THE GULSTONIAN LECTURES,
ON
MALIGNANT ENDOCARDITIS.

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LECTURE I.

MR. PRESIDENT AND GENTLEMEN,—It is of use, from time to time, to take stock, so to speak, of our knowledge of a particular disease, to see exactly where we stand in regard to it, to inquire to what conclusions the accumulated facts seem to point, and to ascertain in what direction we may look for fruitful investigations in the future. With your permission, sir, I propose to do this in the case of that most interesting disease generally known as ulcerative endocarditis, a disease the phenomena of which were first clearly explained by the late Dr. Kirkes, from whose investigations in 1851-52 we date our accurate knowledge of the affection. Some of those who listen to me to-day can doubtless recall, and recall with pleasure, the Gulstonian Lectures of 1851, in which Dr. Ormerod dealt so fully and so ably with valvular affections of the heart; but a reference to them will show how much the past twenty-five years have done to widen our view of cardiac disease, more particularly in regard to the effects of emboli, and the association of valvular inflammation with grave constitutional disorder, and the probable connection of the disease with the presence of micro-organisms. By the labours of Drs. Ogle, Wilks, Simpson, Moxon, Bristowe, and others in this country, of Charcot, Vulpian, and Lancereaux in France, and of Virchow and a host of observers in Germany, a large amount of material has been accumulated; and we may assume that the etiological, clinical, and anatomical characters of the disease have been fairly well ascertained, and that we have got about as far towards a full knowledge of the affection as the ordinary means at our disposal will permit. The inquiry now enters upon another stage, and it remains for experimental investigation to determine, if possible, the relation of the endocarditis to those diseases with which it is most frequently associated. This being the case, the present time has seemed to me a favourable opportunity to summarise our knowledge to date; and, for this purpose, I have reviewed the records of over two hundred cases, which, from the description of the symptoms and lesions, were evidently of the type of malignant endocarditis; and these, with the considerable experience I have had at the General Hospital at Montreal, may perhaps enable me to give a somewhat more comprehensive account, in some respects, than has yet been attempted.

In discussing the subject of endocarditis, we are met at the outset by difficulties of nomenclature and classification. The designation acute may be used to indicate those forms which are accompanied by proliferation of, and exudation upon, the endocardial surface, with or without loss of substance, as opposed to chronic, in which there are sclerotic changes without vegetations. Subdivisions of the acute form have been arranged on an anatomical basis, as the terms plastic, papillary, verrucose, fungous, ulcerative, indicate. On the other hand, from an etiological point of view, the forms of endocarditis are as numerous as the diseases in which it occurs, and we constantly hear the expressions puerperal, rheumatic, scarlatinal, etc. Some speak of primary and secondary forms; while, from a clinical standpoint, they are arranged in two classes, simple and grave. Anatomically, there appear to be no very essential differences in the various forms of acute endocarditis. Between the small capillary excrescence and the huge fungating vegetation with destructive changes, all gradations can be traced, and the last may be the direct outcome of the first; the two extremes, indeed, may be present in the same valve. They represent different degrees of intensity of one and the same process. A classification of cases, based on the ordinary macroscopic characters of the inflammatory products, into watery or verrucose and ulcerative, will, in many instances, group together cases widely different in their clinical aspects; and, contrariwise, a clinical subdivision into cases of simple and cases of malignant endocarditis by no means of necessity implies that the lesions in the former case are all of the plastic or warty variety, and in the latter of the ulcerative or destructive. The term ulcerative has come into very general use to describe the grave form, and it expresses well an anatomical feature present in a large proportion of cases; but in others it is very inapplicable, as there may be no actual loss of substance, and no more destruction than occurs in the verrucose form; and, on the other hand, there may be great destruction and ulceration from causes of an entirely different nature. The numerous other terms employed—septic, infectious, diphtheritic, mycosis endocardii, arterial pyæmia—while each expressing some special feature, and so far suitable, have never come into very general use. On the whole, it seems to me that the names simple and malignant, which we use often to separate the milder and severe forms of many diseases, might appropriately be employed in describing the cases of acute endocarditis; the simple being those with few or slight symptoms, and which run a favourable course; the malignant, the cases with severe constitutional disturbance and extensive valve-lesions, whether ulcerative or vegetative, the term being more clinical than anatomical.

