THE CARDIAC RELATIONS OF CHOREA.

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The heart symptoms of chorea demand special consideration as among the most important and peculiar features of the disease. Chorea is rarely a fatal disease in children, and hundreds of cases may be treated without
Osler, The Cardiac Relations of Chorea.

Before discussing the probable nature of these murmurs it will be well to study the anatomical condition of the heart in fatal cases. Fortunately these are rare. I have inspected three cases.

Case I.—S., a girl, aged eleven; had had acute rheumatism. Admitted to the Montreal General Hospital, under Dr. George Ross, with acute chorea, and died of an intercurrent pneumonia. The movements had almost ceased under hypodermics of arsenic. The autopsy (No. 455 post-mortem records Montreal General Hospital) showed slight hypertrophy of the heart, somewhat thickened mitral curtains with numerous irregular warty vegetations just inside the auricular margins. Two of the aortic segments also presented bead-like vegetations below the corpora Arantii.

Case II.—T. R., a boy, aged eleven, had chorea in May, 1880, and a second severe attack in July of the same year. No rheumatism. No heart murmur. About the 20th of February, 1881, there was a recurrence, and on March 8th he again came to the general hospital to see Dr. Molson. About the 10th he began to get feverish and extremely restless. On the 14th the temperature rose above 104° F., and he became comatose. The left arm seemed powerless; the right arm and leg were constantly twitching. On the 16th the temperature reached 106° F., and there were cutaneous ecchymoses. He died on the morning of the 18th. The autopsy showed very extensive mitral valvulitis, the vegetations large, soft, grayish-white in color. No chronic affection of the valves. The spleen and kidneys contained many recent infarcts. The brain and membranes healthy, with the exception of a spot of grayish-red softening in the right corpus striatum (lenticular nucleus) about the size of a cherry. It was no doubt embolic, though the arteries of the perforated space were carefully examined for emboli without success.

Case III.—Emma M., aged eighteen, admitted to the Montreal General Hospital, under Dr. George Ross, and died in five days of exhaustion. There was no rheumatism, and the attack had followed a fright five days before admission. Here, too, the only important lesion was on the mitral valves—a row of soft warty vegetations on the auricular face just within the free margins.

The statistics of fatal cases of chorea have been collected by Sturges and Raymond. Of eighty cases, representing the combined experience of Guy’s, Bartholomew’s, St. George’s, and St. Thomas’s Hospitals, Sturges states that there were only five with the heart valves and pericardium reported healthy.

Excluding the London cases from Raymond’s table of 79 cases, there are left 34, in only 19 of which there were specific statements as to the condition of the heart, and in every one of these endocarditis was present.

I have found the reports of 15 additional cases, which, with the three

here given makes 18, in 16 of which there was mitral endocarditis. We may say that of 115 fatal cases of chorea, with notes of the state of the heart, in not more than 10 was this organ found normal, and in the great proportion of the cases the lesion was acute mitral valvulitis.

One other point must be considered before we speak of the nature of the heart murmur. In what proportion of the cases is there a history of rheumatism? In 35 of the 120 cases, 29.1 per cent., there was a note of articular affection, either acute or subacute, or of pains which might be regarded as rheumatic.

Much has been written in explanation of the heart murmur of chorea; an idea of how much may be gathered from the fact that a discussion of the theories which have been advanced occupies twelve pages in Hayden's work on Diseases of the Heart. We are concerned chiefly with the apex systolic murmur, universally recognized as the most frequent and characteristic sign of implication of the heart in chorea. Speaking generally, we meet with such a murmur in mitral endocarditis, or in relaxation of the ventricular walls, such as occurs in anaemia and fevers, and it is attributed to regurgitation through the mitral orifice, owing to absolute insufficiency, in consequence of the endocarditis, or to relative insufficiency when the normal valves are unable to close an orifice enlarged as a result of relaxation of the heart muscle. In chorea a special theory of muscular-papillary spasm has been advanced to account for the mitral murmur.

It would be fruitless to re-discuss, in all its aspects, a subject so well and ably presented in various works, particularly in those of Hayden and Sturges. That there is such a condition as spasm of the papillary muscles resulting in a “want of correspondence between the fibres of the ventricle, which obliterate the cavity and those which close the valve,” is a plausible hypothesis unsupported, so far as I know, by any clinical or anatomical facts, while the general immunity of involuntary muscular organs in chorea speaks strongly against it.

