Osler’s Museum Specimens of Heart Disease*

Their Nature and Significance

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MEDICAL HISTORY

There are 55 of Osler’s autopsy museum specimens on display at the McGill Pathology Institute. Of these, 23 are examples of heart disease, including ten cases of endocarditis. A review of related publications reveal that Osler was one of the first to stress the significance of micrococci and prior scarring of valves in infective endocarditis, and the differences between congenital and acquired bicuspid aortic valves. His review articles on other heart diseases, such as cardiac thrombi, tuberculous endocarditis and congenital heart disease, contributed significantly to the general understanding of these entities during the latter part of the nineteenth and the first part of the twentieth centuries. It is concluded that Sir William Osler played an important role in the development of current concepts of heart disease.

Sir William Osler’s activities as a physician have permeated all areas of medicine, both during his lifetime and in the past 50 years. The magnitude and excellence of his careers as a clinician at Philadelphia and Baltimore and as Regius Professor in London have overshadowed the first ten years of his professional life at McGill Medical School. Osler acted in the capacity of pathologist at the Montreal General Hospital from 1876 to 1884. During this period he laid the foundations of his diagnostic expertise by performing and studying 786 autopsies.

Osler’s autopsy experience involved more than dissection. He utilized the material extensively, in teaching, for publications and by the collection of approximately 200 museum specimens. According to Maude Abbott, nearly one-third of his publications on clinico-pathologic topics and over 50 percent of the original museum specimens were related to the circulatory system. Fifty-five of Osler’s specimens have survived and are on display at the McGill Pathology Institute. Thirty-eight are examples of cardiovascular disease and 23 of these are of diseases of the heart (Table 1).

A study of Osler’s heart specimens provides an opportunity to evaluate his contributions to our knowledge of heart disease and to examine some concepts of heart disease in the late 19th century. His museum specimens were obtained from the autopsies he performed himself.

Table 1—Existing Osler Heart Specimens at McGill Pathology Institute

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. Specimens</th>
<th>Museum No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibrosis of myocardium</td>
<td>2</td>
<td>6, 7</td>
</tr>
<tr>
<td>Fatty heart with rupture</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Acute purulent pericarditis</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Pericarditis ( fibrinous)</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Subacute tuberculous pericarditis</td>
<td>1</td>
<td>11</td>
</tr>
<tr>
<td>Hypertrophy of heart</td>
<td>1</td>
<td>12</td>
</tr>
<tr>
<td>Mitral and tricuspid stenosis</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>Pulmonary stenosis</td>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>1</td>
<td>15</td>
</tr>
<tr>
<td>Acute infective endocarditis</td>
<td>7</td>
<td>16, 17, 18, 19, 20, 23, 26</td>
</tr>
<tr>
<td>Subacute infective endocarditis</td>
<td>1</td>
<td>22</td>
</tr>
<tr>
<td>Chronic infective endocarditis</td>
<td>2</td>
<td>24, 25</td>
</tr>
<tr>
<td>Bicuspid aortic valve</td>
<td>1</td>
<td>21</td>
</tr>
<tr>
<td>Ball thrombus of heart</td>
<td>1</td>
<td>27</td>
</tr>
<tr>
<td>Secondary carcinoma of heart</td>
<td>1</td>
<td>28</td>
</tr>
</tbody>
</table>

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Records of Osler’s Autopsies

Osler performed approximately 100 autopsies a year from 1876 to 1884. Most of the protocols were written by himself in five volumes. Some were recorded by student clerks from his dictation. In 1890, while at Baltimore, he borrowed these volumes to assist in the writing of his textbook of medicine. Only the first three volumes were returned and are now catalogued in the Osler Library at McGill as a significant part of Osler’s records.

From the existing records it is evident that Osler did a thorough, fairly detailed and competent autopsy. The language is terse, non-verbose and clear. There is little of the flowery rhetoric of his later years. The descriptions are readily understandable to a modern pathologist. Weights and measurements are abundant and include heart weight, valve circumferences, and thickness of ventricular walls. These protocols are modern in their methodology.

