A POST-GRADUATE CLINICAL LECTURE on MITRAL STENOSIS, with SPECIAL REFERENCE to RECENT INVESTIGATIONS: Delivered at the London Hospital

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Notes
A POST-GRADUATE CLINICAL LECTURE
ON MITRAL STENOSIS,
WITH SPECIAL REFERENCE TO RECENT INVESTIGATIONS.
Delivered at the London Hospital

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In the course of clinical lectures which I have delivered this session I have considered the clinical and pathological signs of mitral stenosis as evidenced by the cases under our observation in this hospital. On this occasion I shall confine myself chiefly to a review of the investigations which have been made in recent years towards the elucidation of some of the problems presented by this form of disease.

We will, in the first instance, examine the patients who will serve as object lessons, and I will in the briefest terms state the circumstances with regard to these which I wish you to note.

CASE I.—C. M., aged 37, female. From 18 years of age suffered from anemia, palpitations, and shortness of breath. For weeks before admission had hemoptysis, two attacks. No rheumatic history. Pulse-rate 75 irregular. Slight thrill at apex. Long purring presystolic bruit, finishing up downwards and rougher bruit in character; double shock sound during diastole heard at apex. Pulmonary second sound accentuated. Patient improved very much while in hospital, her chief trouble being indigestion. Temperature normal; no albumen in urine: quantity passed normal in amount.

CASE II.—E. P., aged 29, female. Patient had right hemiplegia. She could not write, but recognised her name. The heart examination showed all the signs of uncomplicated mitral stenosis, presystolic murmur, and thrill, with embolemic dangers. She discharged herself on being cleared up, so that on discharge patient could walk well. The patient could copy print, etc., but could not write anything original. Inspection of each hand was absent; she could only say "No." We had no doubt that this patient when admitted was suffering from embolism of the left middle cerebral artery. Nutrition was re-established in the arm served by the branches that supply the speech centres; in these a destructive lesion persisted.

In the cases shown to you there is no physical clinical sign of mitral regurgitation; the lesion, so far as the evidence indicates, is obstructive only. The morbid condition of the mitral orifice that is defined by Duriez's and other French writers as "pure mitral stenosis." This is said by these observers to be characterised by a slow, gradual, progressive narrowing of the mitral orifice, evidenced:

(1) Anatomically by a funnel-shaped transformation of the mitral valve due to the complete union of the two curtains, the resulting membrane forming a funnel retaining a smooth and polished appearance;

(2) Clinically by the following signs: (a) Thrill felt near the apex; (b) a prolonged, muffled murmur of low pitch, heard travelling the atrioventricular diastole, followed by a short and rougher bruit in the presystolic period; (c) a reduplication of the second sound.

I am unable to accept these views in their entirety. In the first place, a progressive narrowing of the mitral orifice even in young subjects does not invariably result in the funnel-form of mitral stenosis. For example, Hayden recorded the case of a boy, aged 7, in whom there was no rheumatic history, but who died from cerebral embolism, the result of mitral stenosis of button-hole form. In this case Dr. Hayden distinctly states that many of mitral regurgitation did not exist. It is quite clear that the funnel form of valve change cannot with confidence be predicted in the so-called pure mitral stenosis; conversely the funnel mitral in its typical form I have found in old subjects in the degenerative period of mitral fibroid changes.

Now as to the etiology of this slowly developing obstructive disease at the mitral orifice. Duriez has said that pure mitral stenosis is not a rheumatic affection. With this statement, I think, we means agree. I have never myself observed nor found any record of observations indicating that mitral stenosis is a congenital malformation; but in very rare cases I have witnessed examples in which other congenital anomalies of the heart have been in association with a fringe of vegetations around the mitral orifice, and with thickening, which, I could have no doubt, represented an early stage of mitral stenosis. Congenital malformation is one thing and intracerebral endocarditis another. The vegetations observed had exactly the characters of those of endocarditis of the rheumatic form. Duriez thought that rheumatic endocarditis always resulted in mitral insufficiency or mitral insufficiency and stenosis combined, and not in pure mitral stenosis. In his cases it said that the mitral orifice was unequal with hard edges often cretaceous, in the form of crescent or slit, whilst in the other there was only a polished funnel; the first was complicated by disease of the other orifices; the second was uncomplicated and was as the case of an expressive volcano. These observations also do not accord with my experience. In my Lettomanian Lectures (1883) I gave reasons for the conclusion that there was an intimate association between mitral stenosis and rheumatism, but with its insidious rather than its pronounced forms. Rheumatic fever, especially repeated attacks of the disease, is more frequently attended with mitral regurgitation—the slight, insidious, sometimes clinically inde- testable forms of rheumatism by mitral stenosis.

