CLINICAL LECTURE ON A CASE OF ULCERATIVE ENDOCARDITIS.

Delivered at The General Hospital, Birmingham.

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[REPORTED BY MR. J. HAZELWOOD CLAYTON.]

GENTLEMEN,—At our last meeting I showed you in the wards a youth suffering from malignant endocarditis of the right side of the heart, with a communication between the pulmonary artery and the aorta. He lay in a typhoid condition, having embolism of the lungs and kidney, and also an enlarged spleen. The patient, J. V., was 17 years of age; he had never been a robust boy, having always been thin and pale; but, with the exception of the ordinary diseases of infancy and childhood, he had never had any serious illness until the present one. He had never had rheumatism, nor did he inherit any tendency to it. He joined in sports, as other boys, and worked without interruption. Though somewhat delicate, he was considered to be free from disease, and he never showed any signs of dyspnoea or lividity on exertion.

He was admitted on September 8th last. It was during the first fortnight of July last that it was noticed that his health seemed failing; he looked pale and exhausted when he returned from work. He was an engineer's draughtsman, and was very ambitious of getting favourably marked the designs he was from time to time submitting for examination. His condition was thought to be the result of overwork and overanxiety. In the middle of July he was seized with pain in the chest and shortness of breath, and was confined to his bed; the surgeon called in pronounced his affection "heart disease."

On admission to the hospital he was very pale and extremely emaciated. He said that for seven weeks he had had pain in the cardiac region, considerable dyspnoea, dry cough, and profuse night sweats, and had been wasting away. On examining the heart, its area of dulness was somewhat increased, the apex beat was in the sixth interspace. At the apex both sounds were loud and distinct, accompanying the first sound was a murmur increasing in intensity towards the base. At the base various murmurs were heard, all having their maximum intensity on the left of the sternum over the pulmonary area. A loud double murmur distinguished itself from the others by its loudness and vibrating character. The murmurs were scarcely audible over the vessels of the neck, nor were they conducted down the sternum. Over the second left interspace, an inch and a half from the edge of the sternum, an intense thrill was felt, and here the double murmur had its maximum intensity; there was no pulsation of the jugulars, nor sign of lung disease. His appetite was good, his tongue was red and raw, the spleen was felt to be enlarged; the urine normal.

September 17th. There was a great increase of dyspnoea; a patch of consolidation was found at the base of the left lung.

October 3rd. The consolidation at the left base persisted, but was more limited; another patch was found at the right base.

October 15th. The dyspnoea, cough, and general condition were worse. He complained of much pain in the right chest at the back, and there was an increase of consolidation there. He expectorated blood-streaked sputa. The cardiac murmurs were louder.

October 27th. He had severe pain in the left lumbar region.

October 28th. The urine was albuminous, and lumbar pain severe.

November 2nd. The urine was highly albuminous and contained casts; the feet were oedematous.

November 19th. His general condition had gradually got worse, the dyspnoea had increased, and he died of exhaustion.

While in the hospital his temperature was for the most part of the time 102° to 104° at night and normal in the morning. Respirations varied from 30 to 40 per minute. Pulse 110 to 140. The urine was free from albumen until October 28th. The oedema of the feet, which appeared on November 2nd, had gradually disappeared.

NECROPSY.—*Heart*, 15 ounces in weight. On opening the right

ventricle and pulmonary artery, soft polypoid vegetations were found attached to the cusps of the pulmonary valve, and above growing from the intima of the anterior wall of the vessel a large mass of them was found partially occluding the lumen. The area covered by these measured roughly 1½ inch longitudinally and 1¼ inch in breadth, reaching above the ductus arteriosus, around which they were aggregated. The duct was not closed, its patency being congenital. Small portions of vegetations appear to have been detached and carried by the force of the circulation through the duct into the aorta, impinging upon the posterior and left extremity of the transverse part of the arch, where the inner lining was the seat of acute endocarditis, and of vegetations similar to those found in the pulmonary artery. At this point there was a distinct bulging of the wall, which was firmly attached externally to the lower part of the trachea by fibrous adhesions. Tricuspid, mitral, and aortic valves normal. Left ventricular wall a little thickened.