In one sense, the initial reaction on surveying the autopsy protocols is one of disappointment. Only rarely are there notations of the patient’s signs, symptoms or occupation. Occasionally even age and sex are not recorded. There is no mention of microscopic findings and no clinical-pathologic correlations. These are, simply, the protocols of a thorough, conscientious and hard-working autopsy technician. However, one must examine all of Osler’s activities before it becomes apparent that his orientation to pathology was far more than descriptive. His protocols were merely the initial recording of data, to be followed by printed post-mortem reports and many publications.

Osler’s two Pathological Reports of the Montreal General Hospital represent the first such publications in Canada. Although they are a selection of his autopsy protocols they provide a clue to his true orientation. Many contain clinical histories, a few have extensive discussions of the cases, and some have detailed microscopic descriptions. An example of a microscopic description in his Post-Mortem Records is diphtheritic myocarditis. Evidently Osler kept separate records of his histologic studies, none of which can be found today. However, his papers related to autopsy case material are readily available.

Osler’s publications during his McGill period consisted largely of short descriptions of individual specimens. However, he did publish detailed reviews of several heart diseases, including hypertrophy, fibroid disease, and infectious endocarditis. The last two examples contain microscopic descriptions. These represented major contributions to the medical literature of his day. Osler’s most significant scientific work was in the field of cardiology, including endocarditis, valvular stenosis, myocardial fibrosis, cardiac hypertrophy, pericarditis and congenital heart disease.

Osler and Endocarditis

Endocarditis is represented by ten of the existing Osler specimens. All have involvement of the mitral and/or aortic valves. Seven are labeled as acute endocarditis (Fig 1), one as subacute and two as chronic (Fig 2). Eight are also designated as recurrent, presumably on the basis of pre-existing thickening of the valve cusps or leaflets. In one specimen the basic lesion is a bicuspid aortic valve with superimposed endocarditis.

Osler included several cases of endocarditis in the Post-Mortem Reports of the Montreal General Hospital. He also published descriptions of individual specimens. However, these do not contain any interpretation or assessment. His first major paper on endocarditis was in the Archives of Medicine of 1881, in which he refers to 57 cases of endocarditis, 13 of which had meningitis and 22 pneumonia. Included is one case occurring in a cow. His classification at that time was primary (of no obvious cause), secondary (to some inflammatory focus) and recurrent (engrafted on chronic valve disease). He clearly distinguished infective endocarditis from “rheumatism.”

In the paper of 1881, Osler also described the histologic finding of micrococci which he consid-

Figure 1. Museum card description: acute infective endocarditis. Acute infective (recurrent) endocarditis of aortic valve with destruction of anterior, and fibrous tag projecting from left posterior cusp. Man, age 60 years. Anasarca and purpura. Multiple infarcts of spleen. Note thickened valves from previous attack of simple and thrombotic malignant character of recent destruction process. Ref. MGH PM 16/1876, Presented by Dr. W. Osler (museum no. 18).

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He reviewed in detail the clinical and pathologic findings of 209 cases, including his own and published cases. He suggested a clinical classification of simple and malignant on the basis of mild and severe. Osler listed many possible complications including perforated cusp, ruptured chordae, spread to chamber, emboli, infarcts, miliary abscesses and retinal and skin hemorrhages. He again stressed that many cases are due to microorganisms spreading from the lung and occur on sclerosed valves.

Another term used by Osler for sclerosed and deformed valves was chronic endocarditis. In 1889 Delafield\(^2\) listed two types, following acute endocarditis or a chronic lesion from the first. Osler, in discussing the paper, recognized that its clinical effects were due to "gradual failure in the compensation of the heart." In the first edition of Osler’s Principles and Practice of Medicine,\(^2\) there is a more complete list of causes including acute rheumatic fever, alcohol, syphilis, gout, and prolonged and heavy exertion. Considered in their entirety, Osler’s writings on endocarditis are not only impressive, but also indicative of a considerable influence in his day.

