but merely that lymph has transuded from the vessels, and in this lymph there is a considerable number of lymphocytes, probably obtained from the tissues as well as from the capillaries. There is no doubt a digestion leucocytosis, but it is not what has been usually described.

BATHS.

These arterioles and capillaries offer an extensive area for the practice of the bath physician; if he cannot here cultivate fresh fields and pastures new, he is not worth his salt. A little washing does every one good, especially those who only take an annual tub. The nature of the bath depends on the objects which you want to accomplish. Heat relaxes and cold contracts the arterioles, effleurage causes dilatation, but pétrissage and tapotement rather contract the skin arterioles and dilate those of the muscles. The light or hot-air bath—if not overdone—followed by gentle massage and a cool douche, has often a very stimulating effect. The warm alkaline bath has a sedative, and the carbonic acid bath a stimulating, effect on the nerve endings. In the Nauheim bath the increased specific gravity has a primary effect of raising the peripheral resistance, which is afterwards followed by reaction, and the effervescence has a stimulating effect, but it is a piece of absurd twaddle and unreasoning credulity to attribute any specific effects to the chloride of calcium; fortunately this salt is not absorbed, otherwise it might work irreparable mischief. Baths of all kinds are, I think, powerful agents for good or evil, and should be used with care and discrimination. In advanced arterio-sclerotic changes they do good in great moderation, but it is extremely easy to work mischief. There is no use in telling your patients, as is often done, that they will feel the benefit of the baths when they return home; this, in my opinion, is equivalent to saying that the patient would have been better without the baths as administered at your particular spa. Any beneficial effect should be observed under administration. I do not like this hope deferred, and I think under such circumstances the patient on his return home should thank God for his escape.

THE ARTERIOLES AND CAPILLARIES OF THE MUSCLES.

The arterioles are supplied with vaso-dilator nerves and are thus reciprocal to those of the splanchnic area. The capillaries are arranged in a fine longitudinal network, from which lymph readily transudes. Dr. George Oliver has shown that during the height of the digestive flow of lymph tension exercises of the muscles do not further raise the arterial pressure. The lymph in the limbs is not concentrated and is readily absorbed. Sir Lauder Brunton and Dr. F. W. Tunnicliffe showed that after the cessation of muscular contraction the intermuscular arterioles dilate and thus lessen the resistance. Healthy muscular exercise is necessary for the maintenance of a vigorous circulation, and when such exercise cannot be taken, we should use such substitutes as massage, passive exercises, and douches.

THE CEREBRAL VESSELS.

As the circulation in the brain is carried on in a closed box, and owing to the atmospheric pressure, the total amount of blood varies very little. The arteries and arterioles are thin-walled, the inner coat is well formed, there is a moderate amount of muscle fibre in the middle coat, the external coat is rather defective, but it is strengthened by a lymphatic sheath. The presence of vasomotor nerves is not generally acknowledged, but Dr. Alexander Morison says that he discovered them. Owing to stress and strain these vessels are liable to atheromatous and calcareous degeneration, and in the smaller vessels there are frequently miliary aneurysms. The capillaries are small, short, well-supported vessels, which seem to be able to bear a considerable amount of strain. When the body is inverted—not a desirable method of treatment for any affection, except that of syncope—the carotid arteries contract down to about half their usual calibre, and thus the quantity of arterial blood is lessened, but its kinetic energy increased. The arteries are subjected to great strain from higher potential, but in the capillaries this is largely converted into velocity. The cerebro-spinal fluid, which represents the lymph, is very deficient in proteid, from which we may infer that under

ordinary circumstances the capillary velocity is relatively great and pressure slight. In cases of meningitis the proteid in the fluid is increased. In alcoholic plethoric individuals there is frequently a deficiency in lime salts, transudation takes place into the ventricles, and so serous apoplexy is not such an uncommon event as textbooks make out. In cases of high arterial pressure the arteries are very liable to atheromatous and calcareous degeneration; such cases are often terminated by the rupture of a degenerate vessel or miliary aneurysm.

The treatment is that of the cause; frequently a timely free blood-letting saves the patient. Turkish baths are especially to be condemned in such cases, and other forms should only be used with great circumspection.

THE CORONARY VESSELS.

