perfect meshwork, spreading through the adventitia (Plate X, Fig. 2). Elastic fibres were made out in some instances in the nodule, more commonly as a definite layer between it and the underlying connective tissue (Plate IX, Fig. 2). Occasionally there was a proliferation of elastic fibres through the thickened intima. Only rarely were defects in either elastic coat seen in the inner (myocardial) side of an artery. In these cases no alteration corresponding to the nodule under discussion was noticed in the remaining layers nor in the surrounding tissue. No intrinsic changes in the elastic fibres suggesting degeneration were made out; the only microscopical deviation from the normal was the apparent mechanical separation and the disappearance of the fibres. Several sections containing arteries not surmounted by nodules were stained in the same manner, and although irregularities in the elastic coats were found, there was not in any case the marked alteration described in the elastic membrane as existing beneath the fibrous thickenings.

Sections of the diffuse areas of epicardial thickening, known as "milky patches," showed the appearances described by Ribbert.* No relationship of these to blood-vessels was observed.

* Virchow's Archiv, 1897, cxlvii, 207.
The foregoing microscopical observations indicate a relationship between the fibroid nodules and the demonstrated weakening in that part of the arterial wall immediately beneath the nodule.

In considering the etiology of this certainly distinct lesion, one must recall the diseases with which it may be associated.

(1) There was a distinct history of syphilis in one case (III); in three it was denied (I, II and V), while there is reason to be quite sure that there was no luetic infection in the fifth case (IV). The nature of the histological changes does not correspond to that of a syphilitic affection. The arterial alterations produced by lues consist in an endarteritis associated usually with a periarteritis, or the direct inclusion of the vessel in surrounding gummatous material. In the sections examined the arterial walls were unaffected, except in the manner already described, and in no instance were caseous areas seen in the adjacent tissues.

(2) As far as the gross appearances go, the nodules might possibly be mistaken for tubercles. One of the cases reported had suffered from a general tuberculous peritonitis (Case III) for which laparotomy had been performed. In the other cases there was no evidence whatever, clinical or anatomical, of infection with the tubercle bacillus nor was there the slightest sign of any tuberculous structure in any of the preparations.

(3) It will be recalled that blood infection from either bacteria or their toxins was the cause suggested for periarteritis nodosa by the later writers, Fletcher and von Kahlden. They based their hypothesis upon the primary proliferation of the intima which they attributed to the direct action of toxic substances in the circulation. This cellular increase in the intima, as has been shown, is conspicuous chiefly by its absence in most of the sections from our cases, although it may be present, as in Case V. Moreover, cultures from the heart's blood were negative in two instances, while Staphylococcus pyogenes albus alone was found once, as was also in one case Staphylococcus pyogenes aureus. In the fifth case blood cultures taken during life remained sterile. It would be quite impossible to imagine that the nodules could be due to poisonous agents carried in the blood stream.
and producing this fibrous thickening on but one side of the artery, while they so rarely affect a vein and do not injure further the intermediate arterial coats.

We may, therefore, doubtless exclude syphilis, tuberculosis and other infections as essential etiological factors in the production of these supra-arterial nodules.

The evidence seems to me strongly to support the view that the primary, underlying cause is to be found in a weakening of the arterial wall, due usually to defects in one or both of the principal elastic lamellae of the artery, most frequently of the elastica externa. A number of facts in the protocols lend color to this theory. All the cases were in males between 19 and 34 years of age, accustomed, with one exception (Case III), to hard work, irregular methods of life, indifferent nourishment and varying quantities of alcoholic beverages. The heart was hypertrophied in four cases (I, II, IV, V); in one (IV) there were valvular lesions, in two (II and V) arteriosclerosis, in four (I, II, III, V) nephritis, in four (II, III, IV, V) oedema, and in a single case (I) aneurisms existed. These conditions indicate that during life there must have been irregularities in the force of the blood pressure, influenced further, doubtless, by the ingestion of large quantities of fluid.

The duration of the final illness varied from 8 weeks to 2 years. So far as known, no symptoms are attributable to the epicardial nodules.

