SYPHILITIC ANEURYSM OF LEFT CORONARY ARTERY WITH CONCURRENT ANEURYSM OF A SINUS OF VALSALVA, AND AN ADDITIONAL CASE OF VALSALVA ANEURYSM ALONE *

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CORONARY ARTERY ANEURYSMS

The rarity of such aneurysms, exclusive of the false or dissecting forms and those associated with periarteritis nodosa, is stressed by Karsner [1] and further attested by the few reports in the literature. Packard and Wechsler,[2] 1929, in their classical survey, could find only 30 examples, the first of which was recorded by Bougon [3] in 1812. Packard and Wechsler have thoroughly reviewed all reports of this condition, checked duplications and culled out questionable examples, so that we need only to cover the literature since 1929 and summarize briefly the salient facts elicited by these authors. They placed coronary aneurysms in two etiological groups: (1) mycotic-embolic, associated with bacterial endocarditis involving the aortic valve; and (2) arteriosclerotic, related to coronary sclerosis and long continued hypertension. The average age of the first group was 27 years, as compared with 57 years for the latter. There is no mention of syphilitic coronary arteritis as a cause of aneurysm of these vessels and mesaortitis was present in only 3 of the 30 collected cases. Rarely, indirect trauma due to strain may have been an etiological factor, but direct injury had an insignificant rôle, apparently on account of the protected anatomical site of the coronary vessels. In nearly all cases the aneurysms were single and located within the first inch of the left coronary artery, which was involved about three times as frequently as the right. In 4 hearts both coronaries were attacked. About 50 per cent of the aneurysms ruptured. There were no pathognomonic symptoms or signs that permitted an ante mortem diagnosis of the condition.

Cox and Christie,[4] 1930, described a fusiform aneurysm 2.5 by 4.5 cm. involving the right coronary artery. This was associated

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with cardiac hypertrophy (500 gm.), old and organized thrombosis of the anterior interventricular branch of the left coronary artery, myocardial fibrosis and aneurysm of the abdominal aorta and of the right common iliac artery. Microscopic study of the aorta revealed only marked arteriosclerosis. Apparently no histological study was made of the coronary aneurysm. The lesion occurred in a white male 65 years old with a long history of vascular hypertension and of a paralytic "stroke."

Vogelsang,[5] 1930, reported an aneurysm 6 by 5.5 by 4.5 cm. involving the anterior interventricular branch of the left coronary artery and situated in the anterior wall of the left ventricle. There were also gummatous myocarditis, syphilitic aortitis and gummatous pneumonitis. Distal to the aneurysm the artery was thrombosed. He included no microscopic description of the aneurysm, but expressed the opinion that in view of the gross and microscopic lesions elsewhere the aneurysm was probably also of syphilitic origin, and that the trauma of a 9 foot fall which the man, a 38 year old seafarer, sustained 4 months prior to his sudden death, might have been an etiological factor.

Halpert,[6] 1930, described an arteriovenous communication between the right coronary artery and the coronary sinus, with aneurysmal dilatation of both, in a man 54 years of age who showed no cardiac disturbances during life and died of gastric carcinoma. From gross and microscopic investigation he concluded that the lesion probably was congenital.

Thus, to date (May 1934), 33 acceptable cases of coronary aneurysm have been recorded, and to this total we desire to add another, the only one of its kind among 5896 autopsies performed by the pathology department of the University of Oregon Medical School.

Aneurysm of the Aortic Sinuses of Valsalva

This is another uncommon site of aneurysm formation, but is less rare than in the coronary arteries. The comparative rarity of such lesions, together with the complications peculiar to their anatomical location, warrant the recording of further instances. In our autopsy material 2 cases have been encountered, one complicated by aneurysm of the left coronary artery, the other occurring in conjunction with syphilitic aortitis.

Valsalva aneurysms of congenital,[7] syphilitic,[8, 9, 10] arteriosclerotic,[9] mycotic,[9, 11] and indeterminate [12] etiology have been described as have dissecting or false aneurysms. Often these rupture, usually into the pericardial sac, the chambers of the heart or into the great vessels, but may burst externally after eroding the chest wall, as in the case of Sheldon,[13] in which the pericardial sac had been obliterated by fibrous adhesions, presumably incident to rheumatic disease. Without rupturing, these aneurysms may burrow into the myocardium of the atria, ventricles or interventricular septum and in this way produce stenosis or insufficiency of one or more valves. Another complication, present in one of our cases, is heart block from encroachment upon the atrioventricular bundle, while still another, exemplified by our Case 1, is extension of the aortic disease to an adjacent coronary artery with the formation of an aneurysm in this vessel as well.

Most frequently the anterior sinus is involved, due, according to von Krzywicki,[9] to its unsupported position superior and somewhat anterior to the membranous interventricular septum. Gray [14] considers that the regurgitation of blood, directed chiefly against the anterior aortic wall, is a factor also.

The actual incidence of Valsalva sinus aneurysms is difficult to ascertain since most authors who have made statistical studies have not only failed to distinguish between true and dissecting aneurysms, but in addition have not made clear as to whether or not their figures are based only upon adequately described and complete autopsies. In many compilations the location of the aortic aneurysms is given only within wide anatomical limits. It is probable that some Valsalva aneurysms have been included with those of the ascending aorta and it is possible that others are buried in reports with misleading titles.