The arteries and arterioles which supply the heart closely resemble the cerebral vessels in being thin-walled, rather deficient in muscular fibre, and in having very few, if any, vasomotor nerves. They are also exceedingly prone to atheromatous and calcareous degeneration of the intima. Newell Martin, Roy and Adami, and Alexander Morison found some evidence of vasomotor nerves, but, on the other hand, Schäfer, Dixon, Brodie, and Elliott have failed to get any response to adrenalin, which acts on all muscular fibre innervated by the sympathetic. Recently Dr. W. H. Harvey has found that the pituitary gland contracts these vessels and causes atheroma in them. These arteries are terminal, and this would seem to be one of the very few mistakes which Nature has made, as we might be inclined to think that here, if anywhere, a free anastomosis is desirable. We must, however, remember that the two sides of the heart are two distinct organs only linked together for convenience and mutual support. Perhaps, also, Nature is more concerned with the species than with the individual, and to her the loss of a person from a blocked coronary artery is a matter of no great moment. The terminal portions of the arteries which are imbedded in the muscle remain fairly healthy as a rule, so when a coronary is blocked the heart may be supplied with blood from the venous side. In cases of tricuspid stenosis the coronary veins are much dilated, and form regular sinuses in the cardiac muscles.

I have dealt with the treatment of chronic degenerative lesions of the heart and with the pulmonic circulation in previous addresses.

I have not spoken about the climate because in this country we have got to take it as we find it, but, such as it is, if it were not for the greed of the hotel proprietors, it could be had at a very reasonable price. Moreover, there are the advantages that the atmosphere is frequently washed, the sewers flushed, and the sanitation, as a rule, perfect.

REFERENCE.

¹ BRITISH MEDICAL JOURNAL, 1906, vol. ii, p. 401.

THE CRESCENDO MURMUR OF MITRAL STENOSIS.

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In the extensive investigation of the physical signs and symptoms of cardio-vascular disease which is going on at the present day the rhythm of the crescendo murmur of *a-v* stenosis is apparently playing a very important part. Theories to explain perverted action of the heart are being promulgated; interpretations of jugular pulse tracings, normal or abnormal, deductions for prognosis, and indications for treatment are being made, books are written, and papers published, all being based in part or wholly on the supposed fact that the crescendo murmur of *a-v* stenosis is presystolic or auricular systolic in rhythm. As I have been of the opinion from 1897, when I first wrote on the subject, and am more strongly of the same mind now, that this theory has not only never been proved, but that it is fallacious, I think it worth while to remind observers of the alternative view, and to draw attention to some recent work, because it provides most valuable evidence as to the actual time of onset of this characteristic murmur.

In the first place, however, I should like to review the main theories concerning the rhythm of the crescendo murmur of mitral stenosis (for convenience I will speak of

mitral stenosis only as being the *a-v* lesion most commonly met with).

RHYTHM OF THE CRESCENDO, OR PRESYSTOLIC MURMUR.

There are two chief theories :

1. That the murmur is a direct current murmur, and occurs whilst blood is passing from the auricle into the ventricle, and is caused in part or wholly by systole of the auricle.
2. That it is a regurgitant murmur, and due entirely to systole of the ventricle.

Definition of the Murmur.

To be quite clear, it will be well to define exactly what is meant by the crescendo murmur. It is that murmur which has for its essential characteristics a pitch which rises progressively, and a force which increases progressively, and a sudden termination with an abrupt first sound when at its highest pitch and maximum vigour. It only occupies the terminal part of the interval between the second and first heart sounds, and is probably not more than 0.1 second in duration, and generally less than this. It may occur alone, or be continuous with a true diastolic mitral murmur, with a true systolic murmur, or with both these murmurs.

The A-S or Presystolic Theory.

To the writings of the late Sir William Gairdner is due in great part the almost universal acceptance of this theory at the present day. He assumed from the undoubted position of the murmur in relation to the first sound of the heart that it occurred just before *v-s*, and was coincident in time with *a-s*. But he had no indisputable proof to support his belief that the murmur must be coincident with *a-s*, the only universally acknowledged fact being that the murmur precedes and is terminated by the first sound. Nevertheless, the explanation seemed to be very obvious and appealed successfully to minds of clinicians, as compared with the *v-s* rhythm theory which was supported by Ormerod, Barclay, and Charlewood Turner in the Seventies, and by Dr. Howship Dickinson in 1887.

Gairdner's Case.

In 1893 Gairdner published the notes of a case of tricuspid stenosis which he had followed up clinically and seen *post mortem* in 1872, and which he and his disciples considered to be proof positive that the crescendo murmur is *a-s* in rhythm. I have criticized this case before, but I should like to refer to it briefly again, first, because, even if Gairdner was correct in believing that the crescendo murmur was synchronous with *a-s*, the case affords strong evidence in favour of the *v-s* rhythm of the murmur in mitral stenosis; and, secondly, to point out that there is another possible explanation of the causation of the murmur which altogether destroys the value of the case as the definite proof of the *a-s* rhythm theory which Gairdner believed and which nearly all modern observers assume it to be.

