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ORIGINAL ARTICLES.

RETARDATION OF THE PULSE IN MITRAL INSUFFICIENCY.

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At the meeting of the Association last year, I read before this section a paper on Diminution of the Retardation of the Pulse in Aortic Insufficiency, in which were considered the reality, mechanism and diagnostic value of the phenomenon. In the present paper I wish to present the subject of augmentation of the retardation of the pulse in mitral insufficiency.

This subject in its inception and literature seems to be peculiarly my own. For although none would question the importance of a new diagnostic sign of a lesion so common and grave as mitral insufficiency, and whose diagnosis by means of the old signs is not always infallible; and although I presented to the profession some years ago demonstrations of this phenomenon of mitral regurgitation which to me were conclusive, the announcement has attracted but little attention; my observations have neither been confirmed nor disproved by others, and two writers only have noticed them. Dr. A. B. Isham, in presenting the value of the graphic method from the point of view of examinations for life insurance, accepts abnormal delay of the pulse as a valuable sign of mitral regurgitation.¹ And M. Pélissier, in his inaugural thesis on retardation of the pulse, quotes largely and approvingly from my published reports.² These papers and my own contain all that has been contributed upon the subject.

Convinced myself of the truth and intrinsic value of the observation, and desiring to see the fact accepted and utilized, I have prepared this article, hoping that a new presentation of the subject may lead to new interest in the phenomenon, and stimulate personal investigation of the time-relation between the heart and pulse in real or suspected mitral insufficiency.

Evidences of the reality of the phenomenon are drawn, 1, from a consideration of the mechanism of mitral insufficiency; 2, from experiments on the schema; 3, from experimental observations on man.

Examining the question first from the point of view of the mechanism of mitral insufficiency we shall find a convincing *a priori* argument in favor of abnormal delay of the pulse as a direct effect of this lesion. Under normal conditions with mitral valve intact, the heart is a perfect organic pump, the left ventricle alternately and duly receiving and sending out blood. When the ventricle contracts, the pressure within becomes positive in relation to the arterial and auricular, which insures the prompt closure of the auriculo-ventricular valve and immediately thereupon the opening of the aortic valves and escape of blood into the aorta.

When the ventricle relaxes, the pressure becomes negative with respect to the arterial and auricular, and the blood flows freely in from the auricle, but is prevented doing so from the artery by closure of the semilunar valves. Auricular systole raises somewhat the diastolic intra-ventricular pressure, but the moment of beginning systole finds a considerable excess of pressure on the side of the artery. The time required to overcome this excess after the beginning of ventricular systole measures the duration of the ventriculo-aortic or presphygmic interval, which under the normal conditions will give a normal value.

Thus all goes smoothly on; the rhythmic succession of contraction and relaxation, of valvular opening and closure, of inflow and outflow of blood, and all the events of a cardiac cycle in harmony with each other in time and status and movement.

But when the mitral valve is permanently open the harmony is no longer preserved. The ventricle fills in diastole even more promptly and completely than in normal conditions, in consequence of falling into it of blood from the surcharged auricle; but in systole the blood is first sent backwards through the open valve into the relaxed auricle. This process fills the auricle and pulmonary veins and dams back the blood towards the lungs. The retrograde current, however, is soon arrested by meeting the direct current from the right ventricle, whose systole is synchronous with the left, and whose tricuspid valve is intact. Thus the pressure within the left ventricle and auricle (now one cavity) finally becomes positive with respect to the aortic pressure, and for the remaining part of the systole blood escapes into the aorta. From this it follows that the time and work of each

¹American Journal of the Medical Sciences, July, 1882, p. 119.
²Paris, 1882.

systole is consumed in first driving the blood backwards, and only a part of the systole is effective in driving the blood into the general arterial system. The effect of this condition of things upon the arterial pulse is to postpone the time of its appearance and lessen the volume of its wave; the cardiac systolic portion is shortened relatively to the length of the cardiac systole. If the heart maintain its vigor the pulse may still feel sharp to the fingers and its sphygmogram give a steep ascent and pointed summit; yet it quickly vanishes to the fingers, and, as shown in the trace, it lacks duration, as compared with the duration of the systole which has sent it forth. If the heart passes into irregular action there will be systoles without arterial pulsation, and pulsations presenting a great variety in form and in time. The many and grave ulterior pathological sequences I will not follow, as the immediate mechanism is all that concerns the present study.

