Mitral Stenosis – A Clinical Study, with some conclusions founded on an observation of 50 cases

by

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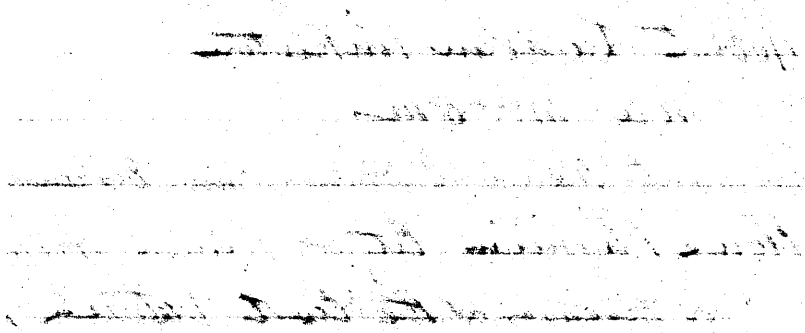

Bramwell, "Diseases of the Heart and Thoracic Aorta (1884) p. 444

Mitral Stenosis has for long had special attraction for me as the subject of a Thesis, partly because of the certainty that, once hearing the murmur, we have to deal with primary valvular disease at the mitral orifice, partly because of its comparatively recent recognition and, no doubt, partly from the fact that, as an old student and clinical clerk of Prof. Gairdner's, - the first in this country to demonstrate the rhythm and cause of the murmur - I had, as it were, a traditional right to follow out this line of investigation.

For the last 17 months I have been examining and making notes on patients in the wards of the Chest Hospital at Victoria Park and in connection with the practice at the Surrey Dispensary. I have now a series of 50 cases - not selected but taken as they came - some of them with other valvular lesions as well, but all of them with obstruction at the mitral orifice, for in all, the characteristic murmur was heard.

It is not uncommon and according to most authorities is essentially a disease of youth or early adult life. Age My own experience is that it is most frequently developed (or at least detected) between the ages of 15 and 25. I found the ages of my patients as follows:-

[1] Hayden "Diseases of the Heart & Aorta (1845) p 990

[2] Broadbent on "Mitral Stenosis" in the International Journal of the Med. Sciences 1886 p. 58

Under 15 there were 13 cases or 26 per cent
Between 15 & 25 " " 26 " " 52 " "
Over 25 " " 11 " " 22 " "

My youngest patient was 9 and my oldest 48.

Then, with reference to the time when the symptoms of Heart Disease first began, my figures are as follows:—

Symptoms began under 15 in 23 cases or 46 per cent
" " between 15 & 25 in 22 " " 44 " "
" " over 25 in 5 " " 10 " "

In a few cases cardiac symptoms had always been present. The latest at which they began was 42 years of age.

Sex. It is generally held that Mitral Stenosis is much more common in females than in males. 66.6% of Dr Hayden's cases were female and Dr Broadbent's were 75% female. I was surprised therefore to find my cases exactly equal — 25 of each sex.

Aetiology.

The obstruction is brought about by an adhesion of the cuspids of the valve and a narrowing of the lumen, due to endocarditis of the lining membrane of the valve. And this endocarditis may be produced mainly by the following causes:— Rheumatic Fever, Rheumatism (subacute or chronic) Chorea and Scarlet Fever. In a certain number of cases, the cause of the endocarditis is obscure and may possibly be congenital. We know how very liable to Heart Disease those patients are who have suffered from Rheumatic Fever — more

1 Sansom: "Lettsomian Lectures on the Treatment of Some Forms of Valvular Disease of the Heart" 1883. p. 99

2 Sansom: "Lectures on Diseases of the Heart in Childhood" Medical Times & Gazette. 1879 Vol ii p. 413.

3 Fagge on "Valvular Diseases of the Heart" in Reynolds System of Medicine Vol iv (1879) p. 609

than a third developing it at some time after, if not at the time. Between Rheumatism, chorea and Scarlet Fever there is strong affinity, as evidenced by the fact that they often exist together or succeed one another in the same subject. And endocarditis may be produced by any one of them. Now according to De Sanctis' Statistics it is much more common for mitral stenosis to follow from the endocarditis produced by a chronic or obscure cause than from that produced by acute Rheumatism & the tendency to stenosis in children increases with the obscurity of the cause.

2 "It would rather appear that the disease which produces mitral regurgitation runs a quicker, while that producing mitral stenosis runs a slower course. The one associated with acute disease tends to retraction of the curtains of the valve while the other attended with adhesions of the curtains and slow fibrous proliferation gradually forms the funnel-shaped or slit like aperture, obstructing the flow from auricle to ventricle".

Most observers are agreed that the numbers of cases of mitral regurgitation is much greater than the number of mitral obstruction or of obstruction combined with regurgitation. It is therefore a matter of surprise to read Dr Hilton Fagges[3] statement, that in 6 years he examined in the post-mortem room only 12 cases of purely regurgitant disease, as against 67 in which the orifice was contracted. Such a record must be quite exceptional.

As I have not a series of cases of regurgitation to compare as to cause

and frequency with those of stenosis, I cannot criticise those statements, but go on to examine the aetiology of the cases, in which stenosis was present.

Of course it is difficult in many cases to say when the heart disease was developed, for it may have arisen during a former attack of a disease, which may produce endocarditis, and only shown symptoms during a later attack. I therefore only attempt an approximation; and I have called "The Cause" of the Heart disease that attack, during which, or after which, cardiac symptoms first attracted the attention of the patient.