Right ventricle and pulmonary artery cut open from the front; A, aortic aneurysm; B, vegetations around orifice of ductus arteriosus; C, vegetations on wall of pulmonary artery; D, vegetations upon pulmonary valves; E, orifice of right branch of pulmonary artery.

Lungs.—Right lung, 20 ounces. Upper lobe, about the middle there was a blackish infarct projecting on the surface, and, on section, plugging of the arterial branch leading to it with an embolus was seen. Middle lobe emphysematous. Lower lobe collapsed and condensed; an aneurysmal bulging of a branch of the pulmonary artery supplying this lobe was seen, completely filled and occluded by pale laminated clots about the size of a walnut. Towards the free border of the base two or three cuneiform infarctions were cut across, well-defined by a raised yellowish-white border; their central zones were of a dirty reddish-brown colour, necrotic, and softened; small branches of the artery leading to these were plugged with emboli. Left lung, 18 ounces. Upper lobe, apex, and margin emphysematous; the lower corner affected with lobular pneumonia. Lower lobe, collapsed, more or less carneified, tough, and leathery in consistence; the base adherent to the diaphragm.

Liver—44 ounces, slightly "nutmegged" on section, soft and fatty.

Spleen—15 ounces, swollen, rather flaccid; no infarcts presented on the surface, but, on section, there were one or two yellowish points resembling small infarctions in its substance.

Kidneys.—Left, 4½ oz., indurated, density increased, almost infarcted throughout, and atrophic. Only small breadths of the red colour of the cortex seen here and there, the rest of the kidney being of a uniform dead yellowish-white colour; and, though the form was preserved, all structural details were lost to the naked eye. Right, 6½ oz. No infarcts; the capsule stripped well; the surface finely

mottled all over with ecchymosed spots. Section showed a red mottled kidney (acute nephritis). It was pretty firm in consistence, and cut with some resistance. Cortex apparently not increased, greyish red, mottled and streaked with hyperæmia, the medullary cones being hyperæmic.

Endocarditis as a primary affection very rarely occurs. It is found occasionally as a secondary affection in fevers, pneumonia, septicæmia, diphtheria, Bright's disease, gout, syphilis, and it is a frequent complication of acute rheumatism and chorea. My clinical clerk, Mr. Hazelwood Clayton, finds that, of forty-three cases of acute rheumatism admitted into hospital under my care during the last three years, seventeen had valves damaged by previous endocarditis, and two developed endocarditis while in the hospital. The onset of rheumatic endocarditis is rarely marked by any great constitutional disturbances, and it is impossible to say how much of the constitutional disturbance which does exist is due to rheumatism, and how much to endocarditis. The onset may be marked by a slight temporary rise of temperature, or by cough, which may be accompanied by the expectoration of streaks of blood; or the patient may complain of a little pain in the chest and shortness of breath. Often the affection is first suspected by auscultation, an endocardial murmur, the only reliable sign of the disease, being heard. The constitutional disturbance soon passes by, and the patients leave the hospital with more or less damaged valves, the left side of the heart being the one almost exclusively affected. The endocarditis of chorea, when apparently of non-rheumatic origin, is similar in kind, less severe, and not so serious in its after-effects upon the heart. During the last twenty-five years it has been recognised that there are certain cases of endocarditis which separate themselves off from the cases of which I have been speaking by clinical and pathological features into a distinct class. Cases of this class present some differences, but there is so much in common to them all as to justify their being arranged together in one group under one appellation. Of the terms which have been applied I would mention malignant, ulcerative, septic, diphtheritic, and infective; but the term malignant commends itself by aptly including all the cases. Many have been the workers in this field of research—Stenhouse-Kirkes, Bouillaud, Virchow, Bamberger, Charcot, Vulpian, Lancereaux, Trousseau, Leudet, Ogle, Wilks, Moxon, Bristowe, Sidney Coupland, Bramwell, and Prudden. In 1885 Dr. Osler, of Philadelphia, in the Goulstonian Lectures for that year, summarised our knowledge of the subject, having collected and analysed 209 cases. Malignant endocarditis seems to attack most frequently persons in the middle period of life, males more than females, the constitutionally weak more than the robust. Sclerosed valves constitute a tendency to it, as do, I believe, malformations of the heart. The endocarditis of septicæmia (puerperal or traumatic), of acute osteitis, and of pneumonia is especially likely to be of this form. It may be engrafted on acute rheumatic or choreic endocarditis. I believe that it affects the right side of the heart proportionately more frequently than does rheumatic endocarditis, and occurs more frequently unconnected with other diseases than does non-malignant endocarditis. Of Dr. Osler's 209 cases, in 45 there was no history of any other disease with which it could be connected.