As a probable explanation, then, of the origin of these fibroid nodules it is suggested that there is a weakening of the arterial coats, mainly of the outer elastic coat, on the side toward the nodule. While this defect may be congenital, it is more probable that it is acquired through poor nutrition combined with sudden alterations in blood pressure from the causes already indicated. This loss of elasticity, it may be supposed, is compensated by a fibroid thickening, situated not in the vessel wall, but beyond it in the epicardial tissue.

For such an explanation an analogy is to be found in the views now generally held of the formation of aneurisms, first advanced by Rokitansky and later supported, with certain minor modifications, by
Eppinger, P. Meyer, Manchot, Thoma and others. These authorities consider the primary change to be a giving away of the media, especially of the elastic lamellae and fibres, with subsequent bulging out of the arterial wall. When the weakening takes place slowly, cellular proliferation occurs in the intima and adventitial coats and the danger of aneurism is lessened by the thickening of the arterial wall. Tears in the elastic lamellae are not uncommon, and may occur without the formation of aneurism. The high pressure in the coronary arteries would seem to render these vessels particularly exposed to such injuries, when the nutrition of their walls is impaired. Of especial interest in our cases is the demonstration of more frequent and pronounced defects in the outer elastic lamella than in the internal one.

It will be recalled that in no case was there an increase in the surrounding connective tissue corresponding to the inner or myocardial side of the artery and it is to be conjectured that here the heart muscle affords sufficient support to prevent stretching of the vessel walls with such ruptures of the elastic lamellae as were observed on this side. Each pulsation must produce expansion of the artery, which is most marked at the point of least resistance, i.e. at the outer or epicardial surface. Excessive or irregular expansion can easily be thought of as injuring more or less the elastic coat. In such cases there follows a still greater protrusion outwards of the vessel wall with each pulsation. The significant localization of the defects in the outer elastic lamellae upon the epicardial side of the arteries indicates the greater exposure of the artery upon this side to injury, and this may be due to the lack of the support which is afforded to the myocardial side of the artery by the surrounding tissues. The firm resistance offered to the expansion of the vessel on the inner side by the ventricular wall is an important factor in favoring increased bulging in the free semi-circumference. The absence of this counter-support probably accounts for the fact that similar nodules are rare upon the auricles.

**SUMMARY AND CONCLUSIONS.**

1. Fibroid nodules seated in the epicardium directly over branches of the coronary arteries of the heart are not uncommon. They may be present in large numbers and are found most frequently upon the
surface of the ventricles, but may occur over the auricles and even on the outer surface of the ascending aorta. They are rarely observed over the coronary veins.

2. While often resembling in gross and superficial appearances the nodules described by various writers under the name of "periarteritis nodosa," they differ from these in essential respects. They are seated outside of the adventitial coat and lie within the epicardium. They are composed of dense, fibrous, sclerotic tissue, poor in cells. In earlier stages of their formation they are richer in cells, both fibroblasts and lymphoid cells.

3. These supra-arterial nodules bear no definite relation to endarteritis, although they may be associated with this condition.

4. There were found with great regularity in the arterial wall immediately beneath the nodule, changes, which indicated a weakening of the wall in this situation. In some instances the muscular coat was thinned and degenerated, but the most common and important change was reduction and often disappearance of the elastic lamellae and fibres, the outer elastic lamella being the one most frequently and intensely affected. These lesions were often limited to the segment of the arterial wall adjacent to the epicardium, the inner or myocardial segment of the same artery being free from similar alterations, or presenting them only in a slight degree. It is suggested that the absence on the outer or epicardial segment of the firm support afforded to the artery on the inner or myocardial aspect by the surrounding tissues renders the former more liable to damage to the elastic tissue resulting from irregularities and increase of blood pressure associated perhaps with defects of nutrition.

5. In consequence of the weakening in the arterial wall the artery would tend to bulge at the affected spot toward the epicardium were this tendency not restrained. The formation of the dense supra-arterial nodule of fibrous tissue over the weakened area holds this tendency in check and may therefore be regarded as an adaptive or compensatory change.