Rheumatic Fever was the cause in	24 cases	(13 male & 11 female)	= 48%
Rheumatism (subacute or chronic) was the cause in	6 "	(2 male & 4 female)	= 12%
Chorea was the cause in	5 "	(2 male & 3 female)	= 10%
Scarlet Fever " " "	7 "	(3 male & 4 female)	= 14%
Herpes Zoster " " "	1 "	(female)	= 2%
No Cause could be detected	4 "	(2 male & 2 female)	= 14%

Now to examine the different causes individually:-

Rheumatic Fever

In 16 out of the 24 in which Rheumatic Fever was the cause, cardiac symptoms arose during the illness or convalescence. In the remaining 8, the period of onset varied from 3 months to 18 years. Again in 8 of the 24, there was more than one attack of Rheumatic Fever. In 3, the cardiac symptoms arose during the first attack; in 1, during the seco

and in 2 during the third. In the remaining 2 cases, symptoms arose respectively 12 and 18 years after a second attack.

Rheumatic Fever associated with Chorea

There was also a history of chorea in 5 of the 24 cases, in 2 of which it occurred with the Rheumatic Fever. In one / the chorea was 5 years after the Rheumatic Fever and here the cardiac symptoms developed a year after the Fever and 4 years before the chorea. In the other 2 cases the chorea was previous to the Rheumatic Fever. In one of them the symptoms developed with the Fever and 3 years after the chorea; in the other, 16 years after the chorea and 12 years after the Rheumatic Fever. We may conclude that Rheumatic Fever is much more potent than chorea in causing Valve disease, for when they are associated it is the Rheumatic Fever that is the cause.

Rheumatism

Of the 6 cases in which Rheumatism was the cause, there was a history of subacute Rheumatism in 3, and in 3 a history of slight or chronic pains. In 5 the cardiac symptoms followed pretty close on the rheumatic pains. In the 6th, there was a history of chorea and scarlet Fever in childhood, with subacute Rheumatism at 18 and cardiac symptoms at 21.

Chorea

Of the 5 cases, in which Chorea was the cause, 3 had no Rheumatic history whatever. One of these had not had an acute attack of chorea, but volunteered the information, that all her life she had been afflicted by "fidgets" in the arms & legs and unable to keep still.

The other 2 developed cardiac symptoms 1 year & 4 years, after the chorea. Of the 2 cases in which there was some rheumatic history, one had chorea and cardiac symptoms at 5 and subacute Rheumatism at 7. The other had slight pains in the joints during an attack of chorea at 5; and developed cardiac symptoms at 11.

Scarlet Fever — Of the 7 cases in which scarlet Fever was the cause, there was no trace of Rheumatic history in 5; in 2 there subsequently developed Rheumatic pains, but after the onset of the cardiac symptoms. In only one case did the symptom develop with the scarlet Fever; in the rest, not till years after. In these 5, although there was no history of rheumatic pains, still there may have been rheumatism latent. It is easier to think this, especially as in all these cases the scarlet Fever occurred in childhood, when rheumatism may be present attacking the endocardium, but not the joints, than to come to the conclusion that the scarlatinal virus was itself the cause of the endocarditis.

Herpes Zoster. The case in which Herpes Zoster was the cause developed cardiac symptoms with the disease. This is a disease which is often associated with the rheumatic diathesis.

Idiopathic Cases — Take lastly the 4 "idiopathic" cases, the word merely meaning that we do not happen to know the cause. It is interesting to note that in 1 of them there was a family history of Rheumatism and in 2 of the 4, one or other joint

1. Fagge in Reynolds System of Medicine Vol IV. p. 614
2. " " " " " " " 624

had died of Heart Disease. Cardiac Symptoms had in 2 of them been always present; in the other 5 they came out about 16-17, in the case of the females at the onset of Menstruation.

It is at first tempting to consider that these cases with no obvious cause may be of congenital origin But congenital cases are rare and are almost always accompanied by Tricuspid Stenosis as well. In none of them did I hear the murmur of Tricuspid Stenosis. Dr Fagge[1] went against the theory of possible congenital origin for cases of obscure causation. "Even when shortness of breath and other symptoms of cardiac defect have existed from childhood, it appears to me to be more likely that the Stenosis is due to Morbid changes arising in the years since birth, than to malformation or disease occurring in the short period of intra-uterine life, especially since in the foetus the left side of the heart is so situated as to be very little susceptible of morbid action." And further on, he says[2]:- "It would appear, therefore, that the Mitral Valve is very liable in Children and young subjects to undergo those changes which lead to Stenosis, either as the result of a spontaneous, Morbid, Chronic process or else as the consequence of some disease, other than Rheumatism or Chorea, the tendency of which to produce Endocarditis is as yet unknown."

The Family History was noted as follows:—

Good or unimportant in 31 cases or 62%
Family History of Heart Disease " 10 " " 20%
" " " Rheumatism " 8 " " 16%
" " " Chorea Movements " 1 " " 2%

Antagonism of Phthisis Pulmonalis

In 6 of the cases there was a well marked hereditary tendency to phthisis pulmonalis and in each of them it was interesting to find at the same time some family history of heart disease or rheumatism.

In a case, in which a brother and sister died of phthisis, the father has Rheumatism.

In one, in which the mother & 2 aunts on mother's side died of phthisis, the father suffers from Heart Disease.

In one, in which a sister and an uncle on the father's side died of phthisis, an uncle on the mother's side died of Heart Disease.

In one, in which a sister and several relations on the father's side died of phthisis, a brother has heart disease.

The last case is not so striking. In it the mother and a brother died of phthisis; one sister suffers from "weak heart."

