Mitral stenosis : a clinical lecture, delivered at the Hospital for Consumption and Diseases of the Chest, 1889 / by T.D. Acland.

With the Author's Compliments.

MITRAL STENOSIS.

A CLINICAL LECTURE.

DELIVERED AT THE HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST, 1889.

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MITRAL STENOSIS,

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THE history of the discussions which have arisen regarding the symptoms produced by stenosis of the mitral orifice have been so well told by Hilton Fagge,[1] that only a very brief outline of questions raised or solved previously to 1870 will be given in this lecture. Subsequent additions on various disputed points will alone be considered in detail.

It is now forty-six years since M. Fauvel[2] described the characters of a murmur heard in cases of mitral stenosis. He seems to have clearly recognised an important clinical fact, which was then little appreciated, and barely if at all understood. The name had previously been devised by M. Gendrin,[3] but he did not throw any light upon the diagnosis of the disease. Long before this the lesion and its symptoms had been recognised, as is shown by the following description taken from Dr. Forbes' translation of Laennec's "Diseases of the Chest," 1829, p. 648. "The hand applied to the region of the heart feels the pulsation strongly, and accompanied by the purring vibration. The vibratory sensation is not continuous, but returns at regular intervals. The stethoscope gives the following results: contraction of the auricle extremely prolonged, accompanied by a dull but strong sound exactly like that produced by a file on wood. The sound is attended by

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[1] On the Murmurs attendant on Mitral Contraction, by C. Hilton Fagge, M.D. Guy's Hospital Reports, 1871, p. 247.
[2] Archives Générales de Médecine, 1843, vol. i., p. 1.
[3] Leçons sur les Maladies du Cœur, 1841.

a vibration sensible to the ear, which is evidently the same as that felt by the hand. *Succeeding* this, a louder sound and a shock synchronous with the pulse point out the contraction of the ventricle.....From these signs ossification of the mitral valve is confidently made."

In 1854 Dr. Stokes, in his work on Diseases of the Heart and Aorta,[1] puts the question whether the state of our knowledge of the signs of cardiac disease, and of vital acoustics in general, justify us in making an absolutely positive diagnosis, not only of the seat of the murmur, but also of the nature of the disease and of the calibre of the orifice. This question, he says, must be answered in the negative; and he continues: "We must receive as unproved and calculated to throw discredit on the science of diagnosis all those rules and descriptions of special phenomena supposed to apply not only to almost every pathological change of the valves, but every possible combination of those changes. In ordinary cases of mitral murmur we cannot say whether the murmur is constrictive or regurgitant, or constrictive *and* regurgitant, and we must reject a large proportion of descriptions of phenomena which, although the changes they are supposed to indicate are familiar to anatomists, are themselves of doubtful value. To the inexperienced, the detailed descriptions of such phenomena as the intensification of the sounds of the pulmonary valves, of constrictive murmurs as distinguished from non-constrictive, of association of different murmurs at the opposite sides of the heart, of pre-systolic and post-systolic, pre-diastolic and post-diastolic murmurs, act injuriously: first, by conveying the idea that the separate existence of these phenomena is certain, and that their diagnostic value is certain; and, second, by diverting the attention from the great object, which, it cannot be too often repeated, is to ascertain if the murmur proceeds from an organic cause; and, again, to determine the vital and physical state of the cavities of the heart." To add weight to these arguments a passage of Dr. Graves'[2] is quoted, in which that great clinical teacher says: "As at each motion of the heart valves are opened and valves are closed, a morbid change of sound may be produced by any change of structure which permanently prevents the complete opening or shutting of the valves; and consequently the

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[1] Philadelphia, 1854.

[2] Clinical Medicine, p. 922.

sound may arise either from changes of structure obstructing the advancing blood, or from changes permitting regurgitation; in other words, it is impossible to judge at the moment a sound occurs which of these is the cause." Since the time at which this was written, there has been a running fight between those who believed in the possibility of diagnosing with certainty the existence of mitral stenosis by the occurrence of the murmur called by Dr. Gairdner[1] auricular-systolic, but by most observers pre-systolic, and those who, with Dr. Barclay,[2] believed that the murmur characteristic of mitral stenosis was post-systolic, and therefore regurgitant.

The object of my present lecture is to show cases of mitral disease in which there is reason to believe that an accurate diagnosis *can* be made; and it is proposed to lay before you such grounds as may seem to justify the belief that in the great majority of cases it is possible to say with certainty during life what is the condition of the mitral orifice.

Mitral stenosis is more common in women than in men. This fact is well illustrated by the presence here to-day (out of twenty-five cases taken indiscriminately) of at least four times as many women as men suffering from the disease.

Causation.— It may occur as a congenital affection, or may be acquired as the result of chronic inflammation in connection with rheumatism, or with calcareous or atheromatous changes. It is often, however, not possible to trace any history of an acute onset. Two cases are shown to-day in which, though there is no history of articular rheumatism, the patient has suffered from chorea; and two *post-mortem* specimens in which the same record is given have been lent me by Dr. Kidd. (*a*) The obstruction may be due to cohesion of the edges of the valves, forming a funnel-like aperture projecting into the ventricle, and having at its apex a narrow slit; (*b*) a mass of vegetations may help to block the way, as shown in one specimen; (*c*) there may be general thickening and puckering of the valves and of the valvular orifice, with thickening and coalescence of the chordæ tendineæ. The size of the

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[1] A short account of Cardiac Murmurs. Edinburgh Journal, 1861, vol. vii., part i., p. 438.

[2] *The Lancet*, vol. i. 1872, p. 283. Turner, St. Thomas's Hospital Reports, 1876. Dr. W. H. Dickinson, *The Lancet*, 1888.

aperture varies from that which will barely admit the tip of the little finger up to that which admits three fingers—*i.e.*, the normal aperture.