Clinically the case showed a murmur which began "immediately after the second sound, continued diminishing throughout the pause, and then went on crescendo up to the first sound, at which it stopped abruptly." The obstruction was caused by a pedunculated tumour in the right auricle, which, from its position, was forced during auricular systole into the tricuspid orifice and interfered with the flow of blood through this valve. In Gairdner's opinion the tumour "could only have been obstructive during ventricular diastole and auricular systole, and could by no possibility become the physical cause of a murmur of regurgitation." He also further believed that the murmur occurred "under circumstances which render it impossible to suppose that there should have been any difficulty or delay in the closure of the valve, any more than in a normal heart. And even had there been regurgitation, the obstacle, not being fixed but movable, would not have been in the way any more than the ball valve of a syringe is in the way of the current generated by suction."

In the first place, then, let us assume that Sir William Gairdner was correct in his supposition that the terminal part of the murmur, namely, the crescendo, rising-pitch portion, was caused, in part or wholly, by the contraction of the auricle, and we must ask the question, How were

the characteristics of the murmur, namely, rising pitch and increasing force, produced?

The rising pitch could have been produced, as I shall explain later, by the tumour being forced, partly or wholly by the systole of the auricle, further and further into the orifice of the valve, until it finally completely obstructed the latter. Whilst this was going on, that portion of the murmur which was undoubtedly produced by the obstruction to the passage of blood into the ventricle, would become progressively of higher and higher pitch synchronously with the progressive obliteration of the blood-way into the ventricle, and would terminate when at its highest pitch. If not in this way, how was the rising pitch produced?

My point can be seen at once, if whilst air is being sucked in or blown out through the pursed-up lips the tip of the middle finger be more or less rapidly used to block up the air passage in the lips. A rising pitch "murmur" is produced at once. If the air be blown or sucked with increasing force during the insertion of the finger a complete crescendo character is at once imparted to the "murmur."

This explanation of the production of the crescendo murmur in Gairdner's case, paradoxical though it may seem, is strong evidence in support of the early *v-s* rhythm of the crescendo murmur of mitral stenosis, as I shall show later. The crescendo or increasing force of this murmur must, if the latter were due to *a-s*, have been caused by a progressively vigorous, suddenly terminating contraction of the auricle.

But there is another possible explanation of the causation of Gairdner's crescendo murmur, and so, adopting a legal method, I will submit alternative pleadings. There are two disputable points in Gairdner's interpretation of the significance of the case :

(1) There is no analogy between the ball of a ball-valve syringe and the tumour in the auricle which interfered with the normal action of a flap valve.

(2) It is not impossible that the crescendo murmur was caused by blood escaping through the tricuspid valve at the onset of ventricular systole before the tumour, which was forced into its orifice by auricular systole, could be dislodged and the valve curtains come together in competent apposition. I may illustrate this criticism by the simple experiment of placing the tips of two of one's fingers into the mouth between the lips whilst the atmospheric pressure in the mouth is increased, and then suddenly withdrawing or blowing out the fingers and allowing the lips to come together and prevent the escape of air. During the withdrawal of the fingers a greater or less amount of air escapes through the lips, according to the way the fingers are removed from the lips, and gives rise to a bruit, which is of ascending-pitch type, owing to its being produced at an aperture of rapidly and progressively diminishing area. It also terminates abruptly with the final closure of the lips, which prevents any further escape of air. The lips in this experiment represent the valve curtains, and the tips of the fingers the foreign body.

Whether Gairdner's interpretation of the rhythm of the murmur or my own be correct, both afford strong evidence in favour of the early *v-s* rhythm of the crescendo murmur of mitral stenosis, because they both depend on the fact that the rising-pitch characteristic was imparted to the murmur at a closing valve orifice.

The V-S Rhythm Theory.

Dr. Howship Dickinson in 1887 wrote an important paper on "The Presystolic Murmur, falsely so-called." In this paper, Ormerod's and Barclay's views were supported by very powerful arguments, derived from much clinical experience. It is impossible to do justice to Dickinson's views in a short abstract of his paper, but I may note that one of his strongest reasons for believing that the "presystolic" is really a *v-s* murmur is the frequently repeated observation, that "if a finger be pressed between the ribs, particularly in a thin person and when the heart is not too rapid, and if, at the same time, the double stethoscope be adjusted to the murmur, the beginning of the murmur will be found synchronous with the beginning of the impulse."

As Dickinson looked upon the "short sound which immediately follows the special murmur—up to which the latter runs—as marking the conclusion of ventricular

systole," it is not to be wondered at that his view was not accepted.

