Syphilitic Aortitis

A Clinicopathologic Autopsy Study of 100 Cases,

1950 to 1960

By H. Alexander Heggtveit, M.D.

The pathogenesis of syphilitic aortitis has been recognized for decades and its pathologic features are well documented. Although declining in incidence, it still accounts for the largest single group of aortic inflammatory lesions.

In recent years a diagnosis of syphilitic cardiovascular disease is rarely made. Even in the presence of positive serologic tests and overt clinical manifestations, physicians are often reluctant to diagnose syphilis. Whether the reasons for this are social or scientific, the fact remains that antibiotic therapy has not eradicated the disease. Old, untreated cases still come to light, and tertiary lesions persist in many treated persons. The indiscriminate use of penicillin has masked many early stigmata of syphilis. In addition, a number of reports during the past few years have shown an alarming resurgence in the incidence of primary syphilis.

An accurate clinical diagnosis was established in only 17 per cent of a large group of cases of syphilitic aortitis coming to autopsy from 1950 to 1960. The purpose of this report, a review of these cases, is to emphasize the fact that the cardiovascular manifestations of tertiary syphilis are still a significant, though uncommon, cause of morbidity and mortality.

Materials and Methods

The necropsy files of the Kings County Hospital Center, Brooklyn, New York, a large municipal general hospital of 3,000 beds, were surveyed for the period from January 1, 1950 to December 31, 1960. During this time necropsies were performed on 13,082 patients of all ages including stillbirths. Of these, approximately 55 per cent were male and 45 per cent female. Sixty per cent were white and 40 per cent were Negro.

All examples of aortitis were reviewed. Possible cases of rheumatic or rheumatoid aortitis were carefully studied and excluded from the series, as were atherosclerosis, medial necrosis of Erdheim, and others, unless there was conclusive evidence of syphilitic aortitis as well. A total of 100 cases were found that satisfied the generally accepted morphologic criteria of syphilitic aortitis.

The clinical charts and autopsy protocols were scrutinized in each instance and all pertinent data recorded. Photographs of the aorta and heart supplemented the gross descriptions in 40 cases. Many of the gross specimens were mounted in museum jars or preserved in formalin-filled crocks and were available for study. A minimum of two and as many as 20 (average six) microscopic sections of the aorta were available for examination in every instance. Elastic tissue stains (Verhoeff-Van Gieson) were done on the majority of the aortas. Levaditi stains for spirochetes were done in several instances, but since the demonstration of Treponema pallidum in histologic sections of tertiary aortic lesions is rarely accomplished, no concerted attempt was made to do so in all cases.

Results

Incidence

Of the 100 cases of syphilitic aortitis, 57 were white (43 male, 14 female) and 43 were Negro (30 male, 13 female). The age-sex distribution is given in figure 1. The youngest patient was 30 and the oldest 92 years of age. The average age, 63.3 years, was the same for men and women. The over-all crude autopsy incidence of syphilitic aortitis was 0.76 per cent.

History and Therapy

A history of treated syphilitic infection was
SYPHILITIC AORTITIS

recorded in only 23 cases. Of these, 11 had received bismuth or arsenicals, seven had been treated with penicillin, and 5 had received both penicillin and metallotherapy. In addition, 36 patients, not specifically treated for syphilis, received penicillin for other reasons, and 15 more were given a variety of other antibiotic agents. Histories were inadequate in 13 instances because of the patients’ impaired mental status, state of consciousness, or lack of previous medical records.

Serology

Serologic examination (VDRL and Kolmer) of the blood was positive in 40 instances and negative in 28. In 32 cases the serologic tests for syphilis were either not performed or the results were not available. It should be noted that 15 to 30 per cent of patients with aortic syphilis have negative serologic tests.13 Lumbar puncture was done in 27 instances. In only two of these was the serologic examination of the spinal fluid positive; both of these patients had neurosyphilis.