Results.—The results of mitral stenosis may be so slight as not to attract attention, or may be very grave. When present they may be grouped under the following heads :—

I. *Abnormal symptoms.*

(*a*) *General.*—1. Palpitation. 2. Dyspnœa, without signs of pulmonary disease. 3. Pain.

(*b*) *Symptoms referable to condition of systemic circulation and due to disease of the left side of the heart.*—1. Irregularity. 2. Rapidity. 3. Feebleness and lessened volume of the pulse. 4. Syncope.

(*c*) *Symptoms referable to the condition of the pulmonic circulation and disease of the right side of the heart.*—Especially—1. Tricuspid regurgitation and pulsation of veins in the neck. 2. Congestion of pulmonary vessels, leading to—(*a*) red induration and infarcts in the lungs ; (*β*) hæmoptysis ; (*γ*) enlargement of liver and general venous engorgement of organs and tissues ; (*δ*) dropsy.

(*d*) *Symptoms referable to obstruction of systemic vessels by emboli.*—1. Hemiplegia from obstruction of cerebral vessels. 2. Gangrene from obstruction of peripheral vessels.

II. *Abnormal pathological conditions.* Of these the most noticeable are hypertrophy and dilatation of the left auricle,[1] in connection with which a thrombus is often found in the auricular appendix. In consequence of the backward pressure, the walls of the pulmonary veins are often thickened and the pulmonary capillaries are varicose. While the right side of the heart is dilated and hypertrophied, the left side frequently does not undergo corresponding enlargement. There are, however, many cases in which there is hypertrophy of the left ventricle, though it is seldom great, except in those cases in which aortic or mitral regurgitation is associated with mitral stenosis. It should be noted that in mitral stenosis the hypertrophy and dilatation of the left auricle is generally greater, but

[1] I am indebted to Mr. Taylor for a very remarkable specimen of this condition, taken from a case under the care of Dr. Roberts. It shows the left auricle so greatly distended that it has exerted pressure backwards on the left bronchus, producing flattening and narrowing of that tube and collapse of a considerable area of the left lung. There are two similar specimens in the Guy's Hospital museum.

that of the left ventricle is generally less, than in uncomplicated cases of mitral incompetence. This fact has an important bearing upon the question as to whether the murmur which occurs in stenosis is direct—*i.e.*, obstructive or regurgitant. If the murmur be due to regurgitation of blood from the ventricle into the auricle, it would be reasonable to expect that conditions similar in kind, if not in extent, would be found in regurgitation, both with and without stenosis of the valvular orifice; this is not, however, the case, as has been said above. Again, in cases of mitral obstruction, in which the auricle might be supposed to have extra active work thrown on it, hypertrophy is more marked than in simple regurgitation. In the latter cases great dilatation of the auricle is frequently found. It is a generally recognised fact that a very effective cause in producing dilatation of the cavities of the heart is undue distension during diastole, and that if nutrition is well maintained, obstruction during systole tends rather to cause hypertrophy. In simple mitral regurgitation there is no obstacle to the backward pressure on the left auricle, which is consequently often found considerably dilated. In mitral stenosis, on the other hand, where there is an obstacle both to the forward and the backward flow of blood, there is more tendency to hypertrophy, the active work of the auricle being in such cases increased, whilst owing to the constriction of the passage, back pressure during ventricular systole is necessarily diminished. The secondary complications are even more marked than in mitral regurgitation, and the whole course of this disorder is often attended by more grave symptoms. This may be due to the fact that hypertrophy of the left ventricle is less common in cases of mitral stenosis than in those of mitral regurgitation, and that the compensation for the defective valve is therefore less perfect.

Physical signs.—The physical signs due to constriction of the mitral orifice are often complicated by those due to regurgitation, but in so far as they can be separated, they are: (*a*) Alterations in the area of cardiac dulness. In some cases the apex beat is felt in the sixth or seventh intercostal space, in the nipple line; but it is more usual to find a diffused undulating impulse spread over an increased area in both directions. (*b*) The apex beat is nearly always feeble—often, indeed, imperceptible. (*c*) When the murmur is present, a rough purring thrill is generally felt over

and localised to the region of the left ventricle. This is felt distinctly to precede the maximum apex beat.

Auscultation.—On listening over a limited area just inside the apex beat, a long rough murmur in most typical cases is heard, which precedes and runs up to a sound which is synchronous with, and terminates abruptly at, the moment at which the maximum apex beat is felt. Whatever be the mechanism of this murmur, it is admitted on all hands to have certain characteristics. (*a*) The area over which it is heard is often, even generally, limited to a space some inch or so around the apex beat. It is for the most part not conducted in any direction, though there are exceptions in which it is conducted as far as the posterior border of the left scapula. (*b*) The rhythm of the cardiac sounds is suggestive even where the murmur is, as it often is, temporarily absent. The first sound of the heart seems to hesitate, and then jump, and terminates very sharply with a loud snap, which is synchronous with the maximum of the apical impulse, and precedes the carotid pulse by an instant. It is neither reasonable nor desirable to time the murmur by the radial pulse, as is often done. This must infallibly lead to errors in estimating the time of the various phases of the cardiac cycle, since the radial pulse occurs after the apex beat by at least one-sixth of a second. (*c*) The murmur is very loud and rough, and, more than any other cardiac bruit, is liable to rapid alterations in its character and loudness. Notable instances of this are not infrequent. Two have come under my own care, in both of which the murmur varied so much as to be at times inaudible, and at others so loud as to make it seem incredible that but a moment before it could not be heard. In one of these cases, when first seen, the murmur could be heard dying away as the heart's action came to rest after exertion. It is therefore important, in all cases where mitral stenosis is suspected, to examine the patient both standing up and lying down, at rest and after exertion. If this is done, in many cases in which the sounds are at first obscure the murmur characteristic of mitral stenosis will be produced. (*d*) Accompanying the murmur, and produced by the same cause, is a strong purring thrill, which is more marked than in any other form of cardiac disease. (*e*) The action of the heart is (owing to defective nutrition rather than dilatation of ventricle) extremely liable to become rapid, feeble, and irregular, such conditions being often marked by a rise