There is a well known antagonism between Mitral Stenosis and Consumption. I have seen, though very rarely, cases in which incompetence of the Mitral Valve of Rheumatic origin was associated with phthisis pulmonalis, but never Mitral Stenosis or Obstruction. And in the cases I have mentioned, in spite of the strong family tendency, there was not a trace of consumptive disease

[1] Dawson, Lettsomian Lectures p. 60
[2] Hayden Textbook p. 941
[3] Balfour "Diseases of the Heart & Aorta" (1896) p. 125

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Mitral Obstruction Alone	9 cases	or	18%
" " and Regurgitation	28 "	"	56%
" " and Tricuspid Regurgitation	2 "	"	4%
" " and Regurgitation and Tricuspid Regurgitation	1 case	"	2%
" " and Aortic Regurgitation	1 "	"	2%
" " and Regurgitation and Aortic Regurgitation	1 "	"	2%
" " and Aortic Regurgitation and Obstruction	3 cases	"	6%
" " and Regurgitation and Aortic Obstruction & Regurgitation	5 "	"	10%

Pericardial Friction was heard in 16 cases

Shape of the Valve

Whatever disease, then, may have originated the endocarditis, its effect on the valve has been the same. The cuttains become adherent, there is thickening and loss of elasticity. The mitral orifice becomes smaller. Sometimes it is found at death not very much contracted, but often so small as scarcely to admit the passage of a quill. Two kinds of valve are usually described — the "button-hole" mitral, which looks like the slit for a button and the "funnel" mitral, which is like an inverted cone, projecting into the ventricle. Among the Sanborn's, their relative frequency is 20 of the former to 2 of the latter; according to Dr Hayden, 2 13 to 2.

The obstruction to the circulation may be complicated by regurgitation. Indeed Dr Balfour[3] holds that it must always be present in the "button hole" variety of valve, whether the murmur be heard or not. I heard it in 85 cases or 92%. Then there may be other valvular lesions in the same case. On the opposite page I have tabulated the cardiac complications as I found them. *

Effects on the Circulation

What are the effects of mitral stenosis on the circulation? The blood is prevented from going freely into the ventricle, and the pressure is raised in the auricle, which dilates to some extent, hypertrophies in order to overcome the obstruction and force the blood forward. The pressure is raised in the pulmonary vessels and back to the right

'Broadbent on "Mitral Stenosis" in the International Journal of Med. Science 1880
p 69

Ventricle, which hypertrophies and, acting more powerfully, still further increases the pressure in the pulmonary vessels. There is thus a continued congestion in the pulmonary vessels and a condition of brown induration ensues in the lungs.

In time, the right Ventricle is not equal to the work; it dilates; the tricuspid valve leaks; Then come the serious effects common to the later stages of all forms of Heart Disease. What Dr Balfour calls a transfusion of pressure from the arteries to the veins.

The left Ventricle having less blood to propel does not dilate and hypertrophy as in Mitral Regurgitation, unless indeed this be present also in considerable degree or aortic disease be a complication

There is more tendency to embolism in this disease, for the blood getting dammed up in the auricle may form clots, and a bit of clot, getting through the Valve, may be carried as a plug to almost any part of the systemic circulation. Also a clot from the dilated right Ventricle may cause embolism in the pulmonary artery, resulting in the haemorrhagic infarction of the lung.

Appearance

There is nothing characteristic about the appearance of a patient with Mitral Stenosis. Often they may look fairly healthy. Dr Broadbent

Samoan: Text Book p. 205

Mentions the lips as perhaps too common. I do not think we have here the same tendency to dilated capillaries as in regurgitant disease. In a large proportion of my cases, I have noted the appearance as "thin, delicate-looking" anaemia; and, indeed, it often so much resembled that of early phthisis pulmonalis, that in several instances, where haemoptysis was also a leading symptom, I have at first thought that it was a case of consumption rather than heart disease that had presented itself. They are not as a rule so bright & hopeful as patients in an early stage of phthisis and in a few cases, with recent chorea history, they were dull, melancholy and listless. In some of the females the mammary development was defective. They have not however the extreme pallor and unhappy, irritable disposition that we so often find associated with aortic disease.

Symptoms

There are very few symptoms that belong to mitral obstruction as distinguished from mitral regurgitation. Dr Dawson thinks it more liable to variable symptoms and spasmodic troubles."

As we should expect, from the constant high pressure on the pulmonary vessels, some of the earliest and severest symptoms are from the lungs. These patients are very liable to bronchitis and other pulmonary complaints. I found by physical examination evidence of some amount of pulmonary complication from

Pulmonary symptoms

1 Hayden: Textbook p. 804
2 Samson: Textbook p. 910
3 Hayden: Textbook p. 991

4 Samson Lettsomian Lectures p. 93.

bronchitis & oedema or pneumonia – in at least 35 cases or 70%. And 6 cases or 12% had pleural effusion.

Haemoptysis

A most important and often comparatively early symptom is Haemoptysis. This took place in 20 cases or 40%, at one time or another, and varied from a few streaks with the expectoration to several ounces of bright blood. Dr Hayden[1] found it in 44 out of 81 cases and Dr Dawson[2] in 3 out of 17.

Embolism

The accident of embolism, as we saw, is more apt to occur in mitral stenosis than in any other form of heart disease. I had a history of right hemiplegia in 2 cases or 4%, one of whom had had 3 attacks. Dr Hayden[3] had it in 7 out of 81 cases or 8.6%. Besides the middle cerebral arteries the plug may go to almost any in the circulation – more commonly those of the spleen and kidney. In 2 cases I found the spleen large and tender. A clot going from the right side of the heart would cause pain, rise of temperature, cough & haemoptysis with a feeling of dullness. I could not be sure of any cases.