Malignant endocarditis occurs under the following conditions: 1, as a primary disease of the lining membrane of the heart or its valves, either attacking persons in previous good health, or more often attacking the debilitated and dissipated, or those with old valve-lesions; 2, as a secondary affection in connection with many diseases, particularly rheumatic fever, pneumonia, scarlet fever, diphtheria, ague, etc.; 3, as an associated condition in septic processes, traumatic or puerperal. We shall discuss first the anatomical characters, then the clinical features, and lastly the etiological and pathological relations.

The lesions of malignant endocarditis are by no means uniform, and may be vegetative, ulcerative, or suppurative; and these various forms may occur alone or in combination. The belief that there is always ulceration has led to some confusion; and we must recognise that there are cases with the clinical history of the malignant form in which, *post mortem*, the valvular condition has been that of a severe vegetative or verrucose endocarditis. Such a case was a lad aged 11, a patient of Dr. Molson's, from whom I obtained the specimen which I pass round. He had chorea in July 1880, the second attack. Rapid improvement and recovery under Fowler's solution, five minims every four hours hypodermically, took place. There was a slight murmurish condition of the first sound. When seen again on March 3rd, 1881, the chorea had returned, having begun ten days before. The patient improved until the 10th, when he began to be feverish; had exacerbations each evening; temperature rising to 104° Fahr. He became unconscious. There was slight paresis of the left side, and death took place on the 16th. The temperature on the 15th was nearly 106°. There were irregular, soft, greyish-white vegetations on the mitral valve, infarcts in the spleen and kidneys, and a small spot of red softening in the right corpus striatum. These photographs from a case of Dr. Musser's illustrate a more advanced condition of the same kind; the vegetations were larger, more abundant, and some were a little irregular and soft on the surface, but, unless a mass were removed, no actual loss of substance was seen. Even in the smallest vegetation there is some destruction of endocardial tissue, if only of the endothelium and superficial layer; while the larger outgrowths are more deeply set in the valve, or may involve the entire thickness. More commonly with or without vegetations, there is ulceration, the frequency of the occurrence of which has given the name most often attached to this form of endocarditis. The loss of substance may be superficial, involving only the endocardium, or it may be deep and destructive, leading to perforation of a valve, of the septum, or of the heart itself. On the valves, extensive outgrowths usually accompany the process, and may conceal the ulcer or project as fungating masses from its edge, as is well illustrated by this coloured drawing. In many instances, the process appears simply ulcerative, without any vegetations to speak of. In the slightest form, only a superficial abrasion exists, perhaps scarcely recognisable; in others, a process of erosion may go on by which half a valve may be destroyed, or there may be (as shown in this drawing) a deep excavation extending beyond the valves, and destroying the muscle-substance of the heart, leading to perforation of the septum or of the wall of the ventricle. These are well known features, however, upon which I need not dwell. In two instances, I have seen superficial necrotic changes without ulceration or vegetations, circumscribed patches, of the size of a sixpence, opaque yellow-white in colour, resembling the necrotic pleura, over a pyæmic infarct of the lung, or a portion of dead peritoneum at the base of a deep typhoid ulcer. Doubtless, these would in time have formed ulcers. I find this condition noted by one or two observers. Lastly, the process may be suppurative, in which case the deeper tissues of the valve appear first involved, and the endocardium only implicated by contiguity. The occurrence of small abscesses at the base of extensive vegetations is not uncommon, but there are also instances in which the suppuration seems the initial step. The combination of ulcerative and fungating