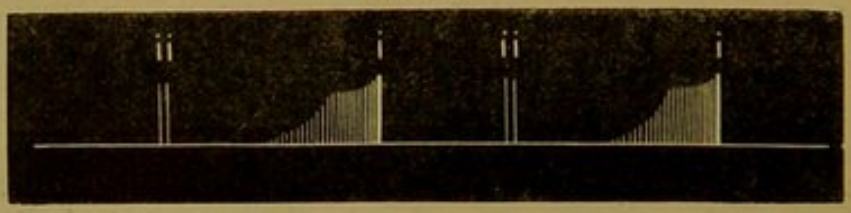
of temperature. (*f*) Owing to the engorgement of the pulmonary vessels, to which there is always a tendency in mitral stenosis, there is a rise of blood pressure in them, and consequently sharp premature closure of the pulmonary valves. This causes a reduplication of the second sound, best heard over the third left costal cartilage, which is due to the want of synchronism in the closure of the aortic and pulmonary valves. (*g*) If a systolic murmur is present, there is often a distinct interval between it and the murmur due to mitral stenosis; and if there is a murmur at all, the longer the murmur the greater probably is the stenosis.

A murmur having such characteristics is universally admitted to be diagnostic of mitral stenosis. It has received much attention from the fact that some observers have insisted, with a skill and persistence which have provoked much discussion, that it is merely a systolic regurgitant murmur, with peculiarities depending on the nature of the orifice through which the blood stream flows. On the one hand, there are those who, naming it presystolic or auricular-systolic, maintain that it is produced by the flow of blood from the auricle into the ventricle through a constricted orifice; and, on the other, those who, mistrusting as it seems their senses, highly trained and acutely observant though they be, have used all their ingenuity in finding out reasons why this murmur should *not* be what it seems to be—a *direct* murmur, due to the obstruction of the orifice—and who insist that it is a sound produced by regurgitation through the orifice owing to the insufficiency of the valves.

There can be little doubt that much of the discussion which has centred round the presystolic murmur, truly or falsely so-called, has arisen from a want of appreciation of the fact that no murmur is liable to greater variations, and that no cardiac lesion gives such protean auscultatory sounds under different conditions. These variations may occur not only in different stages of the same affection, but under different conditions during the same stage. Dr. Broadbent, in his admirable paper on Mitral Stenosis in the *International Journal of Medical Science* (January, 1886), has described three stages, the physical signs of which are not only diagnostic of the condition of the heart in each period of the disease, but which may also under certain circumstances be taken to give evidence of the

physiological conditions under which the heart is acting. These stages are: (*a*) That in which the murmur is loud and ends moderately sharply. Here the thrill is distinct, and precedes the apex beat; the second sound is audible and reduplicated, so that the heart sounds have a galloping rhythm—*root-tata*. (Fig. 1.) During this period there is, as

FIG. 1.



a rule, very little embarrassment of the circulation. (*b*) In the succeeding stage the second sound becomes almost inaudible at the apex. This is due mainly to two facts: (1) that the blood-supply to the aorta is lessened, and hence the rebound which closes the valves is feeble; (2) that the right side of the heart is becoming dilated and hypertrophied, and is therefore coming more to the front, displacing the left ventricle which conducts the aortic sounds. The first sound is very sharp, and resembles a snap more than anything else, and inasmuch as the second sound is feeble, the snap is often taken for the second sound. (*c*) In the third stage the murmur is absent, the second sound is absent, and often all that is heard is a sharp clicking first sound. The action of the heart is often irregular in the extreme.[1] The murmur frequently returns when the patient is placed under suitable conditions of rest and treatment (notably digitalis), and this is a favourable sign, as showing increased cardiac power.

In all these conditions (which I hope to be able to show you to-day) it is evident that there are many sources of error, because, though each one of them is marked by certain definite signs, it is clear that no one description can possibly include them all. When it is remembered that

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[1] Two murmurs during this ventriculo-diastolic period not having the characteristics of the presystolic murmur are described as occurring in mitral stenosis; they are called by Dr. Bristowe *early* and *mid* diastolic murmurs. I have not been able to satisfy myself that they are diagnostic of the affection under consideration. ("On Obstructive Mitral Murmurs," by J. S. Bristowe, M.D., F.R.S., Med. Soc. Proc., vol. xi., 1888.)

any one of these can be, and often is, complicated by mitral regurgitation or disease of the other valves—so that, for instance, an extreme case of mitral stenosis might be marked only by a sharp clicking first sound, no murmur, and no second sound, or on the other hand, by a "systolic" murmur, without presystolic murmur or second sound—it is not to be wondered at that assaults have been made on the very existence of the direct murmur, *i.e*, one caused by the flow of blood from auricle to ventricle, call it what you will, diastolic, presystolic, or auricular-systolic.

A case remarkably illustrative of this condition is now attending as an out-patient (C. D——, No. 8,297), in whom the cardiac rhythm is so irregular and the heart's action so feeble, that, though the heart beat is 152, the radial pulse is only 84. While standing up nothing can be heard over the apex beat except a succession of short blowing whiffs, with no second sound. When lying down, every now and then as the heart gathers strength there is a pause, a hesitation, and then a long rolling presystolic murmur ending with a snap.

FIG. 2.

