Oslerian Pathology An Assessment And Annotated Atlas Of Museum Specimens

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Further variation is encountered in the extent of the description and number of measurements for each case. Some cases have only a few measurements (e.g. nos. 18, 46), others none (e.g. nos. 20, 41). In still others, measurements, especially of the heart, are provided that far exceed the accepted practice 100 years later. In general the more weights and lengths given the more detailed the protocol as a whole. This suggests variation in time available to Osler to perform autopsies and record them.

Table 3

Oslerian Autopsy Protocols[a]

Specimen Number	Protocol Diagnosis
3	Aortic Valve Disease, Acute Ulcerative Endocarditis
16	Pleurisy, Fibroid and Fatty Heart
17	Heart Disease, Pleurisy, Pulmonary Apoplexy
18	Purulent Pericarditis
20	General Tuberculosis (Subacute Pericarditis)
21	Aortic Valve Disease
33	Sacculate Aneurysm of First Part of Aorta
41	Cancer of Stomach
46	Pulmonary Tuberculosis (Multiple Ulcers of Stomach)
53	Chronic Interstitial Nephritis, Pneumonia, Hypertrophy Heart

[a] All protocols are from Osler's original handwritten Autopsy Books, except for number 33, which is from the Montreal General Hospital Autopsy Book of 1902, and performed by John McCrae.

AUTOPSY PROTOCOL FOR SPECIMEN 3

From: Osler's Autopsy Book, 1877, pp. 57-61[43]

Aortic Valve Disease

Ac. Ulc. Endocard

Case 16. June 20/76

George Wells aet 60.

PM 13 hours after death. General description of body. — Body that of a well built man. Skin of face and upper part of trunk slightly icteric. Lower limb oedematous. Feet only to slight degree. Skin of the legs of a dark purplish hue due to numbers of small extravastions. These echymoses punctiform in character also exist in the triangles of the thighs, the flexor surfaces of the elbow joints, a few in the axillae.

Abdomen and Thorax.

On opening the abdominal cavity the stomach is seen much distended in a curved direction towards the right and extending fully an inch below the umbilicus.
The liver extends fully three inches below the costal cartilages. Coils of the small intestine occupy the space between the stomach and pubes. Twelve ounces of a turbid reddish fluid removed from the abdominal cavity.

Thoracic Cavity

Right lung adherent in front to the pericardium and sternum. Left lung free. 3½ ounces of fluid in the left pleural sac. 13 ounces of a clear amber fluid in the right pleural sac. (Drams) VI fluid in the pericardial sac. Heart appears large, fatty at the base & ventricular groove. Marks of attrition at usual site also at base & pulmonary aorta. Over the left ventricle. Cavities all appear distended with blood. Right auricle contains (Ounces) IV of blood and clots. About (Ounces) III of clots & blood in the right ventricle. (Ounces) III of blood in the left ventricle. Right ventricle dilated. Tricuspid orifice admits four fingers. Valves healthy.

Pulmonary semilunar.

Left ventricle large dilated, walls fully ⅝ of an inch thick.

Endocardium clouded. Mitral orifice large, edges of valves covered with vegetations. Aortic orifice semilunar valves covered with vegetations. In the extreme left segment a large mass of vegetation extends down from ventricular surface. In middle segment the vegetations are flat & not so large; the remaining segment to ¼ of its extent completely eaten away & the edges covered with numerous bead-like masses. The remaining portion is perforated near the attachment of the valve. Endocardium beneath the valves much thickened. Coats of the aorta not presenting marked atheromatous disease. Heart weighs (Ounces) XX (Drams) III.
Left Lung — No adhesions, slight cicatrix at apex. Pigmentation moderate in amount. On section a large amount of frothy serum of yellowish (slightly) color exuded. Upper lobe & free edge of lower lobe crepitant. At the base the organ is intensely congested & in a state of splenization.
Right lung adherent over a large portion of its extent & covered with a layer of thickened adhesions. Small puckered cicatrix at the apex. Also very oedematous. Middle lobe on section extremely dark colored, firm to the touch granular looking on section, small portions removed however still float. Spleen (Ounces) VI fissured and lobulated. On section firm. Trabeculae very evident. Two small infarctions. Healing met with in the spleen. Capsule opaque thickened and over the surface small fibroid masses.
Right Kidney (Ounces) V (Drams) I. Firm, capsule detaches with moderate facility. Section pale cortex appears somewhat wasted. Left Kidney (Ounces) VI. Capsule does not detach so readily. Appearance the same. Arteries at the bases of the pyramids very evident and those of the renal arteries somewhat thickened and atheromatous.

Liver —

Firm to the touch, dark yellowish green mottled color. On section presents beautifully the character of ordinary nutmeg liver; tears with difficulty. Weight (Ounces) LVI½. Gallbladder contains a quantity of bile. Ductus communis slightly obstructed.

Stomach —

Mucous membrane covered with a thick tenaceous mucous, and also covered over with small partially decomposed echymoses. Mucous membrane soft easily torn.