I was brought up on the *a-s* rhythm theory, and accepted it until in 1895 I was converted to the rival theory as the result of experimenting in the production of imitation murmurs with a piece of drainage tubing. I found that I could produce a perfect imitation of a crescendo, rising-pitch murmur by blowing with increasing force through a piece of drainage tubing of about $\frac{1}{4}$ or $\frac{1}{2}$ inch diameter of lumen and obliterating the lumen of the tube with the fingers, or by bringing the teeth together on the tube whilst doing so.

As this simple experiment is the key to the whole question, giving, as it does, a bed-rock of fact to build upon, I will state precisely what happens.

PRODUCTION OF AN IMITATION RISING-PITCH, CRESCENDO-FORCE MURMUR.

A sound of crescendo force, rising pitch, and abrupt cessation at the point of maximum vigour and highest pitch is produced if you blow with increasing force through a piece of drainage tubing, and obliterate the lumen of the tube whilst doing so. A similar series of events occurs with water instead of air as the sound-producing medium, as can easily be seen with a Higginson syringe without its nozzle, a binaural stethoscope, and the washbasin of a bathroom.

This "crescendo" murmur varies as regards its force, pitch, tone, and length of duration, according to the force used to produce the murmur, the diameter of lumen of the tube, the capacity for vibration of the wall of the tube, and the rapidity of the obliteration of its lumen. Similar variations of the murmur occur, *mutatis mutandis*, in the *a-v* valves. The murmur is identical in essential characteristics with crescendo murmurs heard in mitral and tricuspid stenosis. A similar sound can be produced in a similar way by phonating the word *w-h-i-p* through the lips, or by placing the tip of a finger between the lips into the mouth, when you raise the atmospheric pressure in the mouth, and then withdrawing the finger more or less suddenly. I can find no other way than by obliterating an orifice at which a murmur is produced, by which you can impart the progressively rising-pitch character to the murmur.

The rising-pitch character of the crescendo murmur of *a-v* stenosis must, therefore, with the greatest probability, if not absolute certainty, be acquired by the act of the valve closing while a murmur is being produced at it.

Gairdner's observations, even if allowed to be accurate, support in a very suggestive way the "closing-orifice" production of the murmur, and also the admissibility of the principle of the "closing-orifice" murmur being applied to the intracardiac conditions.

The character of the crescendo murmur will vary in mitral stenosis according to the condition of the valve. If there be any vibration of the curtains during regurgitation, a rough blubbery or burring sound, will be produced; if the curtains be entirely rigid, only a smooth crescendo rising-pitch sound will be heard.

If the above explanation of the mechanism of production of a rising-pitch murmur in mitral stenosis be fallacious, I shall be glad to be corrected.

Assuming that the murmur be so caused, it could arise during the direct flow of blood through the heart, that is, from *a* to *v* during *v-d*; or by regurgitation, that is, from the *v* to *a* during *v-s*. A stenosed *a-v* valve is of very different character to a normal valve; it is almost invariably so stiffened or thickened that it requires more force to close it than is necessary to float the thin mobile normal *a-v* curtains into competent apposition. Therefore a stenosed valve requires more *i-v* pressure to close it than does a normal valve, and this can only

be produced at a later phase of *i-v* pressure than that at which the normal *a-v* valves are closed. If the diseased valve remains opened during any part or the whole of increased *i-v* pressure—that is, *v-s*—blood will regurgitate through it and cause a murmur unquestionably systolic in rhythm. If the valve is so

distorted and stiffened by disease that the *i-v* force cannot close it at all, a *v-s* murmur is heard throughout the systole of the ventricle; but if the valve is in such a condition that it only requires rather more *i-v* force to close it than a healthy valve does, it will be closed at a later period of *v-s* than normal, and during this delay blood will escape back into the auricle and cause a murmur to which a rising-pitch character is imparted by the act of closing of the valve. The terminal abrupt

first sound is probably caused by the sudden and forcible impact of the hardened, thickened jaws of the diseased valve.

The mitral valve is closed in health when the *i-v* pressure rises above that of the *i-a* pressure through the contraction of the ventricle. In mitral stenosis there is no reason to suppose that anything different happens. No one will maintain that the auricle can raise in the ventricle a higher pressure than that in its own cavity. If any one should hold this view, the onus of proof rests with him.

The preceding arguments can be restated in a few lines: The rising-pitch characteristic of a crescendo murmur is caused at a closing *a-v* valve; the *a-v* valve is closed by *v-s*, therefore the murmur is *v-s* in rhythm.