Sturges thinks that there may be a fatigue paresis of the papillary muscles, similar to that which sometimes involves the limbs, and this weakness and relaxation prevent accurate adaptation of the valve segments. He urges in support the inconstant character of the murmur, appearing and disappearing without apparent cause, and states that it may be synchronous both in its time of arrival and duration with the paresis of the voluntary muscles. I have not been able to trace any such connection, nor have I found in the paralytic cases any special tendency to variability in the murmur. Indeed, so far as my experience goes, the apex systolic bruit of chorea is by no means an inconstant murmur. If muscular incompetency has anything to do with the production of the choreic bruit, it is more likely to be of a similar character to that which occurs in anaemia, debility, and fevers. Here it is the relaxation of

the walls, and particularly the so-called mitral muscle, which induces a condition of relative insufficiency of the segments and permits of regurgitation. There may be in chorea, as is well known, a high degree of anaemia, and in a certain proportion of the cases this explanation of the murmur may hold good, but in the great majority of instances the bruit is detected early when there is neither anaemia nor debility.

I am strongly of the opinion that the apex systolic bruit of chorea is, in at least nine out of ten cases, associated with endocarditis:

1. The extraordinary frequency with which mitral valvulitis is met with in fatal cases. There is no known disease in which endocarditis is so constantly found, post-mortem, as chorea. As the figures above quoted show, it is exceptional to find the heart healthy. I do not know of statistics of any very large number of fatal cases of acute articular rheumatism to place beside these figures, but I doubt if even this disease, so prone to endocardial complication, can be compared with chorea in this respect. Dickinson has raised the question whether these beads of fibrin are not rather the consequence than the cause of the valvular defect, and Sturges holds that this appearance does not represent a true inflammation of the endocardium. Whether a true inflammation or not, I think it must be conceded that the lesion is identical, macroscopically as well as microscopically, with simple or warted endocarditis as we see it in other diseases.

2. The character and location of the murmur are such as experiences in other affections has taught us are associated with inflammation of the mitral segments. I speak of the apex bellow-murmur. Why this should be so generally associated with the presence of a row of small warty vegetations just within the auricular margins of the curtains, not, one would think, seriously interfering with their functions, is a problem to be solved. The condition certainly does not necessitate regurgitation, and the bruit may perhaps, as has been suggested, be due to friction of the roughened faces of the segments.

3. The inconstancy of the murmur and its disappearance on the subsidence of the chorea have been urged against this view. Now we must acknowledge that the bruit may be variable and, indeed, does not necessarily accompany mitral endocarditis. Kirkes, years ago, insisted upon this, and there have been two autopsies in carefully studied cases of chorea in which the vegetations were found post-mortem, and careful examination failed to reveal a murmur (Baxter: Brain, vol. ii.; Frank. Ally. Wiener med. Zeitung, 1879.) The facts which I shall subsequently give suggest that we may during the attack have an endocarditis, not manifest even by a murmur, but which has laid the foundation of future trouble. The disappearance of the apex murmur of chorea—and of rheumatism too—has been repeatedly followed, and if caused by the small vegetations, this is a natural sequence of the changes which go on
in them. At first a soft granulation tissue, they become in time firmer, smaller, and ultimately smooth flat elevations mark the spots. It is not improbable that if we could follow accurately the auscultatory history of a valve affected with acute endocarditis, we should find in many cases that the murmur of the fresh attack disappeared, to reappear when the changes, which it is the misfortune of the acute disease to initiate, have reached a point of interfering with the competency of the valve.

4. In its sequel the cardiac affection of chorea has been supposed to differ from that of other diseases, as none of the injurious after-consequences which attend endocarditis in its other relations...are found to ensue here (Sturgess). A study of any large number of choreics some years subsequent to the disease tells, as I shall show, a sad tale to the contrary and proves that the primary heart trouble is, in a majority of cases, at least, endocarditis.

II. The condition of the heart in choreic patients some years after the attack.

Owing, doubtless, to the difficulties inherent to such an investigation, this line of inquiry has not been followed by many workers. Indeed, as far as I know, Dr. Stephen Mackenzie’s paper, at the London International Congress, is the only one which has dealt with the subject, and he has examined thirty-three patients at periods from one to five years subsequent to the attack. Postal cards were sent to all the choreic patients, in sets of twenty-five, who had been in attendance at the Infirmary since 1876, asking them to return for the purpose of having the heart examined. One hundred and ten came back, a number much exceeding our expectations. All the more recent cases in attendance at the clinics have reached a point of interfering with the competency of the valve.