Dr Dawson[4] puts great importance on rise of temperature as a sign of probable embolism. "In cases under treatment for cardiac disease the one sign, which I have found to indicate the probability of embolism, is a sudden rise in the temperature of the body. The locality of the embolism is not immediately indicated by the symptoms."

In both obstructive & regurgitant disease we have palpitation

[1] Broadbent, Internat. Journal of Med. Sciences p. 559.

[2] Gairdner; Glasgow Med. Journal 1864 p. 290

[3] Sansom; Text book p. 264

[4] Balfour; Text book p. 126

[5] Broadwell; Text book p. 492

[7] Fagge; Guys Hospital Reports (1871) p. 322

[6] Hayden; Text book p. 895

[8] Broadbent; Internat. Journal of Med. &c. (1886) p. 60

and dyspnoea on exertion and after a time we may have oedema

Oedema — I think there is less tendency to mucl oedema in this disease. I found it in 24 cases or 48%, but as a rule it was very little, mostly limited to the feet and that seen in cases with otherwise severe symptoms

Ascites — Ascites is decidedly uncommon. Dr Broadbent thinks it more common than oedema and that it appears earlier in this disease; whereas in cardiac dropsy it is usually later than oedema. I have not found this so.

Enlarged Liver — Jaundice was present in 3 cases. Dr Broadbent thinks great enlargement of the liver common and true pulsation. I found it enlarged, but not greatly, in 10 cases or 20% but no true pulsation

Haematemesis — Haematemesis was present in 2 cases

Epistaxis — Profuse epistaxis was in one case the first symptom.

Pulse — With regard to the pulse opinions have been very various. Dr Gairdner[2] think it regular at first till the atrophy of the left Ventricle; Dr Sansom[3] notes it as very irregular; Dr Balfour[4] as being always irregular, more or less, the intermission of the heart being always below par Dr Bramwell[5], as regular till the auricle get over distended and unable to empty itself; Dr Hayden[6], as regular, till the right Ventricle gives way. Dr Fagge[7] held that in the great majority of cases, in which a pericardial murmur could be heard, the pulse presented nothing abnormal. Dr Broadbent[8] holds that it is regular

at first but may afterwards become very irregular. He also always finds it of moderately high tension.

I have noted it as regular in 24 cases, and in 23 as irregular. The irregularity was as a rule associated with pulmonary trouble and dilated right ventricle. Exercise caused it to run up considerably but did not cause it to become irregular when acting regularly before. I have never observed any increase of tension.

Onset of Symptoms

In 18-20 of my cases cardiac symptoms developed with the disease or convalescence of the disease, that caused the endocarditis; in several the symptoms became aggravated on a second attack of that disease. In the majority, however, the onset was gradual and insidious or due to catching cold and the development of bronchitis, with, of course, increased pressure in the right ventricle and at the same time lowered tone, due to malnutrition during the pyrexia. In 3 cases the altered conditions due to pregnancy first brought out cardiac symptoms. In one they lasted only during pregnancy to return with the next. In another they continued after the first pregnancy; while the third stated that she had haemoptysis, only during pregnancy, but dyspnoea and palpitation at other times. All had easy labours and stated that they made good recoveries.

In 2 of my cases the onset of symptoms was with the first menstruation - the compensation which had before been sufficient, failing to

[1] Bramwell: Text book p. 489

meet the new conditions and requirements.

Physical Signs.

Increased Dulness

One would expect that any enlargement of the heart would be mainly associated with the right side and with the left auricle. In point of fact I found the superficial cardiac dulness enlarged to the right in at least 39 cases or 48%. The apex beat was outside the nipple line in 26 cases or 52%; but here there was also mitral regurgitation or aortic disease. In cases of pure obstruction or complicated with only a slight amount of regurgitation, we should not expect this.

Apex beat

The apex beat was often diffused and indefinite, with pulsation for some distance round the place of maximum impulse. Sometimes there was pulsation all over the praecordium. Epigastric pulsation was noted in 12 cases or 24%. And in several cases there was pulsation over the 3rd left interspace, close to the sternum, due to dilated pulmonary artery or dilated and displaced Conus Arteriosus of the Right Auricle, as Dr Bramwell suggests, rather than the Left Auricle, which is deeply placed

Pulsation

Only 22 of my cases had no abnormal pulsation.

Thrill

On placing the hand over the apex there is generally felt a certain amount of thrill, preceding and leading up the apex impulse. This, when rough and well marked, is almost pathognomonic

[1] Balfour's Text Book p. 128.

[2] Dawson: Textbook p. 116.
[3] Hayden: Textbook p. 991

of the lesion. It was present in at least 40 of my cases or 80%

The Sounds of the Heart

The 1st Sound

The 1st sound is nearly always present at the apex. In only one case have I noted it as absent and here it came back in a few days. It is generally altered in character, becoming flapping and more like the 2nd S. for which it is sometimes mistaken. Usually it gives with the sound, the sensation of a "thump" and in most strong cases, I have noted the "thumping" character. Dr Balfour puts great stress on this regarding it as the last portion of the murmur — of considerable diagnostic value when the typical murmur is absent.

2nd S.

The 2nd S. is sometimes absent at the apex — 18 cases or 36%. It is often, as we should expect, accentuated over the 2nd left cartilage, sometimes a distinct snap being felt. I found accentuated 2nd S in 35 cases or 70%. It is important only as showing tension in the pulmonary circulation. The aortic 2nd S is often weakened.

