AN INQUIRY INTO THE RELATIONSHIP OF CHOREA TO THE MANIFESTATIONS OF RHEUMATISM.

An examination of 263 Cases of Chorea Minor and of 215 Cases of Rheumatism, treated in the Royal Hospital for Sick Children, Edinburgh, during the period from 1900 to 1914.

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INTRODUCTION.

Chorea Minor, Sydenham's Disease or St. Vitus' Dance, was distinguished as a distinct disease in 1686, by Sydenham in his Schedula Monitoria. There he says it is a "sort of convulsion which attacks boys and girls from the tenth year until they have done growing", and this he describes as being the result of "some humour falling on the nerves and such irritation causes the spasm". The name St. Vitus' Dance is misleading, as it was named from the epidemic of Dancing Mania, which occurred in the 15th century in Germany and the Netherlands.

In these pages we shall confine ourselves to cases of Sydenham's Chorea, excluding all cases of habit spasm, tic, Chorea Major and secondary Choreas, as these are totally different affections.

From the time of Sydenham till now many writers have contributed to our knowledge on this affection, but yet the etiology and pathology of Chorea is but imperfectly understood. The chief cause of this is that deaths from Chorea are so uncommon that very little pathological material is to be had.

Some authorities believe that Chorea is a purely functional disorder, among whom is STURGES who looks on it as a "motor disturbance beyond the reach of anatomical demonstration". McCarthy thinks that it "would be a mistake to consider emotional disturbance

as a cause of a clinical syndrome like Chorea". Dickinson believes that it is due to arterial repletion caused by some irritant in the blood, either solid (as emboli) or liquid, and he suggests that the vascular dilatation may be of nervous origin. The older writers thought that Chorea arose from multiple embolism of the basal ganglia. Of this embolic theory Kirkes was the first supporter, and he was followed by Hughlings Jackson and Mackenzie. They thought that while the plugging of large arteries might cause Hemiplegia, the plugging of smaller ones would cause an impaired state, though not necrosis. This theory had to be given up, however, owing to the fact that so few cases of Chorea, seen Post Mortem, had shown this condition.

The belief that Chorea and Rheumatism are different manifestations of one and the same disease has been held in France for more than sixty years. Barlow in 1883 stated that "(1) Chorea should be regarded rather as a symptom than as a disease. (2) Chorea occurs so frequently in connection with rheumatic symptoms in combination and alternation, that we are justified in provisionally regarding it as itself often a rheumatic symptom."

Duckworth maintained that it was a manifestation of the rheumatic habit or diathesis brought on often by fright or emotion. Cheadle thinks that rheumatism is the most common factor. Sachs holds that it is

due to the effect of an infectious agent or its toxin on the central nervous system, causing a fine neurosis.

Recent work of great value has been done by Poynton and Paine in their researches on the cause of Rheumatic Fever. They regard Chorea as being due to the presence of small focal lesions external to the capillaries, caused by the escape of Diplococcus Rheumaticus into these positions. They and other observers like Beattie have been able to produce Choreiform movements in rabbits, into which they injected Diplococci. In a case of Chorea they were able to demonstrate numerous diplococci, in the perivascular lymph spaces of the pia mater, in its capillaries, and also in some parts of the motor area of the brain. In another acute case the diplococci were shown in large number in the mitral valve. Dana, Apert and Wassermann have also isolated diplococci in cases of Chorea.

In this paper we wish to bring forward the results of an investigation into the cases of Chorea, 263 in number, which have been treated in the wards of the Royal Hospital for Sick Children, Edinburgh, during the last fourteen years; and with those have also been collected the cases of rheumatism, 215 in number, which have been in the wards of the hospital for treatment during the same period.

All the cases investigated have not been examined by the one physician, with the result that the reports are not easy to compare. Some are reported much more fully than others and some again only lay stress on very special points. Patients are admitted to this Hospital up to the age of 12 years. Though this does not make any very material difference in our conclusions, still we must remember that in many ways the investigation would be of more value if it were carried on till the age of puberty.

