AN ADDRESS ON CASES OF VALVULAR HEART-DISEASE LONG FREE FROM SERIOUS SYMPTOMS.

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THE discussion which I am asked to open here to-day, in the name of the Collective Investigation Committee of the Association, is one which has bearings upon the whole vast subject of clinical medicine. The subject of it relates to one of the most difficult problems that come, but too often, before practitioners. Large, however, as is the whole subject of prognosis in cardiac valvular disease, limits are set to our discussion by the specific terms of the inquiry we are set to make, and which confine our attention to cases of heart-valve disease which have been for upwards of five years free from serious symptoms.

The fact that such an inquiry as we now seek to make is thought feasible is, in itself, suggestive that the prognosis of cardiac valvular disease has brightened with the general advance of medical science, and such we know to be the case. When, in 1862, Dr. Walshe issued the third edition of his book, on *Diseases of the Heart*, he remarked that the hopeless motto of Baron Corvisart's work on cardiac diseases (*Essai sur les Maladies et les Lésions Organiques du Cœur*; Paris, 1811), published half a century previously,—“*Hæret lateri letalis arundo*,” (Virgil, *Æneid*, Lib. iv, line 73)—“infinitely well-chosen in his time, grows yearly less and less appropriate.”

The advances of the last quarter of a century, in respect of cardiac diseases, as a whole, may fairly be said to surpass those of the previous fifty years. We are not, however, met to discuss this large question. It were easier to do this, perhaps, than to confine ourselves, as we must, to the particular inquiry before us.

In considering cases which properly fall into the category prescribed, we shall find that some of them have been practically symptomless up to the time of recognition.

At the outset, it may be noted that valvular disease of the heart is not always detectable by skilled auscultators; that when it exists, the physical signs of it may be inconstant; and that when murmurs are present, they do not always indicate organic disease. It is certain, for example, that much difficulty sometimes attends the determination of organic as against hæmic or so-called functional murmurs. The inconstancy to which I have alluded is probably associated as much with disease of the mitral, as with that of the aortic valves. In any given instance, the prognosis depends upon an exact diagnosis of the lesion and of the conditions that have induced it.

I think it may fairly be affirmed that we can now better prognosticate the future in any case than was formerly possible, because more attention is paid to the life-history and the diathetic state of the individual, though much still remains to be done in these directions. The mere fact of a murmur, as we all now know, tells little or nothing as to the antecedents or the future of the subject of it. It is literally a “*vox et præterea nihil*.” To verify this much is in the power of any good third-year student. To find its exact significance is a tax on the acumen of the well-trained and observant clinical physician.

I apprehend that the scope of our present inquiry is to discover the conditions under which heart-valve diseases exist, without making their presence known to the subjects of them. What are the conservative conditions which prevent the betrayal of the ordinary symptoms in these cases? Are they constitutionally inherent in the individual, or are they due to concomitant, but inhibitory, states, themselves morbid, or, at any rate, not physiological? We ought, perhaps, to look outside the area of cardiac pathology to find some analogous problem whose elucidation might, perchance, be helpful, and I think we may find one in the morbid condition we recognise in albuminuria. This perverted state of renal function we know to exist, in many instances, without plain indications of its presence till it is suddenly detected in the routine examinations of the ward or consulting-room. How long it may have persisted, we often cannot surmise. Some stray symptom may have led to a suspicion of it, but it may often be

unsuspected. Again, do we not occasionally meet with patients who present the marked hepatic perversion we recognise in the form of glycosuria, who yet present no diabetic symptoms, and enjoy, seemingly, very fair health? It will be conceded that, to determine the precise significance of each of these states, and to found a prognosis concerning them, demands much discrimination and large experience founded on other like cases. Whence comes the constitutional resistance, in such instances, whereby no ravages are wrought, and the patient is saved the ordinary, but grievous, troubles respectively of Bright's disease and of diabetes mellitus? Here lie some problems for future observers. That cases such as I have described exist, have been doomed to a dark future, and have lived on to the surprise of their advisers, is within the knowledge of some, if not of all, of us. It is also certain that many subjects of cardiac valvular disease have been despaired of on the ground of physical signs alone, and have lived a fair life-time in spite of ugly prognosis, and, I may add, too, of ugly treatment.