In order to obtain figures of incidence of various aortic aneurysms in a series of autopsies we have reviewed all of our records and have disregarded 605 protocols because the examinations were incomplete, or the description of the aorta was unsatisfactory. Among these were 13 cases in which one or more sacculations were present. Among the remaining 5896 autopsies 143 individuals had 214 true aneurysms of the aorta distributed anatomically as follows:

	No.	Per cent
Sinuses of Valsalva	2	0.93
Ascending aorta	102	47.66
Ascending aorta and transverse arch	11	5.14
Transverse arch	35	16.35
Arch and thoracic	1	0.47
Ascending, arch and thoracic	3	1.40
Thoracic	43	20.10
Abdominal	17	7.94

In 20 persons there were 26 dissecting aneurysms which are not included in the tabulation. Among those listed were 6 cases of concomitant true and dissecting aneurysm, but the latter have been omitted from the compilation. In the tabulated group the probable etiology was: syphilitic 87.8 per cent, arteriosclerotic 10.3 per cent, mycotic-embolic 1.4 per cent and rheumatic 0.47 per cent. These figures agree fairly well with those of Brindley and Schwab,[15] but in our series syphilis seems to be a somewhat more prominent etiological factor, possibly because only true aneurysms are included.

We have made no exhaustive survey of the literature dealing with aneurysms of the sinuses of Valsalva but wish to call attention to the statistics of Smith[10] and of Lucké and Rea[16] who found 10 cases among 287 aortic aneurysms in 12,000 autopsies collected from various sources.

Case Reports

Case 1. *Syphilitic Aneurysm of the Left Coronary Artery with Concurrent Aneurysm of Sinus of Valsalva:* The clinical history and autopsy record were not available.

Postmortem Examination

The heart and 6 cm. of the ascending aorta had an aggregate weight of 595 gm. The intima of the ascending aorta was wrinkled, roughened, pearly gray to whitish and mottled by irregular yellowish areas. The wall was irregularly thickened, of cartilaginous consistence and in a state of saccular aneurysmal dilatation. The epicardium and endocardium were grossly unchanged. The left ventricle was greatly hypertrophied and dilated. The trabeculae carneae and papillary muscles were much enlarged, elongated and flattened. The mitral leaflets were unchanged. The aortic leaflets felt gristly and had thickened, rolled, rounded edges. The aortic ring had a circumference of 8.5 cm. The left atrial wall was 3 to 4

mm. thick across the pectinate muscles, but between them was almost transparent. There were two right coronary ostia, situated 2 mm. apart. The smaller was less than 1 mm. in diameter and led to a vessel coursing over the conus arteriosus. The orifice of the main artery was slit-like and measured 1 by 3 mm. Both ostia opened 1.1 cm. superior to the upper border of the anterior aortic leaflet. Serial cross-sections of the artery revealed some eccentric thickening of its wall by atherosclerotic plaques which did not close the lumen.

In the left posterior aortic sinus was an aneurysm having a crescentic opening measuring 0.7 by 1.8 cm. Within the anterior wall of the left ventricle this aneurysm expanded, attained dimensions of 3.1 by 3.4 by 2.4 cm. and became filled by a laminated thrombus. Anteriorly and to the right the sac bulged into the right ventricle directly below the posterior leaflet of the pulmonic valve, elevating it somewhat, and producing a triangular stenosis of the valve. The base of the triangle was formed by the bulging aneurysm wall and the opening of the valve was reduced to about half its normal size. The lining of the aneurysm resembled that of the aorta. The inferior wall of the sac was formed by the left ventricular myocardium and did not encroach upon the membranous part of the interventricular septum, but lay somewhat anterior to it. The sac also bulged into the left atrium beneath the anterior leaflet of the mitral valve, elevating it to some extent and producing a slight degree of stenosis. The right ventricle was markedly dilated and its wall was considerably hypertrophied.

The left coronary artery and the Valsalva aneurysm were examined by gross serial cuts, as shown in Figure 3. The left coronary ostium (Fig. 3A) lay 0.7 cm. above the tip of the left posterior aortic leaflet, was oval, measured 3 by 5 mm. and had its long diameter in the superior-inferior direction. It was surrounded by pearly white, wrinkled and greatly thickened aortic intima. Within 5 mm. of its origin (Fig. 3B) the main trunk enlarged to an outside diameter of 2.1 by 0.9 cm. Adherent to the rigid intima was a film of dry, blackish blood clot and even at this point the inferior half of the lumen was occluded by a laminated brownish gray thrombus. The vessel wall was from 1 to 3 mm. thick and fused with the aortic wall. Six mm. distal to the point just described (Fig. 3C) a marked change occurred. The coronary artery was now 2.3 cm. in one diameter and 2 to 4 mm. in the other. The wall had a thickness of 1 to 2 mm. and

over the superior part the lining was coated with blackish blood. Inferiorly the vessel came to a sharp point and here the thrombus observed in the previous block plugged the lumen. In the middle of this segment was the ostium of the circumflex branch of the left coronary artery, also occluded by the thrombus. It must be understood, then, that the dimensions given above represent not only the main coronary artery but also the beginning of its circumflex branch, and because this was cut tangentially the lower part of its lumen appeared pointed. On the right side of the coronary artery lay the Valsalva aneurysm containing a laminated thrombus, most of which dropped out in the process of sectioning. The coronary artery and the Valsalva aneurysm were separated by a hard and fibrous wall only 2 mm. thick. Four mm. distally (Fig. 3D), measuring along the coronary artery, was the beginning of the anterior interventricular branch. The main left vessel was still rather larger than normal, with an external diameter of 1.1 by 0.7 cm., but had decreased appreciably from its size in the preceding block. Its lumen had an undulating outline and the intima was covered by a thin, blackish film. The common wall dividing the artery and the Valsalva aneurysm appeared wholly fibrous and was 2 to 3 mm. thick. At this point the Valsalva sac measured 2.3 cm. in its transverse diameter, 3.4 cm. in the supero-inferior direction and had a wavy and whitish border. In the myocardium of the left ventricle forming the lower border of the aneurysm were many engorged capillaries within a whitish scar. In this block the circumflex artery had fully emerged as an independent vessel and exhibited nothing abnormal. The thrombus occluding the beginning of this vessel did not continue further along its course. The myocardium under the coronary aneurysm was only 6 mm. thick. The anterior interventricular branch, 5 mm. distal to the point previously described (Fig. 3E), was shaped like a bowling pin, with maximum external diameters of 1.2 and 0.6 cm. Its lumen was entirely closed by a dry, blackish clot. The same common wall, still 2 mm. in thickness, separated the vessel and the Valsalva aneurysm, which now measured 3.5 by 2.5 cm. The latter had become more superficial and was underlaid by 1.1 cm. of myocardium. The myocardial scar mentioned above continued into this block. The outside dimensions of the coronary artery were now 7 and 9 mm. In the next block (Fig. 3F), having a thickness of 5 mm., the Valsalva aneurysm had practically left the myocardium