I have to thank the Visiting Physicians of the Hospital, for the use of those cases which have been treated by them in the wards, and Dr. Melville Dunlop for the use of the reports of the cases which were patients in his ward while he was visiting Physician.

Our cases have been placed in three definite groups.

Group (1) includes cases of pure Chorea.

Group (2) " " " Chorea and Rheumatism.

Group (3) " " " pure Rheumatism.

We have examined the reports of the 478 cases and noted those facts which we thought would be of value to us, in our attempt to verify our statements regarding the rheumatic origin of chorea. These notes are given in full on succeeding pages. In our examination we have paid strict attention to the Family History of the patients, and have been able to show the presence of definite rheumatic family history in a great proportion of our cases. The comparison of the sex and age incidence of chorea and rheumatism gives us also useful results. We have tried to find out if there is any definite time of the year when chorea or rheumatism is more liable to occur, and have compared the percentage of cases of chorea occurring in the various months with that of cases of rheumatism. The various exciting causes of chorea have been noted and special investigation was made in 112 cases of chorea to find out if there was any relationship between the occurrence of chorea and any of the infectious fevers. The existence of any nervous tendency in the patients or their parents was also noted. Recurrences have been noted in the cases in the various groups and the percentages of these have been compared. The rheumatic histories of the cases have been gone into fairly fully, and from these we have been able to show the very large percentage of our cases of chorea which have a definite rheumatic history. Heart conditions found in

the three groups are compared, and the heart complications of chorea and of rheumatism are shown to be varied and numerous.

Symptoms of rheumatism such as Nodules, Arthritis, Epistaxis, Erythema, and Tonsilitis have been dealt with separately, and statistics given. From these notes we have tried to draw some definite conclusions, which, though they do not bring out any new points, still may be of interest.

SEX.

Looking into the cases of Chorea in the records of the Hospital we find that out of 263 cases there are 191 females as compared with 72 males i.e. a percentage of 27.3% males. Females are attacked in the proportion of rather more than two to one: according to Osler and in 554 cases collected by him from the records of the Philadelphia Infirmary, 161 were males or about 30%. See in 531 cases found 393 were female, and 138 male or 29% of males were affected. Other authors report even a greater number of females per hundred. Hughes shows his cases as being 27% males while Sturges found that his cases contained only 22% males. Those calculations are based on cases drawn from all ages. Sturges found that in children up to twelve years old his excess of females was not quite so great as in those of all ages, the

per centage being 25% males. Examining a series of patients under the age of eight he found that the number of females was still less giving a result of 30% males. Dickinson found that under eight his results gave 38% males instead of 30% in all ages.

Leonard Guthrie points out that girls are affected three times as frequently as boys but a larger number of boys are attacked between the ages of 5 to 10, whilst more girls than boys are affected between 10 and 15.

If we deduct 3% from our resulting percentage for males over 10 Guthrie's results and ours are practically similar. In considering the cases in Group (3) we find that there are almost as many males affected as females. There are out of 215 rheumatic patients 95 males or 44%.

Still has brought forward a theory to explain why females are more often subject to chorea than males. He considers that the micro-organism of toxin of rheumatism is more likely to produce chorea, when the brain cortex acted upon is that of an excitable child. There is no doubt but that females are as a rule much more excitable than males and this theory, in our minds, is probably the correct one. As we see from the results of Group (3) the number of females affected is almost equal that of males affected. So we must look for some other cause than the rheumatic infection to account for this excess, in the number of females attacked by chorea. This we find in the excitability of the female.

GROUPS (1) and (2).

CHOREA. Per centage of Males at the different Ages.

Total number of cases collected	263	
Number of males . . .	72	27.3%
Number of males over 11 . .	0	0 .%
" " " " 10 . .	8	3. %
" " " " 9 . .	17	6.4%
" " " " 8 . .	31	11.7%
" " " " 7 . .	44	16.6%
" " " " 6 . .	54	20.4%
" " " " 5 . .	65	24.6%
" " " " 4 . .	69	26.1%
" " " " 3 . .	72	27.3%

Number of cases investigated . .	263
Number of cases of pure Chorea .	81
Total Number of Male cases . .	72
Total Number of Female cases . .	191
Number of Male (pure chorea) cases .	18
Total per centage of Males . .	27.3%
Per centage of Males pure Chorea .	6.7%

or 22% of those in Group (1).