In supplying facts to determine the question before us, we must refer to our case-books, and extract such examples as have come before us in which unequivocal signs of valvular heart-disease have been made out, and in which the patient has remained under observation for at least five years, and has presented no serious symptoms. My belief is that those in large practice will find considerable numbers fulfilling these conditions. In most cases, however, the subject of the disease will have come first under notice for some trouble distinctly referable to the heart or the circulatory system. We must then try to find the probable starting-point of the disease. In doing this, we should probably all think foremost of some antecedent attack of rheumatic fever. I shall presently go more into details respecting the points which must be weighed here. Next, I think we should seek evidence of possible congenital malformations, especially amongst younger patients. As guides in this ætiological search, we must always have regard to the age, previous illnesses, occupation, and family history of the patient. In more advanced life, we are to bear in mind the possible existence of valvulitis as part of the cardio-vascular disturbance of chronic renal disease, and as part of more general senile and sclerosing degenerations. In adult life, we must not forget the influences of strain, alcohol, and syphilis. To these, and to some others, I propose now to address myself more in detail; and, for better method, I will take these in the following order: (1) Congenital malformations and endocarditis in early and in adult life; (2) Rheumatic valvulitis in its widest aspect; (3) Valvulitis in the adult as the result of strain, or of strain together with alcoholic intemperance and hard-living in general, including arteritis and valvulitis traumatica; (4) Valvulitis in connection with renal disease, and as associated with general arterio-capillary fibrosis; (5) Valvulitis in connection with anæmia; (6) Syphilis in relation to valvulitis.

When all these shall have been considered, we shall hardly have exhausted the subject before us. There remain other causes capable of provoking cardiac murmurs which, however, are hardly likely to furnish many examples fulfilling the conditions imposed in this inquiry.¹

1. I consider first the cases of valvular disease due to congenital malformation. The large majority of these are inconsistent with viability, or with continuance of life beyond a few months or years. Cases of morbus cæruleus may reach adult life, but they are naturally attended with many symptoms and risks. None of these claim our attention. In some cases, however, we meet with evidences of congenital cardiac malformation, unattended by cyanosis, or by very little. Sometimes the general bodily development is satisfactory, though, most commonly, such patients are stunted in growth, and slim. In the less marked cases, the first-noticed signs of imperfect health may be the very slow general growth and development. The child is backward, and so much so that the rachitic state may be suspected; but, on careful examination, no signs of rickets are to be found. A little dusking of the fingers and toes, and a slight clubbing, may be appreciable, both here and at the end of the nose, but not always. In any such case, it is proper to examine the heart, and a murmur will not uncommonly be found; albeit, no symptoms of cardiac disturbance have obtruded themselves. With care, such children may grow up, and if their constitution be good, they seem to do fairly well in life. They may pass through the exigencies of school-life, and may even take part in athletic exercise. The prognosis is naturally better for girls than for boys. The period of puberty is trying for

¹ Of scarlet fever as a direct factor in the production of endocarditis, I have no experience. When such occurs, there are always associated rheumatismal symptoms, of which the nature is hardly, as yet, determined. Measles is alleged to cause endocarditis, and to lay the foundation of valvular disease. Having never met with an instance, I have grave doubts as to this.