and had come to lie chiefly in the subepicardial fat. Its dimensions were decreasing, being 3 by 2 cm. The wall of the sac and the thrombus contained within it were identical with previously mentioned blocks. The myocardial scar, however, had practically disappeared. The coronary artery and the Valsalva aneurysm continued to share a common wall and the thrombus occluding the lumen of the artery in more proximal blocks was still present. The external diameters of the vessel were the same as in the preceding block although the contour was different. Within the next 5 mm. (Fig. 3G) the Valsalva aneurysm had fully emerged from the myocardium, lay wholly in the subepicardial fat and decreased in size to 2.3 by 1.8 cm. The walls of the anterior interventricular artery and the aortic sinus aneurysm were still in apposition but no longer fused into one. The thrombus mentioned previously continued and the coronary artery measured 5 by 7 mm. Three mm. distally (Fig. 3H), the Valsalva aneurysm terminated blindly in the subepicardial fat, 4 mm. beneath the epicardium, and had not ruptured. The relations of the two aneurysms are shown clearly in the wax reconstruction (Fig. 2).

Microscopic Examination

Microscopically the clots filling the Valsalva sacculation and the aneurysm of the left coronary artery prove to be typical laminated thrombi exhibiting some softening but no organization.

The wall of the Valsalva aneurysm consists almost entirely of hyalinized fibrous connective tissue in which the Van Gieson and Verhoeff stains display persisting remnants of both smooth muscle cells and elastic tissue. Numerous partially or completely obliterated vasa vasorum are collared by abundant plasma cells and lymphocytes. The medial and intimal divisions are not clearly distinguishable on account of the great distortion and fibrosis, while in the inner part of the wall there are extensive atherosclerotic changes and calcification. The common wall separating the Valsalva and coronary artery aneurysms is made up of hyalinized fibrous tissue and granulation tissue containing fragments of elastic and smooth muscle tissue. Here also are small vessels showing obliterative changes and perivascular cuffs of lymphocytes and plasma cells. It appears obvious that this common wall represents a fusion of the walls of the aneurysmal aortic sinus and the left coronary artery.

The left coronary artery wall exhibits changes identical with those just described for the Valsalva aneurysm and these are depicted in Figure 4. The myocardium subjacent to the aortic sinus aneurysm gives histological evidence of pressure atrophy, hyaline degeneration, interstitial fibrosis and contains numerous small and engorged blood vessels. Other sections of the myocardium reveal a distinct hypertrophy of the muscle cells with deposits of lipochrome pigment at either end of the nuclei, slight patchy interstitial fibrous connective tissue increase and multiple small areas of anemic necrosis in blocks coming from the anterior wall of the left ventricle. The intima of the ascending aorta is the seat of typical atherosclerosis with calcification, while the media displays extensive destruction of smooth muscle and elastic tissue. The vasa vasorum of both media and adventitia are greatly narrowed or obliterated by endothelial proliferation and intimal fibrosis and are collared by plasma cells and lymphocytes. Some of these accumulations show early necrosis and are regarded as actual miliary gummas.

CASE 2. *Syphilitic Aneurysm of Aortic Valsalva Sinus:* The patient, a white male, 60 years old, spoke and understood so little English that an adequate history was unobtainable. He had been working "in the woods," presumably at logging, until about the first of November, 1932, when he became short of breath, progressing to orthopnea within 2 months. About the first of January, 1933, there developed a continuous, dull, distressing pain in the epigastrium and right upper abdomen. Swelling of the ankles appeared about a month later. He was weak, "nervous," could not sleep, and had been confined to bed most of the time since the appearance of the edema. Occasionally he had noted substernal distress and tinnitus. He was admitted, walking, to Multnomah County Hospital on Feb. 21, 1933.

On admission the temperature was 95 F, pulse 88, respiration 24 and the blood pressure 160/35. Physical examination revealed slight pallor and cyanosis of the finger nails, but no demonstrable capillary pulse. The retinal blood vessels pulsated and the optic discs appeared hazy. The neck vessels pulsated markedly with systole. The chest was slightly emphysematous. The cardiac impulse was diffuse and heaving. There was slight impairment to percussion between the right scapula and the spine, with bronchovesicular breathing and a to-and-fro murmur. Elsewhere the lung fields seemed normal. To percussion the heart was of the "aortic" configuration, the arch 6 cm. wide, and the left and right borders respectively 14 and 6 cm. from the midsternal line. A loud to-and-fro murmur was audible over all valve areas, loudest at the aortic and mitral areas and transmitted to the axilla and through to the back. No distinct valve tones could be heard. The peripheral vessels were thick-walled and pulsated forcibly. The abdomen was distended. The liver border was 6 cm. below the costal margin and was felt also in the epigastrium. The legs were very edematous.

The urine was negative. Except for a sedimentation rate of 13 mm. in 15 minutes and 37 mm. at the end of 45 minutes (modified Westergren method), and 4 plus Kolmer and Kahn reactions, examination of the blood yielded results within normal limits.