AGE.

The onset of Chorea is said to occur most commonly before the age of 15. Sydenham put it down as occurring in children between 10 and puberty. Osler gives the age incidence in 522 cases showing that 47% occurred in children under 10. See in 191 cases says that 151 were between 6 and 15, 11 were under 6, while 12 were over 21. Dickinson in a record of 71 cases found that 42 were under 10 or about 59%. All authorities are agreed that chorea is very uncommon under the age of 4. Osler points out that cases are found in the literature of chorea under that age, but he is sure that they belong to an entirely different disease and are associated with definite cerebral changes. It would be extremely difficult to distinguish between the jerky irregular movements of an infant, or those seen in cases of meningeal haemorrhage, or the subsequent changes induced thereby. Henoch has only seen one case and that was aged 3; Guthrie also notes one case of a boy aged 3 with definite Chorea.

In the records of the Hospital for Sick Children, Edinburgh, the youngest case is a girl showing definite symptoms of chorea and rheumatism at the age of 3½.

An investigation into the age incidence in the two groups (1) and (2) has been made. The following tables give the facts.

GROUPS (1) and (2).

Statistics regarding the age incidence of Chorea.

There were examined 263 cases of Chorea. Of those 81 were placed in the group (1) as being cases with no history of any rheumatic trouble.

Age	Males	Females	Total	Total %
3		1	1	.4%
4	3	4	7	2.6%
5	4	11	15	5.7%
6	11	21	32	12.1%
7	10	33	43	16.3%
8	13	29	42	15.5%
9	14	36	50	19. %
10	9	28	37	13.6%
11	8	25	33	12.14%
12	0	3	3	1.1%

Ages of cases of pure Chorea.

Age.	Total.	Total%
4	4	1.5%
5	6	2.2%
6	10	3.8%
7	19	7.2%
8	12	4.5%
9	9	3.3%
10	11	4.1%
11	9	3.3%
12	1	.4%

GROUP (3).

Statistics regarding the age incidence of Rheumatism.

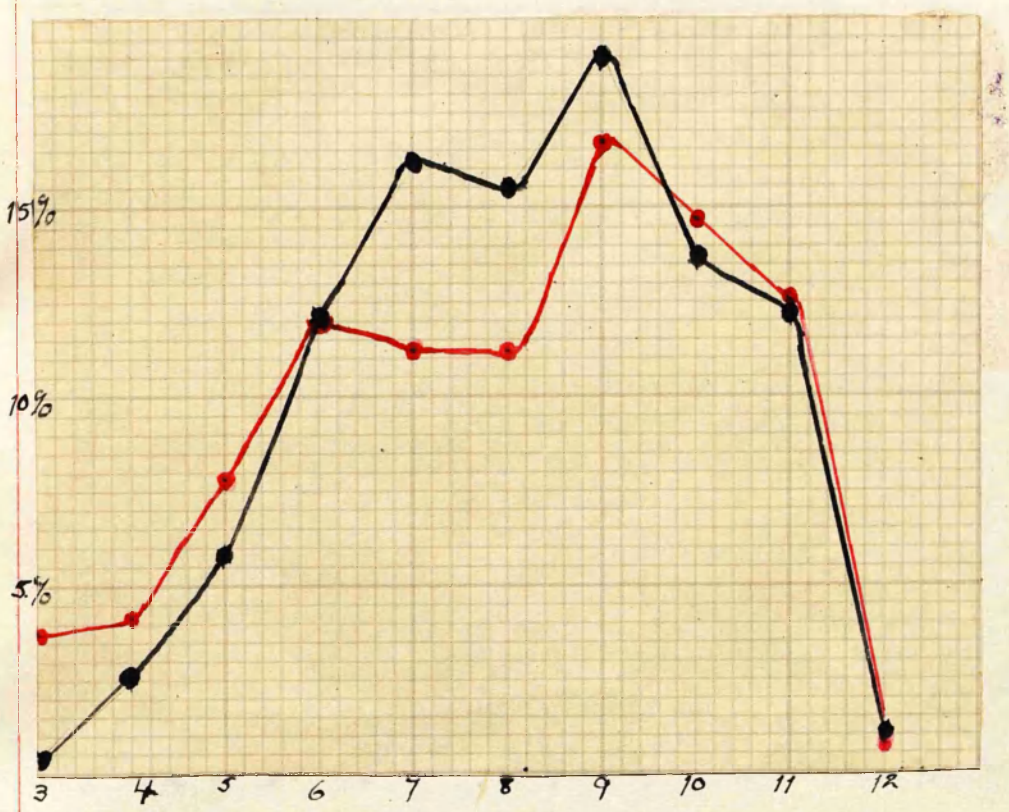
There were examined 215 cases of definite rheumatism.