Some time ago, the author published a paper on the subject of the heart condition in previous attacks. Has attacks of shortness of breath. Status praecox: Impulse is forcible. Dulness increased. Apex systolic murmur heard to posterior axillary fold. Second left accentuated.

CASE II.—Kate L., aged twenty-one. Two or three attacks after 1871; bad one in 1878. In 1882, had inflammatory rheumatism, never any joint trouble before this time. In 1878, note in “impulse strong; apex murmur.” She has had attacks of shortness of breath. Status praecox: Feeble thrill; localized purring presystolic murmur. Loud apex systolic transmitted to posterior axillary fold. Second left accentuated.

1872 (fifteen years). One case. No heart affection.

1874 (thirteen years). Three cases.

CASE IV.—Annie M., aged twenty-five. Second attack in 1883, third in 1885. Had rheumatism just before the first attack. No note of heart in first or second; in 1885, an apex systolic murmur. Status praecox: Loud apex systolic transmitted to axilla; second left accentuated; transverse dulness increased; impulse forcible.


1875 (twelve years). Two cases; one normal.

CASE VII.—Hester G., aged twenty. Original attack very severe; a second in 1879, and one since. No rheumatism. No note of heart in attacks. For two years has had attacks of palpitation and dyspnoea. Status praecox: Impulse forcible. Presystolic thrill; rough presystolic murmur. Loud accentuated second left.

1876 (eleven years). Eight cases; one normal.


CASE XI.—Lizzie H., aged sixteen. Many attacks since 1876, two of them severe. Had rheumatism when four years old. In 1878, second left was reduplicated. Status praecox: No evident enlargement of heart; impulse feeble; no thrill. At apex double murmur, presystolic short, not rough. Systolic not loud, not transmitted to axilla. On
exertion louder. Both very distinct. Second left very loud. Has occasional attacks of palpitation.


CASE XIII.—Jennie A., aged twenty. Second attack in 1878, third in 1879. No rheumatism. In 1879, sound, stated to be normal. \textit{Status praesens:} Impulse not forcible, no apparent enlargement. In fourth left space a rough presystolic murmur; limited in area. At apex a systolic bruit, transmitted to axilla, and heard at angle of scapula. Second left very accentuated. Sounds at apex booming. No symptoms, always good health.

CASE XIV.—Annie L., aged twenty-four. Two attacks since, last one in 1882, when for the first time she had rheumatism. No note of heart. \textit{Status praesens:} Apex an inch outside nipple. Impulse forcible. No thrill. Presystolic murmur, not rough, in fourth space; apex systolic, heard in axilla and at angle of scapula. Loudly accentuated second left. Has had palpitation and shortness of breath on exertion for three years.

CASE XV.—Miriam C., aged nineteen. Two attacks since. Never had rheumatism. Has had heart disease for some years; is now in bed with it.

1877 (ten years). Seven cases; three affected.


CASE XXII.—Rose McF., aged twenty-four. Attack in 1877 prolonged and severe; none since. No rheumatism. In 1877, a faint apex systolic murmur. \textit{Status praesens:} Heart's action violent; impulse forcible; apex outside nipple. Marked presystolic thrill. Presystolic murmur in fourth interspace. Systolic murmur in fifth space, and heard as far as posterior axillary fold. Second sound accentuated at the second left cartilage, and also heard loudly in axilla. Patient is at times very short of breath; has attacks of palpitation and has fainted.

1878 (nine years). Two cases; one affected.

CASE XXIV.—Minnie C., aged fifteen. Attacks also in 1879, '80, and '85. Rheumatism in 1885, never before. In 1878 an apex systolic murmur. No symptoms. \textit{Status praesens:} Impulse forcible; apex outside nipple-line; transverse dulness increased. Apex systolic murmur heard to posterior axillary fold. Double murmur at aortic cartilage; diastolic heard also on sternum. Second left not accentuated.

1879 (eight years). Four cases; all affected.

CASE XXVI.—Fannie N., aged fifteen. Second attack in 1885. Has had rheumatic pains, but no swelling of joints. In 1879 had pain about the heart, and since then had occasional attacks of palpitation on exertion. \textit{Status praesens:} Impulse in fifth a little out. Transverse dulness increased. Presystolic thrill, most marked at apex. Rough presystolic murmur at and just above the apex. Soft systolic at and outside apex beat. Second left much accentuated, and is also very ringing and loud in axilla and at angle of scapula.