Classification of Cases

From a clinical and pathologic point of view, syphilitic heart disease may be conveniently subdivided into four categories: uncomplicated syphilitic aortitis, syphilitic aortic aneurysm, syphilitic aortic valvulitis with insufficiency, and syphilitic coronary ostial stenosis. The term “uncomplicated” implies the absence of the other three complicating syphilitic lesions, i.e., aneurysm, insufficiency, and ostial stenosis. A fifth category, syphilis of the myocardium, is extremely rare and no examples were found in this group of cases.

Thirty-six cases in this series were considered to be “uncomplicated.” The remaining 64 cases exhibited one or more complications as indicated in table 1.

Symptoms and Signs

Some of the major clinical manifestations are tabulated in table 1. The duration of symptoms varied from several hours to 25 years. It is noteworthy that 50 per cent of the patients had symptoms referable to the cardiovascular system for 1 year or less.

Forty-three patients had well-documented histories of hypertension or consistently elevated blood pressure recordings. Persons with either systolic pressures over 150 mm. Hg, or

![Figure 1](http://circ.ahajournals.org/)

**Figure 1**

Age-sex distribution of 100 cases of syphilitic aortitis.
### Table 1

**Major Clinical Manifestations in 100 Patients with Syphilitic Aortitis**

<table>
<thead>
<tr>
<th>Category</th>
<th>Number of cases</th>
<th>No symptoms or signs</th>
<th>Precordial pain</th>
<th>Hypertension</th>
<th>Cardiac enlargement</th>
<th>Congestive heart failure</th>
<th>Accentuated aortic second sound</th>
<th>Systolic, apical</th>
<th>Systolic, aortic</th>
<th>Diastolic, aortic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uncomplicated aortitis</td>
<td>36</td>
<td>11</td>
<td>7</td>
<td>14</td>
<td>14</td>
<td>16</td>
<td>9</td>
<td>12</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Aortic aneurysm</td>
<td>23</td>
<td>1</td>
<td>11</td>
<td>12</td>
<td>11</td>
<td>12</td>
<td>13</td>
<td>8</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Aortic insufficiency</td>
<td>6</td>
<td>0</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>5</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Coronary ostial stenosis</td>
<td>11</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>3</td>
<td>7</td>
<td>3</td>
<td>7</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Aneurysm and insufficiency</td>
<td>9</td>
<td>0</td>
<td>6</td>
<td>3</td>
<td>4</td>
<td>6</td>
<td>4</td>
<td>2</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Insufficiency and ostial stenosis</td>
<td>7</td>
<td>0</td>
<td>2</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Aneurysm and ostial stenosis</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Aneurysm and insufficiency and ostial stenosis</td>
<td>7</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>100</strong></td>
<td><strong>14</strong></td>
<td><strong>35</strong></td>
<td><strong>43</strong></td>
<td><strong>46</strong></td>
<td><strong>55</strong></td>
<td><strong>35</strong></td>
<td><strong>38</strong></td>
<td><strong>21</strong></td>
<td><strong>16</strong></td>
</tr>
</tbody>
</table>
diastolic pressures exceeding 90 mm. Hg, or both, were considered to be hypertensive.

Paroxysmal nocturnal dyspnea was a prominent clinical finding in only 10 instances, including three patients with uncomplicated aortitis. Aneurysmal pressure effects such as dysphagia, tracheal tug, and hoarseness were found in 11 patients with aortic aneurysm. Thirteen cases of aortic insufficiency exhibited a variety of features other than those noted in Table 1 and widened pulse pressures. These included "water-hammer" pulse, "pistol-shot" arterial sounds, capillary pulsation, Duroziez's sign, and precordial thrill.

Venous pressures were elevated and circulation times were prolonged in 18 of 23 patients in whom these procedures were carried out. Cardiac decompensation was present in every one of these 18 cases.

