
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

a death. By far the most serious fact in the clinical history of the disease is the occurrence of endocarditis; but here the danger is remote, not immediate, and lies in the changes which an acute valvulitis may initiate.

A satisfactory study of the cardiac relations of chorea must embrace the condition during the attack, and the subsequent heart history after a period of years. The first question has engaged the attention of many workers, and an attempt is here made to work out the second on a scale not hitherto attempted.

I. Condition of the Heart during the Attack.

Oftentimes the extreme jactitation renders the examination of a choreic child difficult or even impossible. I make it a rule to examine the bare chest. Auscultation through the clothing is not trustworthy, as soft murmurs, readily audible with the stethoscope, may easily escape detection. It is a good plan to let the child lie quietly on a lounge for some time, and make the first examination in the recumbent position when the heart's action is less rapid. Subsequently the effect of exercise and of the erect posture may be tested.

In chorea, as in rheumatism, the evidences of cardiac disease must be sought for, as it is rare to hear complaints of either palpitation, pain, or other symptoms which would direct attention to the heart.

The cardiac disturbance is indicated by the presence of murmurs, alteration in the rate or rhythm of the heart's action, and by pain.

A murmur at one or other of the cardiac areas is by far the most common sign and is present in a considerable number of all cases. Of 410 cases in the records of the Infirmary for Nervous Diseases, there were 120 which presented a heart murmur at the time of examination. In at least 40 cases there was either no note or an imperfect one, and in very many the exigencies of out-patient work prevented a very thorough examination. It can safely be said that in over one-third of the cases a heart murmur was detected, and I have no doubt that this number would have been much increased had each child been stripped and special attention given to the auscultation of the heart.

Of the 120 cases, 113 presented the apex systolic or mitral murmur, in 7 a basic, and in 3 both apex and basic. In 15 cases the heart's action was noted as rapid, and in 6 as irregular. Pain was not a frequent complaint and was noted in only 6 or 7 cases.

It is common experience that the special indication of heart trouble in chorea is the presence of a soft systolic bruit, heard best at the apex or over the body of the ventricles and not often propagated to or beyond the mid-axilla. Basic systolic murmurs are usually associated with anæmia or debility. Diastolic and presystolic murmurs rarely, if ever, occur in acute 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. 465 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. B., 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 3d 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 15th the temperature reached 105° F., and there were cutaneous ecchymoses. He died on the morning of the 16th. 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,[1] 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[2] and Raymond.[3] 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,[4] which, with the three

[1] Canada Medical and Surgical Journal, vol. xi.

[2] Chorea. London, 1881.

[3] Dictionnaire encyclopédique des Sciences Médicales.

[4] Mackenzie (Trans. Inter. Med. Congress, 1881), six cases, five of endocarditis. Donkin and Hebb, 1 case, valves normal (Med. Times and Gaz., 1884, ii.). Baxter (Brain, vol. ii.) one case. Morell-Lavellée (Revue des Maladies de l'Enfance, 1884), one case. Frank (Allg. Wiener med. Zeitung, 1879), one case. Maixner (Med.-Chir. Centralblatt, Wien, 1882), one case. Koch (Deutsches Archiv f. klin. Med., Bd. xl.), four cases

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 anæmia and fevers, and it is attributed to regurgitation through the mitral orifice, owing either 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 musculo-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 paretic 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 anæmia, 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 anæmia, 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 anæmia 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, microscopically as well as macroscopically, with simple or warty endocarditis as we see it in other diseases.

2. The character and location of the murmur are such as experience in other affections has taught us are associated with inflammation of the mitral segments. I speak of the apex bellows-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. Allg. 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" (Sturges). 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, so 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.[1] All the more recent cases in attendance at the clinics have been excluded—all, indeed, after March, 1885, so that the study is based upon 110 cases in which the examination was made *more than two* years subsequent to the attack of chorea. In each case, as it came, reference was made to the original notes, questions asked concerning subsequent attacks, and rheumatism, and the heart examined in the recumbent and erect postures, at rest and after exertion.