Brain —

On cutting thro the soft parts on removal a large amt of blood escaped. Skull cap thick & deeply grooved for the meningeal arteries. Pacchinian depressions along the longitudinal sinus. Small clot in the longitudinal sinus. Pacchinian bodies large. Membranes very adherent along longitudinal sinus. Dura mater thick & opaque & very adherent to the skull. Large amount of serum quite (Ounces) III (Drams) III escaped on removing the brain. Sulci deep. Arachnoid over them cloudy. Surface of brain somewhat pale veins not very full. Brain substance tolerably firm somewhat pale & glistening. Ventricles contain a few drachms of fluid, walls a little soft. Sylvian arteries slightly atheromatous. Pons Medulla & ganglia at the base healthy.

Intestines —

Nothing abnormal noticed about the intestines. Appendix vermiformis small & attached along its whole length.

Annotation for Protocol 3

In summary, this was a case of acute bacterial endocarditis with valve cusp destruction and cardiac hypertrophy (565 gms). Heart failure was evidenced by cardiac dilatation, hydropericardium, hydrothorax, ascites, peripheral edema, nutmeg liver (1582 gms), and congestive splenomegaly (168 gms). The two infarcts of the spleen were related most likely to emboli from the valvular vegetations. Mild icterus could have been caused by septic hemolysis. Terminal events were right middle lobe pneumonia and acute gastric dilatation. Incidental findings include pleural adhesions, apical lung scar, fibrosis of splenic capsule, and atherosclerosis of cerebral and renal arteries.

Although the description accompanying the museum specimen describes cusp thickening due to a previous attack,

this is not evident in the specimen nor described in the autopsy protocol. In general, acute bacterial endocarditis involves previously normal valves.

Although this autopsy represents a typical example of the pathologic manifestations of acute bacterial endocarditis, finer points are not mentioned. These include splinter hemorrhages of the nail beds and subcutaneous nodules which occur in some cases. These skin nodules are now referred to as "Osler's nodes", which were described in his Gulstonian lectures on endocarditis[84].

AUTOPSY PROTOCOL FOR SPECIMEN 16

From: Osler's Autopsy Book, 1877, pp. 212-214[43].

Case LXVII **Pleurisy, Fibroid & Fatty Heart.**

H.H.W. aet 65

Body that of a tall, fairly well built man. Face somewhat emaciated. Feet ankles and legs swollen and pit on pressure.

Thorax and Abdomen:

About a pint & a half of clear fluid in the peritoneum. Viscera in normal position but the liver is pushed down 1½ inches below the ribs in the mammary line.

Fully 2½ pints of a clear citron yellow fluid in the right pleural sac. The parietal pleura on this side was much injected red in colour & covered by some few flakes of lymph. The chest was tapped (for sake of convenience), and on removing the sternum a definite pocket was found from which the fluid had been removed. The upper wall of this compartment was composed of thin membrane of lymph, recent but of considerably tenacity. Another of these pockets existed towards the upper and posterior part of the pleural cavity. The visceral layer was also covered in places with recent lymph and in the fluid removed there were some flakes. There was no fluid in the left sac and the lung was only adherent at one spot towards the apex base.

Pericardium: Covered by a small amount of fat, and the tissue over it in the ant. mediastinum was infiltrated & gelatinous. Membrane thickened, opaque. (Ounces) II of citron coloured fluid removed from it. Patch of attrition over the lower & anterior part of the left ventricle.

Heart looks larger, & the cavities are distended with blood and grumious clots extend into the veins. In the right auricle the appendix contains a very firm ante mortem clot, colourless & intimately adherent to the musculi pectinati. Right ventricle appears dilated. Tricuspid orifice large, about 5½" in circumference. Valves healthy. Pulmonary semilunar also. Coagula in left auricle which extend into pulmonary veins. In the left ventricle besides a large black coagulum, there is a larger ¾" thick of yellow stratified fibrin adhering to endocardium on its anterior aspect & to the septum. About the inner half of the muscular wall of the ventricle at this point has lost its normal colour & aspect, & is pale, almost white at least light grey. It is tough, crunches under edge of knife, is anaemic looking. The muscular wall external to this looks healthy. The general aspect of the cut ventricular walls was not — except at the spot above (?) —remarkable, the colour was dark reddish brown. The columnae carnae at their bases are whitish owing to degenerative changes which can be seen through the tissue membrane. Towards their apices they are firm, evidently fibrois & cut with difficulty on dividing the septum.

Mitral valves are healthy, the orifice of natural size. Aortic semilunar thickened at bases & at the insertion of one of them is a thin narrow calcareous ring. Their free edges are normal; the valve is competent.

The aorta from the arch to its bifurcation presents numerous raised patches of atheroma, ranging in size from a three pence to a shilling. On section of one a thick mass of yellow material is seen immediately beneath the intima.

Spleen, slightly smaller than natural & presents two deep irregular fissures at the anterior border. It is exceeding firm & dense, cutting crisply and with resistance. The trabeculae are very evident; the pulp solid, dark brown in colour, (?) not visible.