It produces a typhoid condition, multiple embolism, enlarged spleen, nephritis, ecchymoses, and acute aneurysms. The endocardial affection may be suppurative, ulcerative, or vegetative, or a combination of these processes. It is a very fatal disease; some cases recover, as we occasionally in the *post-mortem* room find the effects of ulceration, in the form of perforations of the septum and valves, in hearts of persons who have died of other disease. The duration of some cases, especially those of a pyæmic type, is sometimes a matter of days, and often only a week or two; while those of a typhoid type last commonly for many months.

Typhoid Condition.—This condition is characterised by weakness, emaciation, night sweats, quick pulse, and elevations of temperature, the temperature being subject to considerable diurnal variations, being several degrees higher at night than in the morning, the morning temperature being often normal. There may be vomiting, delirium, and jaundice, in some cases rigors, which may be repeated at regular or irregular intervals. The typhoid condition declares itself at the commencement of the disease, preceding embolism and other complications, and lasts usually to the termination of the disease.

Embolic Thrombosis.—The organs most liable to be thus affected are the lungs, spleen, and kidneys, when the right side of the heart is affected; the brain, kidneys, and spleen when the endo-

carditis is on the left side of the heart. The diagnosis of malignant endocarditis has to be founded on the presence of endocarditis and a typhoid condition, without aught else to account for this latter condition. The supervention of multiple embolism confirms the diagnosis, and the picture is completed by enlargement of the spleen, nephritis, capillary ecchymoses, and acute aneurysms. Sometimes the diagnosis has to be made solely on the typhoid condition.

A middle-aged man called on me; he had been wasting and sweating at night for about three months. On the supposition that he was suffering from subacute rheumatism, under the advice of physicians, he visited appropriate health-resorts. Three weeks before I saw him it was discovered that his temperature was 102° F. in the evening and normal in the morning, and this had been constantly the case up to the time he consulted me. He had a mitral systolic murmur, which he said had existed for ten years, and was first noticed when he had rheumatic fever; his heart had caused him no trouble; there was no physical sign of recent endocarditis to be detected, no evidence of tubercle, none of the abdominal symptoms of enteric fever, no rheumatic joint pains, no wound to cause septicæmia. The surgeon with whom I saw the case subsequently, under whose care he placed himself, suggested that the sclerosed mitral valve might be the seat of some fresh mischief. Week by week the typhoid condition deepened; he had cerebral and renal embolism and nephritis, and he died of exhaustion six months after I saw him. Here was a case in which the typhoid condition was the only guide to the diagnosis until embolism supervened. Many cases of malignant endocarditis have been diagnosed, as enteric fever from the typhoid condition, as pyæmia and intermittent fever from repeated rigors.

Pathology.—Bacteriologists have found micro-organisms in malignant endocarditis, to which some say the disease is due. Dr. Coupland has figured them in the *JOURNAL* of 1885, p. 944. Dr. Crooke found micrococci in the vegetations in J. V.'s case, though means were not at our disposal for cultivating them. We must, however, wait for further evidence before we assume that mycosis endocardii is the cause of malignant endocarditis. It may be that the endocarditis is excited by an impoverished state of the blood, or by some chemical alteration in it, or by the presence in it of some animal alkaloid, and that the malignant character is determined by a lowered vitality of the tissues, the microbes, when they exist, being altogether secondary. Bacteriologists, in assigning a germ-origin to the disease, are supported by the fact that malignant endocarditis is clearly in a sense an infectious disease. Particles separating from their original site in the heart not only contaminate the blood, but carry with them the materies morbi, and set up the same pathological condition in distant parts, and give rise to suppurating infarcts and acute aneurysms. Dr. Dreschfeld¹, in a case of non-ulcerative malignant endocarditis, found streptococci in the scrapings from vegetations. He injected a cultivation of these streptococci into the jugular vein of a rabbit, and found well-marked vegetations on the mitral and aortic valves, in which were found the same streptococci and infarcts in the spleen and kidneys. Dr. Prudden² states that *staphylococcus pyogenes aureus*, the only identified bacteria of ulcerative endocarditis, injected into the blood of a rabbit will not produce ulcerative endocarditis unless the endocardium be previously injured; and he suggests as an explanation of the frequent association of ulcerative endocarditis with sclerosed valves, the increased vulnerability to bacteria of endocardium devoid of epithelium.