Reduplication of the 2nd S. over the sternum and 2nd left Cartilage is regarded by Dr Sansom[2] as "strong presumptive evidence of the existence of Mitral Stenosis." He found it in about 1/3 the cases and Dr Hayden[3] in 33%. I have noted it in only 13 cases or 26%, but, of course, I may have overlooked it in others. This also is due to increased tension in the pulmonary artery, which records more than the aorta, with the closure of the valves.

Murmurs

The Auriculo-Systolic murmur

The one pathognomonic sign of Mitral Stenosis

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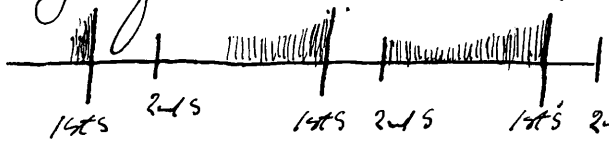
It must be mentioned that Dr Sansom in his Text book [2] says that the murmur of aortic regurgitation may so closely resemble that of mitral obstruction, as to be indistinguishable from it and that a similar thrill may be present, so that when aortic regurgitation exists we cannot diagnose mitral obstruction. Still, I think, the pseudo character of the murmur and the Carp 1st S. will help us. And we have also the tendency to pulmonary trouble and haemoptysis. In one of my cases, in which double Mitral and double aortic murmurs were diagnosed, the autopsy confirmed it.

[2] Sansom: Text book p. 200

[3] Broadbent: Internat. Journal of Med. Sc. p. 66

is a rough, murmur, heard at the apex, running up to and closed by the 1st S. If it be one heard we may expect, with certainty, to find the lesion after death. If we have not heard it we may, from other signs, suspect the existence of Mitral Stenosis, but we cannot diagnose it. My cases exhibited very variety in the length of this murmur, from the shortest limit preceding the 1st S. to one that was heard right from the 2nd to the 1st S. e.g.



Localisation of this murmur

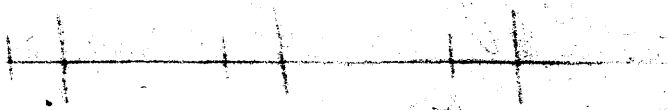
The murmur is remarkably localised at the apex. It was seldom heard outside of an inch round the apex, best. Dr Sansom[1] holds that it is best heard just internal to the apex and Dr Broadbent[3] seems to be of the same opinion. Most other observers refer it exactly to the apex. In none of my cases was it best heard internal

Quality

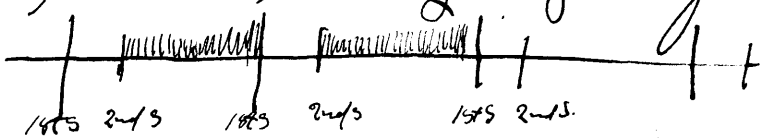
The quality of the murmur is characteristic. It is rough, churning vibrating and runs up crescendo to the 1st S. It is vocalised in various ways. Dr Balfour imitates it by a series of rrrs and a b. (rrrrrrb) This is the Auricular Systolic Murmur of Dr Gairdner, named with reference to the systole of the auricle and the presystolic murmur of other observers named with regard to the systole of the Ventricle

The history of the association of this murmur with Mitral Stenosis forms interesting reading in Dr Foggo's paper in Guy's Reports for 1841

[1] Fagge: Guy's Reports for 1871. p. 252
[2] Galabin: " " " 1875 p. 284

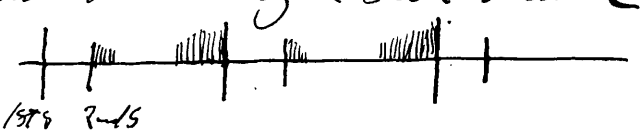


It is important to note that it occasionally, though rarely, happens that the presystolic murmur does not run right up to the 1st Sound. Dr Fagge insisted very strongly on this fact "the presystolic murmur is often quite as distinct from the following 1st S. as an aortic systolic from the following 2nd Sound." Dr Galabin[2] has also observed this and has proved it by cardiographic tracings. I noted this in only 4 cases, in one of which the murmur was very constant and did not run quite up to the 1st S. at any of my examinations of the heart. In the other 3 the murmur varied under the stethoscope, sometimes running up to the 1st S, sometimes stopping short of it, sometimes vanishing altogether for a few beats.



Early diastolic murmur

Sometimes, although not often, we come upon a case, in which a short murmur follows the 2nd S. and is separated from the presystolic murmur by a short interval

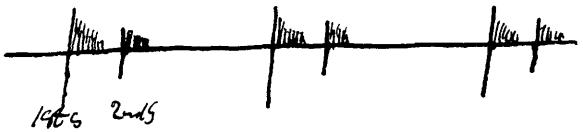


We might theoretically expect to find an "early diastolic" murmur occasionally in Mitral regurgitation; but it is more common in stenosis. I found it in 5 cases or 10%, in all of which a presystolic murmur also existed, and in none of which there was aortic disease. In 3 cases it was heard at the apex and conducted into the

[illegible] on Obstetrical Mutual Murmurs." Proceedings of the Med. Society (1888) p. 44

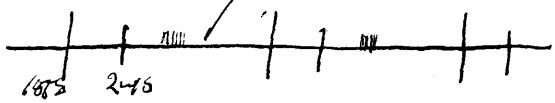
[2] Galbraith; Textbook. p. 134

axilla. In one case heard in the axilla (not at the apex) and conducted round to the vertebral column. In one case it was heard at the level of the 4th left cartilage.
In 4 it was soft & blowing in character: in one, rough.
In 2 cases a systolic murmur developed, while the patient was under observation; and the early diastolic became lost; while in another, a systolic murmur was present from the beginning and both systolic and early diastolic were heard in the axilla:—