outgrowths is, perhaps, the most common condition. The vegetations vary a good deal in appearance and consistence. Soft greyish-white masses, with roughened friable surfaces, to which thin blood-clot adheres, are numerous; or there may be large cauliflower-excrescences, with deep jagged fissures; or, again, long, pendulous, stalactitic masses. In the latter form, we often see, as Dr. Moxon pointed out, the effects of friction, and such a long vegetation from an aortic cusp may produce, by contact, a whole series of smaller outgrowths along the ventricular wall. The pressure of the valves against each other, and the action of the blood, tends to loosen and break the vegetations, and one can sometimes see where masses have been torn off, either entire or by a gradual process of disintegration. Considering the force with which the valves come together, it is curious that the soft vegetations, occupying, as they generally do, the lines of closure, can resist the constant compression to which they are subjected. Some vegetations present a remarkable greenish-grey or greenish-yellow colour. Changes in a conservative direction may go on when the disease is much prolonged. Fibroid induration may take place in the deeper parts, while the superficial portions remain unchanged and necrotic, perhaps also becoming a little harder and shrinking. Such a process can be seen in this specimen of endocarditis from an ox, in which there were most extensive vegetative and destructive changes. Not unfrequently the vegetations are gritty, from the deposit of lime-salts, which may take place in very acute cases, and is not necessarily an indication of age. It is interesting to note how often inorganic material is deposited in the neighbourhood of micro-organisms, as here on the endocardial outgrowths, in the tonsillar crypts, and about the tufts of actino-myces. Two conditions must be distinguished from the lesions of malignant (mycotic) endocarditis: the atheromatous degeneration in sclerotic valves, which leads to ulceration and extensive destruction of segments, a process which has nothing in common, except in its effects upon the valves, with the acute ulcerative changes above described, but is similar to the atheromatous processes in the aorta. It must not be forgotten, however, that an acute mycotic process may be engrafted, and indeed, often is, upon old sclerotic valves, the seat of atheromatous changes. The firm white globular thrombi of the auricular appendices, and of the interstices of the columnæ carneæ of the ventricles, have sometimes an appearance closely resembling endocardial outgrowths, and when softened in the centre and ruptured, the resemblance may be very close indeed. It is possible that the granular *débris* of an atheromatous abscess or a softened thrombus may possess irritating properties when discharged into the blood.

Histological Characters.—The study of a small fresh endocardial vegetation shows it to be made up of cells derived from the sub-endothelial layer, round and fusiform, which, by their proliferation, have produced a small nodular projection on the surface of the endocardium. Varying with the rapidity of the growth, the mass will present the characters of a soft granulation-tissue or a tolerably firm fibrous outgrowth. Usually, the round cells predominate; but there may be many elongated spindle-formed cells, with three or four processes. What part the endothelium plays in this growth, has not been determined. Tiny outgrowths may be seen, in which the process appears to be entirely subendothelial; but usually, before the mass attains any size, the smooth surface is lost, and there is deposited upon it a cap of fibrine in the form of a granular, sometimes stratified, material, of variable thickness. Though this resembles an ordinary coagulable exudation, it is probably deposited directly from the blood, and is of the nature of a thrombus. Upon and in this layer may be found, sometimes in large numbers, those remarkable little bodies which have long been known, when collected together, as Schultze's granule-masses, and which have of late become prominent as the blood-plates of Bizzozero and the hæmatoblasts of Hayem. Occasionally, they are very abundant; and I have seen soft warty vegetations composed (superficially) in great part of them. As their connection with endocardial and endarterial outgrowths has not, so far as I know, been referred to, I may be permitted to call attention to these two drawings, which further illustrate this point. The first represents the aorta from an old man dead of carcinoma, in which, just above the bifurcation, three irregular masses are shown, one nearly an inch in length, which projected fully a quarter of an inch from the intima of the vessel. They were attached to atheromatous ulcers, were soft greyish-white in colour, and were composed exclusively of the elements of Schultze's granule-masses, with fibrine-fibrils, and here and there a few white corpuscles. The second drawing illustrates a small aneurysm of the aorta, which has perforated the œsophagus. On the wall of the sac, the artist has represented a number of irregular whitish lines, which were narrow elevated ridges, also made up microscopically of these small discoid elements, the connection of which with fibrine-formation has been strongly insisted upon by Bizzozero. Scattered in and beneath the fibrinous exudation are numerous small granular bodies, which have the appearance and reaction of micrococci.