If the above sketch of the mechanism of mitral insufficiency be truly drawn, the conclusion inevitably follows that abnormal delay of the pulse is an essential phenomenon of the lesion.

EXPERIMENTS ON THE SCHEMA IN ILLUSTRATION AND EVIDENCE.

Passing next to the results of experiment, I will first offer those obtained with the schema. It is well known that the mechanical action of the circulation can be well imitated by means of a good working schema. With such a device connected with the transmission tubes of an apparatus for simultaneous inscriptions, traces were obtained at the same time from the interior of the ventricle and interior of the aorta. The intra-ventricular pressure in diastole was maintained at 4 inches, and the intra-aortic at 20 inches (water manometer). The ventricle was compressed and decompressed in a uniform manner for all the experiments. The conditions were varied only with respect to the mitral valve. The instant of ventricular impulsion and that of the aortic wave were signaled by the ascents of the traces, and the time-difference between these ascents was easily computed by the accompanying chronogram. Experiments were first made with the mitral valve intact, representing normal conditions. Fig. 1 is a fair example of the results. Delay of the aortic wave, $\frac{1}{16}$ second.

Fig. 1.

Fig. 2.

Fig. 3.

Another series of experiments was made with a large mitral opening, of which Fig. 2 shows an average result. Aortic delay, $\frac{2}{9}$ second—more than three times as long as the result with intact valve.

A third series of experiments was made with a comparatively small mitral opening, with result shown in Fig. 3. Aortic delay, $\frac{1}{9}$ second, which is seen to be intermediate between the other two.

These results well illustrate the retarding effect of mitral insufficiency on the time of the pulse wave, and the additional significant fact that the retardation is in proportion to the size of the mitral opening. Indeed, in view of the careful manner in which the apparatus was prepared and the experiments conducted, together with the analogy in all essential points for our purpose between the schema and heart, these results may be admitted, also, as veritable demonstrations of the phenomenon and phases under consideration.

EXPERIMENTS ON MAN.

The subject remains to be further elucidated by the results of experimental observations on man.

In order to appreciate chronometric discrepancies between the heart and pulse, it is essential that the normal cardio-arterial time-relations should be well established. This has been done. The interval between the beginning of ventricular systole and the carotid pulse has been found, after a great many observations and measurements, to average about $\frac{1}{12}$ second with pulse-rate at 72. The normal range of variation with this pulse-rate may be placed between $\frac{1}{11}$ and $\frac{1}{13}$ second. It has been determined that the interval bears a relation to the pulse-rate, so that a frequency of 120 would give an interval perhaps not more than half as long as a frequency of 60 per minute. Also investigations have shown that the causes of the variations with the pulse-rate are changes in the mode of ventricular systole and variations of the arterial blood-pressure relative to the ventricular. In frequent pulsations, the systoles being quicker and the arterial pressure relatively lower, the cardio-carotid interval would be correspondingly shortened. The rule is this, the cardio-carotid interval for a pulse frequency between 60 and

taneous cardio-arterial traces (with accompanying chronogram in fifths of seconds), from a number of patients presenting diagnostic signs of mitral regurgitant lesion are here produced.

The cardio-arterial intervals in all these cases of mitral insufficiency are abnormally great; greater than those obtained from persons in health, or from those affected with any process of disease which does not involve pulse-retarding lesion of the heart or aorta. In the absence of such lesion, no series of cases with

corresponding pulse frequencies can be produced which will show such an amount of pulse-retardation.

Figures 6 and 7 are from Jennie M., aged 11 years. She had suffered with chorea and recurring rheumatism, and her heart had become seriously affected. It was enlarged, and emitted a loud murmur, which was distinguished as mitral regurgitant. Fig. 6 was taken during an acute attack, in the presence of fever, pulse-rate 140. Fig 7 was taken after subsidence of acute symptoms, pulse-rate 114.

Fig. 6.

Fig. 7.

Some months subsequently the girl experienced a new attack, with grave cardiac implication, under which she died. The post-mortem revealed pericarditis old and new, with adhesions and liquid effusion; general enlargement of the heart, especially hypertrophy of the right side; notable insufficiency of the mitral valve, the result of inflammatory thickening and contraction; the other valves normal.