The results summarized, are as follows: In 43 cases the heart was normal, in 54 there were signs of organic disease, and in 13 there was functional disturbance.

The tables which I have prepared are too full for publication, but the following abstracts of the cases affected will be of interest:

1871 (sixteen years). Two cases.

Case I.—Laura C. R., aged twenty-five. Several attacks subsequent to 1871. Never had rheumatism until February, 1887. No note of heart condition in previous attacks. Has attacks of shortness of breath. *Status præsens:* Impulse is forcible. Dulness increased. Apex systolic murmur heard to posterior axillary fold. Second left accentuated.

[1] It speaks well for the stability of the artisan class in Philadelphia that so many of the postal cards reached their destination. Comparatively few were returned from the Post-office with the comment—*Removed; cannot find.*

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 is "impulse strong; apex murmur." She has had attacks of shortness of breath. *Status præsens:* 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 præsens:* Loud apex systolic transmitted to axilla; second left accentuated; transverse dulness increased; impulse forcible.

Case V.—Bertha G., aged twenty-five. A second attack in 1880. No rheumatism. In 1880, a soft systolic murmur. *Status præsens:* Impulse not forcible. Loud apex systolic murmur propagated to axilla. Very ringing and accentuated second left. Has palpitation and attacks of shortness of breath.

Case VI.—Charles M., aged twenty-eight. Second attack in 1880. Had pains in joints before second attack. No note of heart. Is strong and well, no subjective symptoms. *Status præsens:* Soft apex systolic murmur, not heard in axilla or in pulmonary area. No increase in dulness. Second left accentuated.

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 dyspnœa. *Status præsens:* Impulse forcible. Presystolic thrill; rough presystolic murmur. Loud accentuated second left.

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

Case IX.—Annie T., aged seventeen. Since 1876 three attacks, last in 1885. No rheumatism. In 1885, a soft systolic murmur. Complains that she does not lie comfortably on left side. *Status præsens:* Impulse forcible, outside nipple. Apex systolic loud, heard well in axilla. Second left accentuated.

Case X.—Robert P., aged twenty-one. Second attack in 1879. No rheumatism. No previous note of heart. *Status præsens:* Action rapid, impulse diffuse. Dulness not increased. Blowing systolic murmur just above apex, not heard in axilla; disappears on exertion. Second left accentuated.

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 præsens:* 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 XII.—Ida L., aged eighteen. Three attacks since 1876. No rheumatism. No note of heart in 1879. No symptoms. *Status præsens:* Beat forcible; dulness increased. Loud apex systolic murmur, heard at angle of scapula and very distinct along left margin of sternum. At aortic cartilage a soft systolic bruit. Second left ringing and accentuated.

CASE XIV.—Jennie A., aged twenty. Second attack in 1878, third in 1879. No rheumatism. In 1879, sound, stated to be normal. *Status præsens:* 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 XV.—Annie L., aged twenty-four. Two attacks since, last one in 1882, when for the first time she had rheumatism. No note of heart. *Status præsens:* 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 XVI.—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 XVII.—Andrew G., aged twenty-one. The attack followed acute rheumatism. In 1878, a soft systolic murmur. No symptoms. *Status præsens:* When recumbent sounds clear. Erect and after exercise well-marked apex systolic, not transmitted. Second left ringing, accentuated, and reduplicated. No enlargement of the heart.

CASE XX.—Mamie L., aged fifteen. Rheumatism (acute) four weeks before onset of chorea in 1877. No attack since. In 1877, "mitral murmur." No symptoms. *Status præsens:* Impulse forcible, beat outside nipple line. Transverse dulness increased. Loud apex systolic murmur, propagated to posterior axillary fold. Second left very accentuated.