Roentgenographic Findings

Seventy-one patients had chest x-rays reported. Of these, 43 had radiographic evidence of cardiomegaly and 21 showed calcification of the thoracic aorta. Chest films were performed in 34 of 40 patients with aortic aneurysms. A radiologic diagnosis of aneurysm was made in 14. An additional seven cases were described as showing dilatation, widening, or uncoiling of the aorta. The diagnosis of aneurysm was confirmed by barium swallow in three cases, by angiography in two instances, and by both of these technics in another case. An angiogram failed to demonstrate the aneurysm in one instance, as a result of mural thrombosis in the lesion. Aneurysmal shadows, visualized radiographically in 3 other cases, were interpreted as mediastinal neoplasms. One of these patients received radiotherapy.

Electrocardiographic Findings

Electrocardiograms were made in 72 patients. Abnormal tracings were obtained in 60 cases. Electrical evidence of myocardial ischemia was found in 43, left ventricular preponderance in 21, and right ventricular preponderance in five cases. Bundle-branch block was observed in seven patients and various degrees of atrioventricular block in six. Fourteen cardiograms showed atrial fibrillation. Premature ventricular contractions were noted in 13 and premature atrial contractions in seven patients.

Pathologic Features

It is not within the scope of this paper to give a detailed account of the pathologic alterations in syphilitic aortitis. Certain morphologic aspects of this group of cases warrant special attention, however.

Microscopically, all cases showed chronic mesoaortitis with patchy destruction of musculo-elastic medial tissue and replacement by vascularized connective tissue. Endarteritis obliterans of vasa vasorum with perivascular plasma-cell and lymphocytic "cuffing," adventitial fibrosis, and intimal atherosclerosis were present in all cases. Microgummas were noted in 20 aortas and giant cells were a prominent feature in three cases.

The changes were invariably found in the ascending portions of the aortas and extended distally for varying distances. In only nine instances was the abdominal aorta affected, and in seven of these the lowermost extent of the lesions remained above the level of the renal arteries. In 66 cases the intimal surfaces of the thoracic aortas exhibited the characteristic wrinkles, ridges, and pearly plaques of syphilitic aortitis. Aortic atherosclerosis was apparent in all cases and undoubtedly obscured the classical "tree bark" appearance in many instances. The degree of coexisting atherosclerosis was categorized as minimal in 11 cases, moderate in 28, and severe in 55 aortas. In six cases the grade was not indicated. The severity of the atherosclerotic process in the thoracic aorta was stated to be equal to or greater than the extent of abdominal atherosclerosis in 60 cases. In 24 instances the reverse was true. In 16 cases the relative intensity of the atheromatous aortic lesions was not recorded. Grossly visible calcification of the thoracic aorta occurred in 37 cases and intramural calcium deposition was seen microscopically in another 33 instances.

In none of the aortas was mention made of significant stenosis or narrowing of the brachiocephalic branches by either the atherosclerotic or the syphilitic process.
Aortic Aneurysm

Aneurysm of the aorta was the most common complication of syphilitic aortitis. A total of 51 aortic aneurysms were found in 40 patients. Thirty-three individuals had solitary aneurysms and seven had multiple lesions. The segmental localization of the 51 aneurysms is given in Table 2. Excluding the four aneurysmal outpocketings of the sinuses of Valsalva, 34 of the aneurysms were saccular and 13 had a fusiform configuration. Twenty-two saccular aneurysms and four of the fusiform type contained laminated mural thrombus. On the basis of microscopic examination, the four aneurysms of the lower abdominal aorta were considered to be primarily atherosclerotic in origin.

Aneurysmal rupture with fatal hemorrhage occurred in 14 patients. The left pleural cavity was the site of rupture in five instances; two of these also entered the esophagus, which alone was eroded in two other cases. Rupture took place into the mediastinum three times and into the retroperitoneal tissues twice. The trachea and right pleural cavity were each penetrated once.

Bony erosion, a result of aneurysmal pressure and pulsation, was noted in three cases. The vertebral bodies were involved twice, the sternum and ribs once.

In addition to those with actual aneurysm formation, 17 cases had nonaneurysmal ectasia or diffuse dilatation of the thoracic aorta.