Fig. 8 is from Mary M., next younger sister of Jennie, also aged 11 years at the time of observation. She became affected with chorea and sub-acute rheumatic symptoms, and when first seen her heart was notably enlarged, and there was present a distinct apex systolic murmur. She had slight fever, pulse-rate 114. Carotid delay shows $\frac{1}{10}$ second against $\frac{1}{19}$ which would be about her normal interval.

Fig. 8.

Fig. 9.

Fig. 10 is from Emma O., who presented an apex systolic murmur, which supervened during an attack of scarlet fever, and persisted after recovery from that disease. At the time of observation, June 22, 1883, the murmur and pulse delay were the only indications of cardiac lesion. She remains well to the present time.

Fig. 10.

Fig. 11.

Fig. 12.

Fig. 13.

Fig. 14.

Fig. 15.

Fig. 16.

Fig. 17.

Fig. 18.

Fig. 19.

Fig. 20.

Figures 10 and 11 are from Mrs. E., aged 30 years. She was taken with præcordial soreness and a dry, hacking cough, and, when seen a few days afterwards, betrayed a marked apex systolic murmur. Tracings show the cardiac-carotid interval to be $\frac{1}{9}$ second with pulse-rate at 80, and the cardio-radial interval to be $\frac{1}{6}$ second.

Figures 12 and 13 are from Lizzie R., aged 24 years, hospital case, presenting apex systolic murmur. Delay as shown.

Fig. 14 is from Geo. B., aged 27 years, presenting apex systolic murmur. Delay as shown.

Fig. 15 is from J. B., aged 28 years, hospital case, with diagnosis of mitral regurgitation and cardiac hypertrophy. At the time the traces were taken he was under a febrile paroxysm. Indications and delay as shown.

Fig. 16 is from J. T., aged 50 years, a hospital patient, with cardiac enlargement and apex systolic murmur. Pulse delay and pulse-rate as shown.

Fig. 17 is from Sam. T., aged 28 years, who presented the signs of mitral insufficiency.

Fig. 18 is from J. C., a hospital case, with well-marked signs and symptoms of cardiac hypertrophy and mitral regurgitation.

Fig. 19 is from Chas C., aged 18 years, with indications of mitral insufficiency.

Fig. 20 is from a man in health, aged 54 years. The first traces were taken when the circulation was quiet, pulse 82; the second traces when the circulation was accelerated as consequence of exercise, pulse 130. In this figure the pulse delay represents the normal amount. It measures, in fractions, a little

less than $\frac{1}{13}$ second, with pulse at 82, and a little less than $\frac{1}{21}$ second with pulse at 130.

Having thus shown that the mechanism of mitral insufficiency includes increased delay of the pulse as one of its essential phenomena, and demonstrated the constant relationship between the lesion and phenomenon, both on the schema and man, I submit the proposition as proven, and the fact as true beyond a doubt.

DIAGNOSTIC VALUE.

In undue retardation of the arterial pulse on the systole of the ventricle we have a special and highly diagnostic sign in mitral insufficiency. In this lesion, when pure and effective, the sign is always present, and fails to manifest only when the lesion is so slight as to cause no functional disturbance, or when complicated with large insufficiency of the aortic valves. In the condition of harmless regurgitation through the mitral valve the retardation of the pulse remains normal, because the conditions are virtually normal; in the complication stated the retardation is abnormally diminished, for the reason that the aorta ventricle and auricle are as one cavity, with blood-pressure in equilibrium at the end of ventricular diastole, and the beginning of systole starts a current forwards into the artery as well as backwards into the auricle. Thus in mitral, complicated with free aortic insufficiency, instead of abnormal delay, there is abnormal precipitation of the pulse. These are the only exceptions to the rule, that the sign is present when the lesion is present.

What other conditions besides the prime one considered are capable of determining undue retardation

of the pulse? Slowness of ventricular contraction, and relatively high arterial pressure are known causes of prolongation of the presphygmie interval, but these causes are considered to produce only the normal extreme of prolongation.