Since the time when the diagnosis of mitral constriction became possible, various observers have been active in denying the existence of a direct murmur as distinguished from a regurgitant. Of these the foremost was Dr. Barclay, and he has lately been followed by MacVail, Dickinson, Turner, and others, who have all endeavoured to prove that this so-called direct murmur is, in fact, regurgitant. The main arguments on which this view rests are:—

I. That inasmuch as the pulmonary veins have no valves and no means of closing the outlet backwards, the left auricle could exert but little power in driving the blood forwards from left auricle to ventricle, and that therefore it is scarcely possible that one of the loudest and roughest murmurs heard in cardiac disease could be produced by the contraction of the auricle.[1] To this it may be replied

[1] Barclay, *The Lancet*, vol. i., 1872, p. 283.

(*a*) that the loudness of a murmur does not by any means bear a constant ratio to the force producing it, but depends on the method of its production and the adaptability of the instrument to produce particular forms of vibration. This requires little illustration : a familiar example will suffice. Turn an ordinary spring water-tap full on, nothing is heard but the rush of water through an orifice ; turn it on so that a minute stream only is allowed to pass, vibrations are often produced sufficient to shake the house. Here the same force precisely is at play ; the conditions for producing sonorous vibrations only are altered. A further illustration of this point may be drawn from a very remarkable case of aortic regurgitation, due originally, as I am inclined to think, to the rupture of a valve, which has lately been under my care. Aortic regurgitant murmurs are often softer than a whispered "who" ; but in this case B flat was sounded so loudly and clearly that the patient himself could hear it. Dr. Fowler has recently shown me a similar case in which the murmur could be heard more than three feet from the patient. In both cases there is a well-marked thrill perceptible over the base of the heart. The objection, therefore, raised on this point cannot be admitted ; and it may be granted that the mere loudness of the murmur is no argument against it being caused by a feeble force. (*b*) The auricle is spoken of as though, even when backed by competent pulmonary valves and the tension of the whole pulmonary circulation, it were a feeble thing. It must, however, be remembered that even if the murmur were *regurgitant*, the pressure backwards which has to be met before the aortic valves will open must be equal to the pressure in the systemic circulation. But an attempt will be made to prove that the force producing the presystolic murmur is not a feeble one, and that all the conditions are present which would tend to produce a loud, rough, "churning" murmur, such as that which is so often associated with mitral stenosis. The force available for producing a direct murmur is not, as stated by Barclay, only that of a contracting auricle ; since a powerful factor in the mechanism of circulation is no doubt the expansile force of the ventricles. It is my belief that the whole secret of the characteristics of the murmur in mitral stenosis depends upon this force, which is, indeed, very considerable. It has been estimated by Goltz and Gaule[1] in the dog at 52 mm. of mercury

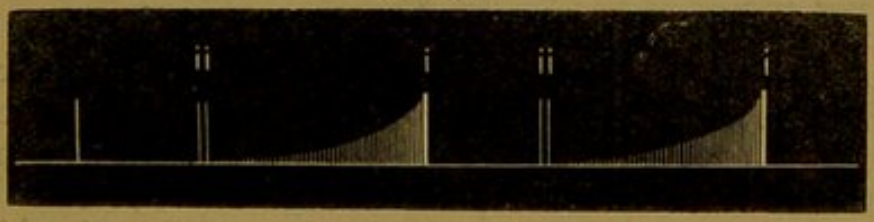
[1] H. Rolleston : The Endocardial Pressure Curve. *Journal of Physiology*, vol. viii., No. 5, p. 281.

when the thorax is closed, and 23 mm. when opened. The significance of this is enormously increased when it is considered that the positive pressure of the right ventricle is only 60 mm., so that, in fact, the suction power of the left ventricle, supposing the entrance to it is obstructed, is almost equal to the contractile power of the right ventricle.[1] "When the entrance is obstructed" is said advisedly, for the above measurements are taken from the healthy heart, and under conditions where the potential vacuum is immediately occupied by the in-pouring blood; and therefore, except in theory, it does not exist. A similar view of the mechanism of the early and mid-diastolic murmurs is given by H. Rolleston,[2] who summarises his conclusions by saying that while the presystolic is due to a *vis a tergo* of the auricular systole, the early and mid-diastolic murmurs result from the

FIG. 3*a*.

vis a fronte of the expanding left ventricle and its suction-pump action. As far as it goes this is quite in accord with the opinions expressed above; but it does not go far enough, for early and mid-diastolic murmurs are not diagnostic of mitral stenosis, in fact, are often present when the orifice

FIG. 3*b*.



is even larger than normal. And it should be added that cases are far from infrequent in which the exact rhythm of the murmur depends on the position of the patient. For instance, in K. V—— (Brompton Hospital, 4231), the

[1] Michael Foster's Text-book of Physiology, fourth edition, p. 151.
[2] The Causation of Mitral Diastolic Murmurs, by Humphrey Rolleston, M.B., St. Bartholomew's Hospital Reports, vol. xxiv., p. 197.

rhythm while she was standing up was, diastolic murmur, pause, presystolic murmur (Fig. 3*a*); when lying down, a characteristic rolling presystolic murmur (Fig. 3*b*) alone was audible. So that, although the suction force of the ventricle is in all probability concerned in the production of the murmurs, the murmurs are not themselves characteristic of mitral stenosis. When there is an obstructed mitral orifice the pressure in the pulmonary circulation and in the left auricle is high; consequently, at the commencement of ventricular diastole there is sufficient blood under high tension, aided by the suction power of the ventricle, to force its way through the obstructed orifice and to fill the distending ventricle, until towards the end of diastole. At this point the auricular systole comes into play, the ventricle tends to expand more rapidly than it can be filled, the auricle contracts, and all the conditions necessary for the production of the loud rough murmur are brought into existence. It is recognised that in the mechanism of its production the presystolic murmur differs from all others. The differences seem to be as follows. Not only is the blood flowing through a constricted orifice, but it is flowing into a cavity which has a constant tendency to expand more rapidly than it can be filled. This explanation of the mechanism, which is not inconsistent with any physical or physiological fact, has been overlooked entirely by Dr. Dickinson, who says "that the current from auricle to ventricle is seldom, if ever, strong enough to give a thrill along with a direct mitral murmur; if a thrill accompanies a mitral murmur, I should regard this as a proof that the murmur is due to the action of the ventricle, and is truly systolic and regurgitant."[1]

