3. The character of the disorder is established as definitely infectious in:

Group 2.—Scarlet fever, smallpox, dengue.

4. An infectious origin is presumed on good analogical evidence in:

Group 3.—Acute articular rheumatism, acute arthritis deformans, Still's type, and the erythema group.

5. When the infectious diseases with acute joint symptoms and known etiology and also those infectious diseases with established identities but still doubtful etiology have been taken out, there remains another group which contains acute articular rheumatism, acute arthritis deformans and the erythema type of lesions. That specific toxic agents are responsible for these conditions seems a matter of no doubt, and that these agents differ from each other seems likely.

6. The final diagnosis of acute articular rheumatism can be made only when Groups 1 and 2 have been excluded and when there are clinical differences clearly separating the diseases in question from the other members of Group 3.

7. There are cases in all groups which can be diagnosed only by the lapse of time, and some which can not at present be differentiated at all.

CARDIAC COMPLICATIONS OF ACUTE RHEUMATISM.*

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In presenting this paper I have accepted the title given me, although I believe that in so stating the occurrences of cardiac involvement in rheumatism we misstate the real condition of affairs and thus tend to create a false conception. Cardiac complications of rheumatism are but one manifestation of a symptom of the disease, and the involvement of the heart, in any attack of acute rheumatism, should be accepted as one of the possible and unfortunately very probable symptoms that will be manifested in the course of any attack.

This is equally true of endocarditis, pericarditis and myocarditis and in my opinion there are but few attacks of the disease which do not show some involvement of the valves, the muscle or the surrounding envelope of the heart.

It is a familiar observation in children to see the cardiac involvement be the main manifestation of the disease, and the joint symptoms of slight intensity. In adults, it has probably occurred in the experience of nearly all of us, to have observed a pericarditis or endocarditis manifest itself several days before the joint symptoms became evident. It certainly, therefore, seems wrong to consider as complicating occurrences lesions which occur with so great frequency and which may be the primary or even the principal manifestation of the disease.

The frequency with which rheumatism attacks the heart is usually given as from 25 to 33 per cent. In the cases recorded by Church there were 889 in which 494 or 57.5 per cent. showed signs of old or recent endocarditis; Thompson records 706 cases, occurring in the Presbyterian Hospital of New York, in which the cardiac manifestation occurred in 329, or 46.6 per cent. In 500 cases admitted in the last three years to Bellevue Hospital 253 showed recent or old endocarditis, or 50.6

per cent. We have, therefore, a total of 2,095 cases in which 1,076, or 51.3 per cent., showed cardiac manifestations. What percentage of the others here quoted were other forms of infective arthritis and not genuine acute articular rheumatism it is impossible to say. But in the 500 cases in Bellevue Hospital, just quoted, there were no cases included in which the small joints of the hands and feet were involved, and always two or more of the large joints showed an inflammatory arthritis. This was done to reduce to a minimum the chances of including any forms of arthritis, except the genuine acute articular rheumatic affection.

It seems safe to conclude that half the cases of acute rheumatic fever will show cardiac involvement. The age at which rheumatism occurs modifies greatly the liability to cardiac involvement; under ten years of age the liability is the greatest. The figures of Church give over 80 per cent. as the occurrence for this decade. The liability decreases with each decade, until over forty years of age the percentage is but 34 per cent. The liability to cardiac involvement in the first attack, for all ages, is 50 per cent. Here, too, taking the figures of Church, the age at which the attack occurs manifests its influence, in children under ten, 75 per cent. showed acute endocarditis and the percentage diminished until in patients over forty endocarditis was present in 12.5 per cent. In 244 first attacks his figures show 124 cases of acute endocarditis, or 50.8 per cent. In 177 cases in my statistics 89, or 50.6 per cent., showed acute endocarditis. Osler shows that while the liability to endocarditis diminishes as age advances, it increases directly with the number of attacks, while of 116 cases 58 per cent. had endocarditis in the first attack, 33 per cent. in the second attack and 71 per cent. in the third attack.

