Of the cardiac affections, endocarditis is much the most common, the mitral area being especially vulnerable. Endocardial inflammation generally begins early in the rheumatic attack —in the first week usually, but may occur in the second, the third, or even the fourth week. The more severe the rheumatic attack, the greater the liability to the endocarditis. If the patient escapes for the first week, and is under suitable care and medication, some believe that the heart should be secure from attack. It is the general opinion that endocarditis is proportionately much more liable to occur in second, and still more so in third, attacks of rheumatism, even although the successive attacks be less severe. There is a very probable fallacy in this view. No doubt in many cases of rheumatism there occurs inflammation of endo- or pericardium, or both, without manifesting any signs of its existence; permanent thickening of the endocardium may, however, result, and become at the affected points more vulnerable to the rheumatic poison in subsequent attacks. This offers the only reasonable explanation of this greater liability to cardiac implication in repeated attacks of rheumatism, as otherwise the liability should decrease with advancing age, and lessening in severity in the recurrent attacks. The truth of this is further borne out by the experience we have probably all had of cases who, having convalesced from rheumatism, have passed out of our hands without any signs of cardiac lesions that could be detected, and who some time later showed unmistakable evidences of heart disease, it may be, of a most serious nature. The greater frequency of heart disease in several attacks of rheumatism was believed by Sibson to be due to the increased strain thrown on the heart by the severity of the disease.* The fibrous structures subject to most strain seem to be most liable to attack; the increased labor of the heart may, therefore, induce inflammation of its fibrous structures.

In children, as with rheumatism, so it is with its cardiac phenomena, they are nearly always mild and trivial; all may disappear for a season, yet they too often recur, soon to persist, until the valve injury becomes serious, and, finally, fatal. In the rheumatism of children the slightest causes may induce relapses. They frequently tax the patience of the physician, and too often shake the confidence of the parents in his skill and treatment. In these recurrent attacks lies the danger to the child, as with each he becomes increasingly liable to disease of the heart. If the heart becomes once affected, the lesion is sure to increase with each relapse.

Such cases of rheumatism call for the most judicious management perseveringly carried out until the rheumatic condition has been wholly eradicated.

In endocarditis the inflammatory infusion takes place into the fibrous tissue of the membrane; the suface changes follow later. As compared with the serous membranes, as the pericardium and pleura, the inflammatory process is very circumscribed; this is owing to its slight vascularity. The reason hitherto assigned by most authors for the frequency with which the mitral valve is affected, and the rarity of the aortic, has been the greater strain to which the mitral is subjected. Later authors* give another cause which seems, on the whole, to be more potent, namely, the fact that the central parts of the mitral segments have some vascular supply while the aortic segments are quite non-vascular. The onset of endocarditis may be accompanied by pyrexia and an appearance of illness and distress in the child's face, even while at play; or the heart's action may be tumultuous with dyspnœa, restlessness, and anxiety from imperfect circulation. But such symptoms occur only in the severer cases. Valve disease gives no physical sign of its existence until it results in some deformity of the valve which either impedes or disturbs the current of blood in its passage through the orifice to whose margin its segments are attached, or impairs the functions of the valves so as to permit a reflux of blood through the orifice which they guard.

Sibson† says we are warranted in assuming that, in a considerable number of cases, the active stage of endocarditis is passing away at the time of the appearance of a murmur. As a general principle, it may be stated that the milder the endocardial inflammation the longer will a murmur be in appearing, and *vice versa;* in many mild cases, certainly no murmur ever appears. It is

* Reynold's System of Medicine.

*Ziegler's Pathology.
†Ibid.