Age.	Total.	Total %.
3	8	3.7%
4	9	4.1%
5	17	7.8%
6	26	12. %
7	24	11.2%
8	24	11.2%
9	36	16.7%
10	32	14.8%
11	27	12.5%
12	2	.9%
13	1	.45%

Cases were found where the ages were under 3 i.e. 11 months, 2 years, 2 years and ten months, and two years and eleven months.

Age incidence of Chorea Chart.

Compared with age incidence of Rheumatism.

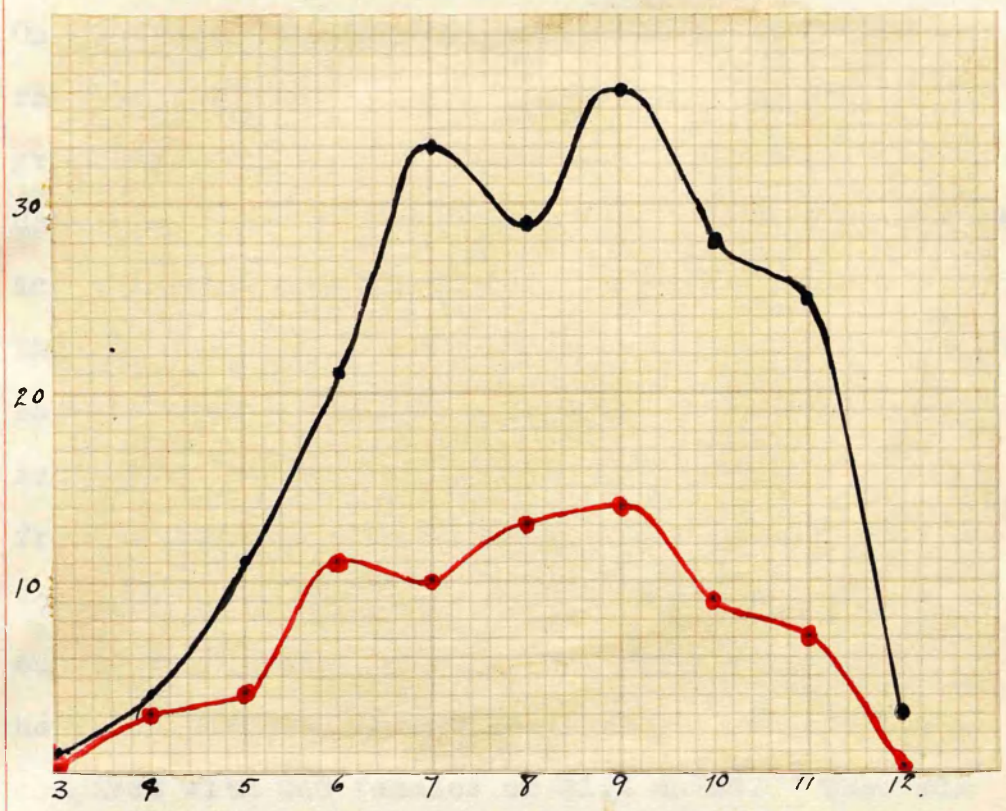


Years.