Mid-diastolic murmur

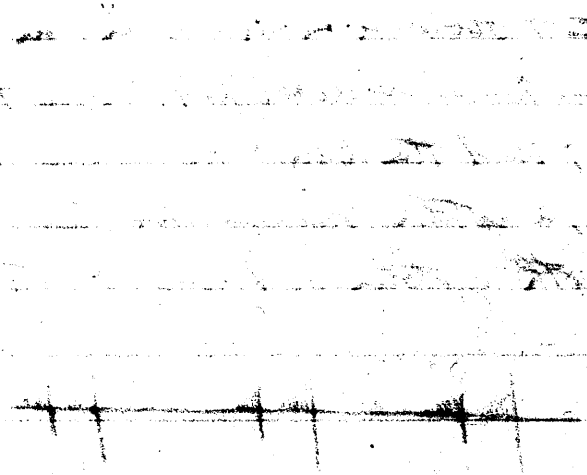
A murmur called "Mid-diastolic" has been described by Dr Bristowe[1]. "It is isolated in the diastolic period and nears the 2nd than the 1st"



He thinks it is often Mistaken for a reduplicated 2nd S; but, he says, it is longer than this and independent of reduplication at the base. I have searched for it but never found it.
No doubt all these murmurs are isolated parts of the long murmur, which sometimes runs from the 2nd S to the 1st.

The murmur of regurgitation

Is there any special character about the murmur of Mitral regurgitation, that we so often find present in Mitral Stenosis?
Dr Norton[2] holds that supposing it does not go past mid-axilla



Broadbent: Internat. Journal of Med. Sc. p. 66–92

but is here replaced by a thumping 1st S., then, even in the absence of a presystolic murmur at the apex, we may diagnose the lesion; for a murmur of pure regurgitation, although it may become fainter as we pass round, yet is not replaced by any of the elements of a 1st sound. The quoted cases in which this diagnosis was confirmed post-mortem, or by the development of a presystolic murmur at that apex. I do not think there is any thing characteristic about this murmur. In 2/3 of the 35 cases in which I heard it, it was conducted round the vertebral column, while in 14 it was not heard beyond the posterior fold of the axilla, and I did not observe that replacing of the murmur by a 1st S. described by Dr Barlow.

These murmurs are very variable and the presystolic is the most erratic of all murmurs. It may vanish for a few beats or for days. It may alternate with a systolic or be present for some time along with it. It often vanishes during illness to return with convalescence or may disappear without apparent cause, nothing in the patient's condition being found to account for its departure.

Dr Broadbent's stages

Dr Broadbent has endeavoured for clinical purposes to divide the course of the disease into 3 stages and this he does by auscultation alone.

1st Stage. There is a presystolic murmur and the 1st S. present at the apex and accentuated & possibly reduplicated at the pulmonary area

In this stage there are practically no cardiac symptoms and even a severe illness or an attack of bronchitis may be passed through without embarrassment to the circulation.

2nd Stage. The 2nd sound is absent at the apex, and the 1st is sharp and clicking, due to the ventricle contracting on insufficient blood. The sound the 2nd is normally heard at the apex to be the aortic, and it disappears because (a) the enlarging right ventricle comes to overlap and push back the left which does not also enlarge and (b) the aorta has less blood and therefore cannot produce much of a 2nd S.
This is the stage of cardiac symptoms associated with embarrassed circulation

3rd Stage. No presystolic murmur at apex, only the loud 1st S, possibly the murmur of tricuspid regurgitation. He thinks that the establishment of tricuspid regurgitation so lowers the pressure in the pulmonary circulation that it cannot cause a murmur, even when reinforced by the auricular contraction. I do not think that this disease lends itself kindly to division into stages. And as Dr Broadbent admits the murmur may come back under treatment, thus shifting the patient into stage 2nd. Then again the 2nd S, which at first was not heard at the apex, may be discovered on a subsequent examination. Also the 1st S. is not always present for in one of my cases a rough murmur at the apex changed into a soft blowing one and a few days later the murmurs were replaced by a sharp 1st S.

Compare Dr. Allan's "Clinical Lecture on Metrit & Stenosis" Lancet 1889 Vol ii p. 180

Balfour; Text book p. 128.

I should be inclined to estimate the patient's condition, not by auscultation alone, but by an observation, for some time, of his general symptoms, - by his tendency to wasting - to easily raised dyspnoea - to pulmonary troubles - to oedema - and all the symptoms & signs of a failure of the right side of the heart.

Explanation of the Phenomena

What is the Explanation of the Altered Heart Sounds and the Murmurs?

Sharp 1st Sound

The sharpness of the 1st S may be due to the fact that the Ventricle contracts when not quite filled with Blood, and the contraction of the muscular substance causes the sound. We have a similar sharpness of sound in palpitation. Also may be in part due to the closure of the tricuspid and when, it can close, to that also of the mitral valve. And it is possible, as Dr Balfour suggests, that the slight thump with the 1st S, is the tail-end of the Murmur.

Accentuated 2nd S.

The accentuation and reduplication of the 2nd S, we have seen, to be due to increased tension in the pulmonary circulation.