An electrocardiogram taken on the day of admission showed the auricular and ventricular rates to be each 71. T_1 was inverted, R_1 slurred, Q-S prolonged and the P-R interval prolonged to 0.27 second. T_2 was inverted, R_2 slurred, and P-R prolonged. R_3 was slurred and notched, P_3 and T_3 were questionably diphasic and R_3 of low amplitude. The interpretation was: "Delayed A-V conduction, coronary type T-waves, myocardial damage."

Under a regimen of bed rest, digitalis, sedatives, bismuth and iodides, the patient improved and 3 days later the peripheral edema was gone. On February 27th a roentgenogram of the chest showed increased hilum shadows, pleural thickening on the right, with obliteration of the costophrenic angle, and a greatly enlarged cardiac shadow with a blunt apex and a somewhat widened and sclerotic arch.

By March 2nd there was no dyspnea or cyanosis. The man's condition remained unchanged until April 15th, when he had diarrhea and abdominal distress. Six days later he suddenly became cyanotic and died.

The final clinical diagnoses were: (1) Aortic regurgitation on the basis of syphilitic destruction of the aortic ring; syphilitic aortitis; hypertrophy and dilatation of the right and left heart with cardiac failure, functional capacity II–B, and peripheral edema. (2) Mild chronic hypertrophic emphysema.

Postmortem Examination

No. 163–4–33. Examination of the abdomen revealed slight ascites, a non-specific diphtheritic, hemorrhagic and ulcerative enterocolitis and proctitis, and chronic passive hyperemia of the liver with acute periportal hepatitis, and chronic passive congestion of the spleen and kidneys.

The left pleural cavity contained about 3 liters of clear fluid, which compressed the lung inferiorly and posteriorly. The right lung was bound to the chest wall by dense adhesions at its apex, and laterally and inferiorly over its middle and lower lobes. Some of these adhesions, which were fibrous in nature, obliterated the right costophrenic sinus and encapsulated some yellowish white, cheesy and hyaline material. The space occupied by the heart was greatly increased, having a maximum transverse diameter of 19 cm. All cardiac chambers were much dilated and more than half of the anterior presenting surface was formed by the right ventricle and atrium. The papillary muscles and trabeculae carneae of both ventricles were appreciably elongated, thickened and also flattened.

With the heart opened there could be seen immediately inferior to the pulmonic valve a rounded, bulging area forming a sort of

shelf on the septal wall of the right ventricle and producing some stenosis immediately proximal to the valve. Over the inferior half of this projection the endocardium was pearly white and the superior border of the whitened area was quite sharp, due to the fact that at this point the interventricular septum had been reduced to a hyaline state and formed one wall, here only 2 mm. thick, for the Valsalva sinus aneurysm to be described in more detail presently. Several of the chordae tendineae of the anterior cusp of the tricuspid valve were attached to the aneurysm wall which lay immediately anterior and to the left of this valve. The aortic valve measured 9 cm. in circumference and its leaflets were somewhat rigid, with thickened and rolled margins. The commissures between the leaflets were widened, the distance between the right and left posterior leaflets being 1 mm., while the commissures separating the anterior and left posterior leaflets and the anterior and right posterior leaflets were each 3 mm. At a point 4 cm. superior to the aortic ring the aorta had a circumference of 12.5 cm. The ostium of the right coronary artery was slit-like, measured 1 by 2 mm. and was situated 6 mm. superior to the tip of the anterior aortic leaflet on the superior aspect of the shelf-like margin of a semilunar aneurysm 2 by 1.3 cm. in size, which occupied the right posterior sinus of Valsalva and extended into the interventricular septum for a distance of 2.8 cm. (Fig. 5). In its development the aneurysm left behind a narrow and firm ridge separating it from the attachment of the right posterior aortic leaflet. The lining of the sac was rough, grayish white, mottled by yellowish atheromatous plaques, of cartilaginous firmness and displayed over a part of its blind end a thin, grayish red thrombus. The wall forming the blind end lay in a concavity hollowed out of the muscular interventricular septum. The pars membranacea septi appeared to have been displaced to the left to form most of the left wall of the aneurysm, which, however, did not stop here but continued downward, cupping out for itself a bed in the muscular interventricular septum. Along the right border of the shelf bearing the right coronary orifice was a deep and narrow vertical groove communicating directly with the Valsalva aneurysm. The ostium of the left coronary artery measured 2 by 3 mm. and opened 2 cm. superior to the upper boundary of the left posterior aortic leaflet. Serial cross-sectioning of the coronary arteries revealed them to be macroscopically unchanged. The wall of the en-

tire aorta was irregularly thickened and distorted and the vessel was quite tortuous. The intima, from the root to the bifurcation, was whitened, thickened and wrinkled longitudinally. Toward the bifurcation was an increasing amount of atheromatous change with ulceration and calcification. The vessel cut with leathery resistance and was unduly adherent to the structures about it. In the anterior wall of the abdominal aorta was a small saccular outpouching. The heart and entire aorta weighed 940 gm. The myocardium of both ventricles was distinctly hypertrophied and pinkish, with a slight yellowish mottling. The anterior wall of the left ventricle seemed to be elongated. Fibrotic patches were noted at the tips of the various papillary muscles. The mitral valve ring was 11.5 cm. in circumference and its leaflets, particularly the anterior one, were flecked by atheromatous patches.

In the lower lobe of either lung were several rubbery, yellowish to grayish, sharply circumscribed, grouped nodules with polycyclic outlines. The bronchial and pulmonary arterial walls were thickened and some of the latter displayed atheromatous plaques.

