

HALL (J.N.)

An Undescribed heart: Mummified.





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**AN UNDESCRIBED HEART-MURMUR.**

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IN quite an extended search in the literature of diseases of the heart I have been unable to find any note of a murmur such as is here described.

Mrs. C., a widow, thirty-two years of age, a housewife, has borne four children and has had two miscarriages, the last being only a month ago. Ten years ago she had an attack of acute rheumatism confining her to bed for five months. She has had several slight attacks since. She lost considerable blood at the last miscarriage, and is rather anemic. Upon August 6th, when I first saw her, the cardiac area was enlarged to the mammary line, the apex-beat under the nipple, in the fifth space, and the cardiac action decidedly feeble. An apical systolic murmur, propagated to the mid-axillary line was heard, with accentuation of the pulmonary second sound. There was moderate edema of the feet, and marked dyspnea; the respirations were fifty per minute while sitting in the chair, the pulse 90, and feeble. No pulmonary edema was found.

I exhibited the patient at the clinic as an example of mitral regurgitation, in spite of the fact that the murmur was not propagated to the back. The feeble action of the heart seemed to me sufficiently to account for this, while the lack of such transmission was in my mind more than counterbalanced by the presence of edema and particularly the marked dyspnea, especially upon



the slightest exertion. In fact, I take decided exception to the dictum of Cammann and others, that it is necessary to hear a mitral murmur in the back before deciding that it indicates regurgitation. I believe I have seen several examples that were exceptions to this rule.

The woman was given iron and a laxative, and returned on August 29th. Her condition was then about the same, excepting that the anemia was a little improved, and the murmur followed the first sound and the apex-beat, instead of being synchronous with them as before. The cardiac action was still so feeble that digitalis was prescribed, and a few days later the patient was re-examined.

The edema of the feet at that time had practically disappeared. The pulse was 70, and of much better strength. The apex-beat was easily visible and palpable. The murmur followed the first sound, and the visible beat, and was transmitted to the mid-axillary line. The respirations reached 50 per minute five minutes after slowly ascending a short flight of stairs. The basic second sound came just at the termination of the murmur, but at the apex was obscured by it. The case was exhibited to many students, and to Drs. McLauthlin, Lobingier, and Hopkins, of the University of Colorado. There was no room for disagreement as to the facts, viz., that the apex-beat and first sound, with a distinct movement imparted to the stethoscope, all preceded the murmur. The only explanation of this state of affairs which I can conceive, is as follows:

The mitral valve was incompetent, and regurgitation occurred through it, giving rise to the murmur, as is usual in such cases. The right ventricle, somewhat hypertrophied, gave rise to the apex-beat and a normal first sound, and displaced the stethoscope firmly applied to the apex region, forming the first element in a reduplication of the first sound of the heart. The second part of the reduplication was made by the left ventricle,

but instead of a distinct heart-sound a mitral murmur was heard, owing to the regurgitation through the mitral valve. I believe this explanation sufficiently accounts for all the phenomena observed.

Barr has reported a somewhat similar case in which the left ventricle contracted first, with a mitral murmur, and was followed by the normal contraction of the right ventricle. Sansom reports a case in which, over the ventricles "a murmur tailed off from the second reduplicatory sound," at the apex only the murmur being distinguishable. The case later developed a presystolic murmur, and the reduplication disappeared, which would appear to confirm his explanation that it was due to a presystolic flap of the mitral valve. It obviously differs from the case we are considering.

George Johnson believed that reduplication occurs from the contraction first of the hypertrophied auricle, followed by that of the ventricle. It is not reasonable, I think, to suppose that the auricle could possibly cause the distinct apex-beat, even if it could cause the sound. Sansom has stated, nevertheless, that in certain cases of mitral stenosis the auricle may cause a distinct impulse. There has been, I believe, no evidence of mitral stenosis in this case, and I consider it opposed to Johnson's theory.

Hayden believed that the reduplication occurred from a resolution of the first sound into a muscular sound and a valvular element, the latter occurring after the former. It seems to be conclusively proved that the valve closes early in systole, however, and I certainly fail to see any bearing upon the present case. Potain's theory that reduplication is generally only apparent, being in reality due to a presystolic flapping of the mitral valve in association with a normal first sound, evidently would not apply here, as the apex-beat accompanied the first sound.

I hope to be able to follow the case, and shall note any further developments.