The larger vegetations, more characteristic of malignant endocarditis, consist of a granular material composed of altered and dead tissue-elements, fibrinous exudation, and colonies of micrococci; the deeper parts present the appearance of a granulation-tissue, while at the attachment in the valve there is always more or less infiltration and increase of the cell-elements. The granular substance is structureless, and resembles diphtheritic exudation, the resemblance at times being so close that one can readily understand the application of the term "diphtheritic" to the inflammation. It may be distinctly laminated, and, with a high power, fine filaments can be seen, though usually the granules conceal all appearance of structure. Strands of translucent material may occur throughout the mass, as if portions had undergone a sort of hyaline transformation. In some instances, this is very marked. Pale spheres filled with granules also occur, and may be very abundant. They have been described as colonies of micrococci; but some regard them as altered endothelial elements. I have seen them too numerous to be explained on this view. At the attachment of the vegetation, there is a zone of tissue deeply infiltrated with leucocytes, and deeper still the tissue-elements of the valve present an increase of nuclei and cells. The destruction of tissue appears to result in two ways: first, a gradual extension inwards of the necrotic process, doubtless induced by the micrococci; secondly, the softening and separation of valve-tissue caused by the rapid development of leucocytes at the base of the vegetation.

The micrococci are constant elements in the vegetations. All granules of an uniform size met with in the sections are not micro-organisms, nor, indeed, are all which stain by some methods recommended for the detection of these bodies. By far the most satisfactory method is that of Gramm (*Fortschritte der Medicin*, Band i, Berlin), in which the section, after staining in gentian-violet, is transferred for a few minutes to a dilute solution of iodine and iodide of potassium, and then to the alcohol, when it is found that the colour has been extracted from all tissue-elements and nuclei, leaving only the micro-organisms stained. They vary a good deal in number and arrangement, and may be scattered singly in the granular substance or arranged in groups. They are usually very numerous at the deeper part of the vegetations, just where the structureless material joins the granulation-tissue, and they may penetrate deeply into the substance of the valve. Sometimes the smaller vegetations seem made up exclusively of them. Several of my specimens appear to confirm the view of Klebs (*Archiv für Experiment. Pathologie*, Band vi), that the micrococci lodge first on the endocardium, and penetrate into the substance, often as distinct columns. In their immediate vicinity, there is a zone of necrosis, and beyond this an accumulation of leucocytes and signs of reactive inflammation. The micro-organisms found in connection with the malignant endocarditis are not all of the same kind. Klebs distinguishes two forms, one met with in septic, and the other in rheumatic, cases. In some instances, the micrococci are all arranged in zoogloea-like masses; in others, particularly the septic cases, they are in chaplets. Some present distinct capsules. Small elongated bacilli have also been found; I have seen them in one instance, short stout rods, often joined in pairs. Delafield and Prudden (*Text-book of Pathological Histology*, New York, 1885) have recently noted the presence of bacilli in the vegetations of a very acute case of malignant endocarditis. Cornil, in a recent lecture (*L'Abeille Médicale*, No. 51, 1884), stated that the bacillus tuberculosis had been found in the vegetations on the valves in cases of phthisis, and expressed the opinion that before long we should have accurate knowledge of a variety of micro-organisms in endocarditis depending upon the nature of the primary disease. By culture-experiments alone can we hope to have the question settled.

The following figures give an approximate estimate of frequency with which different parts of the heart are affected. The aortic and mitral valves were affected together in 41 cases, the aortic valves alone in 53, the mitral alone in 77, the tricuspid in 19, the pulmonary valves in 15, and the heart-wall in 33. The right heart is rarely affected alone; this occurred in only 9 instances, in 5 of which the tricuspid, and in 4 the pulmonary, valves were involved. The valves are most often attacked along the lines of closure, as in the simple endocarditis; the auricular faces of the mitral flaps and the ventricular surfaces of the aortic cusps suffering most severely. Mural endocarditis is most often seen at the upper part of the septum of the left ventricle, just below the aortic ring, in which situation some of the most extensive and deep cardiac ulcers occur, leading to perforation of the septum

Next in order is the endocardium of the left auricle on the postero-external wall, as noted by Lepine (*Bull. de la Soc. de Biologie*, 1869).