The question as to the immediate exciting cause of the new growth of tissue offers the same difficulties as that pertaining in general to
similar growths of connective tissue. Some would doubtless attribute it to direct stimulation from the pressure and shock of the impinging artery, others to defects in the tissue, and still others to a disturbance of the neighborhood relations of the part. It is not deemed necessary to enter into a discussion of these various hypotheses.

I take pleasure in expressing my thanks to Dr. Welch for examining my sections and to Dr. Flexner for constant advice and help.

**Protocols of the Cases.**

**Case I.** Man, G. J., aged 45, colored. Clinical history not obtainable.

*Anatomical Diagnosis.* Enlargement of middle lobe of prostate gland; purulent cystitis, pyoureter, pyelonephritis, pyonephrosis.


Additional notes. Pericardium shows "milk patch" over right ventricle. The remainder is smooth except along the ramifications of the branches of the coronary arteries over both ventricles, where grayish-white, semi-translucent nodules, varying in size from an ultimate tubercle to one of a millimetre in diameter exist. These may be scattered at intervals along the vessel or in the case of the flatter and more opaque ones may almost coalesce throughout the entire length of the vessel. Apparently there is no bulging inward of the vessel wall corresponding to the thickenings. The valves are delicate. The left ventricle is thickened, not dilated. Weight of heart 370 grammes. No arterial nodules detected in any of the organs.

*Bacteriological report.* Heart and lungs, Staph. pyog. albus.

**Case II.** Man, S. C., aged 54, colored, coachman.

*Clinical history.* Patient came to hospital complaining of shortness of breath, cough and swelling of the legs and abdomen. Has led a rough, exposed life. Used tobacco and alcohol freely. Fifteen months before admission had attack of dyspnoea, which has been repeated many times since, together with oedema of face and of extremities and ascites. Much difficulty in micturition. Urine contains albumin and hyaline casts. Died eleven days after admission, in which time 3800 cc. of fluid were drawn from his abdomen.

*Anatomical Diagnosis.* Arteriosclerosis. Aneurism of the thoracic and abdominal aorta. Cardiac hypertrophy. Supra-arterial coronary
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Additional notes. Pericardium generally smooth. Following the course of the coronary arteries and their branches are small grayish-white nodular thickenings from a pin's head to a millet-seed in thickness. Vegetations are present on the aortic segment of the mitral valve. A bulging is seen in the transverse arch of the aorta and a second dilatation in the abdominal aorta below the superior mesenteric artery.

Bacteriological report. Cultures from heart, lungs, peritoneal cavity, liver and spleen are negative.

Case III. Man, W. D., aged 43, colored.

Clinical history. Admitted to hospital complaining of swollen abdomen and dyspnea. One brother died of phthisis. History of syphilis, gonorrhea and abuse of alcohol. Patient felt well until 6 weeks before admission when he began to complain of headache, muscular pain, and weakness followed by cough, dyspnea and ascites. He was transferred to the surgical side, laparotomy was performed and a general tuberculous peritonitis was found. Death occurred 18 days after operation, about 10 weeks after the onset of symptoms.


Additional notes. Heart small. The coronary arteries generally are marked out by grayish-white elevations fusing together over them, forming tortuous opacities. The epicardium between these thickenings is normal except for some milky patches. The intima appears smooth.

Bacteriological report. Cultures from lungs, heart, kidney and liver show Staphylococcus pyogenes aureus.

Case IV. Man, J. H., aged 19, white, florist.

Clinical history. Patient admitted to hospital complaining of cough, dyspnea and palpitation. Three months previously had suffered with edema of ankles, but resumed work until a month before admission, when the symptoms complained of made it necessary to stop. On admission there were marked evidences of aortic and mitral disease with loss of compensation and cardiac enlargement. He died at the end of three weeks after admission, during which time the symptoms had become worse.

Additional notes. Visceral pericardium smooth except for a few granules of fibrous thickening at the outer edge of auricle and over the right ventricle where there are a number of minute, rather translucent white granular spots, slightly elevated, lying over the course of vessels.

Posterior segment of chordæ tendineae of mitral valve shortened and thickened. Aortic valve thickened along its free edge. No nodules in organs.

Bacteriological report. Cultures from peritoneal cavity, heart, spleen and mitral valve, negative.