the former, but may be surpassed; some dyspnoea on exertion, or inability for prolonged exercise, may be all that is complained of, but not even this in some cases. Acute illnesses may be tided over, of which pertussis, measles, and scarlet fever, are the most trying; and, in one of these, it may be, or on some other account, if the heart be examined, a murmur is detected. It may be very loud, and cause alarm or anxiety to the observer. In some cases, the subjects of malformation may enter upon the active duties of life with their special trouble undetected. I have met with several of such instances. The following, I believe, to be an example. J. A., aged 41, a well-grown man, came under my care lately in St. Bartholomew's Hospital. When he was 18 years old, he was enlisted in the Royal Marines, and served for four years; he was then discharged from the service on account of palpitation. He had scarlet fever at 9 years of age. When he was 29, he was admitted to St. Bartholomew's for dyspnoea and palpitation. At the age of 33, he had hæmoptysis for the first and last time. He had never had rheumatism or syphilis, so far as he knew. For twenty years past, he had been subject to dyspnoea. Examination of his heart revealed no obvious enlargement, the apex-beat in natural situation, and a loud, rough, systolic murmur heard all over the chest and both sides of the back. The murmur was loudest in the area of the conus arteriosus and pulmonary artery, the second sound being but faintly audible over the right ventricle. The pulse was locomotor, but not otherwise of aortic type. There was clubbing of fingers and toes, no dropsy, and but the slightest appearance of cyanosis. The kidneys were healthy. The diagnosis here was congenital stenosis of the pulmonary artery. I could not satisfy myself as to an imperfect ventricular septum, albeit it might have been expected in such association, and because of the widely diffused convection of the murmur, especially posteriorly. Had this existed, I should have expected more marked cyanosis, and generally graver symptoms. Now, this man had been accepted for the hard service of the Marines, and had gone through the most trying part of his military training—the first two years, and only failed after four years' duty. It is to be supposed, therefore, that his cardiac murmur had been undetected when he passed as a recruit, and he probably had it all his life. The worst feature of his case, on his last admission, was the presence of pulmonary consolidation and softening at both apices, also a laryngeal ulcer; and though bacilli were not found after several examinations, I have no doubt, from his symptoms and temperature-chart, that he is the subject of tuberculosis, which we know to supervene commonly in association with congenital cardiac disease when this happens to permit viability, and consists mainly of pulmonary arterial stenosis. The prognosis, in such a case, has reference, henceforth, to the condition of the lungs rather than of the heart. Tuberculosis is apt to proceed, but there exists evidence to show that its progress is somewhat inhibited by the presence of the cardiac disease.

Another example is the following. In 1883, I was consulted about a gentleman, aged 27, one of a family of five. He was known to have had heart-complaint from the age of 10 years. He was tall and spare; his general health was fair; he was never robust, and was not long able to remain at a public school. His chief complaints were of occasional languor and attacks of faintness, the latter commonly associated with some dyspepsia, and called by him "bilious." He was able to shoot, dance, and play tennis. As a child, he was frail. There was no cyanosis, and no clubbing of the digital extremities. The cardiac dulness was two inches transversely; the apex struck in the left sixth space just inside the nipple. A loud systolic murmur was heard all over the præcordia, loudest at the lower sternal region, and upwards to the right in the aortic area; it was not heard behind. The impulse was forcible; there was no thrill. The second sound nowhere accentuated. The pulse was small and regular.

These physical signs seemed significant, mainly, of aortic stenosis, the forcible impulse being, however, somewhat contradictory of this, according to Traube's teaching. There was clearly hypertrophy of the left ventricle; the urine was healthy; there was no rheumatic history of any kind.

I believed this to be a case of congenital defect. Since I saw this patient, he has led an active life, travelled much, has married, and become a parent. The slight syncopal attacks occur at intervals, especially after long exertion, and any gastric derangement seems also to determine them. In this case, the cardiac disorder has been known of for twenty years, the patient being now thirty years of age.

I should like to refer here, parenthetically, to a point respecting the prognosis in cases of congenital cardiac disease, the subject of the exanthemata, or of continued fever. It is a matter of interest, and but little is known about it. Are these patients ill-fitted to bear acute disease of this kind? I have known them, even when very cyanotic,

to pass through measles, enteric fever, and pertussis. Scarlet fever seems to be particularly dangerous, even in a mild form.

Pertussis and measles are naturally dangerous, owing to pulmonary complications. At the London Fever Hospital, Dr. Cayley informs me that they do not appear to have had any patients with cyanosis suffering from scarlet fever or enteric fever in a severe form, though the results have not been favourable. In one case, the attack of scarlet fever was very mild, but the patient succumbed from the state of the heart and lungs. One case of enteric fever, the subject of cardiac malformation without cyanosis, died after a long, but not very severe, attack. There was found, after death, an opening from the aorta into the pulmonary artery. All the valves were healthy and normal.