The 1st Murmur

The majority of observers have followed Dr Gairdner's theory that the murmur of Mitral Stenosis is a direct one. The blood, passing through a contracted orifice, from a cavity where the pressure is high into one where it is much lower, gets thrown into the condition of a fluid Vein; and the Main Cause of the pressure being sufficiently raised to produce this fluid Vein is the contracting auricle, which to

1 Galabin: Guys Hosp. Reports 1845 p. 284

2 Rolleston: "On the Causation of Mitral Bruit." St Bartholomew's Hosp. Reports 1868 p. 199

3 p. 200

overcome the obstruction begins its systole earlier and acts more powerfully. (We often find its hypertrophied post mortem) And Dr Galabin, in the paper already quoted, has proved that this is so, for in his cardiographic tracings of this disease, the rise which is due to the contraction of the auricle, which is usually situated just before that due to the contraction of the Ventricle, is here much greater and occurs earlier than usual. He has also shown, that in those cases, in which there is a slight pause between the murmur and the 1st S., the rise in the tracing due to the auricular contraction falls just before that due to the Ventricular systole.

I think that this is the cause of the rough murmur, in spite of Dr Roberton's objection that it is contrary to all physiological experience to have the auricle contracting earlier than usual and to have an appreciable interval between the contraction of the auricle & Ventricle. We have seen that a presystolic murmur may become diastolic, even under the stethoscope, and still preserve its characteristic roughness. But besides the contraction of the auricle we have in the elastic dilatation of the Ventricle a most important factor in the causation of that difference, of pressure in the two cavities, which leads to the production of a fluid vein. Dr Roberton in his paper quotes the experiments of Goltz and Gaule, by which the negative pressure in the left Ventricle of a dog

[1] Galabin; Guy's Hosp. Reports 1875 p. 290

[2] Giacchard; Lancet 1889 Vol II p. 150

was estimated by the manometer at −50 m.m., while the positive pressure was 20 m.m. in the auricle at contraction. The left Ventricle by its elastic dilatation sucks the blood through the narrow orifice from the cavity where the pressure is high into that in which it is negative. As the negative pressure is greatest at the beginning of diastole, this explains the early diastolic murmur and the earlier part of a long presystolic murmur, which occupies the interval from 2nd to 1st sound. While the increase in sound, heard at a particular part, is due to the contracting auricle adding its share of pressure. Dr Colabin[1] found nothing in the tracing to indicate early auricular contraction in the case of the early diastolic murmur.

The early diastolic murmur

Dr Acland[2] puts much stress on the dilating power of the left Ventricle. In debility, the disappearance of the murmur may be explained by the fact that the contracting power of the Ventricle is less and therefore its elastic dilatation less; so it follows, that the difference of pressure is less between the two cavities, and then the addition of pressure in the auricle, due to its contraction, fails to produce the fluid vibration.

Disappearance of the murmur in debility

With regard to the Hardness of the murmur, it is important to remember that hardness has no relation to force. All that is required is that the blood should be thrown into the state of vibration sufficient to produce the sound. It is not necessary that there should be any great hypertrophy

Hardness

Goodhart; Guy's Hosp. Reports 1878 p. 257

Dickinson; Lancet 1884 Vol ii p 657

of the auricle and a rigid obstruction at the mitral orifice, although one almost instinctively thinks of this. Dr Goodhart records a case in which he heard a murmur, presystolic in rhythm "short, rough and almost grating." And, at the autopsy, the mitral orifice was nearly blocked by large soft, greyish, fungating vegetations. It was a case of ulcerative endocarditis and there was no evidence of any chronic thickening of the flaps and its affection of the chordae tendineae

Localisation

Localisation of the murmur and thrill at the apex is due, no doubt, to the fact that the particles of blood, thrown into a state of vibration, come directly against that part of the ventricle, which impinges on the chest wall.

Length

The length of the murmur depends generally on the difference of pressure in the two cavities and, therefore, partly on the amount of stenosis, and partly on the elastic dilatation of the ventricle and early contraction of the auricle.

The Auricular Theory

I do not intend to criticise the position of the observers who hold that the characteristic murmur is regurgitant. Dr Dickinson, in an ingenious paper on "The Presystolic murmur falsely so-called," gives the latest contribution to that side. The ventricular systole is held to begin early, before the 1st S. Dr Acland & Dr Bowditch in the papers already quoted have taken up the various points. This one fact apart from all others is conclusive. The negative pressure being so great in the ventricle at the beginning

Delivered before the Med. Society. Reported in Lancet 1899 Dec 23rd.

of diastole, the early diastolic murmurs are admitted as direct. Now in the case of a long murmur which lasts from 2nd to 1st S, we must suppose this to be all direct too. Otherwise the long murmur changes in mid-career from diastolic to Ventricular systolic, or else the Ventricle contracting obliterates the normal diastole.

Prognosis

It is difficult in a chronic disease like this to keep many patients for a long period under observation. The London poor change their abodes so often and, as symptoms arise, drift from one charitable institution to another, as it suits their convenience or as they hope to obtain relief. They get into the records of several hospitals; some of my cases had been lectured on at different Medical schools and they were acquainted with the name of the disease. Then, again, we must remember their conditions and surroundings at home. Sir Andrew Clarke in a Paper on "The Prognosis of Mitral Regurgitation" says that a man with a Large hospital, but not a large private practice is very apt to give a more unfavourable prognosis. This applies with still greater force to Obstructive Mitral disease. What is the outlook for a patient with Mitral stenosis and how does it compare with that of a patient with Mitral incompetence? The outlook is ultimately a gloomy one and, although we cannot lay down the precise amount

Broadbent: Intrumt. Journal of Med. &c. 1886 p 44

of obstruction that would be an equivalent evil to a given amount of regurgitation, I think that obstruction is the more serious lesion of the two; and, the more I have seen of the disease, the more unfavourable my view of it.
We know that it is mainly a disease of youth — the time for vigorous tissue change and power of adaptation; yet this fact is ominous — we do not find it in the old. Only 10 of my cases were over 30. The oldest was 48. Cardiac symptoms had been present for varying lengths of time — 2 years to 21 years. Some of them were seriously ill, one dying at 31.
Now in contrast with this we have often found old people with mitral regurgitant disease of long duration. I had a patient lately who died at 81 with general oedema and the signs of mitral regurgitation. She had a history of cardiac symptoms for over 20 years, and had had 5 attacks of Rheumatic Fever at different periods of her life. I have 2 others under observation at present — aet 69 & 66. The former with a history of Rheumatic Fever in childhood, has had cardiac symptoms all her life, for which she had to be treated in St Thomas' Hosp. at 40, when she was informed that she had Heart Disease. The other had Rheumatic Fever at 14 and was then told she had Heart Disease.
Dr Broadbent found the average age of death in 53 cases of Mitral Stenosis to be 33 for Males and 34 for females and mentions 2 cases who died at 66 and 68.