The local effects of the ulcerative changes are important. Perforation of a valve-segment is extremely common; sometimes there is a clean-cut, punched-out hole, with scarcely any irregularity of the edges; more frequently, however, there are great fungous vegetations which completely close and conceal the perforation. Erosion of the chordæ tendinæ is frequently met with, and an entire group passing to the papilla may be destroyed, the ends curled and encrusted with vegetations. Ulceration of the heart-muscle, leading to perforation of the septum or of the wall of a chamber is a much less frequent occurrence. I have collected notes of eleven instances; three of the septum close to the aortic ring. Ulcers at the aortic ring perforated the left auricle in three instances, the right auricle in one, and the right ventricle in one. In a remarkable case of Dr. Stephen Mackenzie (Pathological Society's *Transactions*, vol. xxxiii), the left ventricle was perforated by an ulcer at the apex. In a case of Dr. Curnow (*Lancet*, 1883, vol. i), the ulceration extended between the coats of the aorta, and then perforated into the lumen of the vessel, and in one of the Montreal cases there was perforation of an aneurysm of the aorta by ulceration, an instance of extensive ulcerative endarteritis with the production of multiple aneurysms. Another common result of ulceration is the production of valvular aneurysm. The anterior flap of the mitral valve is most frequently affected, and then the aortic cusps. In the records of the cases which I have reviewed, I was surprised not to find this condition noted oftener, only in about 12 per cent. of the cases; but, in very many cases, the record of the anatomical condition was meagre. I shall not refer further to this interesting point, as Dr. Legg has dealt with it very fully in a recent lecture at this College (Bradshawe Lecture, August, 1882). I may observe, however, that the atheromatous ulceration is also a frequent cause of aneurysm of the valves.

It was Sir James Paget (*Medico-Chirurgical Transactions*, vol. xxvii), I think, who first referred to the frequency with which sclerotic and malformed valves are attacked by acute disease. Chronic valvulitis is met with in a large number of cases of malignant endocarditis. The records which I have examined give only a percentage of about twenty-five; but the condition of the valves, except as regards ulceration, was often omitted, and thus represents a very much smaller percentage than actually occurs. In more than three-fourths of the Montreal cases, sclerotic changes were present; and Dr. Goodhart found (Pathological Society's *Transactions*, vol. xxxiii), in a series of sixty-nine cases, that sixty-one presented old thickening of the valves. In very many of the cases, the condition of fusion of two of the aortic cusps was present. This abnormality is almost invariably accompanied by sclerotic changes, and to the existence of these is probably due the frequency with which they are attacked by ulceration. In seventeen instances of fusion of two of the aortic cusps of which I have notes, there were ulcerative changes in eight, in two or three of an atheromatous nature.

In a few instances, the aorta is involved with the heart. The most frequent site is the sinuses of Valsalva, the vegetations growing through the segments spread on to the aortic wall; or it is affected by friction. It is rare for the vegetations to extend into the arch. I have met with one remarkable instance of ulcerative endocarditis in which there was also ulcerative endarteritis, involving the arch and producing multiple aneurysms. The specimen which I here demonstrate was taken from a man aged about 30, who had been the subject of syphilis, and was known to have had aortic incompetency for some time. He was admitted to the General Hospital, Montreal, on June 4th, 1880, with diarrhœa, chills, headache, cough, and fever. Temperature 104°. There were signs of pneumonia at the left base. He became delirious, a low typhoid condition supervened, with chills at intervals, and death took place on July 1st. The aortic valves were curled and hard, and presented extensive recent vegetations; the arch of the aorta presented four aneurysms, three small, not larger than cherries, and one of the size of a billiard-ball. The small ones were not noticeable as aneurysms from the internal surface, but presented the appearance of fresh fungous vegetations, on separating which little slits could be seen leading to saccular dilatations of the middle and outer coats. The large aneurysm was thin-walled, with no laminated fibrine, and presented at the edges of the orifice and over the whole lining membrane of the sac many greyish-green vegetations, some of which had perforated the sac and caused a rupture into the pericardium. It may be presumed that, in this instance, the ulcerations led directly to the production of the aneurysms, certainly in the case of the smaller ones; and the larger sac presented a condition of mycotic endarteritis unique in my experience of aortic aneurysms.

