THE CAUSATION AND RHYTHM OF THE CRESCENDO MURMUR OF MITRAL STENOSIS.

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DEFINITION.—By the crescendo murmur of mitral stenosis, I mean that abnormal heart sound generally called "presystolic," and less often "auricular systolic," which is practically diagnostic of obstructive narrowing of the auriculo-ventricular valve orifices, especially on the left side of the heart. Though the true crescendo murmur is diagnostic of stenosis of the mitral and tricuspid valves, several observers have described a murmur which they call presystolic, as occurring in other cardiac lesions, and even in dilatation of these same valves. I shall deal with this latter murmur in a future paper, and in the meantime will confine my remarks to the true crescendo murmur of mitral or tricuspid stenosis, and especially of the former affection.

NATURE OF THE MURMUR.—The characteristics of the crescendo murmur are known to everyone, but there are one or two points to which I wish to direct special attention. The murmur is short and vigorous, and evidently produced by some strong force. It immediately precedes, and terminates abruptly with, the closure of the mitral valve, an event which is generally marked by an accentuated first sound of the heart, and invariably by the true cardiac impulse. The essential and most peculiar characteristic of the murmur is that it rises progressively from low to high pitch, like an ascending scale of notes, as it rushes rapidly to its abrupt termination. It is also generally of a true crescendo character—that is, it gathers in power of tone coincidentally with its progressive rise in pitch. By the abrupt termination of the murmur at its stage of most complete development, the two important features of rising pitch and increasing force are emphasised.
There are, broadly speaking, two types of crescendo murmur, both of which terminate abruptly with the first sound of the heart, one rough and bur-r-ing or spluttering, and the other smoother and more whiffy in character, the latter type being often overlooked by observers. Some murmurs are accompanied by a tactile thrill, which can be palpated at the apex beat of the heart. The rhythm of this thrill is identical with that of the murmur which it accompanies.

Though of variable duration, the true crescendo murmur of mitral stenosis occupies at most not more than half the interval between the second and first heart sounds (Fig. 1, B, C, D, F, G). But it may appear to be much longer than this, for not infrequently it is apparently continuous with an ordinary diastolic mitral bruit of quite different type, namely uniform or decrescendo. In such cases murmurs

|   | VENTRICULAR   |   | VENTRICULAR   |
|---|---------------|---|---------------|
|   | DIASTOLE      |   | SYSTOLE       |
|   | AURICULAR     |   | AURICULAR     |
|   | DIASTOLE      |   | SYSTOLE       |
|   |                |   |                |
|   | ST            |   | ST            |
|   | 2N            |   | 2N            |
|   | SOUNh          |   | SOUND          |
|   |                |   |                |
|   | ST            |   | ST            |
|   | 2N            |   | 2N            |
|   | SOUNh          |   | SOUND          |
|   |                |   |                |
|   | ST            |   | ST            |
|   | 2N            |   | 2N            |
|   | SOUNh          |   | SOUND          |
|   |                |   |                |
|   | ST            |   | ST            |
|   | 2N            |   | 2N            |
|   | SOUNh          |   | SOUND          |
|   |                |   |                |

16. 1.—Diagram showing the rhythm and the relative duration of the events of a normal cardiac cycle, and of the murmurs of mitral stenosis in a heart beating 75 times per minute. Each numbered space represents 1 second; each cardiac cycle occupies 8 seconds.
may be heard to occupy the whole of the interval between the second and first heart sounds, either as one long continuous sound or with a slight break between the component bruits (Fig. 1, f, g). Some eminent authorities call this whole composite murmur crescendo, presystolic, or auricular systolic, but such a nomenclature is confusing, as they also apply the same terms to the true crescendo murmur of ascending pitch when it occurs, as it does in the majority of cases, quite separately in the latter portion of the interval between the second and first heart sounds, and immediately before the latter sound. But, be this as it may, I am only concerned with the undoubted crescendo murmur of ascending pitch in this paper, whether it occurs alone or as part of a composite murmur, and I shall confine my remarks almost entirely to this peculiar murmur.