Microscopic Examination

Microsections of the septal portion of the Valsalva aneurysm reveal a partially organized and partly softened thrombus at its base. The wall of the sac is formed by a thick layer of hyaline material showing atheromatous changes and hemosiderin deposits. Blending with this is a layer of vascular granulation and fibrous scar tissue containing numerous partially or completely obliterated arteriolar channels surrounded by broad collars of lymphocytes and plasma cells. By means of the Verhoeff stain fragments of degenerating elastic tissue are identified, proving that this portion of the sac represents remnants of aortic media. Separating this layer and the septal myocardium is a thin zone of connective tissue, probably representing aortic adventitia, containing a number of tiny vascular channels, apparently veins. Farther out are degenerated cardiac muscle cells, isolated or split up into small groups and compressed by dense fibrous tissue. On account of the formalin fixation and the time elapsing between death and autopsy, special stains to demonstrate the cells of the atrioventricular bundle were not employed.

Sections of the aorta, taken at various levels from the root to the bifurcation, all exhibit essentially the same changes, namely, irregular thickening, fibrosis, puckering and distortion of all layers, with extensive fragmentation and destruction of medial smooth muscle and elastic tissue with partial replacement of these by fibrous connective tissue of varying age. The aortic vasa vasorum display all degrees of obliterative endarteritis and are surrounded by varyingly dense collars of lymphocytes, plasma cells, Russel-Plimmer bodies and occasional polymorphonuclear eosinophiles. Certain of the cellular accumulations show slight necrosis. The adventitia is the seat of similar changes.

Sections of myocardium from either ventricle exhibit marked hypertrophy and deposition of pigment at the nuclear poles with distinct cross-striations in some cells and an absence of these in others. A few of the cells appear to be atrophic. The interstitial tissue seems to be slightly edematous, a little increased in places and contains occasional lymphocytes and plasma cells but no cellular aggregations resembling gummas. The smaller branches of the coronary arteries are unchanged. Frozen sections stained with scharlach R fail to show any fat in the myocardium.

Microscopic examination of the lower lobes of the lungs reveals compression atelectasis, chronic passive hyperemia, edema, slight emphysema, and numerous pneumoliths. Here and there, in relation to bronchi or larger blood vessels are nodules composed of plasma cells and lymphocytes, surrounded by vascular granulation tissue containing many of these cells. Large areas of incomplete caseation necrosis in which the architecture of lung tissue and blood vessels can still be recognized are present. The necrotic tissue stains pinkish with eosin and there is no evidence of calcification or persistence of nuclear fragments, nor are any giant cells or epithelioid cells seen. No structures resembling daughter tubercles are in evidence. At the edges of such lesions the arterioles have undergone extensive obliterative inflammation. The larger branches of the pulmonary arteries are atheromatous and proliferative intimal changes have reduced their lumens to some extent. In places their walls contain a moderate number of lymphocytes, plasma cells, and rarely eosinophilic and neutrophilic polymorphonuclears. The right costophrenic pleural thickening consists of hyalinized fibrous

tissue and granulation tissue containing patchy accumulations of lymphocytes and plasma cells, forming a capsule about amorphous and hyaline material in which there is much cholesterin.

Discussion

In addition to the previously recognized causes for the development of aneurysm of the coronary arteries, such as mycotic-embolic infection and arteriosclerosis, we offer another, namely, syphilitic arteritis. In so doing we are cognizant of the truth of the generally accepted belief that the coronary arteries are only rarely affected by syphilis distal to their intra-aortic segments. However, in Case 1 there exists a most unique pathological condition which modifies the usual circumstances to such an extent that we have no hesitancy in terming the coronary lesion syphilitic. Undoubtedly the involvement of the left coronary artery was dependent first upon the localization of an active syphilitic aortitis in the left posterior sinus of Valsalva, and secondly the direction of burrowing of the enlarging sac which finally brought it into intimate contact with the main left coronary artery. There must then have been a spread of the *Spirocheta pallida* from the wall of the Valsalva sac to the wall of the coronary artery, with resultant destruction, fusion of the walls of the two juxtaposed structures and, finally, the formation of a true aneurysm in the weakened coronary artery.

As evidence of the syphilitic nature of the lesion we submit the microscopic observation of obliterative endarteritis of the vasa vasorum, perivascular collars of plasma cells and lymphocytes, microscopic sized gummas, destruction and scarring of the media and adventitial fibrosis, all of which are recognized as characteristic of syphilis by Warthin [17] and Moritz.[18] A careful study of the aorta, coronary arteries and myocardium failed to disclose anything that could be interpreted as rheumatic disease, a possibility which, in view of the newer histopathology of rheumatism, as described by Klinge and Vaubel,[19] and others, must be kept constantly in mind in the study of vascular lesions. It should be mentioned also that the vascular changes in the coronary artery of Case 1 were quite different from the commonly observed adventitial cellular infiltration accompanying coronary arteriosclerosis.

Another feature of this case makes it doubly interesting, for in addition to aneurysm of the coronary artery there was recent thrombotic occlusion of the sacculation and the lumen of the vessel adjacent to it. Closure of a main coronary artery near the heart would lead to serious consequences, even in a healthy organ, and in this instance would be even more embarrassing on account of the previously existent and plainly evident aortic insufficiency and dilatation of the left ventricle. Although the record and identifying number of the specimen are missing there is every reason for believing that coronary thrombosis was the terminal event of this person's life. Death evidently supervened shortly after the thrombus formed, for only the earliest indications of infarction of the myocardium were present.

While Vogelsang's [5] example of coronary aneurysm may well have been due to syphilis, one cannot be certain that such was the case because the vessel was not studied histologically. In our case, although spirochete stains were not done, it is felt that the evidence of syphilis is indisputable. With the possible exception of Vogelsang's case the present example appears to be the first of its kind thus far recorded.