Sir William Jenner has suggested that the peculiar condition of blood in these cases may possibly present to the respective fever poisons, or to some of them, a too favourable pabulum, whereby overpowering multiplication of them occurs.

In my experience, it is quite the exception for cyanotic cases to be free from symptoms, oftentimes serious, referable to the heart and pulmonary circulation in the course of their short lives. I believe that cases of simple aortic stenosis may perhaps be most favourably regarded in this category. These may be for many years symptomless. Dr. Peacock recorded several examples illustrating this.²

In one, the patient, an iron-founder, lived to the age of 40 years, and had cardiac symptoms for only twelve weeks before death. In another, a woman, enjoyed good health, had no dyspnoea on exertion, and lived to the age of 76 years, dying incidentally after an operation for strangulated hernia. There was no rheumatic history. The aortic aperture was reduced to a mere slit, but the heart only weighed ten ounces, indicating no great hypertrophy under the circumstances. Corvisart, Stokes, and Graves have all recorded very similar examples, living respectively to seventy-seven, middle age, and fifty-four years. The mitral valves partake of congenital defects, together with the aortic segments, in some cases. Dr. Peacock believed that complete fusion of two aortic valves indicated congenital origin in any given case. (Mr. Barling showed an example of this defect at the meeting. There had been evidence of valvular disease for ten years. I believe the two fused valves were here practically competent; death was, however, very sudden.) Dr. Peacock also thought that cases in which all the sigmoid valves were fused in either the aorta or pulmonary artery were commoner than those in which only two of them were affected; and he found the malformation more common in the aorta when the heart was otherwise natural, only having notes of eight cases in which uncomplicated disease of the pulmonary valves existed.

In many of these cases, the physical signs indicated both obstruction and reflux, the latter probably supervening in course of time from sequential changes in the aperture, as is now well recognised. Congenital tricuspid and mitral stenosis commonly co-exist. Dr. Pearson Irvine (Pathological Society's *Transactions*, vol. xxxi, p. 79, 1880) recorded a case in a woman, aged 37, who had worked as a housemaid, but always had suffered from dyspnoea. There was also aortic stenosis. In many of these cases in adults, there is rheumatic history—50 per cent., according to Dr. Bedford Fenwick's statistics (Pathological Society's *Transactions*, vol. xxxii, p. 42, 1882)—and in 25 per cent. of all cases there was stenosis of the aortic orifice as well.

Professor Gairdner's case of tricuspid obstruction (*Clinical Medicine*, p. 602, 1862), due to a "ball-valve" partial closure of the orifice by a tumour in the right auricle, may be referred to here. The man lived for over ten years after the discovery of the murmur, and died, aged 30 years, not of any cardiac affection, but of pneumonia. The heart was nearly normal in size and relations. I saw it in the museum of the British Medical Association at the meeting in London in 1873.

Consideration of cases such as these seems to teach plainly that, in cases of congenital malformation permitting viability, without cyanosis, we may find life to be maintained over longer periods than we might naturally expect. I cannot suppose that any law prevails here to prevent the access of grave cardiac symptoms, other than may be found to explain similar conditions met with in cases not of congenital origin. The tendency to tuberculosis associated with pulmonary arterial stenosis pure and simple, the lungs being anæmic, or with imperfect ventricular septum, remains to be duly taken note of in any given example. One lesson coming out of a study of these cases is, that we must not believe all cardiac valvular defects in early life, as is perhaps still too commonly conceived, to be of rheumatic

² On Some of the Causes and Effects of Valvular Disease of the Heart. Croonian Lecture, 1865.

origin. I believe, however, in the occurrence of rheumatic disease during fetal life.

Wilks and Moxon record a case of congenital cardiac malformation in which there was history of the mother having suffered from rheumatism during gestation (*Lectures on Pathological Anatomy*, 2nd edition, p. 596, 1875).