The vegetations on the aorta in J. V.'s case, being exactly opposite the opening of the ductus arteriosus, were clearly due to infective particles being carried in the blood from the pulmonary artery by the ductus arteriosus, and impinging on the intima of the aorta. It is fully recognised that sclerosed valves create a tendency to endocardial vegetations. I have so often seen vegetations present in malformed hearts, that I am inclined to think that malformations create this same tendency as sclerosed valves, and probably in the same way, by retarding the circulation through the heart and producing eddies. There is a doubt in my mind whether, if J. V. had not had a patent ductus arteriosus, he would have had endocarditis; whether the malformation was not the predisposing cause, and the impoverished state of his blood the exciting cause, of the endocarditis and its malignancy. A similar case is recorded in the *Pathological Transactions* for 1888 by Dr. Murray. To me it seems probable that the typhoid condition is due to

¹ *JOURNAL*, 1887, vol. ii, p. 887.

² *American Medical Journal*, 1887.

violation of the blood by the inflammatory products of this form of endocarditis, that the spleen is enlarged from persistent pyrexia. The emboli are doubtless crumbs of vegetations or necrosed particles of endocardium liberated by ulceration, and it is reasonable to suppose that morbid dust, as it were, even if not mycotic, getting into the blood from this same source, may be the cause of ecchymoses and nephritis.

Treatment.—When malignant endocarditis occurs as a secondary affection, the treatment resolves itself chiefly into the treatment of the primary disease. There is no established specific treatment of primary idiopathic malignant endocarditis; the indication seems to me to be to regard it as a disease of debility to be combated with rest and restoratives. In the cases under my care antipyretics have failed to improve the general condition and to control the evening rise of temperature. Dr. Sansom³ records a case of recovery under the use of sulpho-carbolate of soda.

ABSTRACTS OF THE MILROY LECTURES ON OCCUPATIONS AND TRADES IN RELATION TO PUBLIC HEALTH.

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LECTURE II.

DUST DISEASES IN GENERAL.

Pathology.—The lecturer commenced by referring to the coloration of the lungs produced by inhaled dust; this was usually black, varying in tint according to the character of the dust, but a case was quoted in which the colour was red, owing to the patient having worked with oxide of iron. The black staining was most marked in coal-miners and charcoal-grinders. Every dirty occupation led sooner or later to interstitial mischief. The earthy and metallic dusts were more provocative of lung-disablement than organic dusts, with the exception of charcoal. Millers and starch-workers suffered from clogging of the air-passages rather than parenchymatous changes; workers in horn escaped, probably because it was an organic material.¹ Woods acted as irritants in direct proportion to their density. Ebony and rosewood had a bad name for provoking bronchial troubles, and the latter is believed to produce eczema. Dust from mother-of-pearl partook of the worst features of mineral dust, producing the same disorganisation and black discoloration as was observed in stone-workers and potters. The dust from cotton was less injurious than that from flax, which also provoked dyspnoea more severe than that caused by any other kind of dust not actually poisonous.

The consolidation of lung produced by dust varied in extent; in some cases scattered nodules, in others larger but still small masses, in others a large portion of a lobe. The indurated portions were mostly very dense, cutting like india-rubber or cartilage, with a slight sensation of grittiness. Nevertheless, at times, scraping and washing the cut surface would dislodge the black matter, and bring into view cut sections of bronchi, and expose a rough spotted surface, resembling that of a nutmeg.² Associated with these serious lesions were enlargement and thickening of the bronchial tubes, and enlargement and pigmentation of the bronchial glands, their true gland-structure being transformed into a fibrous tissue of deep black colour. The indurated masses in the lungs showed a preference for the apices, though found in the posterior part of the upper lobe, and even in the lower lobe; around them the lung was puckered, with bands of fibrous tissue, loose areolar tissue, and areas of emphysema.

