

Dr. Hunter—As I think the pulse comes up, I can't agree with Dr. Rorke that the pulse is characteristic of stenosis. In well marked stenosis the pulse reaches its height slowly and comes down slowly, there is not a great deal of difference in the extreme height and extreme fall. In this case the pulse comes up quickly. No matter how marked a systolic murmur one can get over the aortic cartilage, if we do not get a retarded pulse, I think one must take the pulse as proof of the condition, and not the murmur, and I think it is rather a *celar pulsus* than a *tardus*.

The heart is enlarged considerably downwards and outwards and a well marked systolic thrill on the right side of the sternum, a rough systolic murmur extending up to the neck, as well as a considerable diastolic murmur. In the pure stenosis murmur there should be no marked enlargement of the heart, because it gives a considerable hypertrophy of the heart, which couldn't be made out clinically. On the other hand, where there is a dilatation, one gets dilatation accompanied by hypertrophy. It follows rheumatism, gives a better prognosis.

I never saw a case in which there was so slow a pulse in this condition. I have only seen one case of pure stenosis of the aorta that was diagnosed by a competent physician, Haverdon, of Brompton, once showed a case. The pulse was small, about 60, regular. The heart was not so much enlarged and there wasn't such an area of pulsation here, and the second sound at the aortic cartilage was well marked. If we had so much enlargement of the heart from a pure stenosis condition, I think the man would be in a much worse condition. I tried to make out a venous pulsation. In many cases where the heart seems to be beating slowly it is beating slowly because the venous sinuses are beating at a different rate so on every second or third pulsation the pulsation from the auricle reaches the ventricle. This is due to some interference with the bundle of His. The pulse we have shows a modified Corrigan pulse, rather than a stenosis. In mild conditions of stenosis, considerable regurgitation is obtained, so that going on probabilities, this would have to be, on the other theory, a very marked case of aortic stenosis in order to give no regurgitation, but neither pulse nor sphygmographic tracing bears that out. No condition of the mitral valve could be made out.

As to the history given, if one assumed that in the first attack there was considerable amount of regurgitation and not so much stenosis, one could conceive that the second attack may easily have caused a sufficient amount of stenosis to be of considerable advantage to the heart in diminishing the amount of regurgitation. From that long history after the first attack there may easily be myocardial changes and that may give an explanation of the very slow pulse.

Dr. Rorke said, as to the prognosis, if he lives a careful life it is good till some myocardial changes and if he has myocardial changes and loses his compensation, he will not live a very happy life after that.

Dr. Milroy—There has been a lot of myocarditis with myocardial change which could account for the slow pulse. I think the compensation didn't take place until he had the second attack of rheumatism, and the rest perhaps assisted and facilitated in the compensation, the heart becoming hypertrophied at that time. I think one of the effects of the stenosis of the aortic valve is radiocardial.

Dr. Nichols showed a specimen of ectopic pregnancy. The woman skipped two or three days of her menstrual period, complained of spasms in the lower part of her body, but there was no depression or shock. She had a bloody discharge. Her mother said