Further experience has but confirmed me in these views. Take for example the following case:

A girl, R. W., whom I had long under observation. At the age of 12 she came under my care for suffering attributed to her heart. She had never experienced rheumatic attacks, faints and subacute attacks. A presystolic bruit and also a systolic murmur were heard. She was examined and relieved and then discharged. After then there were thrill and presystolic bruit with a slight systolic blow. After severe lupus complications she died, and the post-mortem examination showed the typical exsudative lesions of rheumatic orifice, having all the characters of rheumatic endocarditis encircling the auroculo-ventricular orifice.

The case is only an example of many others in which we have found rheumatic endocarditis in the subjects of funnel mitral, and I think that there is a chain of post-mortem evidence which shows that morbidity changes having the exact characters of rheumatic endocarditis, bringing about mitral stenosis in greater or less degree, are to be found in subjects who have presented very slight signs of rheumatism or even no signs at all. Clinical evidence goes hand in hand with this anatomical evidence. The rheumatic origin of the case, I described in my Lettomanian Lectures cases in which, after typical and undoubted attacks of rheumatism, there were, first, the development of signs of mitral insufficiency, and then, in the course of years, those of the combined lesion or of mitral stenosis pure and simple.

In a case very carefully observed by my then house-physician, Dr. Needham, the physical signs changed from those of regurgitation to those of stenosis in fifteen days, and thus gave us our subsequently the systolic murmur of mitral regurgitation had quite disappeared, a typical presystolic murmur of mitral stenosis and reduplication of the second sound reestablished.

In December, 1892, I examined a lady, aged 26, who presented all the signs of mitral regurgitation, and in fifteen days subsequently the systolic murmur of mitral regurgitation had quite disappeared, a typical presystolic murmur of mitral stenosis and reduplication of the second sound re-established.

From many such considerations I cannot swerve from my opinion that in a very large proportion of cases mitral stenosis, though unaccompanied by any signs of regurgitation, is a rheumatic disease. The endocarditis which produces the lesion is protracted and progressive, giving rise to a comparatively slow formation of fibrous quasi-cicatrical thick, under the even pressure of the blood, both on the side of the auricle and on that of the ventricle, tends to form the smooth and even membranous funnel which has erroneously suggested a congenital malformation.

Yet I would not assert that pure mitral stenosis must always be in rheumatic endocarditis. Dr. Pierre Teissier has written a valuable memoir containing a condensed report of a large number of cases, in which he maintains that the gradual and progressive narrowing of the mitral orifice due to the effects of rheumatism, is more strongly opposed in this conclusion, which I consider an extraordinary example of specially pleading. Teissier cites my observations, and says, "Sansom is obliged to recognise the fact that mitral stenosis could develop insidiously without the intervention of rheumatism." I did recognise this fact, and pointed it out most strongly, but Teissier would have been more correct if
he had quoted me as saying "without the intervention of rheumatism as clinically evidenced." For I showed that the form of endocarditis as observed in the post-mortem examination was usually just that which we find associated with rheumatism. In a very few cases the lesion, or the concomitant mitral stenosis, must have been of long standing, for the endocarditis might have been a cause of, and not a complication of, the lesion. In one, in fact, the patient died of the endocarditis, which was chronic and of long standing, before the lesion of the mitral orifice was discovered. In another, the endocarditis had been present for ten months, before the patient was admitted to hospital, and the lesion of the mitral orifice was found post mortem in an infant who survived birth only twenty-one hours by Benezet Smith, by Parrot in one who lived fourteen years; and in a case recorded by Gerhardt of an infant only 4 months old. The lesion in cases of this sort has been noticed about the age of 14 or 15 years—men especially at the time of puberty. It is to be remembered that mitral stenosis is much more frequent in the female than in the male.