In Case 2 it is possible that the incipient heart block, not evident clinically but suggested by the prolonged atrioventricular conduction time and the inverted T-waves of the electrocardiogram, may have been caused by digitalis, but we feel that the Valsalva aneurysm, on account of its size and anatomical relation to the atrioventricular bundle, together with degenerative and fibrotic changes in the adjacent myocardium, afford a more plausible explanation for these phenomena. Another factor contributing to cardiac failure was aortic insufficiency, which not only threw additional strain on the left ventricle but also failed to allow sufficient blood to enter the stenosed coronary ostia to nourish the myocardium properly. The right cardiac hypertrophy probably was due to increased pressure in the lesser circulation bed, due both to the stenosis of the pulmonary valve region by the Valsalva sinus aneurysm and also to pulmonary atherosclerosis which, in turn, probably followed increased circulation pressure from left heart failure on the basis of aortic insufficiency and stenosis of the mitral area from the Valsalva aneurysm. The presence of plasma cells and lymphocytes in the walls of the pulmonary arteries suggests that the sclerosis of these vessels may, in part at least, have been the result of syphilis.

The lesions in the bases of the lungs exhibited the characteristics of gummas and bore scarcely any resemblance to tubercles. Although the spirochete stains failed to demonstrate the organism it is our opinion, from the histological structure of the lesions and the known syphilitic nature of the aortic disease, that the pulmonary foci are very probably luetic as well.

The aortic involvement was much more extensive than is usual in syphilis and it is interesting to note that a second saccular aneurysm had developed in the abdominal division of the vessel.

Summary

Two cases of syphilitic aneurysm of the aortic sinuses of Valsalva with unusual complications are described. Such sacculations are distinctly uncommon, forming only 0.93 per cent of the aortic aneurysms in our series of 5896 autopsies.

In 1 case the aneurysm burrowed through the ventricular myocardium until it reached the left coronary artery, where a secondary syphilitic arteritis was established, leading first to aneurysm of the coronary artery and finally to acute thrombotic occlusion. This is the 34th case of coronary aneurysm to be recorded and apparently the first to have a syphilitic etiology.

The uncommon manifestation of Valsalva aneurysm in the second case was incipient heart block, dependent upon the proximity of the sac to the atrioventricular bundle.

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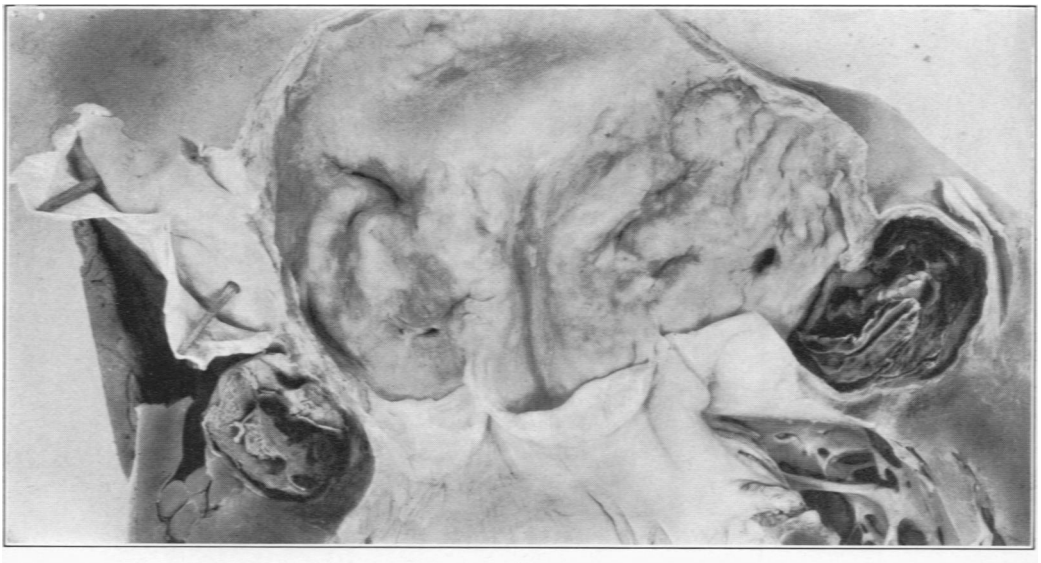
DESCRIPTION OF PLATES

PLATE 169

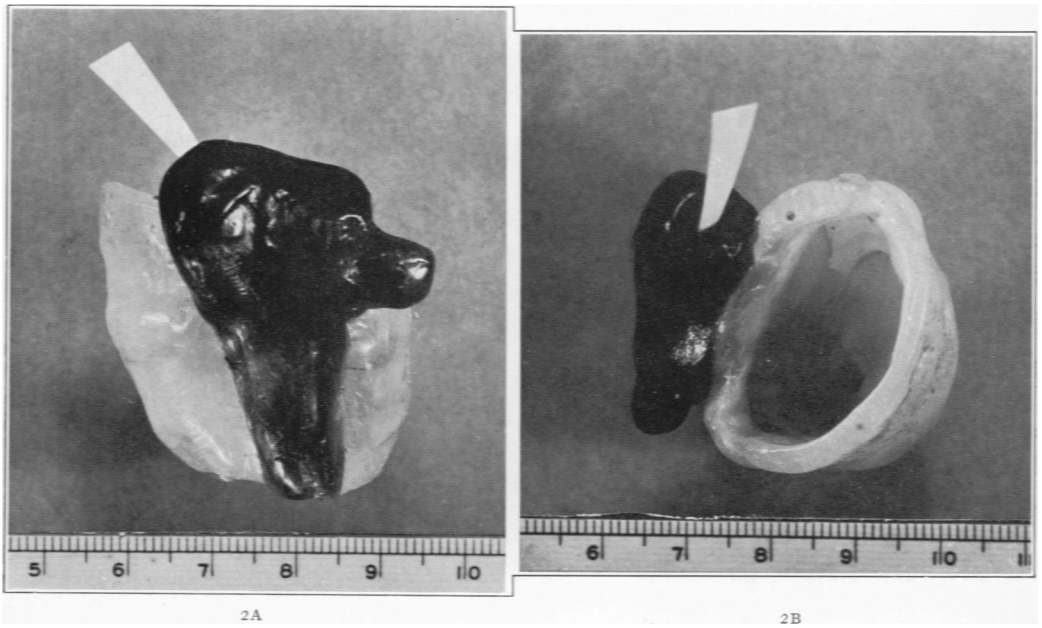
FIG. 1. Photograph of a portion of the left ventricle and aorta of Case 1. To the left are two right coronary ostia and a portion of the thrombosed Valsalva aneurysm. Above and to the extreme left are the right and left anterior pulmonic leaflets held out by props. To the right is the left coronary ostium and the Valsalva aneurysm showing its direct connection with the aorta and its roof formed by the beginning of the pulmonary artery. The groove to the right of the sac lies between the lateral wall of the pulmonary artery and the left anterior leaflet of the pulmonic valve.

