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Analytical Investigation into the Acoustic Phenomena in Mitral Stenosis.

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The most significant acoustic phenomena in mitral stenosis are the diastolic and the presystolic murmurs.

Lewis gave the following explanation of these phenomena: “The diastolic murmurs of mitral stenosis are due to the rapid onflow of blood through the stenosed orifice. Those periods of diastole are occupied by murmurs, during which the velocity of blood reaches a certain grade. When the auricle contracts at the normal time and the heart beats slowly the velocity tends to be greatest in presystole, otherwise it is greatest in early diastole; these are the times at which murmurs are most commonly audible.” Bass explained that the presystolic murmur is caused by the auricular contraction. But Frey observed that the presystolic murmur of mitral stenosis occurs actually in presystole, but too late to be attributed to the auricular contraction, so he considered that the murmur is caused probably by the regurgitation of blood through the mitral orifice after the end of the auricular contraction, but before the ventricular contraction. He considered that the atrioventricular valves do not close perfectly at such a time of presystole. But according to Brockbank and Weitz the “presystolic” murmur does not occur in actual presystole, but after the commencement of the ventricular contraction. They attributed this murmur to the regurgitation of blood through the cicatrized orifice, which could not be closed perfectly at the beginning of systole. These authors explained that the closure of the mitral orifice was delayed owing to the affect of the valves, and so the blood flowed back through the orifice, till the valves closed perfectly. Weitz, Joachim and Weiss observed that in mitral stenosis, the first sound occurred at a later period of systole than in the normal heart.

Another interesting phenomenon in mitral stenosis is the reduplication of the second sound which can be perceived not seldom by auscultation.

Some authors were of the opinion that this phenomenon was due to the dissociation of the contraction of the left and the right ventricle. Schrump considered that it was caused by the delay of the left ventricular systole, in consequence of the prolongation of the period of filling. Pogany considered however, that the ejection from the left ventricle ends earlier than that from the right ventricle. Geigel explained that the ejection phase of the right ventricle is prolonged. Other authors such as Weitz
and Groedel considered that there was no actual dissociation of the two sounds in mitral stenosis, and that the auscultatory reduplication of the second sound was caused by those vibrations of the diastolic murmur occurring at the commencement of the ventricular filling.

Results of the experiment.

The figures here given illustrate various cases of acoustic phenomena in mitral stenosis.

Fig. 1.

![Fig. 1 Image]

Fig. 2.

![Fig. 2 Image]

Fig. 3.

![Fig. 3 Image]

In figs. 1, 2 and 3, no significant vibration can be found in the phase of presystol. The vibrations of the initial part of the first sound do not appear before the commencement of the apex beat, but a number of small rapid vibrations are superposed on them. The duration of them is a little prolonged in fig. 1 and fig. 2. In these cases the interval between the commencement of the apex beat and that of the ejection phase was also prolonged and it was found to be about 0.08 and 0.09 sec. The principal part is very prominent in each case. The second sound shows also a rather prominent principal part. It can be noticed however, that there appear in each case a number of small rapid vibration about 0.08—
0.09 sec. after the beginning of the second sound. In these cases the intense "presystolic" and the diastolic murmurs were perceived by auscultation, and the second sound was reduplicated in the case of fig. 1. and 2. Hence the vibrations occurring in early diastole obviously cause the auscultatory reduplication of the second sound in the case of fig. 1 and 2. and the diastolic murmur in fig. 3.

But in fig. 4, the presystolic murmur is found apparently before the commencement of the apex beat. It precedes the apex beat by 0.06 sec. The appearance of the first sound is almost normal. The interval between the commencement of the apex beat and that of ejection is prolonged (about 0.08 sec.). The diastolic murmurs are found about 0.10 sec. after the second sound.

In fig. 5, the murmur vibrations appearing in presystole are very pronounced. They precede the apex beat by 0.1 sec. and continue till the first sound appears. It is to be noticed that there occur small vibrations after the second sound, and again about 0.08 sec. later. The second group of vibrations is the diastolic murmur, because it falls upon the point, where the curve of the apex beat indicates the beginning of the ventricular filling. Hence the first group must be attributed to the dissociation of the second sound.

Fig. 4.

Fig. 5.

Fig. 6.
As may be seen in these figures, the "presystolic" murmur did not occur always in the actual presystolic phase but was found to appear at various times. Such cases as fig. 1, 2 and 3 cannot be explained by obstruction of the ventricular filling. The fact that the initial part was always affected by small rapid vibrations as in mitral insufficiency can be explained by the regurgitation through the mitral orifice, as Brockbank and Weitz considered. But such cases as fig. 4. and fig. 5. can be explained simply by obstruction of the ventricular filling, as Lewis explained.

Regarding the reduplication of the second sound, the explanation of Schrumpf can not be accepted as correct, because if it is actually caused by the delay of the left ventricular filling, there must be shown at the same time, considerable deformation of the first sound. But in fact there was certainly no such case. The explanation of Pogany, can not be accepted either, because if he is correct, the duration of the right ventricular systole must be about 0.41 to 0.45 sec. as is seen in the figures. Such values are far greater than any we know for the normal duration of the ventricular systole. It is more probable that the occurrence of the second pulmonary sound is delayed as Geigel considered. But Weitz pointed out that such a considerable prolongation of right ventricular ejection is inconceivable. He considered that the reduplication in auscultation was due to the diastolic murmur occurring at the beginning of the ventricular filling. The writer supports the opinion of the last author for the following reasons. In the first place, the interval between the commencement of the second sound and these vibrations in question is almost constant in every case, being found to be about 0.1 sec. If it were caused actually by the dissociation of the left and the right ventricular ejection there might be numerous variations in the interval. In fact, the interval between the two parts of the actually reduplicated second sound, which was observed not seldom in pulmonary infiltration, varied even in a few successive heart beats. In the second place, the writer observed that the interval was not different, even in cases where arrhythmia perpetua was combined. In such cases, as is shown in fig. 6, the intervals were also about 0.08 and 0.09 sec., in spite of the considerable variations of the systolic and the diastolic phases.

In the third place, there was no difference in the interval between the second sound and these vibrations in question, when the reduplication was perceived or when the diastolic murmur alone was perceived. Hence, as already mentioned, the reduplication of the second sound in mitral stenosis is nothing but a very short murmur of the mitral orifice. However, the writer does not overlook a case such as fig. 5 in which the vibrations in question appeared plainly before the commencement of the filling. These vibrations must be considered as caused by the delay of the end of the ventricular systole on either side. But the writer, does not accept this phenomenon as an inevitable consequence in mitral stenosis, because such a phenomenon was found more often in pulmonary
infiltration.

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4) Fahr, Heart 4, 147 (1912).