Kidneys, of normal size. Capsules remove without tearing the substance. Cortex coarse, a little granular, & on the surface points of entrance of the stellate veins are depressed. Much blood in the vessels at the base & in the vasa rectae of pyramids.
Liver, Surface looks a little irregular, organ about normal size. On section tough, the surface markedly nutmeg, the hepatic capillaries being much distended, & the pale interlobular areas small. No stones in gall-bladder. Intestines appear healthy. Brain not examined.

Annotation for Protocol 16

In summary, this was a case of a 65-year-old male with healed myocardial infarction, with resultant cardiomegaly, heart failure, and anasarca. Localized complications of the myocardial scar were adjacent pericardial fibrosis and endocardial thrombosis.

Although the descriptive card with the specimen gives the patient's age as 60 instead of 65, so does an 1880 case report[109] which contains measurements identical to the above protocol. Neither the autopsy protocol or the 1880 report mention the coronary arteries. The description of these arteries and in the card suggests further study of the specimen at a later date, when there was more awareness of such a relationship. By 1892, as evidenced in his textbook[124], Osler was cognizant of the fact that myocardial fibrosis could be the end result of coronary artery disease.

The case report of 1880[109] contains a microscopic description of the fibrosed area (see Chapter IV), not present in the original protocol. It is evident, from this case, that Osler continued his study of some autopsies, and revised his concepts, even after initial publication.

AUTOPSY PROTOCOL FOR SPECIMEN 17

From: Osler's Autopsy Book, 1877, pp. 137-140 Figure 6[43]

Case XLIV **Heart disease. Pleurisy. Pul. apoplexy**

James Watt. aged 39. P.M. 20 hours after death

General appearance.

Body that of a large powerfully built man. Skin of the trunk of an icteroid hue. Upper part of thorax and neck intensely livid. Face swollen skin suffused; bloody fluid oozes from the mouth. On palpation the skin beneath the anterior and back parts of the trunk emphysematous, especially marked about the root of the neck. Legs & lower half of the thighs oedematous. Scrotum swollen and contains fluid. Posterior organs of the body of a dark red colour.

Thorax and Abdomen.

Intestines appear of a dark brown colour. Position of viscera normal. Stomach & intestines distended with gas. (Ounces) III of fluid in peritoneum. Left pleural sac contains 96 ozs of serum slightly tinged with blood. No floculi. Right pleura contains (Ounces) III of fluid. Left lung slightly adherent behind. Right lung free.

Pericardium

(Ounces) II f of-turbid straw coloured fluid. Patch of attrition over right ventricle. Subpericardial fat abundant and tinged lightly with bile.

Heart — A large quantity of blood escaped from the venae cavae on its removal. Organ appears large. Weight — 610 grms. Right auricle much dilated with blood and grumous clots. Endocardium deeply stained.

Right ventricle contains dark clots; walls stained. Tricuspid orifice dilated measuring 5⅞ inches in circumference, and admitting the four fingers nearly as far as the second joints. Valves healthy. Walls do not look thicker than natural ¼" over ant wall. Pulmonary valves normal and competent, circumference of the orifice 3 inches. Left ventricle small &

Figure 6.

The first four pages of Osler's handwritten protocol for autopsy 44, from which specimen 17 was obtained.
[from the *Post-Mortem Book of the Montreal General Hospital for the year ending May 1, 1877*[43].]

partially decolourized clot in the mitral orifice & adherent to the chordae tendinae. Chamber appears much dilated measuring 4½" from apex to aortic ring, and bulges much towards the right ventricle. Endocardium thickened, opaque over septum, not much stained. Walls over middle posterior part ⅞" in thickness. Posterior wall ½". Ventricular septum one quarter of an inch below the aortic valve ½" in thickness. Mitral valves slightly thickened at the edges. Orifice measures 4¼ inches in circumference. Valves competent. A small calcareous mass at the attached border of the anterior segment near the aortic valve.
Aortic semilunar valves. Segments thin, quite healthy & competent. Circumference 2¾ inches at the ring. Diameter of aorta 1" above the valves.
The aorta looks small, & the segments of its valves look smaller than those of the pulmonary artery.
Columnae carnae firm, some fibroid, a few of the thinner ones calcareous.
Substance of the Left vent, & whole heart, looks pale, and on examination is found in a condition of advanced fatty degeneration. A good deal of fatty infiltration also exists between the individual fibres.
The aorta in its course is not atheromatous. Intima healthy. Smaller arteries also are not diseased.

Lungs

Left dark in colour externally. Pleura covered over with shreds of lymph. Apex and posterior parts of the organ in a condition of collapse and only slightly crepitant. Anterior portion of upper lobe the seat of two large apoplexies. The larger one is situated in the (?) over the heart, is 2" in length and extends through the whole thickness. The other, situated above & anteriorly is about the size of a walnut, and is surrounded by a pale condensed wall. The lung tissue about these extravasations is firm airless and in a condition of Pneumonia. Another extravasation occupies the anterior border of the lower lobe & the lung tissue about it is consolidated.
Right lung crepitant throughout, congested, posteriorly a

small consolidated area — lobular Pneumonia — at lower part of anterior lobe in point. Otherwise looks healthy.