CASE XXIX.—Mary G., aged thirteen. Several attacks since 1879; in 1885 a bad one, and now. May, 1887, is in infirmary with a severe attack. Rheumatism in 1885 with chorea, not before; and this time has had swollen joints. In 1885 had systolic apex murmur. \textit{Status praesens:} Impulse in fifth and sixth, outside nipple. Dulness increased. Loud apex systolic bruit propagated to axilla and scapula. Second left much accentuated. Has had attacks of cardiac dyspnea in which she could not lie down. At times severe pain at heart.

1880 (seven years). Five cases; three affected.

CASE XXX.—Ellen McG., aged twenty-three. No rheumatism. No note of heart in 1880. Is anemic; has palpitation, shortness of breath, and at times severe pain at heart. \textit{Status praesens:} Action rapid and forcible; dulness increased. Presystolic thrill all over mitral area. Rough presystolic murmur. Soft systolic bruit just outside apex. Second left is loud but not specially accentuated. Examined again some weeks after a course of iron and arsenic, which had relieved the anemia; murmurs unchanged.

CASE XXXII.—Angela W., aged eighteen. Four attacks since the first in 1880. No rheumatism. Heart, in 1884, said to be normal. Has had pain at heart, and is at times short of breath. \textit{Status praesens:} Action forcible. Soft apex systolic, heard as far as middle axilla, and increased on exertion, not altered by position. Second left a little accentuated.

CASE XXXIV.—Florence B., aged twenty. Rheumatism six months before this attack. In 1880 an apex systolic murmur. Has had since then occasional attacks of palpitation. \textit{Status praesens:} Pulse forcible; apex a little out, but no special enlargement. Apex systolic murmur, heard well to middle axilla. Marked accentuation of second left.
CASE XXXVI.—Louis O., aged seventeen. At least five attacks since 1881. No rheumatism. No note of heart. No symptoms. Status praesens: Apex beat in fourth space in nipple line, having and forcible; dulness increased. Loud systolic murmur at apex heard to posterior axillary fold, but not above fourth space. When recumbent it is heard in second and third spaces as well. Second left very accentuated. No enlargement of heart. Has at times pain in knees in 1882, and lately in shoulders. Heart normal in 1881.

CASE XXXIX.—Frank N., aged thirteen. A second attack in 1884. No rheumatism. Heart said to have been normal in 1884. For some time has been very short of breath, and gets tired on exertion. Status praesens: Precordia bulges. Impulse diffuse; dulness increased. Presystolic thrill in fourth interspace. A blubering presystolic murmur. Maximum intensity in fourth space. Loud blowing systolic bruit; heard also in axilla. Very accentuated second left. Aortic second feeble.


CASE XLI.—Joseph M., aged thirteen. First attack January, 1881; second, October, 1881. No rheumatism. In 1881 a soft systolic murmur. Has had vertigo and rushes of blood to head. Apex in sixth space an inch outside nipple line, is well heard to anterior axillary fold. Loudly accentuated second left. No systolic murmur even when recumbent.


CASE XLIII.—Mary B., aged sixteen. Three or four slight attacks since 1881. In 1881 pains in joints, no swelling. In 1881 an apex bruit. Has had no heart symptoms. Status praesens: No enlargement. When erect, sounds clear; recumbent, systolic bruit at second left, with marked accentuation of second sound.


CASE XLIX.—Jessie J., aged nineteen. Three attacks since. Rheumatism with attack in 1883, and again in 1885. Heart said to be normal in 1885. Status praesens: Beat in fifth space outside nipple. Dulness increased. At apex a soft systolic bruit, not heard in axilla, except after exertion. In fourth space, in localized region, a soft diastolic murmur, not increased toward sternum, not heard at aortic or pulmonary cartilages; it also is intensified by exertion. Has “attacks at the heart,” faints, and gets cold. Has much pain at times and is short of breath.

CASE L.—Tillie M., aged fifteen. Attacks also in 1883 and 1886. No rheumatism, but lately has had pains in shoulders. No note of heart. Has had at times pain at heart and palpitation. Status praesens: Apex beat just within nipple, a little forcible. Apex systolic bruit heard along anterior axillary fold and in middle axilla. Second left accentuated.

CASE LII.—Annie B., aged eighteen. No rheumatism. In 1882 a loud apex systolic bruit. Has had shortness of breath and palpitation. Status praesens: Beat forcible, outside nipple line; dulness increased. Apex systolic bruit heard also in axilla and at angle of scapula; also as high as second rib. Second left loudly accentuated.