Pottain, Landouzy, Broadbent, and others, have pointed out that mitral stenosis in the female is frequently associated with anemia and chlorosis.

That there is some relation between mitral stenosis and tuberculosis I quite agree. Pottain found post-mortem evidence of tuberculosis in 12 cases out of 35 of pure mitral stenosis, a proportion of 35 per cent, and Tsaiiser brings evidence to show that this figure is under the mark. I have myself found very rapid pulmonary tuberculosis evidenced in a case in which there was a funnel mitral, and I have observed clinically other cases in which the pulmonary tuberculosis was evident in the subjects of mitral stenosis. I have not found, however, the coexistence in anything like the proportion quoted by Tsaiiser. Taking the cases in Tsaiiser's abstract with those cited by Percy Kidd and others, I find a total of 37 in which tuberculosis was associated with other lesions, but these were not mitral stenosis. Uncomplicated mitral stenosis was present, therefore, in 16 cases, but in 19 cases the heart was recorded by Gerhardt of an infant prematurely born at the eighth month, and dying at the age of 4 months from acute tuberculosis. In this case the mitral alone among the valves of the heart was affected; it was puckered, and its corde tendineae were fused together and thrown back, with the left ventricle alone affected. The supposed mitral lesion were post mortem; of these 11 presented also the signs of tricuspid stenosis or of endocarditis affecting the tricuspid valve, and 5 others manifested disease of the aortic valve. Uncomplicated mitral stenosis was present, therefore, in 16 cases, but in 19 cases the heart was recorded by Gerhardt of an infant prematurely born at the eighth month, and dying at the age of 4 months from acute tuberculosis.

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From the evidence which I have examined, therefore, I believe that the most frequent cause of mitral stenosis is a form of endocarditis of the rheumatic form and another cause far less frequent, but one operating for the most part in advanced life, namely, a general or widely diffused, slowly developing fibrosis. I recognise, however, that there may be other forms of obscure etiology, though I find that the hypothesis of any tuberculous causation is not supported by anything like sufficient evidence.

It has been shown that in mitral stenosis of the funnel form there has been in some cases a systolic murmur heard in the left auricle. It has also been said that in the presence of mitral stenosis a systolic murmur is heard; only a short systolic murmur is audible. It is the opinion of Goodhart and of Pottain that this systolic murmur may be due to mitral stenosis or to tricuspid regurgitation. Dr. W. Samways has made an inquiry concerning the frequency of systolic murmurs in 70 cases of mitral stenosis examined at Guy's Hospital. In 40 of these a systolic murmur had been audible during life, and in 15 the situation of audibility of the murmur suggested a tricuspid causation. Dr. Samways considers that it is reasonable to conclude that in cases of mitral stenosis, regurgitation through the mitral aperture is less common than has been supposed, a tricuspid reflux having often been mistaken for a mitral regurgitation.

I come now to a very interesting question. Finding, as we do, that in the so-called pure mitral stenosis there is no clinical evidence of mitral regurgitation, how is such regurgitation prevented? In the search for an answer to this question, our attention should first be called to the action of the lesions seen in mitral stenosis on the aortic and pulmonary valves. There are two points to the effect that in some cases of this affection the muscular tissue of the auricle is enormously hypertrophied, in others atrophied to almost complete extinction. I have myself observed post mortem in a child the auricular muscle as thick as that of the right ventricle, and in an aged woman an extreme dilatation of the auricle, the cavity being lined with laminated clot, and in the thin wall muscular fibres scarcely represented at all.

An excellent summary concerning the left auricle in this disease has been published in the graduation thesis of M. Gérard. Some authors—as Laennec, Stokes, and Niemeyer

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—have concluded that dilatation of the left auricle is the direct consequence of mitral obstruction. Others—as Bouil-

taud, Potain, and Rendu—have insisted that dilatation and hypertrophy go together: "the left auricle dilates and hyper-
trophies simultaneously" (Potain and Rendu). The records of post-mortem examinations, however, have not at this stage
determined the exact relation of the hypertrophy and dilatation.