Careful, and certainly more than a cursory, consideration of this question—it occupied very closely my own thoughts for two years before I published my views—and the taking into account

of the clinical variations of the murmur and the pathological conditions of the stenosed valve, auricle, and ventricle; and, finally, both the explanations of the production of the murmur in Gairdner's case will, I venture to think, convince others of the fact that it is the ventricle and not the auricle which alone can produce the crescendo murmur.

APPLICATION OF THE EARLY V-S THEORY TO THE EXPLANATION OF THE CHIEF AUSCULTATORY SIGNS OF MITRAL STENOSIS.

Early Stage—Accentuated First Sound Alone.—The thickened margins of the diseased valve are connected at

Fig. 1.—A, Mitral valve unclosed; B, Mitral valve closed; C, Mitral valve incompletely closed.

Fig. 2.

their junction by mobile hinges, which allow the jaws of the valve to snap together easily and early in *v-s* and before blood has time to regurgitate.

Later Stage—Crescendo Murmur Alone and Accentuated First Sound.—The mobile hinges become stiff and the murmur is formed by blood regurgitating under pressure through the valve whilst its stiffened jaws are being closed by the rising *i-v* pressure developed at an abnormally later stage of *v-s*. (See accompanying diagram, Fig. 2, published in 1899.) An accentuated first heart sound terminates abruptly this murmur, and is due to the collision of the thick hard jaws which guard the stenosed orifice (Figs. 1 and 2, b).

Crescendo Murmur Continued by Ventricular Systolic Murmur.—In this case the former bruit is produced in the same way as when it is heard alone, but the coaptation of the jaws is imperfect, and fails to make the valve water-tight or blood-tight, so a small stream of blood continues to regurgitate through one corner or side of the valve orifice whilst *v-s* persists, and gives rise to the uniform systolic murmur. A more or less abrupt first may divide these two murmurs (Figs. 1 and 2, c).

Crescendo Murmur and Accentuated First Sound, Alternating Systolic Murmur and less marked or no First Sound.—The occurrence of a crescendo murmur and sharp sound indicates that the *i-v* blood pressure is raised by *v-s* sufficiently high to produce a transient regurgitant murmur before it closes the stiff valve with a snap; whilst the production of a *v-s* murmur, but no sound, means that the heart beat is weak, sufficient pressure is not generated to close the valve anything like completely, no crescendo murmur or abrupt first sound can be produced, and an ordinary *v-s* regurgitation goes on, producing the uniform, true, mitral systolic murmur. There is no change of rhythm with the alternation of these murmurs, both occurring during *v-s*, and within a few beats of a heart, often as a result of change of position from lying down to sitting up. The final change to an accentuated first sound only, if a patient walks about, is due to the increased force and quickness of beat closing the valve before regurgitation can take place, or producing a crescendo murmur of too short duration to be heard. This point may also be illustrated by the well-known clinical fact that an overworked ventricle in mitral stenosis, which can only contract feebly and cause a systolic murmur whilst the patient is up and about, will often, on rest in bed, with or without drugs, recover its strength again sufficiently for the production of a crescendo murmur.

Diastolic, Crescendo, and Systolic Murmurs Forming One Long Continuous Bruit.—In this case the diastolic murmur is formed by blood being driven through the stenosed orifice, under those forces which fill the ventricle and by the contracting auricle (when it is able to join in); the crescendo murmur, by blood regurgitating through the closing valve during the earlier half of *v-s*; and the systolic murmur, by blood escaping through a leak in the somewhat imperfectly closed valve during the latter half of ventricular contraction. The two last murmurs may be separated by the abrupt tapping first heart sound, caused by the impact of the jaws of the orifice of the closing valve (Fig. 2, E).

The systolic murmur alone is caused by the *i-v* pressure being unable to coapt the edges of the diseased valve during any part of *v-s* (Fig. 1, A, and Fig. 2, D).

The ordinary diastolic mitral murmurs, early, middle or late, always decrescendo, are produced as indicated in the preceding paragraph but one.

An accentuated second sound heard over the pulmonary area will always be heard in mitral stenosis.

The tone of any murmur will vary according to whether it is produced by a fluid vein, or whether a diseased valve

is thrown into vibrations. In the latter case the murmur will be harsher or more "blubbery."

It will be seen from the above that, in my opinion, a crescendo murmur in mitral stenosis means efficient contraction of the left ventricle, and is of good prognostic import.

Does the *a-s* rhythm theory explain these points as simply or as satisfactorily?

There are a couple of points made by *a-s* theorists in support of their views to which I should like to refer.

Visible Pulsation in the Second Left Intercostal Space.

This argument was especially used by Balfour. He stated that in many cases of mitral stenosis the appendix of the left auricle is greatly dilated and lies close to the anterior chest wall in the second left intercostal space, and when this is the case, contraction of the auricle against the chest wall may be felt by the finger to coincide with the auricular systolic (presystolic) murmur heard with the stethoscope.