Spleen

250 grammes. Two fissures in the ant. border. Organ tolerably firm, much blood exudes. Malpighian bodies indistinct. Pulp of the usual colour.

Kidneys

Right 180 grms. Capsule readily detached. On section much blood exudes. Cortex pale. Malpighian tufts — alone injected. Pyramids red in colour.
Left 175 grms. Presents two puckerings in the upper part of the organ, & these extend for a short distance into the substance. On examination tubules of cortex blocked up with epithelial debris & contains a good deal of free fat & granules in cells.
Stomach and Intestines present nothing abnormal, the larger veins being as a rule full. The former contained about one pint of dirty fluid. M.M. soft.

Liver

1600 grammes. Surface smooth. Organ of good consistence. On section the large veins full. Lobules tolerably distinct and present a very beautiful nutmeg appearance Gallbladder contains a small amount of bile.
Brain. Veins of Pia matter full. Arachnoid a little opaque over the sulci. Nothing unusual about the base. Arteries are not diseased. Substance not examined as organ was kept for dissection.

Annotation for Protocol 17

In summary, this was an otherwise healthy, 39-year-old individual with cardiac hypertrophy (610 gms) and decompensation. Evidences of heart failure were generalized passive congestion, hepatosplenomegaly, peripheral and scrotal edema, hydrothorax, and hydropericardium. Measurements given for the heart valves indicate marked dilatation as well as hypertrophy. A terminal event was hemorrhagic pneumonia.

The cause of the cardiac hypertrophy is uncertain in this case. Possibilities include hypertension, or less commonly, idiopathic cardiomyopathy. The clinical sphygnomanometer was not invented until the end of the nineteenth century[196].

The clinical history of this case is available, being added to the protocol for publication in the *Montreal General Hospital Report* for 1877[17]. The patient had served with the army in India for 19 years and then worked as a blacksmith. Signs and symptoms included dyspnea, slight hemoptysis, vomiting, rales and albuminuria. A brief discussion favors overstrain or prolonged muscular exertion as the cause of cardiac hypertrophy.

Osler also published this case in the *Canada Medical and Surgical Journal* of 1878[126]. The discussion is fuller with some pathologic-physiologic reasoning. Osler stated that muscular exertion affects the circulation by interfering with the free passage of blood through the heart and increasing arterial pressure. He reviewed supporting literature, largely German.

This case is an example of the detailed autopsy examinations and measurements of which Osler was capable. It is also illustrative of the extensive degree to which he used some of his autopsy cases.

AUTOPSY PROTOCOL FOR SPECIMEN 18

From: Osler's MGH Autopsies, 1879-80, p. 37[45]

337 **Purulent Pericarditis**

Body that of a short fairly well nourished woman — rigor mortis present. *In Abdomen* — slight excess of fluid. Pylorus is situated below & 1½ inches to the right of Zyphoid Cartilage. — *On opening Thorax* in chest Pericardium seen to be much distended when opened about 34 ozs of turbid purulent fluid removed. Visceral & parietal layers thickened

& covered with numerous and shaggy masses of lymph — In right Pleura 2 ozs. In left a small amount of turbid fluid. *Heart* — right chambers contain small clot — valves of right side normal — on left side valves a little opaque, left ventricle thickened — muscle substance of a pale brown. Parietal Pericardium a good deal thickened in places 3 to 4 m.m., a good deal of subpericardial fat — particularly at base covered over with irregular fibrinous masses. *Lungs* — Right crepitant except at extreme base which is collapsed — some small Ecymoses over the pleura — Left lung — collapsed at anterior border crepitant. *Spleen* small weighs 100 gms presents nothing abnormal. *Kidneys* a little small-capsules detach readily — blood vessels moderately full small —arteries not specially thickened.
Stomach — (Aorta presents some scattered patches of atheroma) looks normal. Bile duct pervious. Liver of average size — contains a good deal of blood. Small central veins of lobules large.
Uterus small narrow polypus fills the canal of the cervix —projects by one in. attached just above the internal os. Nothing special in large intestines.

Annotation for Protocol 18

In summary, this woman died with approximately 1,000 ccs. of pus within her pericardial sac. The likeliest cause would have been an acute pericarditis due to bacterial invasion from contiguous pleura and lung. However, Osler's brief reference to the lungs suggests only atelectasis and petechial hemorrhage, unless the "ecymoses" were actually hemorrhagic abscesses. Incidental findings included atherosclerosis and endometrial polyps.

This protocol represents Osler's scantiest recording of an autopsy, and his least use of case material. Although the heart was used as a museum specimen, there is no record of publication of this case, or of a paper on the subject. His textbook of 1892[129] refers to 5 Montreal cases of pericarditis associated with pneumonia, but without reference to specific autopsies.

AUTOPSY PROTOCOL FOR SPECIMEN 20

From: Post-Mortem Book MGH Records, 1877-79, pp. 86-87[44]

CL **General Tuberculosis** 8/11/77

I Thompson. aet 5. (Pat. of Dr. Bell)

Body that of a tuberculous looking child. Skin (?), eye (?). Belly protuberant.