Of associated pathological changes, we have, in the first place, those connected with some primary disease, to which the endocarditis is, in the majority of cases, secondary. Thus, in the endocarditis of septic processes, there is the local lesion, a suppurating wound, a phlegmonous inflammation, or puerperal processes of a septic nature. In a very considerable proportion of cases, there is evidence of recent pneumonia; in others, rheumatic affections of joints; and in a few, diphtheritic processes. In the group of primary cases, the lesions are entirely those of endocarditis, local and general. In the second place, there are the extensive pathological changes due to embolism; and these constitute interesting features in the disease, and may produce a very great variety of lesions in every portion of the body. I do not propose to deal very fully with these, but to call attention only to some special points. The cases may be divided into those without any embolic processes, cases in which the infarcts are simple, not suppurative, those in which there are innumerable suppurative infarcts and cases in which some of the infarcts are simple and some suppurative. It is remarkable how variable these embolic features are. They may be entirely absent in well marked malignant cases. They are not necessarily associated with suppuration; indeed, in a very considerable number of cases, they present the characters of ordinary hæmorrhagic infarcts, but in the traumatic and puerperal cases the infarcts are invariably septic. They may be few in number, only one or two perhaps in the spleen or kidney, or they may be in thousands throughout the various organs of the body. When suppurative, micrococci, in my experience, are always present; but the micrococci may exist in the vessels without inducing this change. In severe forms of the disease, hæmorrhages are very frequent upon the skin, and on the serous and mucous surfaces. The cutaneous ones will be referred to again in connection with the symptomatology. They appear, in many instances, to be due to the effect of the poison, just as in other infectious diseases; in others, they are undoubtedly embolic, and a minute necrotic or suppurative centre can sometimes be seen. In the membranes of the brain, I have twice met with extensive superficial extravasation. Litten (*Charité Annalen*, Band iii, Berlin) has called attention to the frequency of retinal hæmorrhages, particularly in the endocarditis of puerperal sepsis. In some instances, there are innumerable miliary abscesses, more particularly in the heart and kidneys. They are often associated with hæmorrhage, and the smaller ones look like little extravasations, but the presence of micrococci and suppuration can be easily determined in stained sections. The spleen is most often the seat of infarction, and next in order the kidneys. The lungs are usually affected when the endocarditis is on the right side, and there may be suppuration or even extensive gangrene, but even with destructive lesions of the pulmonary valves there may be no suppurative infarcts in the lungs, as in a case of Dr. Church (Pathological Society's *Transactions*, vol. xxvi). Or again, as in a case of Dr. Moxon's (*Ibid.*, vol. xix), there may be with aortic valvulitis suppurative infarcts in the lungs, and simple ones in the other organs. The gastro-intestinal canal may present very remarkable changes, due to the presence of numerous infarctions, from the size of a pin's head to that of a split pea. They are slightly elevated, greyish-yellow in colour, often surrounded by a zone of deep congestion or extravasation, and on section may show a suppurative centre. Micrococci are present, as in other miliary abscesses, and in several instances I was able to find small embolic plugs in the arteries of the submucosa. The abscesses may discharge and leave a small ulcerated surface. In the stomach there may be similar minute infarcts, and occasionally larger ones. Carrington (*Lancet*, 1884, vol. i), has described a remarkable case in which there was a gastric ulcer, apparently due to embolic process, in a case of severe endocarditis; and Magill (BRITISH MEDICAL JOURNAL, 1884, vol. ii), a case in which the stomach was intensely inflamed, the mucous membrane at the greater curvature being black, almost gangrenous. The liver may present minute abscesses, and in a number of cases in which there has been jaundice degeneration of the cells has been observed (Schnitzler, *Wiener Med. Presse*, 1865). The serous surfaces are often inflamed, pleurisy and pericarditis being not uncommon complications. The pericardium is most frequently affected in rheumatic cases, in which endocarditis and pericarditis may occur simultaneously. Pleurisy is met with chiefly in connection with the traumatic and puerperal cases, and also with pneumonia, which, as I shall show, plays an important part in the history of this form of endocarditis. The cerebral lesions are of the substance and of the membranes. Embolic softening, simple or suppurative, is extremely common, and in very many cases head-symptoms supervene, and there is paralysis of one side or the other. There may be a single embolus, producing extensive suppuration or red softening, or there may be multiple infarcts in various regions. The meningeal complication of endocarditis has not received much attention. Considering the frequency with which it has occurred in the Montreal cases, five instances out of twenty-three, I was quite prepared to find such a large