The congenital cases that do best are certainly those in which there is little or no cyanosis. Dr. Walshe kindly gives me particulars of two cases in which he believed an imperfect septum to exist between the ventricles. In one, there were no symptoms till the fourteenth or fifteenth year, no cardiac symptoms being even suspected. In the other, seen with Dr. Murchison, the patient was free from any indications of heart-trouble till the age of 17, and then there was only occasional dyspnoea and slight cyanosis. He also met with a case of stenosis of the pulmonary artery which was symptomless till the ninth or tenth year. This patient was remembered to have been a little dusky at the time of his birth. He has frequently seen cases of congenital aortic stenosis in youths presenting themselves for military and other public service, and has also met with some rare cases of mitral regurgitation in the adult, for which he has presumed a congenital origin. Persistence of the foramen ovale seems to be rarely recognised for certain during life. It is often suspected in cases which prove to be pulmonary arterial stenosis. It is very common to find this aperture large enough to admit a small quill in cases which have lived a full term, and have been free from any symptoms. Cases have been recorded in which murmur existed with patency (Hayden and Wolfe, quoted by Sansom in the *Proceedings of the Medical Society of London*, 1877-79, p. 206), and some in which there was no murmur (*ibid.*, Markham, B. Foster, and Mayne) with this condition.

Dr. Latham, in his classical work (*On Diseases of the Heart*, p. 241, London, 1846), gives the histories of two cases of (probably congenital) disease in young ladies who had no serious symptoms for twelve and nine years respectively after the discovery of systolic murmurs; also one of a boy aged nine, famous for gymnastic feats, who had a loud systolic murmur audible all over his chest and posteriorly. He had no untoward symptoms.

2. I pass on to consider cases of rheumatic valvulitis in its widest aspect. It is in this category that most cases with which we are now concerned will fall. The essential characters of these are too well-known to need recital here. It is still not sufficiently recognised that the rheumatic, or, as I prefer to call it, the arthritic diathesis, has other and well-marked characters beyond the joint and cardiac manifestations. Its features may be discerned by those who will study them in their physiognomical as well as in their textural expression. Strong heredity attaches to it. Rheumatic taint may be roused to activity in intra-uterine as well as in extra-uterine life. Thus may be explained some of the cardiac defects met with congenitally. In the motorial neurosis, termed chorea, we have also, as I believe, in the large majority of cases, an expression of the rheumatic habit of body. Hence, we must be prepared to meet with cardiac valvulitis in persons who have suffered no other manifestation of it than the choreic form. When we come to seek evidence of this, and of the former existence of so-called "growing pains," we shall find that the rheumatic habit has much to answer for—far more, indeed, than we should elicit if we only sought proof of a definite rheumatic illness such as rheumatic fever. We have learned of late years to associate the existence of cutaneous and subcutaneous nodules on various parts of the body with co-existing rheumatic cardiac valvulitis, a very nice and useful point in etiology and diagnosis. We must take this wide conception in forming our opinion as to the cause of any cardiac murmur with which we may be suddenly confronted, and where rheumatism may be suspected as the cause of the mischief thus indicated.

Instances are numerous where murmurs are found when unsuspected, significant of valve-lesions which have given no tokens of their presence in past life. Some are thus practically symptomless; but, in most cases, there is commonly to be found an indication of cardiac or pulmonary disturbance, not grave, it may be, but sufficiently marked to prove that all was not right with the patients.

We find aortic disease more frequent in males, and more common in females who have done laborious work akin to men's pursuits. We meet with mitral stenosis more commonly in females. Males are, more than females, subject to valvular disease in general, as shown by statistics, but this undue incidence for the male is shown to be due to the greater prevalence of aortic disease in them. Cases of mitral incompetency sometimes live to full term of years. As much cannot be said for those in which aortic reflux prevails. In some cases of aortic obstruction and regurgitation, life may be prolonged for many years. In reviewing cases due to rheumatic influence, we must not lose sight of

the ill-effects produced in certain cases by myocarditis, leading to softening and dilatation of the heart's walls, whereby fleeting murmurs may be generated, capable, on recovery, of disappearance. Nor can we omit to mention the effects of pericarditis, leading to adhesion of the sac, with general cardiac hypertrophy as the result of this. The latter is more common in children, and, when present, is rarely long survived.