Case V. J. R., man, aged 48, colored, upholsterer. Complained on admission of shortness of breath and swelling of legs. Had always done hard manual labor and used tobacco freely, otherwise fairly good personal history. Present illness began six weeks before admission, when shortness of breath upon exertion was first noticed. This became progressively more marked. Patient had some cough.

On admission there was present general edema of lungs, abdomen, face and extremities with some cyanosis. Heart was enlarged but no organic lesion made out. Radial walls markedly thickened. Urine contained hyaline and granular casts and 0.2-0.7 per cent. albumin. Condition became gradually worse. Cheyne-Stokes breathing appeared and patient died seven weeks after admission, after illness of about three months.

Anatomical diagnosis. Arteriosclerosis; cardiac hypertrophy; thickening of mitral valve; extensive supra-arterial epicardial fibroid nodules; thrombi in auricular appendages; infarct of kidney; chronic nephritis; edema and chronic passive congestion.

Additional notes. Heart weighed 540 grms. Hypertrophy, chiefly of left ventricle, with dilatation. Over the epicardial surface of the main coronary arteries of the left ventricle, as well as their branches and small twigs, are seated, discrete, small, elevated fibroid nodules of a semi-translucent or opaque appearance, resembling often beads strung along the course of the superficial vessels (Plate XI). Similar nodules are found in small numbers upon the auricles and upon the arch of the aorta. In these situations their relation to blood-vessels can also be demonstrated. Several similar nodules occur along the course of the larger branches of the coronary vein (Plate XI).
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The coronary arteries are tortuous and extensively sclerosed. There is a general arteriosclerosis.

DESCRIPTION OF PLATES IX--XI.

PLATE IX.

Fig. 1 (Case II).—Transverse section of small artery in epicardium, surmounted by a fibroid nodule. The fibrous thickening is over about one-half of the width of the vessel. Haematoxylin and eosin staining. A. Intima. B. Inner elastic coat; wavy lines showing only at the sharper turns of the vessel wall. C. Media, slight swelling and degeneration (reduction in the number of nuclei) beneath the nodule. D. Adventitial coat unaltered. E. Normal epicardium. F. Supra-arterial fibroid nodule projecting above the surface of the epicardium and extending to the adventitial membrane. G. Accumulation of small round cells in the epicardium at the sides of the node. H. Nerves in cross-section. I. Portion of a vein evidently not associated with the fibrous nodule. J. Heart muscle.

Fig. 2 (Case II).—Specimen stained in fuchsin (Manclet's method). A. Intima. B. Inner elastic coat unaltered; no break or weakness beneath the nodule. C. Media. D. Outer elastic membrane between the muscle and the adventitial layer. This elastic membrane is thick and firm, and evidently supplying the chief support and giving the elasticity to the vessel wall. The membrane is intact, except at E, where beneath the nodule there is a marked fraying out and disappearance of the elastic fibres with a consequent diminution in the strength of the arterial wall. F, G, H, I as in Fig. 1.

PLATE X.

Fig. 1 (Case II).—Same specimen as Plate IX, Fig. 2, stained by haematoxylin and eosin. A. Intima, with areas of proliferation probably not associated with the fibrous nodule. B. Inner elastic coat. C. Media apparently unchanged. D. Adventitial coat unaltered. E. Normal epicardium. F. Fibroid nodule projecting but slightly above the surface of the epicardium and extending to the adventitial coat beneath. G. Groups of small round cells at the sides and beneath fibrous nodule. H, L, M, N as in Plate IX, Fig. 1.

Fig. 2 (Case III).—Transverse section of larger artery surmounted by a pronounced fibrous thickening, somewhat pedunculated and projecting abruptly from the surface. Same staining as Plate IX, Fig. 2. A. Intima. B. Inner elastic coat, very firm, unaltered. C. Media. D. Outer elastic coat, compact only for a small portion of the circumference on the myocardial side. From this point on each side the fibres may be seen to separate into a network and to become diminished in number until at J they cannot be demonstrated.

PLATE XI.

Heart of Case V.—The supra-arterial epicardial nodules are well shown over the coronary arteries and their branches. To the left are seen a few nodules upon a large vein.