In the accompanying diagram of the rhythm of the murmurs of mitral stenosis, I have departed slightly from the arrangement of lines of various length, thickness, and closeness together, by which Gairdner 1 first represented schematically the varying intensity, rhythm, and duration of these murmurs.

The varying length of the lines in my diagrams represents the comparative areas of the stenosed orifice during the production at it of the different types of murmur; the thickness of the lines and their closeness together indicate the force of the murmur; and, finally, the elevation of the lines above the base line represents the rise or fall in pitch which the crescendo and mitral diastolic murmurs undergo. Thus the diagram of a crescendo murmur (Fig. 1, n, v), interpreted according to the above explanation, signifies that this bruit begins at the very onset of ventricular systole (the line 5), becomes rapidly more and more vigorous, and of higher and higher pitch, as it rushes to its abrupt termination with the accentuated first sound of the heart, and that the orifice at which it is created is open to its widest extent at the beginning of the murmur, and closed, or almost completely closed, at the appearance of the accentuated terminal sound.

Exactly the opposite sequence of events is met with in the development of the early diastolic decrescendo murmur, as indicated by the diagram (Fig. 1, f, g), whilst the later diastolic murmur, part of which is produced (Fig. 1, f, g) in the period of auricular systole, is created at an orifice which remains as widely open as possible during the course of the murmur, and is the result of the action of forces which vary in intensity, and which generally die away more or less before the onset of ventricular systole.

The ordinary systolic murmur of regurgitation in mitral stenosis (Fig. 1, e) is of a lower pitch, and produced at an orifice of greater area of leakage than is the case when the systolic

1 "Clinical Medicine," 1862, p. 574.
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murmur continues the abruptly-terminating crescendo murmur (Fig. 1, c, f).

CaUSATION AND RHYTHM.—Two theories are advanced in explanation of the rhythm, and, to a less extent, of the causation of the crescendo murmur.

1. The presystolic or original and orthodox view is that "the murmur is due to the extrusion of blood through the stenosed aperture, under pressure by the auricle, aided by the aspirating force of the ventricle, during the period of diastole of the heart; in most cases it suddenly ceases at the moment of the production of the first sound of the heart; it manifests a heightened intensity at its close, due to the contributory impulsive force of the auricular systole; the first sound which terminates it is usually very short and abrupt, and is due chiefly to the sudden tension of the tricuspid valve and of the walls of the right ventricle." 1

2. The early systolic or other theory is, that the murmur arises from blood escaping through the mitral valve, before it is closed by ventricular systole, and that it is early ventricular systolic in rhythm.

This latter theory, though looked upon as in a way heretical, is gaining more and more supporters; and, though brought up in orthodoxy myself, I have been obliged to join the smaller band of unbelievers. Some of my reasons for doing so were given in a previous paper, 2 and the object of the present communication is to mention further arguments which appear to me to bring more support to this early systolic rhythm theory of the crescendo murmur.

It will be advisable for me to sketch briefly the main points of my former paper. In it I started with an entirely new explanation of the causation of the ascending pitch characteristic, which is so typical of the crescendo murmur of mitral stenosis. The theory I then stated is, that the crescendo murmur of mitral stenosis is produced by blood regurgitating through the stiff, rigid orifice of the narrowed valve, whilst this orifice, which resists closure, is being rapidly diminished in area and finally obliterated by the action of a strong force, which on its part increases progressively in strength with the duration of the bruit.

The main point in this theory is, that the ascending pitch of the murmur is imparted to it by the blood rushing through an orifice which is being gradually diminished in area of lumen, and to illustrate this contention I described a simple experiment with a piece of drainage tubing about 1½ in. long and ½ in. to 3 in. in diameter of lumen. If the observer will place such a piece of tubing between his teeth, and, with his lips closely applied to it, blow through it, he will produce a sound or bruit;

1 Sansom. "Diseases of the Heart and Thoracic Aorta," 1892, p. 378.
2 Med. Chron., Manchester, June 1897, p. 161.
and if, whilst producing this bruit, he brings his teeth together and obliterates the lumen of the tube (Fig. 2), the character of the bruit will change from low to higher pitch, until it finally is cut off when at its stage of highest pitch, by the obliteration of the lumen of the tube. The movement of the teeth must not be too rapid, but occupy, to begin with, about 1 to 1½ second, in which case the changes in pitch are very noticeable. At the same time, by varying the rapidity by which the tube is obliterated, the character of the resulting bruit varies within limits similar to those of a natural crescendo murmur.