**Osler and Valvular Stenosis**

Four of Osler’s museum specimens are examples of valvular stenosis—mitral and tricuspid stenosis, pulmonary stenosis (Fig 3), aortic stenosis and mitral stenosis with a ball thrombus. As early as 1877\(^2\) he published with R. P. Howard an account of two cases of mitral and tricuspid stenosis. He did not consider the changes to be of congenital origin because the foramen ovale was closed. He suggested that they were the result of rheumatism and

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**Figure 2.** Museum card description: chronic infective (recurrent) endocarditis of aortic valves. Fusion of anterior and right posterior cusps with ulceration and perforation of these, and extension of the inflammatory process upon the subjacent mural endocardium, and through the undefended space into the right auricle, with perforation of septal cusp of the tricuspid valve. Calcification of inflammatory products. Presented by Dr. W. Osler (museum no. 24).

**Figure 3.** Museum card description: pulmonary stenosis. Atresia of inflammatory origin. Presented by Dr. W. Osler (museum no. 14).
chorea in early life. In contrast are Coats' textbook of 1883,15 and Delafield and Prudden's of 1889,16 both of which contain a simple statement that mitral stenosis is caused by chronic endocarditis. In Osler's textbook of 189222 there is an excellent clinical and pathologic account of both mitral and tricuspid stenosis with a detailed discussion of pathologic-physiologic changes that is almost modern in content. Of interest is Osler's paper on tricuspid stenosis in the Medical News of 188224 in which he attributed small granular kidneys to the valvular stenosis.

One of the existing specimens is an excellent example of a ball thrombus of the left atrium associated with mitral stenosis (Fig 4). In the Johns Hopkins Hospital Reports of 189125 Osler described two cases, one seen at Montreal and the other at Baltimore. He suggested that these thrombi were not likely to produce any special symptoms. However, in 189726 in a similar paper in the Montreal Medical Journal, Osler indicated that sudden death may be caused by plugging of the stenosed mitral orifice by the ball thrombus. He was considerably antedated by Coats15 who mentioned obstruction of the valve by an auricular thrombus and described retrograde congestion and a small left ventricle.

Less prominent in Osler's writings are pulmonary and aortic stenosis. Osler's Montreal General Hospital autopsy no. 1765 described a case of pulmonary stenosis in a four-month-old infant. The pulmonary valve had fused cusps leaving an opening of only 9-10 mm. Both the right and left ventricles were hypertrophied and there was thickening of the endocardium of the right ventricle. Osler did not publish any account of pulmonary stenosis. In the Canada Medical Record of 1878-79,27 there is a mention that Osler presented a specimen of congenital stenosis of the pulmonary artery with enlarged ductus arteriosus before the Montreal Medico-Chirurgical Society.

Osler did not publish any papers on aortic stenosis although his Principles and Practice of Medicine28 provided an excellent clinical and pathologic description, with suggested causes similar to those for mitral stenosis. However, Osler's contribution to the understanding of valvular stenosis was relatively minor.

**Osler and Myocardial Fibrosis**

Two of Osler's specimens are labeled as fibrosis of the myocardium (Fig 5). One heart is from Montreal General Hospital autopsy no. 674 in which Osler described emaciation, edema, ascites, serious

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**Figure 4.** Museum card description: heart—ball thrombus. Round ball thrombus in left atrium in mitral stenosis, embolism of abdominal aorta and thrombosis of left external iliac. The thrombus the size of a golf ball lies free across the mitral orifices. Note jagged end of old thrombus in appendix. Woman, age 44 years. History of rheumatism and repeated failing compensation. Gangrene of left leg from embolism of right iliac artery. Ref. MGH 18/90. Surg. Case Book F. XV. P. iii. Presented by Dr. Wyatt Johnson and Dr. W. Osler (museum no. 27).

**Figure 5.** Museum card description: heart—fibrosis of myocardium. Localized fibrosis of myocardium in atheroma and thrombosis of the coronaries. Man, age 60 years. History of repeated anginal attacks extending over ten years. Death from failing compensation. Note complete occlusion of transverse branch of right coronary with dense sclerosis in area of heart muscle supplied. Presented by Dr. Osler and Dr. Howard, 1877 (museum no. 7).
atrophy of fat, large dilated heart, thrombus of left ventricular endocardium, gray alteration of the left myocardium and granular kidneys. A more detailed clinical and pathologic description of this case is given in a paper on “A Case of Fibroid Disease of the Heart,” published in the Canada Medical and Surgical Journal of 1880 by Dr. R. P. Howard, Professor of Medicine at McGill. Osler provided gross and microscopic descriptions for this paper.