1. There are good reasons to believe that mitral stenosis independent of insufficiency is capable of causing a delay which may exceed the normal limits. The mechanism, and experiments on the schema favor this view; besides, not long since I traced a case presenting the presystolic murmur and other clinical features of pure mitral stenosis and found the cardio-carotid interval unduly long. If this fact should become established, (of which I have no doubt) it will be found that the delay, unless in extreme narrowing, will not be as great as in mitral regurgitation, and that the sign will not manifest at all in moderate or even considerable contraction, for it would require a very small opening to prevent the ventricle from duly filling during diastole, which would be requisite to insure the production of the sign. Even admitting the fact of the relationship, the sign can have nothing like the diagnostic significance in mitral constriction that it has in mitral regurgitation.

(2.) Rarely, increased delay may be produced by a "locking" of pathologically changed aortic valves. This condition is uncommon, but may obtain when the valves are stiff and thickened, and, notably, when loaded with calcareous masses. They close with a species of locking and are forced open only after a prolonged effort of the ventricle.

(3.) It has been well established that an intervening thin-walled distensible aneurism of the ascending aorta may cause increased delay of the distal pulse, but that if the aneurism have resisting walls, even though it be large, there is no increased delay. Evidently, if the pulse could be traced on the proximal side of the pouch, which is impracticable in man, the delay would not obtain.

The conditions have now all been named in which there is a possibility of undue delay of the pulse being produced by other causes than mitral insufficiency. With the latter all are serious organic lesions of which abnormal delay of the pulse may be a common product.

The sign then, although always present in pure, effectual mitral insufficiency, is not of itself pathognomonic of this lesion, since it may be present, also, in other organic conditions. Here the stethoscope comes beautifully to the aid of the graphic instrument, and in the union of the apex systolic murmur of the one and the pulse-delay of the other, our diagnosis is perfected.

The graphic sign compared with apex systolic murmur.—Systolic murmur located at the left apex, is the physical sign above all upon which turns the diagnosis of mitral insufficiency. Yet all know there may be free regurgitation without murmur, and insignificant harmless regurgitation with loud murmur, and that no direct relation subsists between the amount of regurgitation and the intensity of this auscultatory sign. Also is it well-known that apex systolic murmur may be distinctly present and the

mitral valve be functionally perfect. These points do not compare altogether favorably with the point we have stated as true concerning the graphic signs. The comparison is well shown by apposition.

<i>Abnormal pulse delay.</i>	<i>Apex systolic murmur.</i>
Always present in pure, harmful, mitral regurgitation.	Sometimes absent in very free regurgitation.
Always absent in harmless regurgitation.	Usually present and often very loud in harmless regurgitation.
The amount of delay directly proportional to the amount of regurgitation.	No certainty that the intensity of the murmur is directly proportional to the amount of the regurgitation, but often it is inversely so.
Present in other conditions besides mitral regurgitation, but these are organic and serious.	Present in other conditions besides mitral regurgitation, but these, if organic, are harmless.

Although the graphic sign has greatly the advantage in the comparison, its value and utility are most manifest when it is obtained and considered in connection with the auscultatory. Alone it is not competent to declare the presence of the lesion, but in association with systolic murmur at the left apex it speaks with positive import. Neither sign singly is conclusive of the lesion, but the two combined could be produced by no other. One locates the lesion, the other determines the extent of its damage.

An important point is that the diagnosis as made by the aid of the graphic sign is certain and immediate at any stage of the case, while the diagnosis as made by all other indications, exclusive of this, is not certain until confirmed by the development of pathological sequences. When, as often happens, other signs raise the question of the existence or not of harmful regurgitation, the graphic comes in with its prompt and positive decision.

RÉSUMÉ OF CONCLUSIONS.

1. Abnormal retardation of the arterial pulse, notably the carotid, on the systole of the ventricle is a real phenomenon of mitral insufficiency.

2. It is present in all cases of pure, harmful, mitral insufficiency, and is absent only in insignificant, harmless, regurgitation, or in regurgitation complicated with aortic insufficiency.

3. It measures by the amount of retardation the amount of the regurgitation.

4. It may be present in two other conditions, probably a third, all organic; hence this sign, notwithstanding its positive value, is not of itself pathognomonic.

5. In conjunction with apex systolic murmur its presence is conclusive of mitral insufficiency.

6. Compared with apex systolic murmur, this sign is more positive and appreciative, and distinguishes, which the latter does not, between harmful regurgitation and harmless conditions.

7. By the aid of this sign a positive diagnosis may be made at once in any stage of the case, and without waiting for the development of sequences and symptoms.