FIG. 2A. Wax reconstruction of Valsalva (white) and left coronary (black) aneurysms viewed anteriorly. Natural size. A white marker has been placed in the ostium of the artery. To the right is the beginning of the circumflex branch. The flatly oval shape of the coronary aneurysm is well shown in this view.

FIG. 2B. Wax model viewed from above showing the cavity of the Valsalva aneurysm. The white area toward the base of the coronary artery is a highlight.



1



2A 2B

PLATE 170

FIG. 3. Case 1. Drawing of the two aneurysms. Actual size. The distal face of each segment excepting Block 1 is depicted. Thus if H were superimposed upon G and so on, the aneurysms would appear as they were before sectioning.

FIG. 3A. Ostium of left coronary artery.
FIG. 3B. Block 5 mm. distal to ostium of left coronary artery.
FIG. 3C. " 11 mm. " " " " " " "
FIG. 3D. " 15 mm. " " " " " " "
FIG. 3E. " 20 mm. " " " " " " "
FIG. 3F. " 25 mm. " " " " " " "
FIG. 3G. " 30 mm. " " " " " " "
FIG. 3H. " 33 mm. " " " " " " "

a, ostium of left coronary artery; *b*, pulmonary artery; *c*, left atrium; *d*, mitral valve cusp; *e*, Valsalva aneurysm; *f*, left main coronary artery; *f'*, anterior interventricular branch of left coronary artery; *f''*, circumflex branch of left coronary artery; *g*, left ventricular myocardium; *h*, myocardial scars; *x x'*, and *x' y*, indicate boundaries of areas from which blocks for microscopic study were taken.

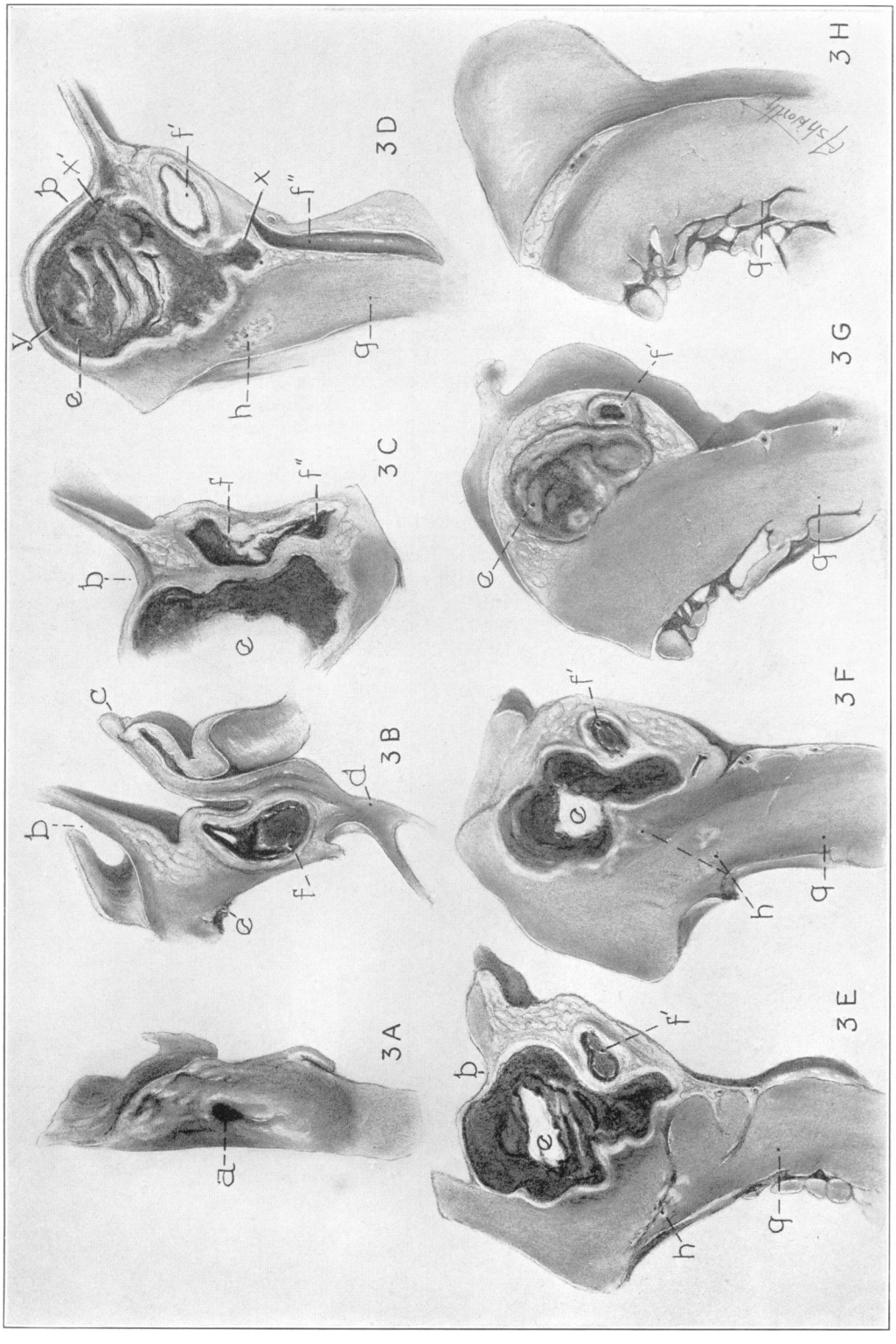
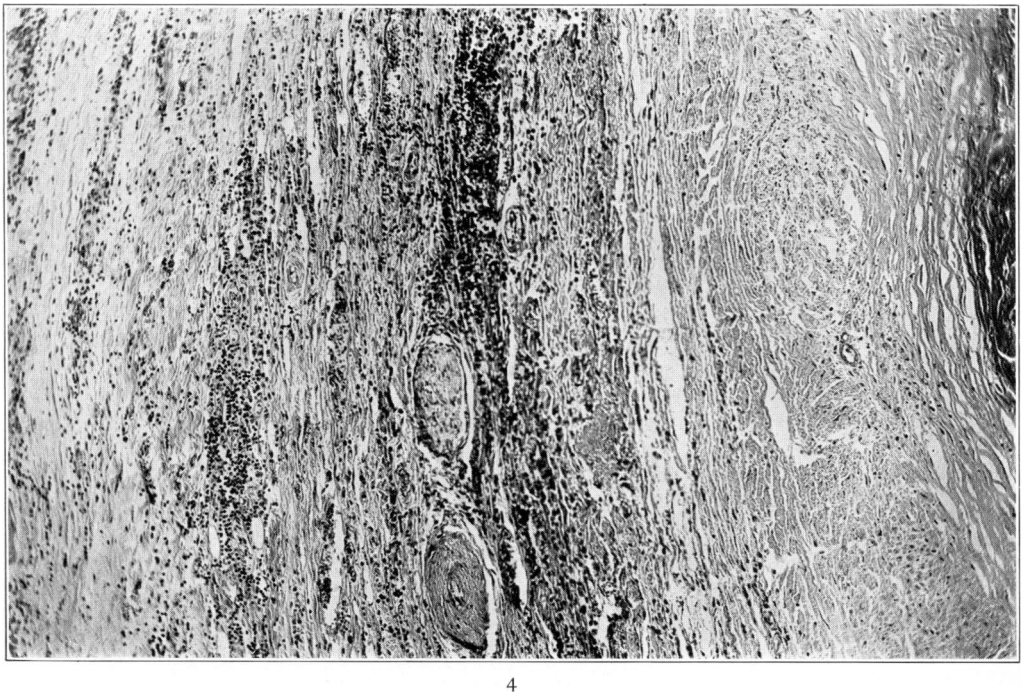