Abdomen — About a gallon of fluid in peritoneum. On the parietal layers of the membrane are numerous tubercles ranging in size from a pins head to a pea, very firm, dense and fibroid. None in the peritoneum nor intestines themselves, but on the portion over the mesentery is an eruption of exceedingly fine ones — like little grains. On surface of liver and diaphragm they are also numerous. In thorax right lung intimately adherent; left free.

Pericardium — Small quantity of fluid in sac. Parietal & visceral layers much thickened and rough. The membranes are irregular, not covered with the usual villous exudations, but uneven strands of firm tissue; a dozen or more flat slightly elevated caseous masses exist in both leaves. They are friable, yellow white in colour, and very superficial. Nothing abnormal in heart itself.

Lungs — Right tore in removal being very closely adherent at apex. It is heavy and slightly crepitant and in section contains much blood and serum. Miliary tubercles are scattered through the organ, chiefly at the apex. They are small, recent looking, here and there occurring in groups. There are no cheesy masses in the lung itself, but on extending the section towards the root of the organ 5 large caseous bronchial glands are seen projecting into the lung tissue. No trace of normal lymphatic tissue remains in these glands, they are (?) friable & yellowish in colour. The left lung is crepitant throughout, contains a few miliary tubercles. No nodules. Bronchial glands caseous but not so enlarged as in the other side.

Spleen Half a dozen round caseous spots, the size of large peas, in the substance of this organ. No miliary tubercles.

Kidneys Eight or ten tuberculous nodules in these organs, which otherwise appear normal. Two supernum spleens.
Liver Numerous miliary tubercles up on the capsule and on suspensory ligament. Organ of full size. On section dark in colour. Central veins full. Not amyloid (this condition was suspected)
Stomach and intestines appear healthy. Brain could not be examined.

Annotation for Protocol 20

In summary, this 5 year old male died of disseminated tuberculosis with a predominantly miliary type of involvement of lungs, liver, spleen and kidney. Serosal lesions were present in both the peritoneal and pericardial layers. Involvement of the latter was of longer duration, as suggested by general thickening of the layers. The caseous lesions described in the hilar lymph nodes were considered by Osler as the commonest source for pericardial spread[107]. Incidental findings were accessory spleens.

Osler does not state on what basis he ruled out amyloid of the liver at autopsy. His textbook of 1892[269] refers to the frequency of amyloid change in the liver, spleen, kidneys and intestines in tuberculosis. The characteristic gross appearance and staining reaction with iodine is described in another section[270]. Presumably Osler ruled out amyloid in this case on the basis of absence of these criteria.

A synopsis of this case was included as one of 17 examples of tuberculous pericarditis in Osler's definitive paper on the subject[107]. This child is referred to in the section on the chronic adhesive form. Such a chronic type would be unusual in a 5 year old child, unless the disease was acquired very early in life.

AUTOPSY PROTOCOL FOR SPECIMEN 21

From: Post-Mortem Book MGH Records, 1877-79, pp. 68-69[44].

Case CXXXV **Aortic Valve Disease**

AB. aet. a stout well built young man, had been a blacksmith

General appearance. Body that of a young, well developed man. Feet & legs edematous. Face suffused, eyelids puffy. Skin of upper part of body slightly yellowish, and mottled. *Thorax and Abdomen.* About a pint of clear fluid in abd cavity. Position of the viscera normal. In the thorax, the pericardium with enclosed heart seen to occupy a larger space than usual. Lungs universally adherent. Small amount of clear fluid in pericardium. Heart — Right aur. and the venae cavae are much dilated being filled dark serum-coagulated blood. Eustachian valve persists and is cribriform. Cavity would hold a large orange. Right ventricle, considerably dilated. Length from pulm. ring to apex 13 CC. Col. carn. strongly developed. Wall ½ CC in thickness. Pul. semilunar valves normal. Circumference of ring 9½ CC. *Left auricle* dilated. Endocardium opaque. Mitral orifice admits a ball 14 CC = 5½" in circumference. Left ventricle enormously dilated; length from aortic ring to apex 14 CC. Wall near septum ¾" thick. Toward apex 1½ CC. Mitral valves little, fat all thickened. Chord. tend. appear numerous, not shortened. Mus. papil. look flattened out, apices slightly fibroid. Surfaces & margins of some of the columnae carnae also fibroid.

Aortic ring measures 8 CC = 3¼", and is apparently guarded by only two valves, between which there is an irregular interval. The posterior segment is large, 3½ CC along its free border, where it is slightly thickened; the body except for one central spot, is translucent. Right and left segments appear to have merged together, looking like a single large imperfect valve having a free border 3½ CC in length. The end of the right nearest the post segment is quite loose, only anchored by a cord 1 CC in length which is attached close to

the posterior valve in a line extending in the curve where the The united valves are thick, especially at edges, and they have not the normal length, the orifice of the right coronary not very covered. The separation between the sinuses of Valsalva is quite distinct, but the raphe between the segments only extend to their bases. On the ventricular side a (?) marked groove indicates the separation. Weight 690 gms, 1 lb. 8½ oz.