CASE LIV.—Bessie P., aged thirteen. Second attack in 1883. Rheumatism in hands and feet with first attack. Heart said to have been normal. Status praesens: Impulse forcible. Apex in sixth space an inch outside nipple line. Slight presystolic rumble at apex. Loud systolic murmur in second and third interspaces, not so marked at apex. Second left loudly accentuated. No symptoms. Status praesens:

CASE LV.—Harriet H., aged eight. No rheumatism. Died of heart disease with dropsy, November 8, 1883.


CASE LX.—Fannie S., aged eleven. Second attack in 1883, third in 1884, and fourth in 1885. Rheumatism in 1883; severe attack. In November, 1882, a basic systolic murmur, which persisted in 1884. In June, 1885, there were hypertrophy and evidence of aortic and mitral disease. Died of cardiac dropsy, July 11, 1886.

CASE LXI.—Catherine B., aged thirteen. A second slight attack in spring of this year. No rheumatism. No note of heart in 1882. Status praesens: Impulse forcible, at and a little outside nipple line. Dulness
increased. Feeble presystolic thrill. Loud apex systolic murmur, propagated to axilla. In fourth space just within nipple, a rumbling presystolic murmur. Second left very accentuated. Had at times severe pain in heart; no shortness of breath.

1883 (four years). Fifteen cases; eight affected.


CASE LXVI.—Nellie H., aged nine. Second attack in 1884, third in 1885. No rheumatism. No note of previous heart-condition. Status praesens: Apex beat diffuse, maximum in sixth space, one inch outside nipple line. Dulness increased. No thrill. Loud apex systolic murmur transmitted to angle of scapula. Just below and inside the nipple a soft presystolic bruit. Second left much accentuated. In December, 1886, the child had a sharp attack of cardiac dyspnea.

CASE LXVII.—Edward R., aged twelve. Second attack in 1885. No clear history of rheumatism; has had pains. No note of heart. Status praesens: Beat in fifth, just outside nipple line. Dulness increased. Just above apex, in localized region, a presystolic murmur; louder in recumbent posture. When breath is held, soft apex systolic murmur. Second left much accentuated.


CASE LXXX.—John D., aged eighteen. Second attack in 1886. In 1887 slight rheumatism. In 1884 soft murmur at base. Status praesens: Apex outside nipple line. Cardiac shock in right side. At fourth left and up and down the sternum is a long-drawn diastolic murmur, of maximum intensity on sternum, opposite fourth cartilage. Heard at right carilage and at xiphoid. No nortic systolic bruit. Second left very accentuated. Posture did not alter the murmurs. He had no heart symptoms.

CASE LXXXII.—Kate H., aged fifteen. Rheumatism very badly at the time. Heart said to be normal. Status praesens: Apex beat forcible, outside nipple line. Cardiac shock over a large area. No thrill. Loud apex systolic murmur propagated along anterior axillary fold. Second left much accentuated. Has no heart symptoms.


CASE LXXXVI.—Lillie D., aged twelve. No rheumatism. Heart normal in 1884. Status praesens: No enlargement. When recumbent, a soft, long, apex systolic murmur, not heard in axilla or in second or third spaces. Disappears when erect. Second dull and loud, not sharp and ringing, like second right.
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CASE LXXXIX.—Ann T., aged thirteen. Several slight returns since 1884. Rheumatism three months after the chorea. No note of heart in 1884. Status præsens: Action rapid, apex a little out from nipple line. Dulness increased. Loud, rough systolic bruit at apex, transmitted to scapula. Second sound very accentuated at third left cartilage. Has "spells" with her heart; has fainted. Is short of breath on exertion.

1885 (two years). Eighteen cases; five affected.


CASE XCVII.—William R., aged nine. No rheumatism. Heart in 1885 said to be normal. Status præsens: No enlargement. First sound not clear, and on exertion a soft systolic murmur at apex; heard also two inches beyond nipple, and as high as third rib. Loudly accentuated second left. Has no symptoms.


CASE CI.—Jennie N., aged nine. Second attack in 1886, in which she had rheumatism. Heart in 1885 normal. In 1886 loud apex systolic murmur. Status præsens: Impulse forcible, apex in nipple line. Dulness increased. Apex systolic transmitted to axilla and angle of scapula; heard also as high as second rib. Second left loudly accentuated. Has, at times, throbbing, palpitation, and pain.