Rheumatic endocarditis forms such a large proportion of all the cardiac cases going into a general hospital, that it occurred to me that it would be of interest to see what proportion of patients, suffering from a breakdown of cardiac compensation, gave a rheumatic origin as the cause of their disease. In 200 cases of chronic endocarditis in Bellevue Hospital 125, or 62.5 per cent., were of rheumatic origin, and 75, or 37.5 per cent., were of non-rheumatic origin. As is well known, rheumatism usually attacks the left side of the heart and usually remains confined to that side, and in the majority of cases its lesions are confined to the mitral valve. In severe cases, however, there is frequently an involvement of the right side, particularly of the tricuspid valve, for involvement of the pulmonary valve is rare. In fact, in severe cases involvement of the tricuspid valve is more common than is usually recognized. Sansom cites 28 necropsies of recent simple endocarditis in which the tricuspid valve was found involved in 50 per cent., but in 83 cases, old and recent, it was involved in only 16.28 per cent. He concludes that acute inflammation of the tricuspid valve is of frequent occurrence in acute endocarditis, and that in severe cases it is usually present. Chronic disease of the tricuspid valve, the result of chronic endocarditis, is of much less frequent occurrence. He draws the further inference, that acute inflammation of the tricuspid valve in many instances subsides and is completely cured.

The preponderance of mitral lesions in any statistics of either acute or chronic rheumatic endocarditis is very striking. In the 89 cases in Bellevue Hospital there was a mitral systolic murmur in 68.5 per cent. of the cases. There were no cases of mitral presystolic murmur, the aortic valves were involved alone in 7.8 per cent., both

* See footnote to Dr. Brown's article, page 739.

aortic and mitral valves were involved in 23 per cent., and in the cases in which both aortic and mitral valves were involved the aortic and mitral systolic murmurs were present in 20 per cent. of the total, and in 78 per cent. of the cases in which both valves were affected. The aortic diastolic murmur was the least common of any murmur. This fact, together with the absence of the mitral stenotic murmur is probably due to the same cause that the processes necessary for the production of these murmurs belong, particularly to the chronic processes, as retraction and fusion of the valves must occur to produce the lesions necessary for the production of these murmurs. It is a well-recognized fact that mitral stenosis occurs most frequently in the subacute and persistent inflammations which extend over a long period of time and are more frequently connected with the chronic myocardial changes than with acute valvular affection. Sansom quotes 300 cases, in which the mitral valve alone was involved in 157, or 52 per cent., the aortic in 40, or 13 per cent. In all of the 300 cases the mitral was affected in 255, the aortic in 129, the tricuspid in 29 and the pulmonary in three. In 283 cases of chronic rheumatic endocarditis recently analyzed by me in Bellevue Hospital, the aortic valves alone were involved in 51, or 18 per cent., and the mitral valves alone in 159, or 56.5 per cent.; both aortic and mitral valves were involved in 73, or 25.8 per cent. The tricuspid was involved in eight instances, but always in combination with other valves.

Considering the murmurs more in detail we find that in the 51 cases of aortic lesions there was an aortic systolic alone in 22, or 43 per cent., an aortic diastolic in five, or 9.8 per cent., and a double aortic murmur in 24, or 47 per cent. In the 159 murmurs at the mitral valve alone 124, or 77.9 per cent., were mitral systolic; 11, or 6.9 per cent. were mitral presystolic and 24, or 15 per cent. were double mitral. These statistics are interesting as showing the relative proportion as to the situation in the valves; they do not show or show but little as to the extent of the lesion which is likely to occur. It is a well recognized fact in cardiac affections that the extent of the damage done in the valve can only be gauged by the amount of hypertrophy and dilatation necessary to compensate the valvular lesion, and the mere record of the compensation of the lesion does not help us in this all-important point. The important point to our patient is for us to be able to estimate the extent of the lesion as judged from the compensation and to make a prognosis as to how long this compensation may be expected to continue. These questions are also intimately involved with the pericarditis and with the intensity of the myocardial changes.

In considering pericarditis in a paper of such short duration that we are only able to consider the various forms of rheumatic cardiac involvement, the ratio in which they are liable to occur in rheumatic fever, and the effect on the heart in general, we can deal but briefly with this portion of the subject. Last year Dr. Davis read an interesting paper before this section on the effects of pericarditis on the heart, and he considered the clinical changes which this condition produced; I will therefore, not touch on this aspect of the question. Pericarditis, like endocarditis, is more likely to occur in children than in adults. In a paper read last year in Boston before the Section on Diseases of Children Dr. Dunn showed that in 300 cases of rheumatism in children, pericarditis occurred in 58 cases. In my 500 cases in Bellevue Hospital, which were practically all adult cases, peri-

carditis occurred in 24. Sibson, in 1877, reported 326 mixed cases in children and adults occurring in the fifteen years ending in the autumn of 1866; of these 63 showed pericarditis.