Aortic Insufficiency

Syphilitic aortic valvulitis with insufficiency was found in 29 cases. Anatomic evidence of aortic valvular incompetence included dilatation of the aortic ring (circumference greater than 8.5 cm.) or widening of the valve commissures, with or without sclerosis or rolling of the cusps. Additional and confirmatory findings were eccentric hypertrophy of the left ventricle with endocardial thickening and pseudovalve formation. Aortic insufficiency was caused primarily by dilatation of the ring in eight cases, to widening and distortion of the commissures in 12, and to both annular dilatation and commissural separation in nine instances.

Coronary Ostial Stenosis

Stenosis of one or both coronary ostia occurred in 26 cases. This narrowing was the result of extension of the syphilitic process in the first portion of the ascending aorta to involve the orifices of the coronary arteries. The subsequent encroachment on the coronary lumen by sclerotic, subintimal, aortic plaques produced the ostial stenosis. The right coronary ostium was affected five times, the left six times, and both orifices were stenosed in 15 cases. The degree of narrowing was considered to be moderate in 11 instances and marked in 15. Mild degrees of narrowing were not included. True syphilitic coronary arteritis distal to the ostia is rare and was found in only one case.

Cardiac Hypertrophy

The hearts ranged in weight from 240 to 1,000 Gm. (average 476 Gm.). Cardiac hy-

### Table 2

<table>
<thead>
<tr>
<th>Segments involved</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinuses of Valsalva</td>
<td>4</td>
</tr>
<tr>
<td>Ascending arch</td>
<td>15</td>
</tr>
<tr>
<td>Ascending and transverse arch</td>
<td>4</td>
</tr>
<tr>
<td>Ascending, transverse, and descending arch</td>
<td>3</td>
</tr>
<tr>
<td>Transverse and descending arch</td>
<td>3</td>
</tr>
<tr>
<td>Descending arch</td>
<td>8</td>
</tr>
<tr>
<td>Descending arch and descending thoracic aorta</td>
<td>2</td>
</tr>
<tr>
<td>Descending thoracic aorta</td>
<td>5</td>
</tr>
<tr>
<td>Upper abdominal aorta (above renal arteries)</td>
<td>3</td>
</tr>
<tr>
<td>Lower abdominal aorta (below renal arteries)</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>51</td>
</tr>
</tbody>
</table>
pertrophy was considered to be present if the heart weight was 400 Gm. or more in men and 350 Gm. or more in women. Forty-three of 73 men had cardiac hypertrophy. The average heart weight for men was 492 Gm. Eighteen of 27 women had enlarged hearts. The average heart weight for women was 433 Gm. The average weight of 43 hearts from hypertensive subjects was 520 Gm. and of 57 hearts from normotensive individuals was 445 Gm. The average weight of 29 hearts with aortic insufficiency was 580 Gm. and of 71 hearts without aortic insufficiency was 435 Gm.

The largest hearts were found in those cases in which hypertension, aortic insufficiency, and coronary ostial stenosis were all present. In cases without coronary ostial stenosis, the hearts from normotensive patients with aortic insufficiency outweighed the hearts from hypertensive persons with aortic insufficiency by an average of 60 Gm.

**Coronary Artery Disease**

Atherosclerotic coronary artery disease was present in 93 cases. Its degree was graded from 0 to 3: grade 3, or severe, represented complete occlusion of a major coronary artery with myocardial infarction; grade 2, or moderate, represented significant narrowing of the coronary arteries; grade 1, was considered to be mild disease without significant narrowing, and grade 0 indicated no coronary disease. The results of this classification are given in table 3.

Over one third of the patients had significant degrees of coronary artery disease (grade 2 or 3). In general, lesser degrees occurred in association with aortic insufficiency than accompanied coronary ostial stenosis. The degree of coronary disease bore an inverse relationship to the severity of the aortic insufficiency or coronary ostial stenosis.