If the above series of changes be carried out rapidly, the murmur so produced terminates with a thud, caused by the collision of the walls of the tube; and this point I used as experimental evidence in favour of the theory, that the accentuated first sound of the heart, which terminates a crescendo murmur, is due to the impact of the walls of the thickened rigid orifice of the valve.

The intensity of the artificial sounds varies with the force

![Diagram](image)

Fig. 2.—A, B, C, D, Progressive stages in the obliteration of the lumen of a tube in which is being produced an imitation crescendo murmur; E, F, condition of the lumen of the tube when a uniform murmur continues the crescendo murmur.

used to produce them, and with the abruptness with which the teeth obliterates the lumen of the tube; and the greater the diameter of the tube, the lower will be the pitch of the note produced by blowing through it, and *vice versa*. If in manufacturing the crescendo murmur, in the way described above, the teeth be moved together more carefully, so that, whilst the lumen of the tube is practically obliterated, its walls are not brought into violent collision, the crescendo murmur will still be heard, but without a terminal thud.

Finally, if the teeth be brought together so as to cut off the greater portion of the lumen of the tube, but to leave a slight area unobliterated (Fig. 2, E, F), the crescendo murmur and a modified terminal thud will still be produced, but running on from the thud will be heard a high-pitched, uniform murmur, which is caused by the escape of air through the “leak” in the tube. This last bruit is an experimental imitation of the systolic murmur which not uncommonly runs on continuously from a crescendo murmur in mitral stenosis. This simple experiment, to
my mind, explains away very clearly what is to many observers a stumbling-block to the acceptance of the systolic rhythm theory
of the crescendo murmur, namely, that it is impossible for even an abnormal first sound of the heart to appear in what is apparently the middle of ventricular systole, and to separate two murmurs, each of which is also produced during ventricular systole.

With the above simple experiment in mind, such an event may easily be explained, and I have shown diagrammatically in Fig. 1, c, f, the rhythm of such a compound bruit in mitral stenosis. The increased vigour which also characterises a crescendo murmur is imitated by increasing the force with which the air is blown through the tube in the production of the imitation bruit. All the above experiments with air bruits can be carried out with the lips, if they be pursed up as if for producing a whistle of low pitch, a method which is frequently adopted by teachers for giving students a good idea of what cardiac murmurs are like.

Having in this way explained the causation of the most important characteristics of the murmur, I went on to consider what must be the part of the heart which can act with sufficient force to develop the murmur, and came to the conclusion, from physiological, pathological, and clinical evidence, that it must be the left ventricle.

I will not go into any further details of the original paper, but beg to refer any one interested in the subject to it.

I have paid a good deal of attention to the same subject since June of 1897, and will now give an outline of the arguments which seem to me to throw fresh light on the subject, and to confirm my adhesion to the "heterodox" theory, that the true crescendo murmur of mitral stenosis is of early systolic rhythm, and is formed by blood regurgitating through the stiff jaws of the stenosed valve, whilst they are abnormally slowly closing under the rising force of intraventricular pressure, generated by the earliest phase of the ventricle's contraction.

**NEW ARGUMENTS IN SUPPORT OF THE EARLY SYSTOLIC RHYTHM THEORY—EXPERIMENTAL.**—1. Although there can be no doubt in my mind that the method of production of an imitation crescendo murmur by air is analogous to that which produces a crescendo murmur in the heart, I carried out similar experiments with water, using an old Higginson syringe with the outlet valve cut off, but with 3 or 4 in. of free outlet tube left. I found that by filling the syringe under water in a basin, and by holding the chest piece of a binaural stethoscope with the left hand to the end of the outlet tube, whilst compressing the ball of the syringe with the other hand, a bruit could be heard which was caused by the flow of water out of the orifice of the outlet
tube, and that this bruit rose in pitch as the orifice of the tube was obliterated gradually by pressing it against the rim of the stethoscope (Fig. 2). Here then is an identical experiment to the drainage tube, but carried out with water as the bruit-producing medium; and, moreover, I found that all the variations of an imitation crescendo murmur which can be produced by air can equally easily be developed by water. So my theory, that the crescendo murmur of mitral stenosis is developed by blood rushing through the stenosed valve whilst it is closing, which was formed as the result of experiments with air, is confirmed by similar experiments with water.

