

borne in mind, that, in many diseases, about the time of convalescence, the patients have a pulse which is below the average frequency in the same persons in health. This is the case in typhoid and typhus fever, pneumonia, and some other affections. This morning there is no fever present, the temperature being 98 $\frac{1}{2}$.

This man has been taking quinine and nitro-muriatic acid. It might, at first, strike the mind as an incongruity in the treatment of this disease to administer an acid, while the predominant feature of the disease is the presence already of an acid in the system, but really there is no incongruity in it. Over and over again we apply remedies and measures which are directly antagonistical, and each will meet its own indication, and often the only proper method of treating certain cases is to meet the indications.

We come now to speak of one of the important events which is liable to occur in the course of inflammatory rheumatism. This disease in itself, as far as the constitutional difficulty is concerned, is not dangerous to life, but there is a danger in connection with certain incidental events and those events which are most likely to occur relate to the heart. There is a special liability to an inflammation affecting one or both serous investments of this organ, and these are the untoward events to be looked after in cases of articular rheumatism. The importance of the disease and the permanent welfare relate chiefly to the occurrence of these complications. There are some other complications which are so infrequent that they do not give us much care, and we will pass their consideration. The cardiac affections are the prominent ones. Endocarditis occurs in quite a proportion of cases, but I do not give you the figures, because I think there has been some looseness and error in making up statistics upon this point. The reason for this I will soon mention. Pericarditis is of much less frequent occurrence than endocarditis, and it may be said further with regard to these complications, that if we have pericarditis we have endocarditis, but the rule does not hold in the opposite direction. Pericarditis involves a certain amount of immediate danger, though a great proportion of cases get well. What we probably have in this case is endocarditis, and first of all we will study the evidence upon which this probability is based. The evidence in this case is not absolute, but it is *probable*. Now you will recollect the fact to which I called your special attention a few moments ago, *viz.*, this patient has not had præcordial pain, or any chest symptoms, whatever, during the progress of his case. The diagnosis of endocarditis is therefore based entirely upon physical evidence. This is the reason why endocarditis is a disease which has been discovered within the last half century, and was never before known. It was discovered by physical exploration, and must continue to be recognized by this means, because it occurs without any subjective symptoms. It is associated probably with some increase of the circulation, but as this increase goes more or less with the rheumatism we cannot draw the inference from this that endocarditis

is present. How are we to determine whether a patient has endocarditis or not, who is suffering with articular rheumatism? We are to reach a positive diagnosis in this way: if the patient be under your observation, and you can determine by auscultation that there is no mitral systolic murmur present at the commencement of the attack, and then in the course of the disease a mitral systolic murmur is developed, you know that the patient has endocarditis. It all depends upon the development of this mitral systolic murmur, and the murmur is the hingeing point. This patient has mitral systolic murmur but the diagnosis is not positive, because the patient had the same murmur when he came into the hospital, and we do not know certainly that the murmur, has been developed since the commencement of the disease. It has probably been developed in this patient since the commencement of this attack, for it is the first attack the patient has had of the rheumatism; he has always been well, and as the murmur is one which does not indicate regurgitation, it is altogether probable that in this case it is evidence of endocarditis. I find here that the apex of the heart is beating in the fourth intercostal space, as it not infrequently does when the body is in a recumbent position. By percussion I determine that the heart is not enlarged. This would not be the case if the patient had had mitral disease for any length of time previous to the present attack, for he would have more or less enlargement of the heart.

Within a certain circumscribed space about the apex of the heart, I get a murmur, and it is not propagated much beyond this quite limited area. It is not proper to call this murmur a mitral regurgitant murmur, because there is no evidence of regurgitation.

What do we look for as physical evidence to show that there is regurgitation? The fact that a mitral murmur is present, is not limited to the apex, is tolerably loud, and is propagated to the left, would be evidence that it was one of regurgitation.

I also get a murmur at the base of the heart, but I attach no special importance to this, because we cannot attach much importance to a murmur at the base of the heart in a case of articular rheumatism. It is very frequently present, and is dependent upon the condition of the blood. It is always present in females, or at least, I believe I have never seen a case of articular rheumatism in a female where this murmur was not present. It is just here I apprehend that a great confusion has arisen with regard to statistics in reference to endocarditis, and many cases have been called endocarditis in which the disease did not exist. I would not make my diagnosis relying upon this murmur at the base, unless I had the mitral systolic murmur at the same time.

Endocarditis is a serious complication, because in it the rheumatism has laid the foundation for the subsequent occurrence of valvular lesions.

We have attenuation of the valves, thickening and calcification of the valves and other valvular lesions, all arising from an endocarditis in connection with rheumatism, and we have not much knowledge of