**Myocardial Lesions**

Recent or healed myocardial infarcts were discovered in 24 hearts. In 17 of these, the lesions were associated with atherosclerotic coronary artery disease. Seven infarcts occurred in cases with coexisting atherosclerotic coronary arterial and syphilitic coronary ostial stenosis. Four of these were associated with grade 1 atherosclerotic disease; therefore the marked coronary ostial narrowing was considered to be the main etiologic factor. The remaining three infarcts were attributed to the

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**Table 3**

**Atherosclerotic Coronary Artery Disease in 100 Cases of Syphilitic Aortitis**

<table>
<thead>
<tr>
<th>Category</th>
<th>Number of cases</th>
<th>Grade 0 coronary artery disease</th>
<th>Grade 1 coronary artery disease</th>
<th>Grade 2 coronary artery disease</th>
<th>Grade 3 coronary artery disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uncomplicated aortitis</td>
<td>36</td>
<td>3</td>
<td>21</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Aortic aneurysm</td>
<td>23</td>
<td>2</td>
<td>14</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Aortic insufficiency</td>
<td>6</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Coronary ostial stenosis</td>
<td>11</td>
<td>0</td>
<td>3</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Aneurysm and insufficiency</td>
<td>9</td>
<td>0</td>
<td>7</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Insufficiency and ostial stenosis</td>
<td>7</td>
<td>0</td>
<td>4</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Aneurysm and ostial stenosis</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aneurysm and insufficiency and</td>
<td>7</td>
<td>0</td>
<td>6</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>ostial stenosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>7</td>
<td>58</td>
<td>18</td>
<td>17</td>
</tr>
</tbody>
</table>
combined effects of grade 2 atherosclerosis and moderate syphilitic coronary ostial stenosis.

Virtually all of the hearts displayed variable amounts of patchy interstitial fibrosis of the myocardium. The most marked scarring was related to grades 2 and 3 coronary atherosclerosis. Coronary ostial stenosis, unless accompanied by marked atherosclerosis, was usually associated with lesser degrees of myocardial fibrosis. The infarcts resulting primarily from coronary ostial stenosis tended to be smaller than those associated with atherosclerosis. Otherwise, it was not possible to differentiate, either qualitatively or quantitatively, the ischemic lesions of syphilitic coronary ostial stenosis from those caused by atherosclerotic arterial disease. Myocardial infarcts occurred in only three cases of aortic insufficiency, and one of these was caused by severe concomitant coronary ostial stenosis. As a rule, lesser degrees of myocardial fibrosis were found in cases of aortic insufficiency than in the various other categories.

No examples of diffuse syphilitic myocarditis or gummas of the myocardium were found in any of the hearts.

**Other Cardioaortic Disease**

Three cases had coexisting chronic rheumatic heart disease with mitral valvular sclerosis. All three had positive serologic tests or a history of syphilitic infection as well as the classical lesions of syphilitic aortitis. None had the segmental distribution, intimal inflammation, microabscesses, fibrinoid necrosis, or concomitant pulmonary arterial involvement seen in cases of rheumatic-rheumatoid panaortitis. One of our cases, a 67-year-old man, had chronic rheumatic endocarditis of the aortic valve with superimposed acute bacterial endocarditis in addition to occlusive coronary artery disease.

Another case, a 65-year-old man, exhibited the typical changes of Erdheim’s cystic medial necrosis with healed dissecting aneurysm of the ascending aorta. This patient also had definite stigmata of syphilitic aortitis and positive serologic tests for syphilis.

Two examples of cor pulmonale secondary to chronic pulmonary disease were present in the group. In one case chronic pulmonary tuberculosis was the underlying disease and in the other instance bronchiectasis and emphysema were at fault. In both cases the syphilitic aortitis was uncomplicated.

**Causes of Death**

The causes of death are listed in table 4. Syphilitic cardiovascular disease was the primary cause of death in 33 cases of complicated aortitis. Death was attributable to rupture of an aortic aneurysm in 14 instances, to coronary ostial stenosis with sudden death in five cases, and to aortic insufficiency with cardiac failure in nine cases. In five other patients, both ostial stenosis and aortic insufficiency played a role. Syphilitic aortitis contributed to death in 14 additional cases. These included three postoperative fatalities subsequent to repair of aortic aneurysms. Arteriosclerotic heart disease and cerebrovascular accidents were responsible for death in 36 cases.