2. Rhythm of the murmur. — Having found that I could imitate a crescendo murmur with water, I carried my experiment a stage further to investigate the rhythm of such a murmur.

My apparatus consisted of two syringes, coupled together by a tube, and represented the left side of the heart (Fig. 3). The auricle syringe (A) is simply a ball syringe with inlet tube and valve, and a short valve-less outflow tube, which is connected with the tube running into the other syringe. The tube represents the auriculo-ventricular ring, and the second syringe (V) the ventricle. This latter syringe consists of an old type of Glover’s lithotripsy evacuator, part of which is formed by a glass chamber. Into the free end or head of the glass chamber the auriculo-ventricular tube screws, so that about 1 in. projects into the chamber and an equal amount remains outside. To the outer end of the tube the auricle syringe is fixed, and on to the inner end imitation stenosed valves are placed, where their behaviour, under different conditions of water pressure, can be clearly watched.

The imitation stenosed valves are best made by shaping out of a piece of grey rubber sheet, $\frac{1}{4}$ in. thick, a slightly conical tube about $\frac{3}{4}$ in. long, and with a diameter at the apex equal to about that of a black-lead pencil. The rubber is cemented into the proper shape by means of the solution of rubber in puncture-mending outfits for bicycle tyres. Rather stiffer, and not quite so satisfactory, but still very fair imitation valves, may be made with $\frac{1}{2}$ to $\frac{3}{4}$ in. of rubber drainage tubing.
about \( \frac{1}{4} \) in. in diameter of lumen. Either of these valves is simply slipped on to the inner end of the metal tube, so that about \( \frac{1}{4} \) in. of it projects beyond the end of the tube. The metal tube is then screwed home, and the valve is seen in position through the glass chamber, and it will be noticed at once that it tends to prevent the flow of water out of the ventricle syringe, but allows a free flow in the opposite direction. In this apparatus, then, the syringe (A) corresponds with the auricle, (v) with the ventricle, and the imitation valve with the stenosed mitral valve. There is no outlet from the ventricle syringe to compare with the aorta, but I have purposely avoided such, as without it a high pressure can be more readily generated, and the results obtained are the same as if an aorta were present.

I will not go into any more detail concerning this apparatus, but will mention the results obtained with it. As before, the experiments were carried out under water, to keep all air out of the apparatus. The object of my inquiry was, to see whether I could produce a crescendo murmur by forcing blood from the auricle syringe into the ventricle syringe, or in the opposite direction. To hear the bruits the chest-piece of the binaural stethoscope must be applied to the glass chamber under water, but I may say that the murmurs are not nearly as distinct as in the former experiment, owing to glass being a bad conductor of sound. To cut a long story short, I found that it was quite impossible to produce any bruit of varying pitch, and nothing at all resembling a crescendo murmur, whilst the water was being forced from the auricle into the ventricle syringe through the stenosed valve; but the moment the current of water through the valve was reversed, by compressing the ventricle syringe, very fair imitations of "blubbery," ascending-pitch murmurs were obtained. If the ventricle syringe be compressed gently—these ventricle experiments are best done after disconnecting the syringes—to imitate a feeble contraction of the ventricle of the heart, water will flow out through the valve, which is stiff enough to resist closure by a slight force of water pressure, and if the syringe be compressed more strongly a bruit of regurgitation ("systolic") will be heard. Further, if more force be used, the escaping water under high pressure throws the valve into "blubbery" sonorous vibrations, which can be felt by the compressing hand as well as heard with the stethoscope. The vibrations of the valve producing this sound can also be seen through the glass of the apparatus. They vary in character with the thickness and stiffness of the imitation valve, for, the harder and stiffer the latter, the louder and rougher is the murmur. Finally, if still more force is used, the murmur assumes a crescendo character, and is heard to rise in pitch and to terminate abruptly with a thud comparable to that at the completion of a crescendo murmur in mitral stenosis.
With the less perfect valves a smooth uniform "systolic" bruit may run on from the thud which terminates the crescendo murmur, in the same way as described with my air experiments, and is caused by the presence of a small leak in the otherwise closed valve, probably at the edge (Fig. 2, e, f). This experimental method of producing an imitation crescendo murmur also shows the probability of the abrupt first sound of mitral stenosis being caused by the impact of the walls of the closing rigid valve, for a clear note of impact can be heard when the sides of the rubber valve are seen to be forced into collision by compressing the ventricle syringe. There is no difficulty in understanding how a note of collision should be heard when the sides of the rigid, often cartilaginous, jaws of a stenosed valve meet abruptly under forcible pressure, even if the colliding surfaces should only travel \( \frac{1}{8} \) in. in approaching each other.