Patients in whom pericarditis develops are usually also afflicted with endocarditis and the heart is inflamed without and within. In Sibson's cases, 9 of the 63 had pericarditis without endocarditis. In my 24 cases 4 escaped endocarditis. In Dunn's 58 cases but 2 did not show some endocardial murmur. In Sibson's cases pericarditis developed in one-sixth from the third to the sixth day after the commencement of the disease; in one half the friction murmur was audible on or about the eleventh day. In only 7 of the cases did the pericarditis show itself so late as the twenty-fifth day and from that to the sixty-third after the onset of the acute rheumatism; in one-eighth of these cases it was evident that the onset of the cardiac inflammation, both internal and external, took place at the very beginning of the disease, and at the same time with the onset of the inflammation of the joints. Dunn shows that primary pericarditis, that is pericarditis without other manifestations, is fairly common in children, and that in 30 of his 58 cases there was pericarditis alone or pericarditis with endocarditis as the sole manifestation of the infection.

In adults, pericarditis as the primary manifestation of acute rheumatism is not common, but it does occur, and I can remember three cases in the last ten years in my service in Bellevue Hospital in which the pericarditis has been the only symptom manifest for several days before the outbreak of the inflammation of the joints.

Sibson also draws some interesting deductions in regard to sex, age, occupation and the occurrence of pericarditis in rheumatics, which show that below the age of twenty-one it occurs in the two sexes in nearly equal proportions, but a little more frequently in the male than in the female. Above the age of twenty-five men were attacked three times more frequently than women. He draws attention to the great frequency of pericarditis in young servant girls. He concludes that in acute rheumatism affecting the female, that youth with labor is nearly always attacked or threatened with endocarditis or pericarditis or both, that youth without labor is thus attacked with comparative infrequency. Mature age with labor is attacked less frequently and much less severely with inflammation of the heart than youth with labor. He shows also that men following indoor occupations seem to be affected in the same ratio as the female patients, and that in men pursuing outdoor occupations, pericarditis occurs much more frequently in those of mature age when their work is hard but not when it is comparatively easy.

It is thus seen that endocarditis and pericarditis are most commonly the recognized manifestation of a general infection in children, and that in young adult life, in those whose resistance to the infection is reduced by overwork, the number of cardiac inflammations are disproportionately large. The liability to endocarditis and pericarditis disappears with advancing age, and only in those whose resistance is reduced by overwork and exposure does the liability to cardiac inflammation become great. When the pericardium is inflamed the inflammation is not limited to the serous membrane, but it extends down to the muscle of the heart, and we have to deal with a rheumatic myocarditis as well.

So far as the permanent effect on the heart is concerned, the pericarditis is much less serious than the

endocarditis, for in the majority of cases the pericarditis clears up entirely, and it is only in those cases in which the inflammation has been so severe that the two pericardial surfaces remain adherent that the permanent results from the pericarditis are at all serious. The rheumatic inflammation of the heart, whether it starts in the pericardium or the endocardium, or begins as an inflammation of the muscular substances of the heart itself, sooner or later, whenever the heart is at all affected, produces an inflammatory condition in the heart muscle, which is being more and more clearly recognized as the greatest danger in the cardiac involvement in acute rheumatism.

Pathologically, the changes in acute myocarditis seem to be acute parenchymatous degeneration of the muscles. This can be recovered from, but when the process is protracted, it can go on into fatty degeneration which may recover or which may go into brown atrophy or fibroid degeneration. This degenerative condition is seen in the old chronic rheumatic hearts in which the inflammation is intense in the pericardium or endocardium. It may extend into the muscular substances of the heart and show a round cell infiltration of inflammatory changes. In the acute stages we see, therefore, a process which is especially prone to diminish the function and produce the dilatation of the muscle. In the chronic changes we have a process which prevents the vigorous hypertrophy necessary for the increased demands of compensation and a muscle in which brown atrophy and fibroid degeneration are prone to develop and weaken its contractile powers.