**Associated Conditions**

Lesions attributable to syphilis, at sites other than the heart and aorta, were found in 10 patients. These included neurosyphilis (four cases), hepatic lobatum (two cases), penile scars (two cases), epididymo-orchitis (one case), and interstitial nephritis (one case).

Some degree of encephalomalacia, slight to

**Table 4**

Primary Causes of Death in 100 Cases of Syphilitic Aortitis

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syphilitic heart disease</td>
<td>33</td>
</tr>
<tr>
<td>Arteriosclerotic heart disease</td>
<td>20</td>
</tr>
<tr>
<td>Cerebrovascular accidents*</td>
<td>16</td>
</tr>
<tr>
<td>Postoperative deaths</td>
<td>8</td>
</tr>
<tr>
<td>Malignant neoplasms</td>
<td>4</td>
</tr>
<tr>
<td>Nutritional cirrhosis</td>
<td>3</td>
</tr>
<tr>
<td>Perforated viscera with peritonitis</td>
<td>3</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>3</td>
</tr>
<tr>
<td>Bronchopneumonia</td>
<td>2</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>8</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

* Two cases of cerebral hemorrhage followed anticoagulant therapy for thrombo-embolic disease.
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severe, was present in 31 of 40 cases in which the cranial contents were examined. A significant degree of pulmonary emphysema was found in 25 individuals and 14 exhibited pulmonary tuberculous lesions in various stages of evolution. Pyelonephritis was observed in 21 and malignant neoplasms in 14 persons. Eight patients were psychotic and seven had nutritional cirrhosis.

Discussion

The limitations of such a retrospective clinical and anatomic study are many. Perhaps one of the most important is the accuracy and consistency with which various data are recorded by different individuals. Considerable caution must be exercised when the incidence of specific diseases and associations among diseases are derived solely from necropsy findings. Any series of postmortem examinations represents a select group of patients which is not characteristic of the population at large. Taking these factors into consideration, however, certain generalizations, comparisons, and conclusions can be made.

The incidence of syphilitic aortitis has certainly declined over the past half century. Prior to 1940, syphilitic heart disease accounted for 5 per cent of all heart disease in white persons and 25 to 30 per cent of all heart disease in Negroes. It has been estimated that cardiovascular syphilis is responsible for 10 to 15 per cent of all heart disease presenting after age 50. These figures are subject to considerable variation depending on the social, economic, racial, and geographic characteristics of the population at risk. There have been relatively few surveys of syphilitic heart disease reported in the literature over the past 10 years. Studies of large series of necropsies are worth while to detect trends in incidence, changing patterns of lesions, and the influence of therapeutic measures.

In a series of 15,000 unselected autopsies, performed at the Philadelphia General Hospital from 1927 to 1937, Welty found 1,040 cases of cardiovascular syphilis of all types. This study is similar in many respects to that of Welty and certain comparisons seem warranted. The institutions concerned are similar large municipal hospitals; are within 100 miles of each other; each study spans an 11-year period; a comparable large number of necropsies was surveyed in each instance; the racial and economic composition of the groups were much the same.

In the space of 23 years that separates these reports, the incidence of cardiovascular syphilis has dropped dramatically from 6.93 per cent to 0.76 per cent; the average age at death has increased by almost a decade, and aortic aneurysm has displaced aortic insufficiency as the major complication of syphilitic aortitis. Presumably these changes reflect the introduction of penicillin therapy and vigorous public health education. The increased proportion of aneurysms is probably related to greater longevity, with the synergistic effect of superimposed atherosclerosis playing an important pathogenetic role, although the factor of hypertension might also have to be considered. For similar reasons, coronary ostial stenosis has become a more prominent complicating factor, and is more often associated with myocardial infarction than in the past.