**Origin of the force which produces the crescendo murmur.**—In my previous paper I argued that the auricle is not always, if it is ever, strong enough to contract with that vigour which is necessary for the development of the murmur. My own experience is, that the auricle is as often as not abnormally weak in mitral stenosis, and quite incapable of producing a vigorous murmur. As further support of this belief, I may quote the following figures, obtained from the post-mortem room, which show the condition of the wall of the left auricle in cases of mitral stenosis.

Sansom\(^1\) found the auricular wall hypertrophied in thirteen cases and dilated in eighteen cases of mitral stenosis, whilst Samways\(^2\) found hypertrophy recorded, in the Guy's Hosp. Rep., London, in sixty-five, and dilatation in sixty-eight, of a total of 173 similar cases. Sansom\(^3\) says that "the muscular fibres of the wall of the auricle may be attenuated to almost extinction"; and Balfour\(^4\) writes: "It does seem remarkable and even mysterious that the comparatively feeble and brief contraction of the auricle should be associated with a murmur so loud and rough as the presystolic murmur," nevertheless the facts in favour of such being the case seem "incontrovertible." Any theory which explains the production of a crescendo murmur must hold good in whatever condition the left auricle may be, and it must be remembered that this murmur not uncommonly occurs in cases in which the auricle is dilated and abnormally weak. On the other hand, there is no question as to the ability of the left ventricle to produce a vigorous murmur like the crescendo murmur of mitral stenosis.

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1. "Diseases of the Heart and Thoracic Aorta," 1892, p. 353.
2. Brit. Med. Journ., London, 1898, vol. i. p. 365.
3. Ibid., 1898, vol. i. p. 1644.
4. "Clinical Lectures on Diseases of the Heart," 1898, p. 118.
DURATION OF A CRESCENDO MURMUR.—The time occupied in the production of these murmurs, as explained by my theory, is only a small fraction of a second. Thus, in a heart beating at the rate of seventy-five per minute, each beat occupies 3 of a second of time. Of this time ventricular systole takes up 3 and ventricular diastole 2. Auricular systole occupies 1 of the time taken up by ventricular diastole. Less than half the duration of ventricular systole fully covers the period which I believe is occupied by the crescendo murmur, i.e. 1/3 or less than 1/2 of a second (Fig. 1, A, B, C, D). But though of very short duration, crescendo murmurs may vary in length, some being more prolonged, whilst others are very short and abrupt.

In its more marked appearances a crescendo murmur is of a drawn-out emphatic character, and may be imitated by producing the murmur in a leisurely way with the lips, as described in a former paragraph. On the other hand, it is often so short and abrupt, that careful auscultation is necessary before a trained ear is certain of its presence. One feels that there is "something" which gives an impression of impurity to and terminates with the abrupt first sound. This type of the murmur can be imitated by making a crescendo sound as rapidly as possible with the lips. Between these two extreme types are crescendo murmurs of different lengths, which vary with certain conditions under which the murmur is produced. These conditions may be arranged somewhat roughly in the following groups:—

1. Rigidity of the valve.—The more rigid and stiff the valve, the longer will closure be delayed, and therefore the longer will a crescendo bruit produced at the valve be. The extreme of this condition is a rigidity which cannot be overcome, with the consequent development of an ordinary systolic murmur of regurgitation.