Lettsomian Lectures / 89

Broadbent: Internat. Journal of Med. & 1886 / 88

Dr Dawson gives the average age in 19 cases as 35. I know of 6 deaths in my 50 cases, but there are probably more. The average age happened to be low viz 18. All of them had pulmonary complications and 4 had pericardial friction.

Why should we expect the prognosis to be serious? Because here the lesion has more tendency to increase and therefore the compensation to fail. The endocarditis, whether beginning acutely or chronic from the first, has caused a certain amount of adhesion between the curtains of the valve, with the formation in time of a ring of connective or fibrous tissue. It is the property of this tissue to contract and, even although all inflammation have subsided, the contraction will go slowly on, for months or years. — encroaching on the blood channel. We see the same process at work in the formation of a stricture of the urethra or oesophagus. Again as Broadbent points out, a chronic inflammation may be kept up by the friction and strain which results from the narrowing of the orifice. Other attacks of endocarditis will tend to hasten the process, and therefore a history of recent rheumatism or the presence of rheumatic pain or a rheumatic family history make the prognosis more serious. It is in young people too that we are apt to find Rheumatism.

Then again, if we have reason to think the contraction

1 Balfour Tait book f. 149

2 " " " 150

3 " " " 147

has begun in early life, that would make us give a more unfavourable prognosis. Dr Balfour has pointed out that if the contraction begin during the development of the body, the ventricle has a smaller wave of blood to send forward and, consequently, the aorta is ill-developed and in a condition of "hypoplasia". All the arterial system suffers in a like manner and, therefore, the nutrition of the body is below par. This accounts for the defective development, we so often notice in this disease. This hypoplastic condition of the aorta & arterial system will according to Dr Balfour[2] constitute a second obstruction to the blood stream and render still more difficult the rehabilitation of the right ventricle should it become embarrassed.

Given a stenotic orifice, the natural course of events would be an accumulation of blood pressure behind it, reaching in course of time back to the veins.[3a] It is to this slow transference of the blood pressure from the arteries to the veins, accompanied by an equally gradual impairment of nutrition that death from asthenia, accompanied and indicated by collections of serum in the cavities of the body, is ultimately due, even in the most favourable cases. This hardly ever happens and death is usually precipitated by an accident.

The prospects of the patient practically depend on the right ventricle, already working at its utmost. We have seen

that a comparatively trivial cause, such as getting a chill and developing bronchitis, may increase the pressure in it, at the same time lowering its tone, so that it fail to meet the demands and dilates, giving rise to cardiac symptoms. These patients, we have seen, are peculiarly liable to pulmonary troubles, and, although they may make good recoveries, yet each attack damages the heart fibre and renders more difficult for future exigencies the preservation of equilibrium. The debilitation and surroundings of the patient, as rendering him more liable to pulmonary troubles has thus an important bearing on the prognosis.

Emaciation and rapid wasting are bad signs, as showing that the nutrition suffers.

Pericarditis is a most serious complication, not only from the possibility of adhesions, but also that degenerative changes may be occurring in the heart substance, due to the formation of connective tissue. I heard friction in 16 cases.

Mitral regurgitation, as we have seen, is very common unless it be slight and the left Ventricle dilating it does not make the prognosis more serious. Aortic regurgitation or Aortic obstruction and regurgitation more than doubles the gravity of the outlook, being both a more serious disease. Tricuspid regurgitation shows that the Right Ventricle is now leaking and is to that extent a bad sign.

' Broadbent. Internat. Journal of Med Dec 1886 p. 81

Even when we are called to see a patient seriously ill and labouring for breath, with all the signs of failure of the right side of his heart — his lungs full of râle & wheeze and his feet swollen, it is often astonishing how soon, after a little rest and treatment, he may be going about again. So the immediate prognosis need not be very bad, especially if it be a first attack. But the tendency will remain. And at length there will come one attack that he cannot battle through. Practically we find death threatens from the lungs.

Treatment

Knowing the strong tendency to pulmonary complications and the dangers they bring, it is our duty on discovering the disease to sound a warning note; so that the patient may take every precaution against catching cold. Exercise in moderation and nutritious diet are indicated and so far as possible avoidance of worry and of occupation that throw a strain upon the heart.

General tonics may be given, if required, and one of the best of these is arsenic from its effect on cardiac nutrition and innervation.

Cardiac symptoms, when they arise, must be treated; and here, as in other forms of heart disease, digitalis is our sheet anchor. Dr Broadbent recommends that it be carefully

watched as it has a special tendency in this disease to cause irregularity of cardiac action. But, so far as I have seen, it is here as reliable as in regurgitation. If the right ventricle be getting distended and unable to cope with the amount of blood, we may endeavour to relieve the engorged lungs by dry or wet cupping or by leeches. But practically the treatment of obstruction is the same, as for incompetence at the mitral orifice.

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