In entering, thus far, into particulars of these cases, I am aware that I seem to depart somewhat from my text; but I am rather concerned now to lay down principles to guide us in sifting the facts which have already come before us, or may do so hereafter. Our clinical facts must be accurate, if we wish to deduce trustworthy prognostics.

Amongst my notes of cardiac cases, I find many in which no serious symptoms occurred till many years had elapsed. I imagine that this is a common experience after rheumatic valvulitis. In many instances, the lesion was mitral, and of obstructive character. I believe that this is often long unaccompanied by serious symptoms. Professor Gairdner informs me that this is also his experience; but many observers are of a different opinion. Thus, Broadbent affirms that it stands in point of danger next to aortic incompetence, "being, indeed, the most serious of the two in early life." (*International Journal of the Medical Sciences*: London, January, 1886, p. 57.) Dr. G. W. Balfour affirms that, in mitral disease, it is rare for the patient to be long without some breathlessness, though no serious troubles may supervene for many years. At any rate, in hospital practice, it is rare for patients to come for advice before the graver symptoms have set in. In most of my cases, the rheumatic attack had occurred some years previously, most often in early life. With respect to many cases of mitral stenosis, I am quite in agreement with Dr. Sansom, as to overt attacks of rheumatic fever forming no part of their history or etiology; but I believe, with him, that the valvular mischief is, nevertheless, often truly rheumatic, the result of a chronic form of the disorder, which leads to stenosis and gradual maiming of the valve. Anæmia is, probably, also an etiological factor in some of these cases.

To speak vaguely of mitral disease, and of prognosis in regard to it, is, of course, too loose a method. The prognosis of mitral reflux, and of mitral stenosis, in pure cases of each, certainly varies in respect of the specific lesion. It is better, I believe, in the former, though I do not necessarily regard the latter, in well-to-do patients, in the second decade of life, with very fearful forebodings.

Where reflux co-exists with obstruction, I think the prognosis much graver. As to compensatory changes in the heart, in mitral stenosis, I would call attention to the fact that there may be limits placed to hypertrophy of the left auricle, by reason of the fibrous toughness of the endocardium, which naturally exists here, and which is often described as morbid. But both hypertrophy and dilatation occur in this chamber. The average age at death, in nineteen cases of mitral stenosis, observed by Dr. Sansom, was 35 years; and of forty-two cases, collected by Dr. Hayden, 37.82 years.

In these cases, compensatory action is secured *pari passu* with the progressing lesion, and no bad symptoms appear, till, with failing health, advancing stenosis, or as the result of undue exertion, they at last supervene. Dr. Sansom is of opinion that compensation is, on the whole, more certain and more persistent in mitral stenosis than in mitral insufficiency. My experience accords with this. In discussing the question before us, much depends on what we consider to be serious symptoms. I apprehend we should be agreed that by these we mean failure of compensation, cardiac dilatation, tricuspid stenosis, dyspnoea, pulmonary congestions, and dropsy. Of minor symptoms we meet with many in such cases, such especially as palpitation induced by overwork, dyspepsia, or emotion; pulmonary congestion evidenced by cough, and even by hæmoptysis, and occasional epistaxis. Some of these rapidly subside after rest and simple treatment. In some instances of rheumatic valvulitis, mitral or aortic, phthisis is associated, and it has seemed to me that the latter disease has been long inhibited by the cardiac concomitant. Drs. Walshe and J. E. Pollock have called attention to this point (*Elements of Prognosis in Consumption*, p. 272; 1865). The latter found that death more often resulted from the cardiac than from the pulmonary disease.

Professor Gairdner records, in his *Clinical Medicine*, some important cases of rheumatic valvulitis, illustrating prolonged immunity from cardiac symptoms, where loud murmurs were present.