2. Rapidity of development of intraventricular blood pressure.—The earlier the stage of ventricular systole at which an intraventricular blood pressure sufficiently high to close the rigid valve is developed, the shorter will be the crescendo murmur, and vice versa. The development of high intraventricular blood pressure depends on the degree of the pressure within the aorta, and on the force of the heart's contraction. With a high intraventricular pressure, or with a strong heart beat, the crescendo murmur runs a short, rapid, and vigorous course, but with the opposite to these conditions prevailing the crescendo murmur will be prolonged and less forcible. If with a very low intraventricular pressure and feeble heart-beat the intracardiac pressure remains too low to close the valve, then the crescendo murmur will be replaced by a uniform true systolic bruit. The latter bruit may often be heard whilst the patient is lying on his back in bed, and yet be immediately replaced by a crescendo bruit when the intraventricular

1 Allbutt's "System of Medicine," vol. v. p. 470.
pressure has been raised, and the force of the heart-beat strengthened by the patient sitting up.

3. The rapidity or slowness of the heart's action—i.e. the length of the ventricular systole—also has some effect in determining the length of the crescendo murmur. The quicker the contraction of the ventricle, the shorter will be the crescendo murmur under otherwise similar conditions, and vice versa. The transition from the more prolonged to the rapid type of crescendo murmur is at times observed in the same patient.

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In some most favourable cases the change from a systolic murmur and no first sound to a deliberate, and then rapid crescendo murmur, with terminal accentuated sound, and finally to simply an accentuated first heart sound and no murmur, may be observed in the same patient during rest in bed and subsequent increased action of the heart induced by the erect posture or by walking about. Such a series of events is to my mind a further confirmation of the theory, that an ordinary systolic murmur of mitral regurgitation is but an imperfectly developed crescendo murmur, and points most forcibly to the conclusion that the latter bruit is caused by early vigorous ventricular systole and not by auricular contraction.

Nature of Ventricular Contraction.—The nature of ventricular systole favours the theory that the stenosed valve is closed in this phase of the cardiac cycle. Contraction of the whole ventricle wall is not simultaneous, but is in all probability of a peristaltic nature. Teaching, to my mind, is not definite in saying whether the wave begins at the base and extends to the apex, or vice versa. For my own part I think that the contraction ought to begin at the apex, for this is the thinnest and weakest part of the ventricle, being often not more than \( \frac{1}{4} \) in. thick (Quain); whereas the rest of the ventricle wall is two or three times as thick. Thus the contraction beginning at the apex will protect the weakest part of the ventricle and prepare it for the strain which it will be subjected to by the completion of systole. It is by this apical contraction, or at any rate by the earliest portion of the peristaltic wave, in health, I believe, that the already apposed cusps of the mitral valve are competently locked, and the regurgitation of blood into the auricle during the rest of ventricular systole prevented. Foster\(^1\) recognises a phase of ventricular systole, in which pressure is being got up

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1 "Text-Book of Physiology," 6th edition, vol. i. p. 264.
before the semilunar valves are opened. The time so occupied is '03 of a second in health. In mitral stenosis, however, the peristaltic contraction is, I believe, too feeble at its onset to close the stiff immobile jaws of the valve, but sufficiently strong to force blood to regurgitate through them into the auricle, and to give rise to the commencement of a crescendo murmur. The more forcible contraction of the rest of the ventricular wall, following rapidly and with increasing vigour, develops sufficient intraventricular blood pressure to close the valve, thus finishing off the crescendo murmur, which is terminated abruptly by the snap of the closing valve.

By this explanation we account for the development of both the ascending pitch and crescendo characteristics of the murmur which we are considering.

**Duration of Ventricular Systole in Mitral Stenosis.**

In health, as stated before, ventricular systole occupies three-eighths of the whole time of the cardiac cycle, the rest being attributed to ventricular diastole; and in a heart working at the rate of 75 beats per minute, each beat occupies '8 of a second of time. If the pulse rate increase in health, the additional beats are obtained without appreciable shortening of the cardiac systole, and almost entirely by reduction of the diastole of the heart.  