But it has been shown conclusively by various observers that the left auricle never occupies the position in mitral stenosis described by Balfour. It lies well back and away from the chest wall, and its contraction never can be felt, but the pulsation to which Balfour refers is caused by the movements of the base of the right ventricle, which lies behind the second costal interspace on the left side of the sternum. Keith says that the left auricle is behind the ascending aorta.

Balfour's clinical evidence, taken with the pathological investigations of other observers—who, I may mention, are all upholders of the presystolic theory—yields an important fact against and not in favour of the theory which he advocated. This evidence, in truth, supports the early systolic rhythm theory.

Again, Sansom and other observers argued that the fact that the crescendo murmur is heard and felt to exactly precede the maximum of the cardiac movement is proof that the murmur is *a-s* in rhythm, whereas it is really in favour of the early *v-s* rhythm.

There is one question which I have previously asked, and as I have never received any answer to it I will put it again. Why is not a crescendo murmur heard at the aortic orifice if that heard in mitral stenosis be caused by blood being forced by auricular contraction through the narrowed valve? It is not unreasonable to wonder why the much stronger ventricle never produces any suspicion of a crescendo murmur when it drives blood through an entirely analogous obstruction—namely, that of stenosis of the aortic valve. Here it must be noted that the action of ventricular systole is that of a gathering force, and that the blood is forced through the aortic valve with increasing vigour.

Here, then, we have practically an ideal condition for the production of a crescendo murmur, and yet one is never produced. On the other hand, it is allowed that a crescendo murmur is produced at times by blood regurgitating through an incompetent aortic valve (Flint's murmur). I have never seen an explanation of how it is that no crescendo murmur is heard in aortic stenosis offered by any supporter of the presystolic theory.

Cardiographic Evidence.

If the early *v-s* theory of the crescendo murmur be true, it is obvious that the first sound of the heart will occur at a later phase of *v-s* than is normally the case, for there will be contraction of the ventricle for a longer or shorter time before the closure of the valve; in other words, there will be a lengthened presphygmic interval (Fig. 1, B). There will also be a longer or shorter time of preliminary movement of the apex beat, during which blood is slipping back through the open valve, before the strong impulse

Fig. 3.—IX, Loud, rough murmur at apex, commencing at the second sound and becoming very loud and harsh towards its conclusion, and leading up to the first sound. XI, Long, very loud, presystolic bruit of rough, grinding quality, commencing immediately after the second sound, and leading up to the first sound. XIII, Rough but not very loud bruit, commencing immediately after the second sound, and leading up to the first sound, accompanied by a thrill. A, Auricular systole; K, Closure of semilunar valves. (Reproduced by kind permission of Dr. Galabin and Editor of *Guy's Hospital Reports*.)

denoting the forcible contraction which drives blood through the semilunar valves. This preliminary movement, synchronous with blood passing back through the diseased valve, and with a crescendo thrill, when one is present, ought, with a good cardiograph, moving more rapidly than those much used now, to impart to the cardiogram a more or less gradual rise, to be terminated suddenly by a wave and notch caused by the jerk of the closure of the stenosed valve, and from this point there ought to be a more vertical upstroke like that of a normal cardiogram. If a crescendo thrill be present the preliminary gradual rise ought to be more or less wavy or serrated according to the fineness or burring character of the thrill. With a prolonged ordinary systolic murmur of mitral regurgitation, and no closing movement of the diseased valve, there will be an abnormally long pre-sphygmic interval, and the upstroke of the cardiogram ought to be abnormally gradual throughout its whole extent.

Many good cardiograms taken from cases of mitral stenosis to prove the *a-s* theory show these points clearly. Thus, Fig. 3 is a reproduction of three adjacent tracings from Galabin's paper on the subject. There are several others of similar nature. The letter *a* is supposed to indicate *a-s*. If it is, what is all the irregularity in XI and XIII tracings before the final upstroke due to but ineffective ventricle contraction? The clinical notes appended to the figure are taken from Galabin's paper.

So much for the early ventricular systolic rhythm theory of the crescendo murmur of *a-v* stenosis, which appears to me to be so unquestionably sound that it has never been shown to be fallacious, and is usually ignored.

I am pleased to see that Dr. Mackenzie, who has known my views from the time I first published them, has at last come to the conclusion that a brief crescendo murmur can be caused by slight regurgitation through the mitral orifice at the beginning of ventricular systole; but he still thinks that it is indisputable that auricular systole can produce a pre-systolic or crescendo murmur.

Recent Investigations.