Some hæmic murmurs, according to the well-known theory, are produced under conditions closely resembling these; only in such cases, the blood is deficient in quantity, and therefore cannot fill up the cavity into which it flows, in consequence of which fluid veins are produced which in their turn give rise to sonorous vibrations. With regard to these undulations, which occur, as a matter of common observation, most forcibly when a fluid is rushing through a narrow orifice into an expanding cavity (cf. filling an ordinary elastic syringe), it should be noted that a thrill very often accompanies the murmurs of aortic regurgitation,

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[1] Dr. W. H. Dickinson, *The Lancet*, vol. ii., 1887, p. 650.

i.e., when the blood is flowing back into a cavity in which, during diastole, there is a constant tendency to diminution of pressure. Again, in thoracic aneurysms, there is often a thrill, and probably from the same cause, viz., the blood rushing under the high pressure of an hypertrophied left heart into a large sac with comparatively slack walls, which give opportunity for the development of large vibrations, comparable in their method of production to the *bruit de diable* of Bouillaud. These observations all tally with the accepted fact of the extreme variability of the presystolic murmur, and of its frequent disappearance when the ventricle is feeble; since, if the explanation here given of the production of the murmur is correct, active dilatation of the ventricle is necessary to allow of sufficient amplitude for the vibrations generated at the constricted orifice to produce audible sounds. And if the ventricle expand feebly or less rapidly than the cavity can be filled, the sounds generated by the vibrations produced at the point of constriction are damped, even to disappearing.

The abrupt first sound, which even without the murmur is sometimes diagnostic, often suggestive of mitral stenosis, is closely allied to the same series of phenomena. It is now generally admitted that the first sound of the heart is due to two distinct causes, viz., the ventricular contraction, and the closure of the auriculo-ventricular valves. If this be so, it is to be expected that any cause which would enable the ventricular wall to commence its contraction with more than ordinary rapidity would (supposing the closure of the valves were entirely out of the question) produce a sharper and more rapid sound than the normal one. Such conditions are found in mitral stenosis; for when there is an obstructed orifice, the dilating ventricle tends to expand more rapidly than it can be filled; the intra-ventricular tension is therefore proportionately diminished. It is, moreover, capable of proof that under increased resistance a muscle contracts more slowly, and inversely, that under lessened resistance it contracts more rapidly, than under normal conditions. It has been shown that in mitral stenosis agencies are at work which tend to diminish intra-ventricular pressure; it follows therefore as a natural result that, apart from other considerations, conditions exist in the heart for producing a short, sharp first sound. That the systolic snap is in part thus produced has been suggested by various observers—Fagge, Mahomed, and Broad-

bent. It may be concisely stated in the words of the latter.[1] "The muscular walls at the first moment of contraction meet with less resistance, and, acting more rapidly, are suddenly brought up and made tense at the moment when resistance is encountered, so producing the sharp first sound....." That this is actually one of the causes of the first sound is in some degree borne out by the fact that in hypertrophy of the left ventricle from aortic regurgitation the first sound becomes long and booming; in dilatation it becomes short and sharp; and in palpitation the brevity of the diastolic interval does not give the ventricle time to fill, and a similar result is effected. With regard to the sharpness of the first sound being in part due to the closure of the auriculo-ventricular valves, it is admitted that the tension in the pulmonary vessels is greatly increased when there is obstruction at the mitral orifice; the tricuspid valves tend therefore to close more sharply than in health. It is not urged that it is impossible for a sharp sound to be produced by the closure of the rigid funnel into which the orifice is often transformed, for in many cases it may be proved after death that the valves have come together with sufficient accuracy to prevent regurgitation, from the fact that their thickened edges are often facetted, and that water injected into the ventricle cannot make its escape. When the valves can come together, the tendency would be entirely for them to close more sharply; since, instead of the curtains floating out from the ventricular walls with plenty of blood behind them to bring them together at the moment of the commencement of systole, they stand out hard and rigid, and, coming together with a shock which is proportionate to the rapidity of ventricular contraction, cause a loud snap, such as may often be produced by suddenly turning off a water tap. If the rapidity of ventricular contraction in uncomplicated mitral stenosis *is* increased, it should be easy to show by the cardiograph that when the first sound is sharp and loud the ventricular contraction is short, and when the first sound is long and booming the ventricular contraction is lengthened. The annexed cardiograms, taken for me by Mr. C. J. Martin, to whose kind help I am much indebted, clearly show that such is the case. The one (Fig. 4) is from E.

[1] Dr. W. H. Broadbent, International Journal of the Medical Sciences, 1886, p. 68.

M——, suffering from mitral stenosis; the other (Fig. 5) from J. W——, suffering from aortic incompetence.

FIG. 4.