Rheumatism

Chorea

Chart showing age incidence of chorea. Sexes compared.



The cases we have looked into lead us to believe that Chorea is more common in the child from aet. 6 to aet. 11 than at any other period of life. The years 7, 8, 9, show a larger number than any other three years, and as is shown by the chart the incidence line rises steadily to 9 and as gradually falls. On examining the accompanying chart we find that the results obtained from the cases of Rheumatism in group (3) very closely resemble those obtained from Chorea. The age incidence in both these diseases is in very close relationship. Garrod says that Rheumatism is rarely seen before 3, but is most common between 4 and 9. He gives 80% as being his results. The number of males and females suffering from rheumatism is practically the same according to Voelcker. In our cases however the number of males out of 397 children, suffering from rheumatism or having a definite rheumatic history, was only 149 as compared with 248 females or 31.% males. When all cases which have a history of chorea are deducted we find that there are 95 males to 120 females, or 44%.

Localities.

The cases mentioned in the literature do not afford any data on this subject at all. Chorea is found in all European and temperate countries, and according to Cranmer does not occur in the Tropics. It is very seldom found in the Negro or in any dark skinned people. Much work has been done on this

point by American authors.

Most of the cases in literature are children who have been in the large Hospitals, and are therefore probably drawn from the lower classes in the towns. In the report of the Collective Investigation Committee the cases were drawn from private practice and showed 72.27% of the cases were from the lower classes.

In our cases we find that the great majority of the children are drawn from the poorer part of the town. Very few cases are from outside Edinburgh and these are mostly from smaller manufacturing towns or mining centres. The histories of the cases give facts pointing to the conclusion that the housing conditions of the children have a good deal to do with the presence of Chorea in those cases.

An investigation into the time of year of the onset of Chorea helps us a little in forming our ideas regarding the cause of this affection.

Seasonal Incidence.

In America much work has been done on the relative frequency of chorea at different seasons of the year. Lewis has given us most of our knowledge on the subject from his study of period of attack of 717 separate attacks of chorea. He comes to the conclusion that there is a seasonal relationship between Chorea and Rheumatism, also that "Weather is one of the most important predisposing causes."

Lewis found that in March Chorea occurred most frequently, while in November its occurrence was

least frequent. "There is a rapid rise in the number in December, while during January and February the number is almost stationary. In May there is a slight rise from April but the number then falls gradually to its lowest point in November."

Other authors however have failed to find any real relationship between the onset of chorea and the season of the year.