First, Is dilatation of the auricle a necessity in comp-

ensated mitral stenosis? Is hypertrophy always to be 
demonstrated? Is hypertrophy of the auricle a provision for 
effecting the maintenance of the auricular and ventricular orifices—the latest sign of a falling compensation? Gérard says: "In

pure mitral stenosis, the heart adapting itself, as it were,

naturally to its diminished labours, the dilatation of the left auricle are often absent for a long period." This is contrary to what

occurs in the combined lesion of insufficiency and stenosis, where the adaptation of the heart is impossible and hyper-
trophy of the left auricle occurs at any rate provisionally.

The evidence is not less evident in the cases of mitral stenosis by means of the cardio-

graph. In 1880 I published some cardiographs showing a very pronounced, in some cases enormous, elevation which could only indicate an inordinately powerful contraction of the auricle and make the ventricle swell in the cases.

Examining the records of 70 post-

mortem examinations of cases of mitral stenosis made at Guy's Hospital, he finds that in pronounced mitral stenosis the characteristic modification of the left auricle is not dilatation, but hypertrophy. In every examination dilatation is associated with hypertrophy, the dilatation is probably not evident during life whilst compensation exists, and it occurs when compensation fails.

Let us turn to clinical evidence. I examined a large number of cases of mitral stenosis by means of the cardio-

graph. In 1880 I published some cardiographs showing a very pronounced, in some cases enormous, elevation which could only indicate an inordinately powerful contraction of the auricle and make the ventricle swell in the cases. In these cases the elevation of the auricle, as perceived by the finger. Dr. Dickinson's inference was that the murmur which was heard could not be pre-systolic and of auricular origin. In my opinion Dr. Dickinson was unwise, though his clinical observations were strictly accurate. I argued that one should not assume that the elevation of the heart's apex, as felt by the finger of the observer is of necessity synchronous with the contraction of the ventricle—that in some cases the hypertrophied auricle extruding its blood contents through a moderately narrowed mitral orifice might be sufficiently powerful itself to cause a pronounced elevation of the apex, the proper systole of the ventricle following. My observations and reasoning thus expressed have been exactly corroborated by Professor Potain of Paris, who, in his work in the Clínique Médicale de la Charité, 1894, has recorded a number of cardiographic and sphygmographic observations of his own and of others who have employed instruments of precision. Potain agrees with Dickinson that the pre-systolic roll (roulement précystolique) coincides with the apical impulse. But this lifting of the apex is not always produced by the ventricular systole, but rather by the auricular systole and the distension of the ventricle which this determines. The elaborate reasoning on which this conclusion is based are detailed in the article on the apex beat of the heart. Potain further states that accumulated proofs leave no room for doubt not only that the left auricle contributes to the impulse of the left ventricle, but that the contraction of the muscle of the auricle does not cease until the complete closure of the mitral orifice, and the commencement of the propulsion of the blood of the left ventricle into the aorta. Dr. Samways has seen in this an explanation of the preven-
tion of regurgitation into the left ventricle in the "pure" mitral stenosis. He contends, from experimental as well as mathematical data, that the auricular cavity being small (its muscular walls by their contraction having nearly emptied it) and the ventricular cavity being large (nearly replete), it is possible that the auricle as a contracting sphere might be the controller of the situation, and force the reflections into the ventricle, in spite of the muscle of the latter being already in contraction.

Dr. Robert H. Woods of Dublin had already shown in 1892 by mathematical demonstration that a small contracting sac can become the controlling element in the circulation. In other words, the contraction of the auricle may be responsible for the mechanical effects—on the area of the orifice, and the shortening and approximation of the papillary muscles. Marc Sée has contended that the papillary muscles of the left ventricle in their contraction are so disposed as to fit more tightly into the upper part of the auricle and ventricle. It is true that these muscles do not contract at the very earliest period of systole of the ventricle; the parietal muscle commences, but is reinforced by the papil-

aries in an extremely brief time. I can only adduce these con-

tractions as evidence that the contraction of the auricle is not to be considered as the sole force, but is an auxiliary force in preventing regurgitation at an early part of ventricular systole. Furthermore, if the auricle can contend successfully for the layer of muscular fibres by which it is surrounded, and the pressure in the aorta overcomes the aortic valves, this is all that the argument demands. Auricle and ventricle would co-operate to do this. Dr. Robert H.