The advocates of the presystolic theory state that the phase of ventricular diastole is abnormally prolonged in mitral stenosis, to the extent, I take it, of the duration of the crescendo murmur, for the early and late diastolic murmurs when present occupy fully the time attributed to the normal phase of ventricular diastole. Now the crescendo murmur lasts for at least '1 of a second (see Fig. 1); and if this period of time be added to that already occupied by ventricular diastole ('5), we have this latter phase of the cardiac cycle taking up '6 and ventricular systole '2 of the total duration ('8) of the whole cycle. This means that the phase of the ventricle's diastole is three times as long as that of its systole. As ventricular systole is the most stable part of the cardiac revolution (Gibson 2), such a division of the cardiac cycle is very improbable. It is much more likely that the time occupied by the different phases of the heart-beat in mitral stenosis is as indicated in Fig. 1, b, etc., i.e., practically as in the normal state of affairs. Gibson 3 in his criticism of my view of the origin and rhythm of the crescendo murmur, groups me with other "early systolic" theorists, as believing that the ventricular systole, when it produces a crescendo murmur, begins earlier than is normally the case. But he has misunderstood my view of the case, whatever other writers may think, for I do not consider that there is any alteration in the time of onset, or even in the duration of the ventricular systole, in these cases. There is plenty of time in the three-eighths portion of

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1 Sherrington, *loc. cit.*
2 "Diseases of the Heart and Aorta," 1898, p. 55.
3 *Ibid.*, p. 156.
the whole cardiac cycle occupied by systole of the ventricle, for the
development of a crescendo murmur of ascending pitch, whether it
occurs alone or is followed by an ordinary systolic murmur of
mitral regurgitation.

The possibility of the existence of a transient systolic murmur,
terminating with the first sound and due to regurgitation through
an incompetent mitral valve during ventricular systole, is acknow-
ledged by Balfour.\textsuperscript{1} In the chapter on curable mitral regurgita-
tion the following passages occur:—"The result of even a slight
dilatation (from muscle failure) is the establishment of so-called
relative insufficiency, not because the auriculo-ventricular opening
itself is dilated—that does happen, but at a later period of the
affection—nor because the segments of the valve are unable to
close the opening, as one alone of these segments is almost sufficient
for this purpose; but because, owing to the separation of the
cardiac walls by dilatation of the (ventricular) cavity, the in-
sertions of the chordae tendineae into the papillary muscles are set
so wide apart, and so far from the centre of the ventricle, that the
trilling pressure of the auricular blood is unable to bring the valve
segments into apposition just before the commencement of the
ventricular systole. But any interference with the instantaneous
closure of the valve, at the moment of ventricular systole, favours
regurgitation, and in these cases this is a constant phenomenon,
varying in degree at different times. In all these cases there is a
more or less impure first sound audible in the mitral area, or a
transient systolic whiff may terminate in an apparently normal
first sound, and in still other cases a murmur is heard throughout
the whole of the systole from its commencement to its end. This
is quite in accordance with the account just given. In slight
dilatation, at the commencement of the ventricular systole, the
valve segments are not in apposition, as they ought to be, and
there is some regurgitation, but this ceases as the systole pro-
gresses and the valve segments are perfectly closed. When the
dilatation is greater, the valve segments never come together, and
the regurgitation persists throughout the whole of the systole."

Such opinions seem to me to be strong evidence in favour of
the early systolic rhythm of the crescendo murmur. For if in
such cases of mitral insufficiency, with mobile valve curtains, as
Balfour describes, the development of a transient systolic whiff,
terminating in a first sound, is possible, why should it not be
equally easy for a similar transient systolic whiff to be developed
at a rigid, stiff, narrowed mitral orifice, which most certainly
requires an abnormally great force of intraventricular pressure
to close it? The transient murmur described by Balfour must
in my opinion be of rising pitch, from the fact that it is formed
at a gradually diminishing area of leakage in a valve. It is thus
an imperfectly developed crescendo murmur.

\textsuperscript{1} Op. cit., p. 168.

(To be continued.)