“A layer of yellow stratified fibrin about k inch thick adheres to the lining of the left ventricle and covers about the inferior two-thirds of its anterior wall, whence it extends somewhat over the septum. On section, about the internal half of the wall of the ventricle at the site of the adhering fibrin, has lost its muscular appearance and color, and is anæmic, of pale grey hue, and tough, crunching under the knife.”

“Microscopical examination of the portion involving the septum near the apex showed it to be made up of ordinary connective tissue fibres, arranged in wavy bundles, with very few corpuscles or fibre cells. In the greater part of the area no trace of muscle substance is found, but at the margins the new growth is seen penetrating between the muscle fibres, some of which are completely cut off and isolated. The endocardium over the spot is thickened. Many of the muscle fibres in contiguous parts presented brown granules and fatty molecules.”

The 1881 paper also included an extensive discussion of “fibroid transformation” of the heart. The prevailing opinion as to causation at that time was a chronic myocarditis, resulting from endocarditis or periocarditis which extended to the myocardium. However, the author suggested that there were three causes—degeneration of the myocardium due to some disturbance of innervation of the heart, primary hypertrophy of myocardial fibrous tissue and fibrosis of inflammatory origin. Eleven years later, in Osler’s Principles and Practice of Medicine, the term fibrous myocarditis was used for the end result of gradual transformation of areas of anemic necrosis due to blocking of a coronary vessel by thrombus or embolus. Nonetheless, Osler cannot be given credit for the initial delineation of the cause of myocardial fibrosis.

Osler and Cardiac Hypertrophy

The one specimen of hypertrophy of the heart in the existing Osler museum specimens is an excellent example of the multiple ways in which he capitalized on his autopsy experiences. The case formed the basis for a museum specimen, a detailed account in the first Pathological Report of the Montreal General Hospital and a review article.

The patient was a 39-year-old man who died about six months after the onset of heart failure. The autopsy protocol No. 444 described a 610 gm heart with no lesions of the valves. The kidneys were smooth and the arteries of the body did not have any signs of degeneration.

The description of this case in the Montreal General Hospital Pathological Report of 1878 is accompanied by a short discussion. Osler emphasized that, “There were none of the common causes present to account for the hypertrophy of the heart—no valvular disease, no arterial degeneration, no chronic renal or pulmonary disease.” He then suggested that the cause may have been overstrain and prolonged muscular exertion, as previously reported in the German literature.

This case was further used by Osler for a paper on a “Case of Hypertrophy, Dilatation and Fatty Degeneration of the Heart, Consequent Upon Prolonged Muscular Exertion,” published in the Canada Medical and Surgical Journal of March, 1878.

It was now noted that the patient was a blacksmith. There is a more comprehensive review of the literature on the effects of muscular exertion on the heart. Osler listed rupture of valves, irritable heart, increased aortic pressure with aortic incompetence and primary dilatation and hypertrophy as effects of overwork. He stated that “severe muscular exertion affects the circulation by interfering with respiration and the free passage of blood through the lungs; the right heart gets over-loaded, the systemic veins full, and thus an obstacle is offered to the outflow of blood from arteries; in consequence of which the left ventricle becomes dilated and must hypertrophy to overcome the increased resistance to the arterial flow.” Another case of cardiac hypertrophy was described in the second Pathological Report of the Montreal General Hospital. In discussing this case he broadened his concept of causation by stating that “All circumstances which tend to produce, and keep up, a state of high tension in the arterial system may lead to dilatation and hypertrophy of the heart.” Thirteen years later, Osler’s Principles and Practice of Medicine contained a still more sophisticated account of cardiac hypertrophy. Causes of left ventricular hypertrophy included aortic valve disease, mitral insufficiency, pericardial adhesions, sclerotic myocarditis, hyperthyroidism, tea, alcohol, tobacco, general arteriosclerosis and hypertension. Right ventricular hypertrophy was considered to arise from valve lesions, lung disease and left-sided heart disease. Thus, in his textbook, Osler attained a currently acceptable concept of cardiac hypertrophy.