Woods makes a statement with which I by no means agree. I know the view is held by many, and I think it that the evidence of the other side should be duly considered. He writes: "There are no valves between the auricle and the lungs, and therefore raising the intra-auricular pressure during con-

traction of the ventricle could only result in the blood follow-
ing the layer of muscular fibres by which they are surrounded, and any great reflux of blood into the veins is thus pre-
vented." The contraction of the circular muscular fibres around the venous orifices and the subsequent contraction of the auricle causes these veins to empty themselves into the auricle; and by their presence and action they prevent any large quantity of blood from passing backwards into the veins when the auricle contracts. No valves are present in the superior and inferior cave in the adult heart, or in the pul-

monary veins. The circular muscular fibres of the auricle play an important part in preventing any reflux of blood during the contraction of the auricles. From these observations you will infer that though there is no membranous valve there is a very efficient sphincter—indeed the valve tending to prevent reflux towards the lungs. I think that this very important quasi-valvular arrangement is too often overlooked. It may be said that the sphincters may become inefficient. No doubt they may; hence some of the important complications of the late stages of mitral stenosis. As evidence of the strain to which the pulmonary veins are subjected, I may cite the observations of Dr. James Barr, of Liverpool, who found well marked atheroma of the pulmonary veins in cases of mitral stenosis.

There are circumstances observed in cases of mitral stenosis...
in children and young persons which have seemed to me to point very strongly to the correctness of the inferences of Potain and Samways. It is the rule to find in the post-mortem examination of such cases that the left ventricle is of small capacity contrasted with the large dilated auricle of the same subject, and with the normal left ventricle of a child of the same age as the subject observed. There cannot be any doubt that the morbid condition of mitral stenosis is of slow pathognomonic. These morbid processes must have commenced at the periphery of the valve curtains, or at the auriculo-ventricular orifice, and it seems to me that unless some cause prevented regeneration, regeneration must have occurred. If such regeneration had occurred, why is it that its usual consequences have not occurred? Surely the enhanced blood pressure within the ventricle would have caused left ventricular dilatation. But such dilatation is conspicuous by its absence. It is in the highest degree probable therefore that regeneration was prevented, and, according to the accumulated testimony, prevented by the action of the auricle.

It is probable that the normal endowment of the auricular muscle is sufficient to prevent the regeneration in the earlier stages of development of mitral stenosis, but in course of time the muscle becomes hypertrophied by the accumulation of the auricle. The auriculo-ventricular muscles become attenuated to almost extinction. Atrophy of the auricular muscle may, however, occur in the earlier stages from various causes of failure of nutrition. My experience coincides with that of Dr. Samuel in establishing as a general rule the doctrine that "the characteristic alteration in the left auricle in mitral stenosis is not dilatation but hypertrophy—dilatation is probably introduced only with the auricular breakdown."

The prognosis in the condition of disease which we are considering is grave. I have found the average age at death of 61 patients to be 32.7 years, but it is to be remembered that my cases included an unusual number of children, for in the stenosis the disease in males is more advanced and probably more than my fair proportion of cases were sent under my notice. The late Dr. Hayden's cases, 42 in number, gave 37.8 years. Sir William Broadbent, 53 cases, gives as the average age at death 33 years for males and 37 to 38 for females.

The most recent inquiry, that by Dr. Samways, as to the post-mortem records of Guy's Hospital during ten years shows that the average age at death, males and females (identically) is 35.6 years; the three pronounced forms of operation of the disease 43.6 years; the milder forms 35.6 years.

The concurrence of testimony, therefore, is to show that the conditions of the disease are grave that life is rarely prolonged beyond 40 years. In the form of disease which is associated with mitral stenosis, the average age at death is much more advanced; but these commence insidiously for the most part at a comparatively advanced age. I have observed such a case at the age of 70.

## Notes and References
11. Roy and Adami, have been able to detect this interval. Célèl Pennebaker and Overend found that the papillary muscles contracted exactly at the same moment as those of the ventricular wall. Lancet, October 10th, 1899.