In one, described in 1862 (*Clin. Med.*, p. 562), in the person of a medical man, a murmur indicating mitral reflux was known to exist for many years. He had rheumatism when a very young man. He

* On the Etiology of Mitral Stenosis, by Sir D. Duckworth. (*St. Bart. Hosp. Reports*, vol. xiii, p. 263. 1877.)

married twice, the second time late in life. He has had many threatenings of a break-down, and once had anginoid symptoms. Professor Gairdner informs me he is still living, and continues his practice. He took much digitalis, and lost his murmur; two months ago he reported himself comfortable and well. I am indebted to the kindness of Dr. G. W. Balfour, of Edinburgh, for the history of a case in which there was one attack only of rheumatic fever, at the age of eleven or twelve years. The patient is a policeman, married, over 40 years of age, living at the top of a high house in Edinburgh. Many years ago, Dr. Littlejohn accidentally discovered an aortic murmur in this case. This man has now a heart, as Dr. Balfour describes it, like an ox, with loud double aortic murmurs, and he is now failing, having only had cardiac symptoms for a few weeks, following a bad attack of bronchitis. This case has been absolutely symptomless for nearly thirty years. I think it is hardly to be doubted that the records of most life-insurance offices would add testimony to the fact that cases of rheumatic valvulitis live on for long periods without the occurrence of serious symptoms. Our proposed inquiry will be largely aided, if medical officers of assurance societies will procure the testimony needed.

Respecting mitral reflux, Sir Andrew Clark reports a case of a gentleman who was condemned to die in less than six months, and who, forty years afterwards, was enjoying good health; and also one where the patient was living, in fair health, forty-two years subsequently, at the age of 85, (*Proceedings of Med. Soc. of Lond.*, p. 49, 1877-79). Dr. Semple (*Diseases of the Heart*, p. 114, London, 1875) has recorded a case of mitral and aortic valvulitis, after five attacks of rheumatic fever, in a lady, in whom the disease has existed for thirty years. She is now in New Zealand, and is, Dr. Semple informs me, still in good health. In the museum at St. Bartholomew's Hospital, are several specimens, taken from patients who appear to have been long free from symptoms of heart-disease. In one there is sclerosis of the mitral valve, with vegetations on it, and on the left auricular wall; a pendulous vegetation is also attached to an aortic cusp. This came from a woman, aged 27, who had attacks of rheumatic fever at the ages of 16 and 23. She had a loud murmur at the apex, audible behind. She had no inconvenience from her heart, and died of acute nephritis with anæmia.

In another specimen there are but two aortic and two pulmonary valves. These and the tricuspid and mitral valves are all thickened. The aortic valves are quite rigid and stenosed. The whole heart is enlarged proportionally in its several parts. This came from a man, aged 30, who had rheumatic fever fifteen years before death. There were no symptoms till a year before his death, when he had dyspnoea on exertion, much increased in the last week of life. Anasarca only existed for three days at the last. Murchison (*Path. Soc. Trans.*, vol. xix, p. 100, 1865) recorded a case of mitral and aortic disease in a man, aged 45, who had rheumatism eight years previously, had drank freely of beer, and went on without any symptoms till within six months of his death, from cardiac failure, rupture of the right ventricle (which was fatty), and rupture of an aortic valve. I have under my care a lady, who has had mitral reflux without any serious symptoms for nearly ten years. The lesion began insidiously. There was gouty history in the family.

Dr. Walshe has met with two or three cases with imperfect second sound at the aortic base, which have suggested imperfect closure of the valves, and which have passed on to perfect reflux. These cases were void of all symptoms. I have seen several of such cases, and have now two of rheumatic origin under my care at this time. This would indicate that incipient aortic regurgitation may be a symptomless lesion, and as much might, perhaps, have been predicated.