Emphysema was also common in the anterior edges of the lung, and was probably in part vicarious, and in part due to the catarrh

consequent on the dust. The larger patches of consolidation were liable to undergo disintegration, commencing in the centre, which, it was believed, might go on to complete breaking down, leaving a ragged cavity. This disintegration was attributed to cutting off of the blood-supply, and such a mass of tissue would react on the surrounding parenchyma as a foreign substance. Dr. Arlidge, however, thought that such cavities should arouse the suspicion of tubercular complication, particularly as the sufferers were exposed to causes of malnutrition, and had often inherited a predisposition, which would render the absence of tuberculosis with such severe pulmonary lesions a marvel. In all cases of dust-produced disease of the lungs there was thickening and adhesion of the pleura, and often some effusion. These pleuritic changes, together with the interstitial fibrous bands in the lungs, produced the local or general shrinking of the chest-cavity so commonly observed in these cases. Greenhow, Kussmaul, Tardieu, and others had shown that the hard black matter contained much silica, and an analysis of the masses from a potter's lung made for Dr. Arlidge by Professor Church, 1875, gave the composition of the ash as silica 47.78, alumina 18.63, and peroxide of iron 5.5. These results have since been confirmed by other observers.

Symptoms.—The most prominent symptom was dyspnoea, to which the term asthma was popularly applied. The early symptoms—tracheal irritation, constriction about the sternum, and shortness of breath—might continue slowly to increase for years before cough and expectoration became troublesome. The dyspnoea was to be attributed partly to spasm of the bronchial muscles produced reflexly by the irritation, partly to the turgescence of the bronchial mucous membrane produced by congestion. The expectoration was at first mucous, then mucopurulent; it was tinged more or less deeply by the dust, and in colliers and charcoal-grinders the sputum was of inky blackness, popularly called "black spit." Colliers at times expectorated solid friable pellets of coal dust mixed with mucus. The late appearance of expectoration, especially of purulent expectoration, was of assistance in the differential diagnosis, as was also the general absence of hæmoptysis; when hæmoptysis did occur, the quantity of blood lost was small; but occasionally, chiefly in metal workers, considerable hæmoptysis occurred in the early stage. In the later stage, when necrobiosis and softening supervened, there might be abundant hæmorrhage and expectoration, but such occurrences were rare. The lung disease produced by dust was not accompanied by fever; the pulse was not accelerated, and, until the stage of free purulent expectoration, there was no hectic. Diarrhoea was not a common complication, nor aphonia; appetite and digestion remained good until the last stage. Such were the symptoms in uncomplicated cases; but, as a rule, they were modified by intercurrent disease, especially bronchitis or pneumonia, which were common causes of death.

Varieties of Organic Dust.—Organic dust was of either vegetable or animal origin; the sources of animal dust were silk, wool, hair, feathers, horns, hoofs, hides, or skins. Silk and wool constituted the leading staples of the textile industry of this country, and the lecturer proceeded to deal with these two industries in the following terms:

SILK MANUFACTURES.

The Better Varieties of the Raw Material.—Silk as received from China, India, Italy, and elsewhere, though in a raw state as wound from the cocoon, requires only a simple boiling in an alkaline soapy liquid for the purpose of removing the gummy substance attached to it to fit it for the future operations of twisting, winding, reeling, and spinning, and finally weaving; and as all these processes are accompanied by little or no dust, where the best silk is in use, they call for no remarks as producers of disease.

Silk Waste.—It is otherwise where silk waste is manufactured. This material is derived from mills where the best silk is employed, of which it represents, in a certain sense, the refuse; also from the cocoons received from foreign lands which from defects have been unfit for the primary simple process of winding off the silk. Moreover, the inferior silks from abroad and the peculiar silk of India, which contain a good deal of foreign matter, rank also in the category of silk waste, and have to be submitted to the same processes to prepare them for trade purposes. The preliminary process is that of boiling these materials in a mixture of soap and potash to remove the "gum," which is an integral element of natural silk. After this the cocoons and the entangled impure silk are placed in machines, consisting of large cylinders

³ *Lancet*, 1886, vol. i, p. 400.

¹ Dr. Greenhow, however, states that the workers suffered from oppression of breathing.

² On Lung-disease from Inhalation of Dust, by J. T. Arlidge, M.D. *British and Foreign Medico-Chirurgical Review*, 1875.