Of the 43 cases in which the heart was found normal, 12 had three or more attacks, 8 had had two, and 23 a single attack. There was a history of rheumatism in 8—i.e., 18.6 per cent. In 6 of these cases the rheumatism was acute. In only 2 cases had there been a murmur noted at the time of the original attack.

From the cases presenting abnormal physical signs, 13 may be separated as examples of functional trouble. They are cases without signs of enlargement of the heart and with localized or variable murmurs. Ten presented soft apex systolic bruits not propagated, in 3 variable with position. In most of these there was accentuation of the second left pulmonary sound, but I do not think much stress is to be placed upon this sign in young persons, as it is by no means uncommon in normal hearts. Particular attention was paid to this point in the examination of all the cases and comparison made between the sounds in the second right and second left spaces. There were 10 normal cases in which the pulmonary sound was distinctly louder than the aortic, and in some instances reduplicated. No note was taken of the murmurs, so often developed in the region of the pulmonary artery during respiration and which are extremely common in thin-chested children. In 2 cases the sounds in this region were clear in the erect posture, but in the recumbent position systolic bruits developed; in both the second sound was accentuated, and in one the area of pulsation somewhat increased. In a third case there was a soft systolic murmur in the second and third spaces in the recumbent position only, with accentuation of the pulmonary sound and the apex beat outside the nipple line. In some of these there may have been organic changes in the valves, but I deemed it best to exclude all doubtful cases.

There remain for consideration 54 cases with signs of valve disease. In 21 cases there had been three or more attacks of chorea.

The facts regarding rheumatism are interesting. In 22 cases, 40.7 per cent., there was a distinct history of articular trouble, sometimes with the chorea, but in 6 cases from one to five years after the attacks. Comparing the frequency of rheumatic affection in this group, 40.7 per cent., with that in the total number of cases, 15 per cent., or with the group of 43 normal cases, 18.6 per cent., we see the influence this disease exercises in producing the heart lesions. We have, however, the larger proportion, 59.3 per cent., of the cases without any history of rheumatic trouble. Of the 21 cases which had had three or more attacks of chorea, only 7 had rheumatism.

In this group there are rather more than 3 females to 1 male, a proportion considerably greater than in the total number of cases.

With reference to the nature and seat of the lesion, there were 44 cases of uncomplicated mitral affection and 4 instances of combined aortic and mitral disease. In 25 cases there was a mitral systolic murmur; in 17 a distinct presystolic murmur, with or without a thrill, and usually with a systolic bruit. Of the aortic lesions Case XII presented a soft aortic direct murmur and a mitral systolic; Case XXIV, a double aortic murmur as well as a mitral systolic; Case LX died of combined aortic and mitral disease; Case LXXXII, presented the unusual combination of an aortic diastolic and a mitral presystolic murmur. The overwhelming proportion of cases, with mitral lesions, is what we might expect from the constancy with which the acute endocarditis of rheumatism and chorea attacks these valves.
There are many points of interest in physical diagnosis which these cases illustrate, but I am only concerned now with the clinical problem of the frequency with which organic heart disease follows chorea.

Not many of the cases had subjective symptoms of cardiac disease. In 14 instances there was complaint of shortness of breath; 16 cases had attacks of palpitation, and in 6 cases there was cardiac pain. Two cases had died of heart disease, 1 was in bed with cardiac dropsy, and in several others there were premonitions of heart failure. The majority illustrated the important clinical law in valvular disease, that the symptoms do not result from the lesion, but from failure in the compensatory action which for years may equalize the circulation and obviate completely the most serious mechanical defect.

A study of these cases justifies, I think, the following conclusions:

1. That in a considerable proportion of cases of chorea—much larger than has hitherto been supposed—the complicating endocarditis lays the foundation of organic heart disease.

2. In a majority of the cases the cardiac affection is independent of rheumatism, and cannot be regarded as in any way associated with it; unless, indeed, we hold with Bouillaud, that in the disease "chez les jeunes sujets le cœur se comporte comme une articulation."

3. As the presence of an apex systolic murmur in chorea is usually an indication of the existence of mitral valvulitis, as much care should be exercised in this condition as in the acute endocarditis of rheumatism. Rest, avoidance of excitement, and care in convalescence, may do much to limit a valvulitis, and obviate, possibly, the liability to those chronic nutritional changes in the valves wherein lies, after all, the main danger.