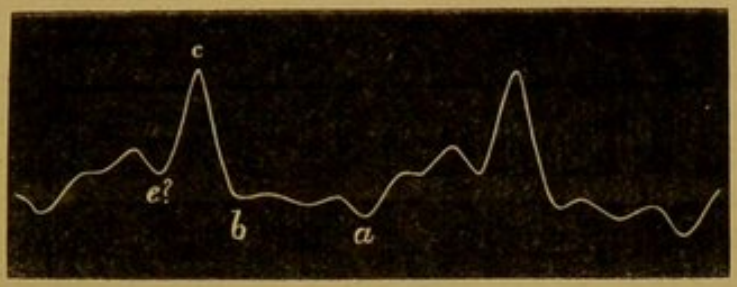
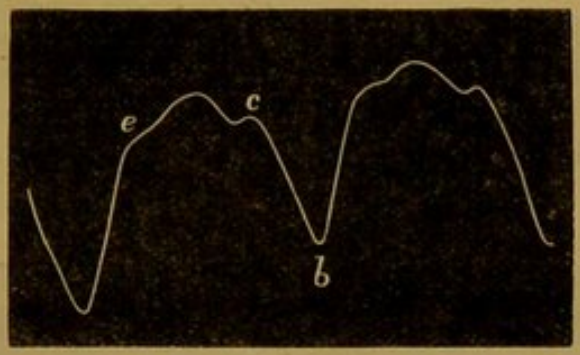


FIG. 5.



a b, Duration of Auricular systole. *b*, *c*, *e*, Duration of Ventricular systole. *e*, End of Ventricular systole.

The various phases which are possible in a case of mitral stenosis make it probable that the view I have expressed as to the part taken by the diastole of the ventricle in the production of the murmur is correct. When the obstruction is slight, the murmur occurs only at the end of diastole and during auricular systole, and the snap is not so loud as in the second phase, where the obstruction is greater, and the tendency to diminished intra-ventricular tension therefore increased. In the last phase the nutrition of the ventricle is much impaired; its suction power is therefore greatly lessened. It is under such circumstances that the murmur is sometimes entirely absent, and the snap very feeble.

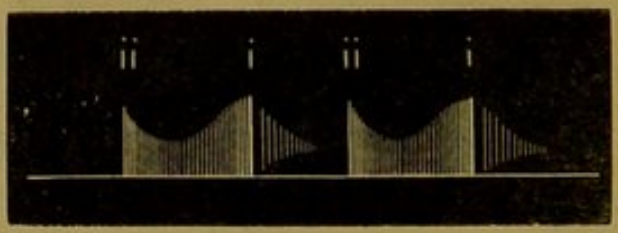
The extreme variability of the murmur is well known; for when the action of the heart is excited, or the ventricular beat becomes stronger under the influence of treatment, a murmur appears which was inaudible before; and conversely, when from any cause the heart's action becomes

feeble, a murmur which had previously been clearly audible disappears. Thus it would seem that the presence or absence of the murmur is directly dependent on the power of the ventricle; and from the facts adduced it would seem probable that it is the suction power of the ventricle, and not its contraction, that gives the peculiar character to the murmur in mitral stenosis.

Thus far on physiological grounds an attempt has been made to prove that there are ample reasons for believing that the murmur in question is produced, at least in a large measure, by the suction action of the ventricle, and that, if this is so, a satisfactory explanation can be given of its roughness, of the thrill, and of the sudden termination of the first sound.

II. The second argument which is brought by those who deny the existence of a presystolic murmur to show the impossibility of this murmur being an obstructive one is, that were it so, when a regurgitant murmur is present as

Fig. 6.



well, there must be an interval between the two, and that if there is no interval between them to mark the change of direction of blood-flow it is impossible that any such change can be taking place. To this the reply is simple, for in the first place there often is an interval between the two murmurs, as in the case of J. F——, in the Brompton Hospital, Victoria ward (Fig. 6), where there existed mitral stenosis and incompetence, and a very loud rolling presystolic murmur, followed after a distinct interval, by a soft, high-pitched, systolic whiff.

Again, if it be true that the sharp snap takes place in consequence of the contraction of the ventricle on the blood, which does not yet entirely fill the ventricular cavity, as well as to closure of valves, it is not remarkable that the direct (presystolic) murmur should only be suddenly terminated by the rise of intra-ventricular pressure closing the valves and putting an end to the flow of blood from the

auricle. That this is a reasonable view may be allowed from the fact, which is acknowledged by all, that there is a tendency for more blood to collect on the right side of the heart than can in the given time gain entrance to the left ventricle. There is also a tendency towards the incomplete filling of the left ventricle, which does not seem to be due to the fact that there is a want of *vis a tergo* to send the blood forward, for the auricle is often much hypertrophied, but that the time requisite for filling the ventricle is too short, and is terminated by some superior force which closes the orifice entirely, and puts an end to the auriculo-systolic phase of the heart's cycle. This force, it seems probable, is the ventricular contraction.

Dr. Dickinson, and those who agree with him, maintain that the sharp sound which terminates the murmur is due to the fact that "the mitral orifice is constricted; that the valve, nevertheless, allows of regurgitation during the first part of the systole, which it is able to close as the systole proceeds, so as to stop regurgitation towards the end of that movement. The mitral valves are often converted into a mere funnel of stiff fibrous tissue; but with the advancing contraction of the ventricle they come together, late, rather than never, and with their closure make the short sharp sound which replaces the normal first sound. The essential features of the so-called presystolic murmur are therefore mitral regurgitation followed by mitral closure. In support of the view I have presented" (Dr. Dickinson's own words are being quoted) "I might adduce much evidence to the effect that, when the so-called presystolic murmur has been heard during life, after death it has been found, first, that the mitral orifice was contracted, and, secondly, that the valve was capable of closure."[1] This latter statement would meet with general acceptance, with the previso only, that if the valves had *not* been capable of closure, a systolic as well as a presystolic murmur would probably have been heard.

These arguments, however, that the presystolic murmur is really systolic, may be rebutted on two grounds—the physiological and the clinical. Taking the latter first, it is difficult to understand how the murmur in dispute can be systolic if it be true that the snap is caused by the closure of the valves in the middle of systole; because, if this were

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[1] Dr. W. H. Dickinson, *The Lancet*, loc. cit.

the case, there should be *no* snap of the valves when there is regurgitation as well as obstruction. But this snap is often clearly present, as in the case of J. S—— (Brompton Hospital, Dr. Roberts), in which there are mitral stenosis and incompetence, and in which the murmur varies very much with position, but is generally a long rolling presystolic murmur, terminated by a snap and followed by a long systolic whiff; and in the case of J. F——, already referred to (see Fig. 6). Neither should the character of the murmur alter, as undoubtedly it does, from a rough purring murmur to a soft whiff, with a well-marked interval between, but should be of the same character all through; whereas, if the explanation of the causation of the murmur and of the snap given above is true, all the physical signs follow almost as a matter of course.