CASE XXIII.—Rose McF., aged twenty-four. Attack in 1877 prolonged and severe; none since. No rheumatism. In 1877, a faint apex systolic murmur. *Status præsens:* 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. *Status præsens:* 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 has had occasional attacks of palpitation on exertion. *Status prsœns:* 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 XXVII.—Lizzie R., aged twelve. Three subsequent attacks, 1880, '83, and '86. Those of 1879, '80, and '83 very severe. No rheumatism. No previous note of heart condition. Has had no heart symptoms. *Status prœsens:* Forcible, diffuse impulse. Apex a little outside nipple. Systolic murmur at apex transmitted to axilla and heard feebly at angle of scapula. Second left very accentuated.

CASE XXVIII.—Rose F., aged thirteen. Second attack in 1881. Heart normal in 1879. Has been short of breath, particularly on exertion. *Status præsens:* Impulse strong. Transverse dulness increased. Rough presystolic thrill. Very rasping presystolic bruit. Maximum intensity in fifth, just within nipple. Second left accentuated and reduplicated. Aortic sounds feeble.

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. *Status præsens:* 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 dyspnœa in which she could not lie down. At times severe pain at heart.

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

CASE XXXII.—Ellen McG., aged twenty-three. No rheumatism. No note of heart in 1880. Is anæmic; has palpitation, shortness of breath, and at times severe pain at heart. *Status præsens:* 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 anæmia; murmurs unchanged.

CASE XXXIII.—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. *Status præsens:* Impulse 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 the attack. In 1880 an apex systolic murmur. Has had since then occasional attacks of palpitation. *Status præsens:* Impulse forcible; apex a little out, but no special enlargement. Apex systolic murmur, heard well to middle axilla. Marked accentuation of second left.

1881 (six years). Sixteen cases; nine affected.

CASE XXXVI.—Louis O., aged seventeen. At least five attacks since 1881. No rheumatism. No note of heart. No symptoms. *Status præsens:* Apex beat in fourth space in nipple line, heaving 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.

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 præsens:* Precordia bulges. Impulse diffuse; dulness increased. Presystolic thrill in fourth interspace. A blubbering presystolic murmur. Maximum intensity in fourth space. Loud blowing systolic bruit; heard also in axilla. Very accentuated second left. Aortic second feeble.

CASE XL.—William P., aged twelve. Second attack in 1883, third in 1885. No rheumatism. Condition of heart not noted. Has no symptoms. *Status præsens:* Diffuse apex beat in nipple line, in fourth and fifth spaces. Transverse dulness increased. In erect posture sounds clear. Recumbent, distinct apex systolic murmur transmitted along anterior axillary fold. In third and fourth interspaces double murmur, the diastolic not rough. Second left very much accentuated.

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. *Status præsens:* Impulse not forcible; dulness slightly increased. No thrill, but loud shock of first sound. Rumbling presystolic murmur, maximum in fifth space in nipple line, is well heard to anterior axillary fold. Loudly accentuated second left. No systolic murmur even when recumbent.

CASE XLII.—Carrie B., aged ———. Second attack in 1884; third in 1886, all severe. No rheumatism. In 1881 heart normal. No symptoms. *Status præsens:* Visible, somewhat forcible, pulsation in third, fourth, and fifth spaces. Erect posture, no murmur; recumbent, systolic bruit at second left, localized. Second sound here loud, sharp, and reduplicated.

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 præsens:* No enlargement. When erect, sounds clear; recumbent, systolic bruit at second left, with marked accentuation of second sound.

CASE XLV.—Marcus Van A., aged eleven. None since. No rheumatism. In 1881 a somewhat loud musical bruit. No symptoms. *Status præsens:* Apex beat in nipple line, fifth space. Impulse not specially forcible. Loud blowing systolic bruit at apex, propagated to axilla and heard well at scapula. Second left accentuated and reduplicated.

CASE XLVI.—Alice W., aged seventeen. Second attack in 1882. Pains in knees in 1882, and lately in shoulders. Heart normal in 1881 and 1882. *Status præsens:* Soft apex systolic murmur, not heard in axilla. Second left accentuated. No enlargement of heart. Has at times palpitation and shortness of breath.

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 præsens:* 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.

1882 (five years). Thirteen cases; ten affected.