Lungs. Right almost universally adherent. It is heavy & oedematous, & contains much blood in dependent parts. The left also adherent except at extreme apex. On section only slightly crepitant. The surface looks like a piece of spleen.

Kidneys R. 220. L. 215 grms. Capsules detach easily. Cortices anaemic & coarse looking, bases of pyramids congested. Organs very firm.

Spleen firm, not enlarged; pulp of a dark red colour. Malpighian corpuscles distinct. Weight 205 grms.

Liver remarkably nutmeged — a little increased in size.

Nothing abnormal in stomach, intestines.

Annotation for Protocol 21

In summary, this male had a markedly hypertrophied and dilated heart, as evidenced by the weight and the increased circumference of valves. The presence of heart failure is suggested by ascites, peripheral edema and generalized visceral congestion. The bicuspid aortic valve may have been responsible for aortic insufficiency with consequent left ventricular hypertrophy and failure. A terminal event was acute pulmonary edema.

This autopsy protocol was included in Osler's *Montreal General Hospital Reports* of 1878[130]. Added was a brief clinical history of a 26-year-old blacksmith with cardiac murmur and signs and symptoms of heart failure for more than a year. A drawing of the valve is also provided (Fig. 5). According to a published synopsis of a paper delivered by Osler[131], eighteen cases of bicuspid aortic stenosis were seen by him at the Montreal General Hospital.

AUTOPSY PROTOCOL FOR SPECIMEN 33

From: Autopsy Book of the Montreal General Hospital for 1902[271]

Autopsy 83, Series 1903 Date — May 5, 03

Name Richard Digby Age 55

Occupation Laborer

Exam made at Magdalen St, PE. St. Charles, Partial Autopsy. Body of a moderate sized man, considerably emaciated: rigid: Externally no marks save two small depressed scars over region of left groin: no scar on genitals. On section, sternum is found adherent to tumor over region of r. 3 & 4th ribs & costal cartilages: This tumor rises up from deeper structure about 7 cm diameter, is firm & whitish. Pericardium — adherent throughout by adhesions which, while easily broken down, are not very recent: Moderately organized. Complete adhesion of left Pleurae throughout: No fluid: considerable fluid in rt. chest, rising half way. Left lung shows considerable oedema: Rt. lung several infarcts, red, of consid. size: Largest 6 x 6 x 6 cm., triangular. Otherwise lung tissue oedematous.

Heart — Much enlarged, especially Left ventricle: no valves seem diseased, save aortic which is mod. sclerosed, but appears competent.

Muscle of l. vent. 1.75 to 2 cm thick: soft, mottled: cavity much dilated.

Tumor comes off from rt. side of asc. aorta (1st part) measuring 7 x 7 x 9 cm. subcircular. 3rd of its cavity filled by organized clot which is easily broken down. Wall of aorta throughout shows mainly ath. ulcers: the rt. sinus is sl. dilated: No rupture has occurred.

Spleen — indurated.

Liver — Firm: op. sect^{n} nutmeg very markedly.

Kidneys — much lobulated & R. shows deep depressions where loss of tissue has occurred. Both large & mod. firm. Caps. not adherent. Neph. vs "progress". Other abd. organs nothing special

Mic. Exam:

Heart muscle. Intense fatty degeneration: all globules small & nearly all intracellular.
Many musc. fibres of large size, nuclei also: fibres fewer than usual & much larger. The larger muscles are size of 20-25-u. diameter or more.

Hyper. & fatty Degen:
Liver — Cells very numerous: centres of lobules (stained spec) almost colorless: Dil. of central veins: Many fragmented nuclei in centres of lobules: peripheral cells about normal.
Kidney — Glomeruli large: much prolif. of cells & many nuclei of comma or flattened shape interspersed: Many such cells between the lobules throught. No exudate in Bow. capsule.
Diffuse interstitial nephritis:
ANATOMICAL DIAGNOSES
Sacculate *aneurysm of 1st part of aorta:* organized clot in it: arteriosclerosis: adherent pericardium: *chronic diffuse myocarditis* (stage fatty degeneration): Hyper. & dil. of heart: infarcts & oedma of lungs: fatty nutmeg liver: induration of abd. organs: chr. diffuse interstitial nephritis (mod.):

Annotation for Protocol 33

In summary, this 55-year-old laborer had a large aneurysm of the ascending aorta with adherence to the sternum and pericardium. Mention is made of atheromatous ulcers of the aorta, but not of syphilitic changes, which would be the likely cause of an aneurysm in this location. Terminal events include pulmonary edema, generalized passive congestion, and infarcts of the lung. The descriptive card accompanying the specimen indicates rupture of the aneurysm into the trachea. Presumably this was not recognized at the time of the initial autopsy.