Osler and Pericarditis

Three of the existing Osler specimens are examples of pericarditis. One is an acute purulent pericarditis, another a fibrinous pericarditis (Fig 6), and the third tuberculous pericarditis. Osler did not publish any papers on nontuberculous pericarditis.
Osler’s specimens is bicuspid aortic valve. The first record of this condition in Osler’s writings is Montreal autopsy No. 1357 in which is described a 690 gm heart with an aortic valve consisting of only two valves separated by an irregular interval. In the Montreal General Hospital pathologic report of 187817 he added a clinical history of shortness of breath, palpitations, edema, slight jaundice and a double murmur at the base.

Osler’s discussion of bicuspid aortic valve is found in two papers, both published in 1886, one in the Transactions of the Association of American Physicians18 and the other in the Journal of the American Medical Association.19 Eighteen cases seen at the Montreal General Hospital are described. Ulcerative endocarditis was present in eight and sudden death occurred in two. Osler believed that the majority of cases were congenital in origin. He favored an anomaly of development as the cause rather than fetal endocarditis because it was associated with other anomalies of the heart and because the two cusps were usually of similar length and had a smooth appearance.

Osler’s18 made a significant contribution by carefully delineating the anatomic characteristics of congenital bicuspid aortic valve with a raphe as compared to acquired bicuspid aortic valve with fusion of two cusps.

“In the conjoint valve there are three points to be noted. The free border was usually straight, oftentimes curled, and in no instance was there any nodular thickening indicative of the presence of a corpus Arantii. The attached border presented, from the ventricular aspect, either the normal contour of a semilunar valve, or, more commonly, a shallow groove, indicative of the junction of two cusps. The aortic side of the valve presented in all the cases a more or less distinct raphe, or fraeurn, dividing, or indicating a division into, two sinuses. This raphe, the representative of the hands which in the normal segments unite them to the aortic wall, was present either (a) as a narrow elevated ridge confined to the aortic wall; (b) as a single band passing for a variable distance on to the valve; or (c) was divided into two distinct portions, which passed out the inner aspect of the valve and were ultimately lost. The sinuses of Valsalva, thus incompletely marked, were usually of equal size, and in sixteen of the cases they gave origin to the coronary arteries.”20

Although there is now some doubt that congenitally bicuspid aortic valves can be readily distinguished from acquired ones,19 Osler’s18 did popularize the fact that there are two etiologic types.

Two other publications are indicative of Osler’s interest in congenital heart disease. In the second Montreal General Hospital Reports of 188010 he described four cases of cardiac anomalies, two of pulmonary atresia, one of premature closure of the foramen ovale with general dropsy, and one of “aorta given off from the right ventricle.” He
suggested that dropy might be the result of disturbed fetal circulation due to the premature closure of the foramen ovale. The case labeled as "aorta given off from the right ventricle" was also discussed by Osler in his chapter on "Congenital Affections of the Heart" in Keating's *Cyclopedia of the Diseases of Children* of 1889.\(^1\)\(^1\) The case was now labeled as transposition of the aorta and pulmonary artery.

“A fetus of the eighth month presented all the malformations above referred to. The heart was not much enlarged; the right auricle was of moderate size; the cavae were normal; the aortic arch valve was large, and the foramen ovale open. The tricuspid valves presented two bead-like hemorrhagic nodules. From the right ventricle, which was larger than the left, a vessel was given off, eight millimeters in width at the root, which passed over a vessel emerging from the left ventricle, crossed the left bronchus, and then descended as the thoracic aorta. Seven millimetres from its origin it gave off a small pulmonary branch to imperfectly-developed lungs, and, just before it reached the spine, the left subclavian passed off vertically to the first rib. The left ventricle was smaller than the right. The mitral orifice and valves were normal. From this chamber a vessel passed up on the trachea without communicating with the vessel from the right ventricle; it then divided into the innominate and common carotid arteries. In the septum ventriculorum was a small orifice, the size of a goose-quill, at the upper and back part of the septum. The semilunar valves in both vessels were abnormal; there were only two on the branch from the left ventricle, and in that one from the right two of full size and between them a tiny imperfect one."\(^4\)