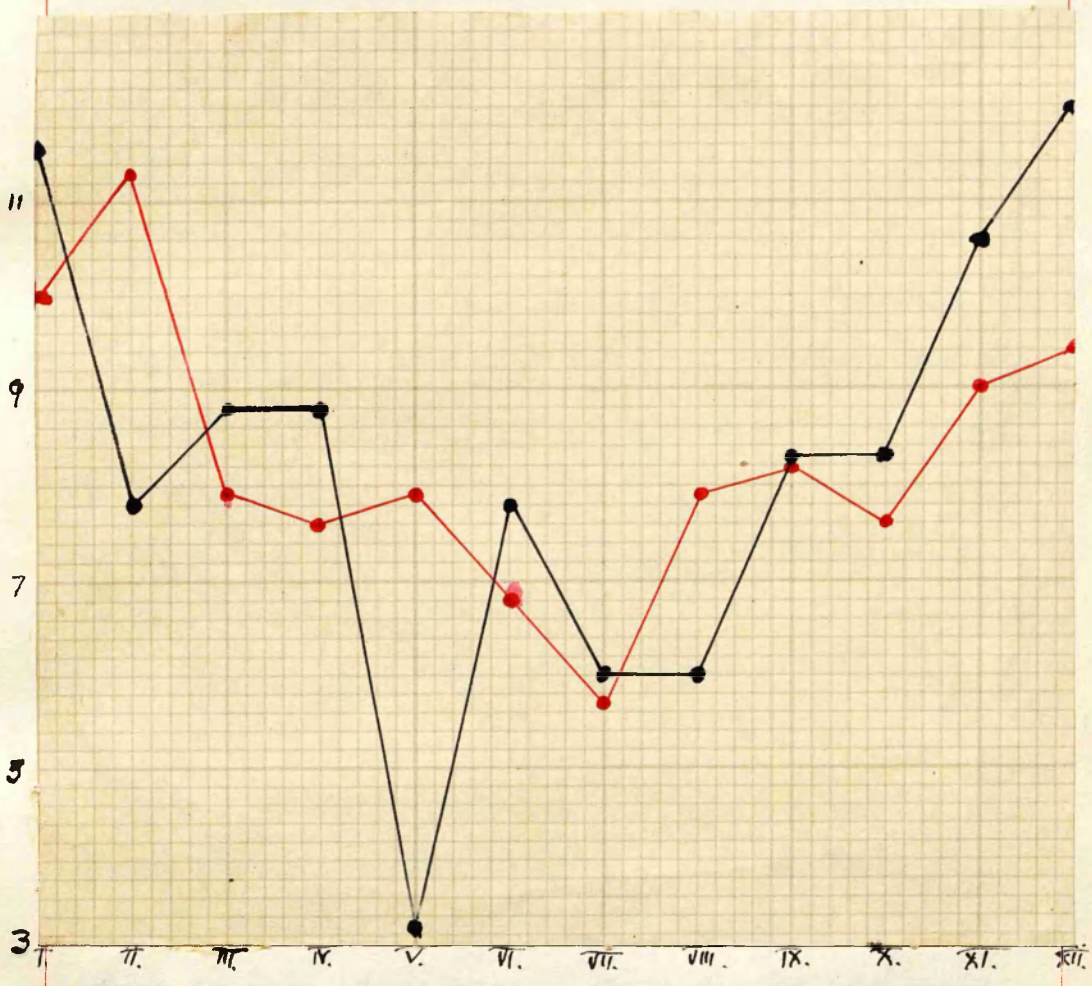
In our cases February shows occurrence of Chorea most frequently while we find that July is the month in which fewest cases are dated, June coming next. There is very little difference in the numbers for the other months. The winter months January, February and December are at the top of the list. Though this investigation in itself is not of much importance the comparison of those figures with those from the group (3) is of distinct value.

There we find that there is a close relationship between the month of the year of the onset of Chorea and the month of the year of onset of Rheumatism.

Months in which disease occurred.

Month.	Chorea		Rheumatism		Total.
Jan.	26	= 9.8%	25	= 11.6%	51
Feb.	30	= 11.3%	17	= 7.8%	47
Mar.	21	= 7.9%	19	= 8.8%	40
Apr.	20	= 7.6%	19	= 8.8%	39
May	21	= 7.9%	7	= 3.2%	28
June	18	= 6.8%	17	= 7.8%	35
July	15	= 5.7%	13	= 6 %	38
Aug.	21	= 7.9%	13	= 6 %	34
Sept.	22	= 8.2%	18	= 8.3%	40
Oct.	20	= 7.6%	18	= 8.3%	38
Nov.	24	= 9 %	23	= 10.6%	47
Dec.	25	= 9.4%	26	= 12. %	51

Seasonal Incidence of Chorea compared with Seasonal Incidence of Rheumatism.



Exciting Causes.

The records of chorea cases are not all definite enough on this point to make any certain advance in our knowledge of the origin of this disease.

Henoch says he has seldom seen chorea come on during polyarthritis, only 4¼% of his cases springing directly from rheumatism. Dickinson on the other hand states that the causal factor in 70% of his cases was Rheumatism. Peacock puts it down at 78.8%. See mentions that in 61 cases out of 128 chorea and rheumatism coincided. Chorea is often mentioned as coming on during the convalescence from rheumatism after all pain had left the joints. Rheumatism is counted as one of the exciting causes by Henoch, Sturges and Osler. At present we shall leave out of account the rheumatic cause as it will be dealt with very fully later and we shall confine ourselves to the other causes which are given in the histories.