Again, it is admitted that in mitral stenosis the blood-supply to the left ventricle and to the systemic circulation tends to be deficient and the arterial tension inclines to be diminished; but if really the murmur of mitral obstruction is regurgitant, the length of the ventricular systole would be increased, because, under ordinary circumstances, the carotid pulse immediately follows the first sound; but in cases of mitral obstruction the long rolling murmur precedes the termination of the apex beat and the carotid impulse, and its length must therefore be added to the time usually occupied by the ventricular systole. The carotid pulse is evidence that the contracting ventricle has overcome the inertia of the systemic circulation. It is reasonable, therefore, to suppose that when the ventricle is acting at a disadvantage—as, for instance, when there is much regurgitation through the mitral orifice—the commencement of systole would precede the carotid pulse by a longer interval than when the valves are competent. In the former case part of the force of the contracting ventricle must be expended in driving the blood backwards, and if this be the case, the greater the narrowing of the mitral orifice, and the less, therefore, the chance of regurgitation, the less would be the interval between the commencement of systole and the carotid impulse. Let it be granted, however, for the moment that the "presystolic" murmur is caused by regurgitation, and that the snap is due to the closure of the valves after the commencement of systole; the carotid pulse, as a matter of observation, follows the snap, and is therefore delayed long after the commence-

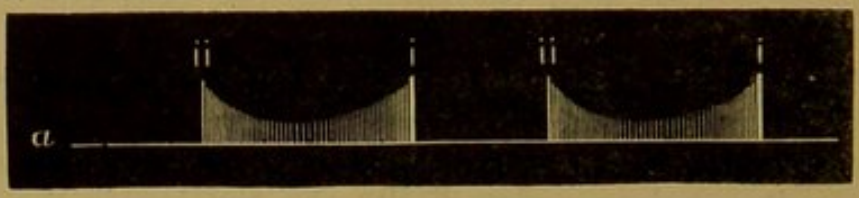
ment of ventricular systole, and takes place only when the margins of the damaged orifice come together. On the other hand, in a case of simple regurgitation, the carotid impulse follows immediately on the commencement of the systole, and although regurgitation is going on all through the systolic period, and the valves do not ever completely close the orifice, the carotid pulse is not delayed appreciably. This fact alone is a powerful argument both against the presystolic murmur being in reality regurgitant, and against the "snap" being other than the commencement of systole.

Again, it is acknowledged that the tension in the right heart is increased, and that the tricuspid valves have an extra strain thrown on them which at times leads to regurgitation. If it were true that the snap was due to the closure of the valves at a period after the true commencement of systole, equal to the length of the presystolic murmur, it ought to follow that the sharp closure of the tricuspid valves should take place at the commencement of the murmur, when, according to this view, ventricular systole begins; and that the closure of the mitral orifice, being delayed by the stiffness of the valves, should follow it, but separated by the length of the murmur. There is no evidence that this does actually occur, but it seems that the right and left ventricular systole are synchronous. From this it follows: (*a*) that the snap is at the commencement of systole, as on other and entirely different grounds has been shown to be probable; (*b*) that the murmur which precedes it is "presystolic."

Supposing the murmur of mitral stenosis to be regurgitant, it follows that, unless the relations of the normal cardiac cycle are very much disturbed, the diastolic interval would be just so much shortened as the systole is lengthened, which put into plain English comes to this: the contracting ventricle, having less to do, takes longer to do it; and the auricle, aided by the expanding ventricle, having more to do and greater difficulties (in the shape of obstruction) to overcome, takes a shorter time, which seems wellnigh incredible. The argument may be reduced *ad absurdum*, for practically the whole diastolic interval is frequently occupied by the murmur due to the obstructed orifice. As may be heard in the case of V. C—— (Fig. 7, (Brompton Hospital, Dr. Roberts), where there is a diastolic ending in a long rolling presystolic murmur. In such cases it follows either that the ventricle is filled during its systole,

which is impossible, or that in spite of the obstruction the auricle can fill the ventricle instantaneously at the commencement of the diastolic period, which is contrary to all that is known of cardiac pathology or physiology.

Fig. 7.

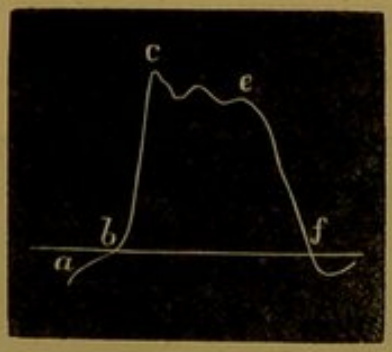


It has been frequently shown by cardiographic tracings taken from cases of mitral stenosis that the diastolic period, instead of being reduced to a minimum and the systole prolonged, the contrary is the case, and that the diastolic period is more prolonged than in ordinary health. The extent to which cardiographic tracings can be used in evidence of the true rhythm of the murmur in mitral obstruction does not as yet make it possible to record with certainty the exact period during the cardiac cycle at which a murmur commences and ends; although recently an elaborate attempt has been made to do so by Dr. Byrom Bramwell and Dr. Milne Murray.[1] The experiment with a piece of cork and a pin made by Dr. Dickinson may be disregarded, owing to the fact that Professors Marey, Galabin, and Landois, all skilled observers, agree that the auricular contraction is represented in the cardiographic tracing; and nearly all cardiograms from cases of mitral stenosis show a rise before the commencement of ventricular systole, as may be seen on the cardiograms (Figs. 4, 10). Rolleston's objection to this, based on endo-ventricular tracings, is not of much weight, since obviously endocardial tracings are not only taken under entirely abnormal conditions, but they are not the tracings of the apical beat, which is of necessity that from which the cardiogram must be taken for clinical purposes. Marey, unfortunately, in his classical work, "La Circulation du Sang," 1881, had not appreciated the murmur which is here designated presystolic, due to the narrowing of the mitral orifice, and he calls the murmur of mitral stenosis "diastolic," and says, in conclusion, it is not known what modifications narrowing of the mitral orifice

