often affected, because its central parts have a good blood supply. The aortic valve has not such a good vascular supply and is therefore more apt to escape. The cardiac murmur, according to Sibson, is not the sign of a commencing endocarditis, but occurs when the inflammation is passing off. The first sign of mitral obstruction is seeming reduplication of the first sound, heard only at the apex; of mitral incompetence, prolongation of first sound heard at apex. Murmurs occur earlier in rheumatism than in such diseases as typhoid. Pericarditis and myocarditis are often overlooked and misinterpreted; they occur most often in first attacks, severe attacks, and between the ages of fifteen and twenty-five years. Severe and fatal cases may occur without any symptoms. The immediate prognosis of the cardiac lesion is favorable, because in young children in whom the lesion is most apt to occur compensation is more quickly established and is more complete, therefore it is but seldom that cyanosis or dropsy are seen. In older patients the prognosis depends on the extent of the sclerotic and atheromatous changes. In the well-to-do the prognosis is better, because they are subject to less exposure and to less violent exertion.

Unfortunately, there is no treatment by which we can prevent rheumatism; the great object should be to save the heart; this may be done by arresting the rheumatic process with alkalies or the salicylates, by promoting excretion in order to purify the blood, by good nourishment in the form of light liquid food, and by rest. Great caution should be observed in the use of alkalies in anæmic cases, for they, by increasing the anæmia, may cause the cardiac lesion. In such anæmic cases iron is preferable. Rest should be insisted on in children, particularly in boys.

Dr. Macallum, of London, said that the endocardium was subject to the same sclerotic changes as the aorta; these were seen chiefly at the valves, but might exist on any part of the endocardium. The myocardium might also be affected, but it is impossible to express an opinion as to its condition, save after microscopical examination. The dilatation of the heart was due, *not* to want of contractility, but of elasticity. Rheumatic patients should have special attention paid to the heart, owing to the round cell infiltration and vascularisation of the endocardium. He believed that for a year after convalescence the patient should be put on a course of alkalies, iodide of potash, and iron; the iodide of potash given for the same reason as in aneurism.

Dr. Sheard, of Toronto, did *not* think that second attacks of rheumatism were more likely than primary ones to produce cardiac lesions. A slight swelling, a tumefaction, as it were, of the valves, he considered the real cause of the first appearance of the cardiac murmur. Each recurring attack increased the deposition on the valves. He did not agree with the reader of the paper in believing that pericarditis was often overlooked. He had seldom seen it *post mortem*; it was easily diagnosed, and hence he was led to believe it of rare occurrence in rheumatism. Endocarditis was the lesion nearly always present. Præcordial distress might be the only sign of it—might occur even before a murmur was to be heard. The heart was best treated by rest. Iron is useless in the anæmia of endocarditis unless the patient was enjoying perfect rest. He believed this to be true of the use of iron in all cases of anæmia, due to any cause whatsoever. Time and again he had seen this in anæmic girls. He preferred salicylates to the alkalies. He would use alkalies for twelve months after the attack.

Dr. McPhedran, replying, said that he failed to see the similarity of argument for the use of potassium iodide in aneurism and in rheumatism. Dr. Macallum, no doubt, thought that it would act as an alterative, removing the cell infiltration. He objected strongly to the iodide in any event, for it tended to produce anæmia, and this was the great thing to avoid. The tumefaction, adduced as a cause of the murmur, he did not consider a sufficient cause, nor did he believe it such an easy matter to detect the cardiac lesion, endocardial or pericardial, in the first attack. His experience, substantiated by *post mortem* evidence, was that pericarditis occurred more generally than was thought to be the case. Time and again in hospital practice he had been chagrined to find, on *post mortem*, well-marked pericarditis, which had not been diagnosed during life. He had not been alone in this, for his colleagues had equally often met the same fate. Præcordial distress, insisted on as a symp-