The above is an excellent description of interruption of the aortic arch, the first account of which has been attributed to Steidle of Vienna in the 18th century.\(^2\)\(^2\)

In addition to individual case reports, Osler’s 21 page chapter on congenital heart disease in Keating’s *Cyclopedia*\(^4\)\(^1\) was a systematic review of the subject. His classification included general anomalies and anomalies of the septa, valves and larger vessels. Only the more common and less complex anomalies were presented. There was no mention of more complicated abnormalities such as tetralogy of Fallot. The major symptoms described were cyanosis, clubbing of the fingers and toes and murmurs. Three categories of etiology were discussed—persistence of normal fetal structures which normally disappear, anomalies of development and fetal endocarditis. Osler’s recommendations for treatment were brief and “largely hygienic.” Osler’s chapter in his own textbook\(^4\)\(^3\) was even shorter, five pages. He stressed that congenital heart disease is of limited clinical interest as it is either not compatible with life or else no treatment is available. Although this statement is not true today, it adequately summarized the status of cardiac anomalies prior to the past few decades.

**Discussion**

Although Osler did not make any “new” discoveries or engage in experimentation in cardiology, he served as a synthesizer, educator and popularizer. From the extent to which he capitalized on his autopsy specimens it is evident that he had a considerable influence on cardiology during the last 25 years of the 19th century. His review articles on endocarditis,\(^1\)\(^2\) cardiac thrombi,\(^2\)\(^5\) tuberculous pericarditis\(^3\)\(^6\) and congenital heart disease\(^4\)\(^1\) were printed in widely circulated and respected publications.

The Gulstonian lectures on endocarditis, delivered before the Royal Society of Physicians of London,\(^1\)\(^8\) did much to familiarize the profession with the clinical patterns and in the significance of associated Micrococi and prior scarring in the valves. His attempts to differentiate between congenital and acquired bicuspid aortic valves\(^8\)\(^9\) are still cited in detail by current articles on the subject.\(^4\)\(^2\)

Osler’s interest in cardiology was sufficiently broad to include a case of endocarditis in a cow.\(^1\)\(^2\) He prepared a greater number of museum specimens of endocarditis than any other entity. According to Maude Abbott,\(^4\)\(^4\) there were 23 such specimens in the original Osler collection.

Osler was too much of a generalist to be labeled a cardiologist. The extent of his activities in other areas was almost as great.\(^4\)\(^5\) However, a detailed review of his work in this field suggests that Osler played a significant role in the development of current concepts of heart disease.

**Acknowledgments:** Dr. D. Bates and staff, of the McGill Osler Library, were instrumental in obtaining information for this article. Dr. R. More, Strathcona Professor of Pathology at McGill, provided orientation and permission for photography and publication of Osler’s museum specimens. Dr. C. Burns, of the Division of History of Medicine at the University of Texas Medical Branch in Galveston, provided encouragement and expert guidance for this project.

**Note on Museum Specimens**

The figure descriptions are reproduced from the cards accompanying each specimen. The number in parentheses is the specimen number in the Osler Museum of the McGill Pathology Institute. Some of the cards were probably written long after acquisition of the specimen by Osler. For example, the description, for “fibrosis of the myocardium” (Fig 5) states that coronary thrombosis was responsible for the myocardial fibrosis, a correlation that was not apparent until long after the specimen was presented by Drs. Osler and Howard in 1877.

The cards contain names of several McGill faculty members of the time. Dr. R. Palmer Howard (Fig 5) was Chairman and Professor of Medicine while Osler was a medical student at McGill. he became Dean of the school in 1882 and died in 1889. Dr. Wyatt G. Johnston (Fig 4) was Osler’s demonstrator in pathology at McGill, and succeeded him as pathologist of the Montreal General Hospital.
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