Paradoxically, in the cases reviewed here, there was no significant difference in the average age at time of death between the treated and untreated patients. The over-all effect of therapy on mortality was difficult to evaluate. All of the histologic features of syphilitic aortitis, seen in the untreated cases, were also present in the treated cases, usually but not always to a lesser degree. It is well known that antisyphilitic therapy does not reverse the cicatricial lesions and their functional consequences. The longevity of patients in this series showed no apparent correlation with the presence or absence of hypertension.

The definitive diagnosis of syphilitic cardioaortitis clinically is difficult to establish. This is particularly true in persons over 50 years of age. The blood serologic tests may be negative or positive with a low titer. A history of a primary syphilitic infection helps in making the diagnosis.

Analysis of the cases recorded in this paper demonstrates that the presence of precordial pain, hypertension, cardiac enlargement, or congestive heart failure is of little assistance
in confirming the diagnosis of syphilitic aortitis, and, in a number of cases may contribute to errors of diagnosis. Accentuation of the aortic second sound and a systolic aortic or apical murmur are helpful in uncomplicated cases, especially when accompanied by submanubrial dullness. Paroxysmal nocturnal dyspnea is uncommon, but when found, favors the diagnosis. Clinical aortic insufficiency, in the absence of other stigmata of rheumatic heart disease, should be considered as suggestive evidence of syphilitic aortitis, particularly when accompanied by a history of a primary lesion or positive serologic tests. Apart from actual demonstration of an aneurysm, radiologic widening or calcification of the aortic arch or both, are also suggestive features.

There are no specific or pathognomonic electrocardiographic changes in syphilitic heart disease. The abnormalities of the electrocardiogram attributable to syphilis result from left ventricular hypertrophy following aortic insufficiency, or are a consequence of myocardial ischemia due to narrowing of the coronary ostia. These alterations are mimicked by other conditions causing enlargement of the left ventricle, and by atherosclerotic coronary artery disease. However, the chances of having an abnormal tracing in syphilitic heart disease are indeed high.

Besides failure to consider the possibility of syphilitic heart disease and to perform appropriate serologic tests, the most important reasons for lack of proper diagnosis were resemblance of the signs and symptoms to other types of cardiovascular disease and coexistence of other types of cardiovascular disease. Syphilitic aortic insufficiency itself may be mistaken for rheumatic heart disease, or rheumatic heart disease may occur in addition to syphilis. Chronic dissecting aneurysms may simulate syphilitic cardiovascular disease and occasionally these lesions occur in conjunction with syphilitic aortitis. More often, the association of atherosclerotic or hypertensive heart disease obscures the presence of syphilitic aortitis, or at least blunts the awareness of the clinician to this possibility. Cases of syphilitic coronary ostial stenosis are particularly prone to be regarded simply as examples of coronary atherosclerosis.

In a clinical study of 547 patients with syphilitic aortitis, Rich and Webster considered 141 cases to be uncomplicated. Admittedly, some of these undoubtedly represented instances of asymptomatic coronary ostial stenosis. They concluded that the diagnosis of uncomplicated syphilitic aortitis can be made with reasonable accuracy. In their opinion the concomitance of atherosclerosis or hypertension does not make the diagnosis of syphilitic aortitis significantly more hazardous. It should be emphasized that all of their patients had been followed in a clinic devoted to the treatment of syphilis. It seems safe to conclude, however, that a diagnosis of syphilitic aortitis can be made in the great majority of complicated cases, and in a significant number of uncomplicated cases.

**Summary**

The major clinical and pathologic features of 100 cases of syphilitic aortitis, autopsied from 1950 to 1960, are reviewed. Decreasing incidence, changing patterns of lesions, and reasons for the frequent failure to diagnose syphilitic heart disease are considered. Aortic insufficiency or aneurysm of the thoracic aorta, or both, accompanied by a history of syphilis or reactive serology are required to establish a definitive diagnosis. Syphilis still remains a real threat, and, until it is eradicated, it will continue to cause clinical and subclinical cardiovascular disease.

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