Of interest is the microscopic description at the end of the protocol. Fatty degeneration of myocardial fibers may have been related to intra-tracheal hemorrhage with shock and hypoxia. The relative lack of staining of the central parts of hepatic lobules was likely due to centri-lobular degeneration

caused by congestion and hypoxia. The renal description is more difficult to interpret. Scattered deep depressions may represent healed infarcts, following emboli from thrombus material associated with the aortic aneurysm. Another possibility is focal chronic pyelonephritis. The proliferation of cells with flattened nuclei may represent fibroplasia.

This autopsy was not performed by Osler. The fact that he added a specimen to his museum nineteen years after his departure from McGill was indicative of an intensive and prolonged dedication to morphologic aspects of disease. This much later autopsy protocol is no more detailed or advanced than those of Osler of a much earlier period. Although Osler did not record microscopic studies in his own protocols, they were performed in some cases, as evidenced by inclusion in latter presentations of the cases.

AUTOPSY PROTOCOL FOR SPECIMEN 41

From: Post-Mortem Book MGH Records, 1877-79, pp. 238-239[44]

CCLXXXI **Cancer of Stomach** 22/2/78 9 hrs Pm

Mrs. Howard aet 45 — Ill 5-6 months with well marked symptoms of C. (?). Moderate emaciation. *general oedema* —not much in trunk.

In *abdomen* Parietal peritoneum in epigastric region adherent to omentum and deeper structures. About two quarts of turbid fluid, with lymph flakes, removed. Parts about margin of left lobe of liver & stomach matted together & the omentum is retracted and puckered.

In *thorax* lungs intact, no adhesions except at apices. *Heart.* of average size, pale; rt. chamber full of dark coffee coloured clots. Valves & orifices normal. Left V. not (?) a small clot, muscle substance pale. Valves normal. Aorta healthy. *Lungs* pale, dark coloured at bases from filling of blood vessels. At left apex a slight puckering & within this a small cavity, size

of marble, with well defined walls & fluid — pus-contents. Rest of organ crepitant. At right apex, puckered spot, but no cavity.
Spleen small, capsule shrivelled. Pulp (?)
Kidneys normal size, capsules detach readily. Organs pale, look fatty. Med. rays very pale, vessels between full. Liver, stomach and duodenum removed together.

Annotation for Protocol 41

In summary, this 45-year-old female died of emaciation and anasarca about 6 months after onset of symptoms related to malignancy. Anasarca may have resulted from hypoproteinemia. The turbid fluid in the peritoneal cavity is suggestive of gastro-intestinal perforation. Although the protocol does not describe dissection of the stomach, the museum specimen is characteristic of an extensive gastric carcinoma that has eroded through the wall of the stomach. The incidental finding of apical scars and an associated cavity is suggestive of healed or quiescent pulmonary tuberculosis.

This is a rather cursory autopsy protocol with no measurements and no recording of the major findings. It is possible, that because of pressures of other duties, Osler elected to complete the dissection at a later date. That he did so is evidenced by the carefully dissected museum specimen.

AUTOPSY PROTOCOL FOR SPECIMEN 46

From: Post-Mortem Book MGH Records, 1877-79, pp. 195-6[44]

Case CCXLVII

(?) vent. **Tuberculosis Pul.**

Dr. (?) aet 55, died suddenly: had suffered for years from dyspepsia & had a cough for some time. A daughter had died of consumption. Body considerably wasted.

Nothing of special note in abdominal cavity. In thorax pleural sac is almost completely obliterated on each side more fully in the left. *Heart:* right chambers moderately full of dark blood and grumous coagula. Tricuspid orifice a little larger than normal. Valves healthy. In left ventricle, Mitral a little thickened at edges. Muscle substance looks dark, not increased. Semilunar valves firm, calcerous at bases. Corp. arantii fibroid & thick.

Aorta is slightly dilated and presents small scattered patches of atheroma and at beginning of descending part a firm calcar. plate. In thor. & abd. part not as ather. but at bifurct. where it lay upon lumbar vert., it was very thick & rigid, from the presence of calcareous laminae.

Lungs — left crepitant throughout, except in certain areas at the apex, which feel firm. On section a good deal of blood and serum oozes from post. parts. At apex is an old fibroid spot pigmented & with stellate cicatrix. In its neighbourhood & scattered more or less through the entire lobe are groups of very firm miliary granulations, grey in colour but very dense & hard. They were chiefly arranged in groups closely set together, 50-80 in each, very few hard (?) & there was no caseous degeneration or lobular inflammation about them. A few were noticed in the middle and lower lobes. In the right lung the tubercles were still more abundant at the apex and through the upper lobe presented the same (?) firm to the touch, in groups yet not coalescing into large nodules. No caseous masses. Post. parts (?) congested & oedematous. Bronchi contain a frothy serum: they are not specially injected.

Br. glands not enlarged.

Spleen a little enlarged, dark in colour.

Kidneys a little firmer than natural — otherwise normal.

Liver healthy.