Dr. Fothergill (*The Diseases of the Heart*, p. 500, 2nd edit., 1875) records two noteworthy cases of aortic disease. In one, a medical man, had thus suffered often for twenty years. He had had serious attacks occasionally, but had recovered from them. He had led a very active life, and the disease made no progress. In the other, a woman over forty years of age, there had been signs of aortic reflux for more than twenty years. She had an enormous left ventricle. It is not, however, stated if these were cases of rheumatic disease. Dr. King Chambers (*Lectures chiefly Clinical, etc.*, p. 307, 4th edit., 1865) has recorded cases of valvular cardiac diseases, of long standing, many of them of rheumatic origin. He seldom refers to the special valves affected, and only mentions that murmurs were present. The histories were taken from private practice, and from insurance examinations. There was evidence, in these cases, of great prolongation of life from the time of the original rheumatic attack, and the periods varied from one to fifty-seven years. In most of these cases, eighteen in number, there were no symptoms, and few that could be called serious, save some anginal attacks in a case of aortic disease, in a lady, which

had lasted seven years. Dr. Chambers remarks, upon this series of cases, that "you know very well that cases like these are not to be met with in hospital practice, or among the paupers in a workhouse." They are, indeed, only found amongst patients of the well-to-do class, who are able to live carefully, without exposure to poverty and hard labour. Dr. Walshe gives me his experience, to the effect that it was very common to meet with examples of mitral reflux, aortic stenosis and reflux, running on for years, without symptoms fairly traceable to valvular defect. He has known cases, observed by himself from the first, of mitral reflux remain functionally innocuous for upwards of twenty years. Cases of aortic regurgitation, which are symptomless, he has not found to be commonly of rheumatic origin.

[To be continued.]

CONGENITAL CLUB-FOOT.

THE PART PLAYED BY THE TARSAI LIGAMENTS IN MAINTAINING THE DEFORMITY; AND THE VALUE OF THEIR SUBCUTANEOUS SECTION IN ITS CURE.

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FOR some years past, I have been much occupied in the study of the anatomical conditions existing in congenital club-foot, and, in conjunction with my friend Mr. Shattock, I contributed to the Pathological Society a paper on the subject, which appeared in the *Transactions* of that Society for the year 1884. I may very briefly say of that paper, that it was commenced without reference to the conclusions at which Mr. Shattock and I both arrived, long before we had finished it. When we commenced the study of club-foot, we were both dominated by what, I believe, is the most widely accepted doctrine as to its mode of causation; namely, that it is either a paralysis or a spasmodic contraction of certain muscles of the leg acting upon the foot. Neither of us was prepared to find the nerve-centres, the nerve-trunks, and the muscles of the afflicted limb in a state of absolute histological integrity; neither of us was prepared to find that, after all the soft parts had been entirely removed from the limb, the deformity would persist in just as marked a degree, and in just as intractable a form, as previously to the dissection. Yet such was the case. My object in this paper is not to merely repeat the substance of what I said at the Pathological Society, but rather to advocate the practical lessons which, I venture to think, this anatomical condition imposes on all who are concerned with the treatment of club-foot.

Brief Abstract of the Anatomy of Congenital Club-Foot.—In a not inconsiderable number of cases of club-foot, which I have had the opportunity of dissecting during the past four or five years, mostly still-born children at term, but including also three cases in children ranging from 6 to 18 months of age, the only constant condition, which I have found, has been inability to overcome the deformity (even after removal of all the muscles), until some of the ankle and tarsal ligaments have been divided. This condition of ligaments can hardly be described as a shortened condition, because such a term would imply that, once long enough, they have in some way or other become shortened; and this is not in accordance with the actual facts. These ligaments are short, but they are not shortened; they are as they have been developed; they are in the same condition as certain of the muscles, that is to say, shorter than normal, owing to the fact that the points of attachment have been unduly approximated during development and growth. Microscopic examination of the muscles shows them to be histologically normal in all respects in early infancy; the nerve-trunks also, and the spinal cord, show a healthy structure; the full supply of normal ganglionic cells was particularly striking in the lumbar enlargement of the spinal cord in the cases examined, and shown at the Pathological Society.

As regards the bones of the tarsus, I may say that, although an exaggerated inclination inwards and forwards of the neck of the astragalus was found in the great majority of cases, yet this condition was not constant; that in one well marked case the inclination inwards was much below the average amount found in the infantile normal astragalus, a fact which demonstrates that this conformation of the astragalus is not only not the essential element of the deformity, but also that it is not even a necessary part of it. The average amount of this obliquity in club-foot is 49°, while the maximum has reached as high as 64° in my own cases. Some authors