severer cases die early; but these are quite the exception, and the number of deaths from heart disease in children is quite insufficient to furnish the solution of the problem. Although we may admit that children having valvular defects (even though not presenting obvious heart symptoms) are likely to resist the attacks of other diseases less successfully than children who are not handicapped in the same way, yet even this would give a very insufficient account of the matter. I think the only explanation at all adequate is that in a very large proportion of cases the mitral incompetence observed in children disappears in later life. I wish it were possible to furnish statistics in support of this proposition. But that is obviously impossible. In the nature of things these mild and favorable cases are just the ones that disappear from our observation the soonest. Physicians to children's hospitals necessarily lose the opportunity of seeing them again in later life. Hence the difficulty of bringing to statistical proof the fact of which I am nevertheless strongly convinced, namely, that mitral regurgitation in children is a condition which is very frequently recovered from.

Now, it is probable that structural alteration of a valve, following endocarditis, may be more readily recovered from in childhood than in later life. But there is another condition equally capable of producing the phenomena observed, and much more readily recoverable—I mean regurgitation through the mitral valve produced by dilatation of the left ventricle. I believe that far less prominence has been given to this condition than it deserves in ordinary clinical teaching. Regurgitation through the tricuspid, arising in a similar manner, is a clinical commonplace. But when distinct evidence of mitral regurgitation is discovered, valvular lesion from old (or recent) endocarditis is usually assumed as the almost necessary corollary. Many authorities certainly speak of mitral incompetence following dilatation, but in ordinary clinical teaching the fact of its frequent occurrence is left in the background. I have shown elsewhere that it takes place in chlorosis much more commonly than is generally supposed. In 400 cases of chlorosis under my own care I found complete evidence of mitral regurgitation (a systolic *bruit* audible at the angle of the scapula, as well as at the apex) in 123. In many of these the graver physical signs were known to have appeared during the time that the chlorosis was getting worse from neglect of treatment; and in a still larger number they entirely disappeared when the anæmia was removed by the administration of iron. It was therefore evident that the mitral incompetence was due to dilatation, and not to any structural change in the valve itself. I cannot doubt that dilatation of the left ventricle plays a very important part in the production of these common mitral *bruits* in children. I am convinced that the small vegetations commonly observed on the edges of the valves in chorea do not explain the bruit which is so frequently heard in the course of that disease, and which is comparatively seldom permanent. For although such vegetations are rarely absent when a *post-mortem* examination is made after chorea, we must recollect that it is only in cases of altogether exceptional severity that death occurs. So that it is rather an unwarrantable assumption that endocarditis is a frequent accompaniment of the ordinary mild type of the disease which recovers. But there is a much stronger reason for doubting the causal relation of these tiny vegetations to the *bruits* in question. It is not at all likely that such vegetations would interfere with the closure of the valve. And, therefore, they would not produce a systolic *bruit*; if they produced any *bruit* at all it would be pre-systolic. But the bruit in chorea is usually, if not always, systolic. Probably these minute vegetations, when they do develop, have nothing to do with the ordinary transient *bruit* of chorea. I think that ventricular dilatation is a more satisfactory explanation than irregular—choreic—action of the musculi papillares. I once observed, in a girl, aged 11, who had suffered from chorea, a *bruit* audible at the pulmonary area only, the first sound at the apex being normal. The *bruit* disappeared under treatment. In another patient, whose age was 24 when I examined her, and who had suffered from chorea following a fright when she was about 10 years old, I found a *bruit* presystolic and systolic at the apex and systolic at the angle of the scapula. Here I should be disposed to attribute the damaged condition of the mitral valve to an attack of endocarditis such as I have above described, although I obtained no history pointing in that direction. This seems rather more probable than to lay the blame of such extensive damage to the chorea directly.

It should never be forgotten that, when signs of mitral regurgitation occur in the course of acute rheumatism, this is not necessarily due to endocarditis and its consequences. In a large number of instances a temporary dilatation of the left ventricle offers the more reasonable explanation. This suggests that all such cases should be treated by prolonged rest, with heart tonics, including iron. Even where actual valvular deformity exists, prolonged rest gives perhaps some chance of complete recovery, and certainly affords the best opportunity for the establishment of satisfactory compensation.

Dr. Townsend said that cardiac disease in children was due in most cases to a rheumatic cause, either hereditary or acquired, and was generally very insidious and not indicated by symptoms. To be found it must be looked for.