Stomach. Immediately below the cardiac orifice, on the post. wall there is a loss of substance ½" in diameter, base whitish firm, edges hard, not much elevated. M.M. about it is a little puckered. On peritoneal surface adhesions had formed with neighbouring parts and a firm mass of fibrous tissue 1/3" in thickness formed the base of the ulcer. On the post. wall of

the fundus, situated 1", 1½" & 2" distant from the main ulcer are 5 smaller ones the largest the size of a three-penny bit. Edges a little firm. Bases greyish white & peritoneal surface a little thickened. M.M. over rest of organs thickened & firm, covered with an adherent mucus. Pylorus normal. Bile flows from Pap. (?).

Annotation for Protocol 46

In summary, this 55 year old physician died of acute miliary tuberculosis of the lungs, apparently arising from more chronic disease in the apices. Also present were extensive pleural adhesions secondary to the tuberculosis. The multiple peptic ulcers of the stomach were related to the history of dyspepsia. The "grumous coagula" in the heart were undoubtedly post mortem clots.

This protocol was reproduced in the second *Montreal General Hospital Reports*[73], where the statement is added that "Examination of the ulcers gave no evidence of tuberculous origin".

AUTOPSY PROTOCOL FOR SPECIMEN 53

From: Post-Mortem Book MGH Records, 1877-79, pp. 121-122[44]

CLXXXIV **Chronic Inter. Neph. Ac. Pneumonia. Hyper. Cord.**

aet.

Body that of an averaged sized poorly nourished man. Legs oedematous. Scrotum swollen, old cicatrixes on left legs, about ankle.

Abdomen no fluid, serosity increased. Position of viscera normal, though stomach extends low down and is vertical. In *Thorax* 75 ozs of turbid serum in right pleura in which flocculi float. None in left.

Pericardium — little (?) subpericard. fat. Very little fluid. Patch of attrition in right vent.
Heart very large, weighs 575 gms — a large red beefy organ. Left auricle greatly distended with blood and clots, which are colourless at upper part. Rt. ventricle large, length from pul. ring to apex 10 C. Ant. wall 4 C. Col. carnae very distinct and developed. Tricuspid orifice 14.5 C. Valves normal. Pul. orifice 9.6 C. Valves healthy. Left auricle full of clots which pass thro. the mit. orifice and fill the left vent. in which cavity there is a large coagulum which is continuous with one in the aorta. Walls greatly hypertrophied. Ant wall midway between apex & base 1.9 C. posterior 1.5 C. Septum 2.1 C. Length of cavity 10. C. Mitral orifice 10.4 C. Valves thin & healthy. Aortic orifice 8.3 C. Valves competent & normal. Rt. sinus of Valsalva very deep. Arch not dilated; intima contains a few patches of atheroma & they also occur scattered over the thoracic and abdominal portion. They present flattened yellowish swellings — not calcareous.
Lungs R. Weighs 1125 grms. Pleural surface injected and covered over with a thin layer of false membrane, a similar one existing in the costal leaf. Upper lobe in its posterior ¾ of its extent is in condition of hepatization. Ant. fourth is collapsed the free border alone crepitant. Middle lobe is collapsed. Whole of lower lobe, with exception of extreme post. border is solidified. On section of these hepatized areas the surface is red, (?) and these mottled with small colourless areas; a large quantity of purulo-sanguinous fluid bathes the section. On close inspection the fine granules can be seen plugging the air cells, most of them still red, a few greyish white in colour. Bronchi are free, do not contain casts.
Left lung weighs 575 grms. Crepitant throughout. Does not contain an unusual quantity of blood in post pts. Ant. region in section even dry. No oedema of the organ. A few spots of collapse in lower lobes; and one or two very dark areas, looking like spots of apop.
Spleen small firm & hard. On section trabeculae distinct.
Kidneys R. 121, L. 120 grms. Organs *small.* Capsules opaque & detach without tearing substance, but show dark red granular surfaces, in which the remains of the primitive

lobules can be plainly seen. Granules are fine but very evident. On section organs congested. Cortices diminished, in places very much so. The columns of Bertini (?), in between the pyramids seem large. Arteries very prominent at bases. On surfaces small cysts are seen here & there (?). Bladder & arteries present nothing abnormal.
Stomach & intestines present nothing of special note. *Large bowel* Mucosa normal. *Liver,* not diminished in size — nutmeg. D.C. chol. patent.
Brain nothing abnormal. Arteries at base a little thickened but not markedly atheromatous.

Annotation for Protocol 53

In summary, this adult male died of lobar pneumonia involving the upper and lower lobes, with associated pyothorax. This was likely a terminal event complicating renal disease, as evidenced by finely granular kidneys, and marked cardiac hypertrophy.

Chronic renal disease may result in hypertension with consequent cardiac hypertrophy. However, the fact that the kidneys were not significantly reduced in weight mitigates against primary renal disease with end-stage kidneys. More likely is primary hypertension resulting in both cardiac hypertrophy and arteriolonephrosclerosis. Osler did recognize that hypertrophy of the heart could be a consequence of "cirrhosis" of the kidney[240], as originally suggested by Bright in 1836,[272], but hypertension was not a generally recognized primary disease in the nineteenth century.