number as twenty-five cases; that is, somewhat over 12 per cent. In the majority of these cases, it occurred in connection with pneumonia. It is almost always cortical, but may extend to the base and involve the nerves, leading in one case, which I saw with Dr. Ross at the Montreal Hospital, to strabismus, and also to ulceration of the cornea from involvement of the fifth nerve. In rare instances the spinal meninges are involved, and the clinical picture may be that of an acute cerebro-spinal meningitis (Hunolle, *Bull. de Soc. d'Anatomie*, 1874; and Heineman, *Med. Record*, New York, 1881, vol. ii). Acute suppurative parotitis was noted in three cases.

LECTURES
ON
THE ANATOMY OF THE INTESTINAL CANAL AND PERITONEUM IN MAN.

Delivered at the Royal College of Surgeons of England.

By FREDERICK TREVES, F.R.C.S.,
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Lecture II.

The Mesentery.—The mesentery, so far as its intestinal attachment is concerned, extends, it is needless to say, from the end of the duodenum to the ileo-cæcal junction. Its upper or right layer is continuous with the under layer of the transverse meso-colon, and with the peritoneum that invests the ascending colon. Its lower or left layer joins with the serous membrane that encloses the descending colon, that forms the sigmoid mesentery, and that descends over the lumbo-sacral eminence into the pelvis. The parietal attachment of the mesentery is liable to considerable variation, and cannot be so readily disposed of. The point at which this attachment commences above is practically constant. It corresponds with the ending of the duodenum, is about on a level with the lower border of the pancreas, and is just to the left of the vertebral bodies. From this point the insertion of the mesentery follows an oblique line that runs downwards and to the right, crossing the great vessels, and then ending in a somewhat uncertain manner in some part of the iliac fossa. The precise manner of its ending will be dealt with subsequently. In an ordinary case, if the mesentery be divided close to the bowel, and all the small intestine be removed, the membrane will appear as a well marked fold, arising by a narrow line from the posterior parietes, and deviating not very considerably from the middle line. It is important to recognise that this attachment does not represent the real root of the mesentery, nor is it any part of the attachment of the median vertical fold of peritoneum, that went to the primary intestinal loop. The real root of the mesentery is in the interval between the transverse colon and the duodenum, where the trunk of the superior mesenteric artery enters.

The lower part of the primary vertical fold is represented by the serous attachments of the descending colon to the parietes. The long line of insertion of the mesentery in the adult is entirely a secondary or acquired attachment. I might recall the fact that there is a time in the history of the development of the intestine when the small intestine, the ascending colon, and the right half of the transverse colon, all form part of a single simple loop, enclosed in a single fold of peritoneum, which is attached to the vertebræ, and has its root in the comparative narrow interval between the transverse colon and the duodenum. At such a time, the parts of the colon named and the small intestine have a mesentery in common. When the rotation of the bowel takes place as already described, when the colon crosses over the duodenum so as to reach the right hypochondriac region, this common mesenteric fold is rotated to the extent of half a circle. Thus it is that what was once the left and under layer of the common mesentery becomes the right and upper layer of the mesentery of the adult, and *vice versâ*.