A complete description of myocarditis in rheumatism, its physical signs, its course and its varying influence on acute and chronic endocarditis is yet to be written. It seems to be true that we must accept the fact that in all severe cases of endocarditis and pericarditis the myocardium is also involved. This can be a general or a local involvement, and the varying course of its intensity and of its local or general involvement, coupled with the varying degrees of involvement in a single valve or in several valves, makes the accurate unraveling of the varying influences at play in any given heart, a very difficult task. We realize from clinical evidences alone that it follows the same liability to be especially prevalent in children and apparently it follows the same laws of occurrence as endocarditis and pericarditis. It may occur in a heart in which neither pericarditis nor endocarditis show any evidences of being present.

This winter, in my wards in Bellevue Hospital, in a strong young adult man afflicted with acute rheumatism in several of the large joints, the heart suddenly became more and more rapid in its action, the pulse rose from 80 per minute, in the morning, in a few hours to 110, then to 130, then to 160, and finally became uncountable. He was restless, cyanotic and suffered from intense dyspnea. This continued for several hours, but gradually subsided and after a few days of rapid action his heart came down to normal. At no time, in this heart, were there any evidences of pericarditis or endocarditis. After ten days or two weeks the man left the hospital apparently recovered, but after a few weeks he returned with a relapse of his rheumatic joint symptoms, and his heart again showed intensely rapid and weakened action. He again recovered from this without any evidences of pericarditis or endocarditis.

Another young man of nineteen, who had an acute attack of rheumatism and showed but a slight mitral murmur, when the joint symptoms subsided, insisted on

leaving the hospital, although I tried to make him stay because I was suspicious that the cardiac processes had not subsided. In forty-eight hours he returned with an excessively dilated heart, his body generally edematous, with a large, throbbing and painful liver, face cyanotic, practically in a state of collapse.

These two cases impressed me as evidences, the first of a pure myocardial inflammation and the second of a cardiac inflammation of slight extent around the mitral orifice and of intense degree in the cardiac muscle. Myocarditis can pursue three courses, it can begin acutely, run an intense acute course and end in death (this is the least common), or it can begin with acute myocarditis, and gradually subside, or go into a long continued chronic course and last for years.

It can not be reiterated too often or emphasized too strongly that the cardiac affections of rheumatism occur in all their intensity without any ratio to the extent of the joint manifestations, and the intensity of the cardiac involvement in both children and adults is not necessarily in any ratio to the intensity of the other symptoms. Many of the symptoms which we have heretofore believed due to endocarditis occur with the myocarditis and must be considered as equally due to this condition. There is the sensation of pressure, of shortness of breath, of smothering oppression, of sensation of pain in the region of the heart, independent of any pericarditis, and these symptoms may all be present or any one or all be absent and still the myocarditis exist. Careful examination may show an increase in the area of the heart dulness or a disturbance of the cardiac rhythm or an insufficiency of the mitral valve as the first symptom. After the acute condition has subsided slight deviations from the normal are more common than severe symptoms. The physical signs of acute myocarditis are first the deepening and dulling of the muscle tone, the reduplication of the heart sounds, and, if dilatation occurs, a still further loss of muscle tone, and the sound becomes slappy and valvular. The reduplication of the sound not infrequently goes on into the so-called galloping rhythm. This is most often best heard in the fourth or fifth space, near the sternum or out toward the apex; the triple sound at the apex generally means myocardial change, and the triple sound at the base a pericardial infection.

After the acute joint symptoms have subsided, it is often noticeable that the patient's temperature does not come down to normal and remain there; there is a continued run of temperature from subnormal to subfebrile, from 97 or 98 to about 100, and it may rise as high as 101. This, in my opinion, invariably means an actively continuing endocardial or myocardial inflammation. The pulse may range near normal or be slightly above. In the subacute forms, in which the degeneration of the muscle is more common than in the acute inflammatory condition, and in the chronic condition, the pulse rate may be distinctly below normal.

Heart disease, in the early half of life, is mainly of rheumatic origin, and we have come, in late years, to realize that the treatment of heart disease has more and more become the treatment of the cardiac muscle. We have now reached the position where we must realize that our knowledge of rheumatic endocarditis is still fragmentary and very incomplete. Heretofore our knowledge has been chiefly focused on the endocardial and pericardial involvement to the exclusion of the myocardial, but to-day we realize that the cardiac involvement of rheumatism includes endocarditis, pericarditis and myocarditis, and the most serious is myocarditis.