3A
3B
3C
3D
3E
3F
3G
3H
a
b
c
d
e
f
f'
f''
g
h
x
x'
y

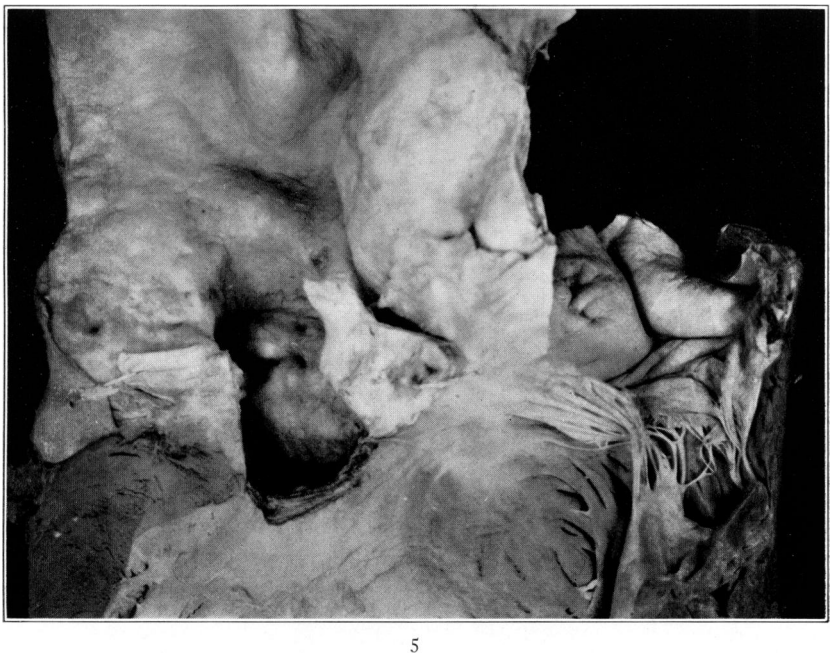
PLATE 171

FIG. 4. Case 1. Low power photomicrograph showing the intima, media and a small portion of the adventitia from the left coronary artery aneurysm near the point where it fuses with that of the sinus of Valsalva. Along the right margin is a small part of the occluding thrombus. The intima is thickened and contains a small capillary. Much of the media has been destroyed and is heavily infiltrated with lymphocytes and plasma cells. A nutrient vessel with marked narrowing of its lumen is shown and above it is a tangentially cut nerve. Note also the cellular infiltration and fibrosis of the adventitia at the extreme left.

FIG. 5. Case 2. View of the Valsalva aneurysm after opening the left ventricle and aorta. A part of the wall of the aneurysm has been cut away and turned to the right in order to display the bed of the sac in the interventricular septum. Near the left border of the aorta is the orifice of the left coronary artery. Note the thickening and distortion of the intima in the ascending aorta.



4



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