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 præsens:* 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 præsens:* Beat forcible, outside nipple line; dulness increased. Apex systolic murmur, heard also in axilla and at angle of scapula; also as high as second rib. Second left loudly accentuated.

CASE LIII.—Mary J., aged fourteen. Attacks also in 1883, '84, and '85. No rheumatism. Heart normal in 1882. No symptoms. *Status præsens:* Impulse forcible. Soft systolic bruit at apex, heard as high as third space, not propagated to axilla. Remarkable accentuation of second left.

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 præsens:* 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.

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

CASE LVII.—Sadie C., aged welve. Second attack in 1885. In 1886 ankles swollen and sore; never had rheumatism with the attacks of chorea. No note of heart in 1882. In 1885 "hypertrophied and loud apex systolic murmur." *Status præsens:* Apex an inch outside nipple line. Impulse forcible. Dulness increased. No thrill. High-pitched systolic bruit at apex, loud also in axilla and at angle of scapula. Very accentuated second left. Has much throbbing of heart on exertion, and has vomited after skipping.

CASE LIX.—Maggie W., aged fifteen. Second attack in 1885. No rheumatism. Heart normal in 1882. *Status præsens:* A soft murmur at apex, not transmitted; increased on holding breath. Second left very accentuated.

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 præsens:* 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. Has had at times severe pain in heart; no shortness of breath.

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

CASE LXII.—James G., aged thirteen. Second attack in 1885, third in 1886. No acute rheumatism; pains in shoulder. In 1886 a systolic apex murmur. *Status præsens:* Apex outside nipple line; large area of forcible impulse in fourth and fifth spaces. Transverse dulness increased. No thrill. High-pitched apex systolic murmur transmitted to axilla and angle of scapula. In fourth space a faint rumble before first sound; second left accentuated and reduplicated. Has no heart symptoms.

CASE LXIII.—Tinnie B., aged twelve. Second attack in 1884, third in 1886. No rheumatism. In 1886 well-marked cardiac lesions. *Status præsens:* Apex beat forcible, outside nipple line. Dulness increased. Loud, rough apex systolic bruit, transmitted to scapula; second left accentuated and reduplicated. Has pain, and at times palpitation.

CASE LXIV.—Henrietta K., aged twenty-one. Second attack in 1884. No rheumatism. In 1883 heart's action intermittent. *Status præsens:* Beat forcible. No thrill. Loud, rough apex systolic bruit heard at angle of scapula. Second left much accentuated. Has great shortness of breath on exertion.

CASE LXV.—Lorenzo D'A., aged eleven. Two slight returns. No rheumatism. No note of heart in 1883. *Status præsens:* Impulse slow, forcible; apex in fifth space, in nipple line. Soft apex systolic murmur, louder on exertion; not heard at mid axilla. Second left much accentuated and reduplicated. Has distress at heart on exertion.

CASE LXVI.—Nellie H., aged nine. Second attack in 1884, third in 1885. No rheumatism. No note of previous heart-condition. *Status præsens:* 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 dyspnœa.

CASE LXVII.—Edward R., aged twelve. Second attack in 1885. No clear history of rheumatism; has had pains. No note of heart. *Status præsens:* 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 LXXI.—Annie C., aged eleven. Bad attack for a month; no recurrence. No rheumatism. No note of heart in 1883. *Status præsens:* Beat at nipple, in fourth space. Transverse dulness increased. Feeble thrill above apex. Rough presystolic murmur in third and fourth spaces; heard also along pectoral fold. Just outside apex a soft systolic. Loudly accentuated second left. Is short of breath on exertion.

CASE LXXIV.—William H., aged fifteen. Still has twitches at times. No rheumatism. No note of heart. *Status præsens:* Apex beat in nipple line. Dulness increased. Feeble presystolic thrill at apex. In second left interspace a loud, *rough*, systolic murmur. In third and fourth spaces a softer bruit. Distinct presystolic rumble above apex beat. First sound reduplicated at apex. Second left much accentuated. Has what his mother calls "asthma spells," particularly on exertion.

1884 (three years). Thirteen cases; ten affected.