In time the cæcum descends to reach its final resting-place in the right iliac fossa. As it progresses it outgrows its serous covering, and in time the ascending colon above it acquires a non-peritoneal surface. This part of the large intestine is no longer a part of a free loop, and what is now the permanent mesentery may appear for a while to come off from the parietes, along the inner border of the now attached ascending colon. As a result of further development, the line of

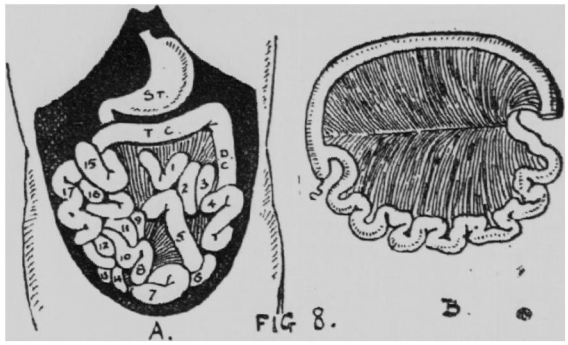

FIG 8.

origin of the mesentery is moved nearer to the middle line, until it comes to occupy the position that is familiar in the adult. As a matter of fact, the isolation of the permanent mesentery appears before the descent of the cæcum, and it may be seen as a separate fold attached to the spine in the fœtus, when the cæcum still occupies the right hypochondrium (Fig. 8 B). While the cæcum is in this position, the line of the attachment of the mesentery, such as it is, appears to be almost transverse, and it may not attain its permanent oblique direction until the cæcum has reached its goal in the iliac fossa.

In a large number of the mammalia, the ascending colon never loses any part of its original complete serous investment. It never, therefore, becomes attached to the parietes, but remains as a part of the great loop of intestine, and still invested in a simple mesentery that is common to it and the whole of the jejuno-ileum. In such animals the right limb of the large bowel remains singularly free; the mesentery of the small intestine retains its primitive relations; it acquires no secondary attachment to the parietes, and its sole root and attachment is in the narrow gap between the transverse colon and the duodenum. This condition is occasionally met with in the human subject. The ascending colon is entirely free up to the hepatic flexure, and is invested by a mesentery, common to it and the small intestine. I have met with two examples of this in one hundred specimens. The condition is of interest pathologically, as favouring the development of a certain form of volvulus of the cæcum and small intestine.

Putting aside this condition, it may be said that the parietal attachment of the mesentery measures, as a rule, about 6 inches; its mode of ending at its inferior extremity is as follows. When an ascending meso-colon exists, the mesentery ends by joining it. The two membranes meet at an angle, often at a right angle, and then the right layer of the mesentery becomes continuous with the left layer of the ascending meso-colon, and the left layer of the mesentery with the right one of the colic fold. When no meso-colon exists, the peritoneum that covers the cæcum is reflected from the hinder surface of that part of the bowel on to the posterior parietes; at this reflection the mesentery ends. Its left layer is continuous, and often in a line, with this reflected membrane, and then passes on into the pelvis, while its right layer is continued on to the ascending colon. As the position of this reflection varies considerably, so the length of the parietal attachment of the mesentery must be varied in proportion, and the same applies to cases where an ascending meso-colon exists.

The length of the mesentery from the spine to the intestine varies in different parts of the canal; its average length may be taken as between 8 and 9 inches. It soon attains its full length, and within one foot of the end of the duodenum is already 6 inches in length.

The longest part of the mesentery is that which goes to the coils of intestine that lie between a point 6 feet from the duodenum, and a point 11 feet from the same part of the gut. Such coils will, therefore, include 5 feet of the intestine, and the mesentery here not infrequently reaches the length of 10 inches. This point is of interest in connection with the position of certain coils of intestine, and to the subject allusion will again be made.

The important part that the mesentery must play in connection with the commoner forms of hernia has, it would appear, been somewhat overlooked. If the fresh body of an adult be opened, and the condition of the viscera and peritoneum be normal, it will be found that it is impossible to drag a loop of small intestine through the femoral canal (artificially enlarged) on to the thigh, or down the in-