Through close attention to other work I have not been able to follow all the work in recent years on cardio-vascular investigation, but I have read a certain amount and cannot see any new and indisputable evidence in favour of the *a-s* theory. Many of the observations are not as satisfactory from a scientific point of view as they might be, inasmuch as the tracings often show too little detail and are taken on too slowly recording surfaces with time marks of 0.20 of a second. In some tracings there

is no time record at all. Much quicker movement and more delicate instruments are required to bring out the niceties of cardio-vascular waves and movements, and so to permit of more accurate estimation of synchronism. We ought to think and observe in 0.02 second at most, not 0.20 second, in this work. Such tracings can as a rule only be taken in hospitals, but if they were obtained cardiographical inexactitudes would be avoided and many knotty points more quickly unravelled, I have no doubt. I quite agree with what Dr. Thomas Lewis has written about cardiographic record making (p. 86). His diagrammatic representation of the cardio-vascular events is drawn in 0.01 second.

The jugular pulse argument, which is used so much now as a basis for deductions in cardio-vascular work, is that a wave synchronous with the *a-s* is present in cases of *a-v* stenosis when a crescendo murmur is heard and marked by hand on the tracing as occurring synchronously with this wave, and that the wave disappears when the crescendo murmur is not heard; therefore the

crescendo murmur must be due to *a-s*. This line of argument has been used by Dr. Lewis to prove, in a case in which, at one time, there was a crescendo murmur which disappeared, that since there was an auricular wave in the jugular pulse at the time there was a crescendo murmur, which was still present when there was no crescendo murmur, there must have been contraction of the right auricle to cause the auricular venous wave with synchronous paralysis of the left auricle. I have been unable to find any indisputable

2nd. Presystolic 1st.
sound. murmur. sound.

Fig. 4.

Diastolic murmur, 1st sound. 2nd sound.

Fig. 5.

From cases of mitral stenosis. In these figures the upper curve is the carotid pulse, the middle the murmur, and the lowest the time record (0.01 second). The straight line in Fig. 5 was caused by a piece of dust. (Reproduced by permission of the publishers of *Pflüger's Archiv*.)

proofs of the statement that the crescendo murmur of *a-v* stenosis is synchronous with a wave in the jugular pulse due to *a-s*.

On the other hand, I say that the crescendo murmur is caused by early *v-s*, and then only when the ventricle is contracting well, and that it disappears temporarily when the ventricle muscle acts quietly, and permanently when the muscle fails. A greater or less degree of ventricular disability accompanies auricular muscle failure, and when the latter is marked, and there is no auricular wave in the jugular pulse, the muscle failure of the ventricle is also so considerable that the *v-v* pressure cannot close the mitral valve, and we get in consequence no crescendo murmur, probably only an ordinary regurgitant murmur. So absence of the auricular wave in the jugular pulse will probably be noted in a case in which the ventricle muscle is incapable of producing a crescendo murmur, as is always so towards the end of *a-v* stenosis.

As part of his argument Dr. Lewis stated that some observations of Weiss and Joachim finally settled the question of the actual time of onset of the crescendo

murmur in favour of the *a-s* rhythm. I was much interested in seeing this opinion, and turned to the paper in question, feeling quite sure that if the observation established anything definitely, it would be the early *v-s* rhythm. I now beg to ask those readers of the JOURNAL interested in the matter to look at the paper in *Pflüger's Archiv* and say what they do tend to prove. I give the essential points here.

Weiss and Joachim have devised a most ingenious instrument called the phonoscope, by which they can record in a wonderful and unmistakable way the normal sounds of the heart and any murmurs louder than the very softest blowing type. They publish several tracings, of which two are appended, showing the murmurs of mitral stenosis. The points they make are:

1. The distance from the beginning of ventricular systole to the rise of the carotid pulse in a normal heart is 0.08 to 0.09 second.

2. In mitral regurgitation this is increased to from 0.125 to 0.15 second, according to the degree of muscle failure present. This fact appeared to the writers to be strange, but they explain it by suggesting that blood escapes back through the incompetent valve at the beginning of *v-s*, and delays the development of sufficient *i-v* pressure to open the semilunar valves. This is exactly what I say happens in the development of a crescendo murmur, causing delay of the first sound (see Fig. 2).

3. The crescendo murmur of mitral stenosis occurs well within 0.15 second of the carotid rise (Fig. 4). Time vibration 0.01 second.

4. Weiss and Joachim make a special note of the fact that if the time of commencement of contraction of the auricle before the rise in the carotid artery (0.20 second) be marked off on the tracings, no noticeable sudden alteration of the curve is seen there (Fig. 5). In Fig. 4 they point out that the first vibrations of the murmur are only 0.13-0.14 second removed from the carotid pulse.