CASE LXXVII.—Harry B., aged thirteen. Second attack in 1885, third in 1886. Rheumatism with attack in 1884. Apex murmur in 1886. *Status præsens:* Impulse feeble, just inside nipple line. No thrill. Dulness not increased. Soft apex systolic bruit; heard well to mid-axilla. Rough presystolic murmur, maximum intensity *at apex*. Both intensified after exertion. Loudly accentuated and reduplicated second left. No palpitation; no shortness of breath. Cheeks are flushed, and he has a *cardiac* look.

CASE LXXVIII.—Ida M., aged fourteen. No other attack. No rheumatism. No note of heart. *Status præsens:* No enlargement. Soft apex systolic bruit propagated along anterior axillary fold. Systolic murmur at second left space. Loudly accentuated left. Has no symptoms.

CASE LXXIX.—George G., aged thirteen. No other attack. Had pain in left hip in 1884. Heart normal. *Status præsens:* No enlargement. Soft apex systolic bruit; not heard in axilla, but well-marked in third left apex. Second left very accentuated, and the diastolic shock here loud.

CASE LXXX.—Nellie M., aged eleven. Right knee was swollen. No note of heart. *Status præsens:* Forcible apex beat in fifth space, one inch outside nipple line. Dulness increased. At apex first sound booming and echoing. In third and fourth left spaces loud systolic bruit; feeble at second left cartilage; not audible in axilla; faintly heard in mid-sternum. Much accentuated second left. Has no symptoms.

CASE LXXXII.—John D., aged eighteen. Second slight attack in 1886. In 1887 slight rheumatism. In 1884 soft murmur at base. *Status præsens:* Impulse just within nipple. Dulness increased. No thrill. At apex a rumbling presystolic murmur. No systolic bruit audible at apex. 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 aortic cartilage and at xiphoid. No aortic systolic bruit. Second left very accentuated. Posture did not alter the murmurs. He had no heart symptoms.

CASE LXXXIII.—Kate H., aged fifteen. Rheumatism very badly at the time. Heart said to be normal. *Status præsens:* 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 LXXXIV.—Henry M., aged fifteen. No rheumatism. Heart normal in 1884. *Status præsens:* Impulse diffuse in fourth and sixth spaces, one inch outside nipple line. First sound at apex booming. When recumbent a soft systolic murmur in second and third left spaces near sternum. Second left much accentuated.

CASE LXXXVI.—Lillie D., aged twelve. No rheumatism. Heart normal in 1884. *Status præsens:* 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.

CASE LXXXVII.—Fannie P., aged ten. Second attack in 1885. Pains in wrists, but no swelling. In 1885 apex murmur, presystolic; soft basic murmur; hypertrophy. *Status præsens:* Forcible apex beat in fifth space, outside nipple. Feeble thrill. Loud, high-pitched apex systolic bruit, transmitted to scapula; and, in fact, all over left chest. Presystolic bruit. At aortic cartilage a rough, systolic murmur. Second left accentuated. Has palpitation at times.

CASE LXXXIX.—Annie 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 XCI.—Lizzie B., aged fifteen. No attack since. No rheumatism. No note of heart. *Status præsens:* Impulse strong. Thrill at apex. Localized systolic murmur at apex, not heard in axilla or on third or second spaces. Loudly accentuated second left.

CASE XCII.—Alice N., aged ten. No rheumatism. In 1885 loud mitral systolic. *Status præsens:* Apex beat diffuse in fourth and fifth spaces in nipple line. Transverse dulness increased. Apex systolic murmur, heard beyond mid-axilla; intensified in recumbent posture. Marked accentuation of second left.

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 C.—Georgie G., aged thirteen. No rheumatism. In 1885 a basic systolic murmur. *Status præsens:* Impulse diffuse, forcible; apex just outside nipple line. Dulness increased. Thrill. At apex loud systolic bruit, propagated to posterior axillary fold. Second left dull, thudding, and accentuated. Heart's action irregular. Has palpitation and shortness of breath.

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 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.