[1] Method of Recording the Exact Relation of Cardiac Sounds and Murmurs. *British Medical Journal*, vol. i., 1888, p. 10.

is able to impress on the cardiac pulsation. Galabin [1] sums up the result of his observations by saying that it appears "that the evidence of the cardiograph is in favour of the view that two totally distinct murmurs may be caused by mitral contraction: first, the auricular systolic bruit, which may either run up to the first sound or be separated from it by a short interval; and, secondly, a diastolic bruit due to venous flow through the roughened and narrowed orifice, which in rare cases may be blowing in character and separated from the succeeding systole by a long pause; and that, thirdly, these two may be merged together into a compound murmur, somewhat rough from its commencement, but much intensified in loudness and harshness towards its conclusion." He further adds, as the final sentence of his paper, that he has "not yet met with any cardiograms which seemed at all to indicate that the auricular contraction had been transferred in rhythm to the preceding ventricular systole, and so formed the termination instead of the commencement of each revolution of the heart," as would in fact be necessary, supposing the murmur to occupy the whole diastolic period, and to be caused by auricular contraction alone. Dr. Galabin expressed his belief that almost all cardiograms of mitral stenosis abso-

Fig. 8.



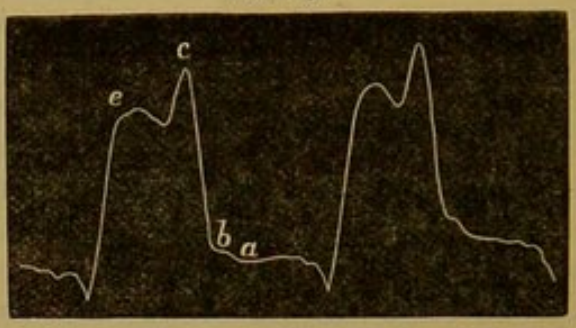
Normal cardiographic tracing, after Marey. (Fredericq: Trace Cardiographique, 1887, p. 84.) *a b*, Auricular systole. *b*, Commencement of ventricular systole. *c e*, Duration of ventricular systole. *f*, Closure of the aortic valves. The letters given in all the tracings have the same significance.

lutely contradict the theory that the murmur and thrill occur during ventricular systole. Marey has shown, by simultaneous tracings, that normal auricular systole ceases

[1] Guy's Hospital Reports, 1875, p. 309.

before that of the ventricle commences; and, in looking at the healthy cardiographic tracing, it is seen that the auricular elevation (Figs. 8 and 9, *a b*) does not lead gradually

FIG. 9.

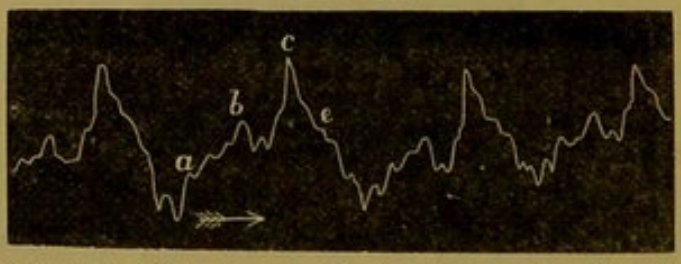


Normal cardiographic tracing from F. O. P., labourer. (Brompton Hospital.) Powerfully acting heart. No evidence of disease.

up to the main ventricular elevation, but is separated from the main up-stroke by a slight fall. Marey has shown that the actual commencement of the apex beat almost exactly coincides with the commencement of ventricular systole, and if the muscular contraction has anything to do with the production of the sound, is synchronous with the commencement of the first sound of the heart.

In cardiographic tracings from cases of mitral stenosis there is seen a long irregular elevation, which is succeeded

FIG. 10.



Cardiogram, from a case of mitral stenosis. (Galabin, Guy's Hospital Reports, 1875, p. 314, Plate iii., Fig. 19.) The fall after *b* shows the interval between a presystolic murmur which occurred before *b* and a systolic murmur which followed the apex beat. "In this case murmur and thrill extended over the whole cardiac revolution except the short interval just preceding the systolic bruit."

by an up-stroke quite sudden and distinct from the first part of the curve, and not shorter than might be expected from

a small ventricle[1] (Figs. 4, 10), and thus the utmost probability is given to the view that the murmur of mitral stenosis occurs during ventricular diastole and auricular systole. It is clearly shown, apart from all theories, that the phase of elevation due to ventricular contraction is not lengthened (Figs. 4, 10), while the diastolic interval (Fig. 4) is in many cases remarkably so.

These facts are entirely in accordance with the views here expressed, for which little claim to originality is made, but which have in some points been amplified—viz.: (*a*) that the murmur of mitral stenosis occurs during the diastolic period; (*b*) that it is not always or solely auricular-systolic; (*c*) that its essential characteristics are due to the fact that the murmur is produced largely by the suction of the ventricle and the passage of blood over a rough surface into an expanding cavity; (*d*) that the snap is not due to the sharp closure of the thickened valves during the systolic period; (*e*) that it is due to the increased rapidity of ventricular contraction, coupled with increased sharpness of closure on the part of the tricuspid, and at times also of the mitral valves.

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[1] Galabin, loc. cit.