Weiss and Joachim do not state what appears to be the obvious conclusion to their work, namely, that the crescendo murmur is *v-s* rhythm, but I have written to them drawing their attention to this, and asking them to examine their records to see if they afford similar support to the early *v-s* rhythm theory, an account of which I have sent them.

In my judgement these tracings are not proof positive that the auricle has nothing to do with the production of the crescendo murmur. As there is wanting on them a definite indication of the exact time of onset of *a-s*, it may be argued that the auricle contracts nearer to the carotid pulse in mitral stenosis than 0.20 second; but, on the other hand, one of the chief doctrines of the modern school of cardiology, advanced partly on the assumption of the synchronism of *a-s* and the crescendo murmur, is that the *a-c* interval (between the onset of *a-s* and the carotid pulse) is often increased in mitral stenosis even to 0.40 second.

In conclusion I submit that I have shown that the *a-s* rhythm of the crescendo murmur is not only highly disputable, but even entirely fallacious, and that the murmur is early *v-s* in rhythm. It would be very instructive for all interested in the matter if Dr. Mackenzie will produce his "indisputable" evidence that the murmur is, in part or wholly, caused by *a-s*, so much of the superstructure of modern cardiology being founded on this belief. Then the two discordant views can be compared by readers of the JOURNAL.

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OBSERVATIONS CONCERNING THE BLOOD IN CHOREA AND RHEUMATISM.

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THE association which is generally supposed to exist between rheumatism and chorea is very difficult to understand, and I think we may fairly say that the question of their actual relationship is hardly settled yet. Rheumatism itself is a disease which has a much narrower interpretation to-day than it had some years ago, and a good many cases formerly classed as rheumatic fever are now known to belong to an entirely different category.

In the *Liverpool Royal Southern Hospital Reports* for 1902 I referred to this fact, and directed attention to a series of cases which closely resembled rheumatic fever clinically, but were quite uninfluenced by treatment with the salicylates, and were found to result from toxic absorption from one or another of the mucous tracts. In chorea we have another disease which, although it frequently occurs in children who have suffered from antecedent rheumatic or rheumatoid conditions and is associated with endocardial and pericardial complications, cannot be said to be more than an ally of true rheumatism, and this supposed alliance rests only upon certain clinical similarities and effects which may possibly result from an entirely independent toxin.

It is upon this point that I have been endeavouring to gain some information recently by means of experiments with the bloods of the two diseases. I have also tried to discover whether any evidence can be found to prove or disprove that chorea is to be regarded as an infective disease. The infectivity of chorea is certainly suggested by the remarkable way in which it tends every now and then to become so prevalent as to appear to be epidemic. In November, 1907, there were 5 cases in the wards, all admitted within a few weeks. In 1908, of 18 indoor cases, May and October furnished more than half the admissions; and during May of this year I have had under my care no less than 8 cases, of which 4 presented themselves in one day. It has more than once happened in my experience that two children in the same family have developed the disease in succession, and on one occasion the return home of a convalescent child was followed by the admission of a sister with whom she slept. On making inquiries, I have sometimes found that two or more children in a school have had chorea in succession or simultaneously, and it seems possible that the old impression that children get the disease by imitation may have arisen through the unsuspected infection of one patient from another.

H. C. Ross's method of telling whether leucocytes are living or dead¹ by examining the blood on an agar film containing atropin and a stain, has enabled us to investigate the effects of placing the leucocytes of healthy people in the plasmata of patients suffering from various diseases. He has shown that if the leucocytes of one healthy person are placed in the plasmata of other healthy persons they live a somewhat shorter time than they do in their own plasma, whereas if they are placed in the plasmata of persons suffering from diseases which are due to or associated with certain toxins in the blood, their lives are greatly shortened. This fact has proved useful inasmuch that it has (so far as we can tell up to the present) placed in our hands a method of estimating to a considerable extent whether a disease of uncertain nature is accompanied by a toxin in the blood, and it was this general principle which led to my suggesting that we might employ it to throw some light on the problem of the relationship between chorea and rheumatism.

I need not enter into all the details of the experiments, but shall very shortly explain their nature:

1. The first research had for its object the determination of the presence or absence of a toxin in the blood of chorea patients. The chorea blood was mixed in a capillary tube with an equal volume of citrated solution, centrifugalized, and the corpuscles removed. To the remaining plasma a small volume of the blood of a healthy person was added. Simultaneously another tube was prepared by adding the same healthy person's corpuscles to another healthy person's plasma. Both tubes were kept at 30° C. and samples of the contained corpuscles were