Over-pressure at school is often given as the cause of the onset of chorea. This is thought by Holt to be an important element and Leonard Guthrie gives it some place also. In a few of our cases only, was there a history of overwork at school: and the months in which most chorea occurs are certainly not those in which the school examinations are held, nor those just succeeding them.

Among the reflex causes may be mentioned Thread Worms and Tape Worms, though some authors do not

believe this. Osler for instance says that he has not found a single case which would indicate any causal relationship between chorea and the presence of worms. Purves Stewart had only one case of Tape-worm causation. In our series of cases three are put down to Thread worms and the history is interesting in each case. They are noted below.

(Case 171. Chorea).

Male, aet. 10½ admitted January, has been suffering for 4M from chorea. F.Rh. As a child had convulsions, and otorrhoea. Thread worms were noticed before the fits. He had convulsions again aet. 5 and once more aet. 7, and before each attack T.W. were noticed in the stools. 4 months before admission he once more had fits and T.W. were seen. The fits were followed this time by choreiform movements and he still has T.W. on admission. There is no sign or history of rheumatism.

(Case 180 Chorea).

Female aet. 8, admitted September, has been suffering for 3 days with Chorea. History shews F.Rh. S.T. Wh.C. Mor. and T.W. in stools the day before chorea showed itself. There is definite sign of rheumatism having been present and on admission child had M.S.$2P^{X}$. She was readmitted for a relapse in April and then showed M.S.; M.D.; M.P.; $2P^{X}$.

(Case 201 Chorea).

Female, aet. 9, admitted in November after 2

weeks' illness. She had passed some T.W.s in the stool and next day chorea like movements were noticed. On admission there is no history of rheumatism, but on examination she is found to have Systolic murmurs over the Mitral, Aortic and Pulmonary areas.

From these cases and from the other cases in literature it seems that it is quite possible for T.W. to bring on chorea in children but these children will be found usually to have some rheumatic symptom as well, and we are sure they would not show symptoms of chorea at all were these rheumatic signs not present.

Fright is commonly brought forward as the cause of an attack of chorea. By fright is meant any shock either mental or physical. The patients come with a history of a fright, accident, operation or some emotional disturbance. In many of our cases there is a history of the child getting into trouble at school and being punished, but these cases we have not counted as cases of fright as the punishment was in all probability due to some of the effects of chorea, such as bad writing, restlessness, blotting copy book, etc. Osler states that in 86 cases 15.5% were said to have followed fright. Cheadle points out that fright is present equally in cases of chorea with a rheumatic history and in those without. Purves Stewart gives his cases as 2% with history of fright.

In the cases we have collected there are 37 in

which a fright was blamed for the onset, that is 1.4%. Of those 37 cases there are 24 which give a definite history of rheumatism or had rheumatism on admission.

From these results we find that in the majority of cases of chorea where there is some history of fright at the onset of the illness, that fright simply acts on a body and mind previously infected with rheumatism bringing on the choreiform movements.

Causes other than fright.

Imitation. Many cases of this are mentioned in the literature but most of them turn out to be simply cases of hysterical disorder. Cheadle goes out of his way to explode the theory of imitation "this time-honoured fallacy". He states that no child ever became choreic from this cause. In a medical ward there are always one or two chorea cases and yet no case is noted where a child has developed chorea in hospital. One of this description occurs amongst our cases and is worth notice.

(Case 168 Chorea).

Female, aet. 8¾, admitted December, duration 2 weeks. There is previous history of Wh.C.; Mor.; S.T. Two days before the onset of the sumptoms of chorea, a scholar at school, who was very choreic, fell, and the patient became very excited.

This is the only case in which imitation is said to be the cause but there is a history of S.T.s and the case may be considered one of fright.

Scarlet Fever. Among the cases under consideration there are 9 in which there is a direct origin with definite history of the chorea coming on immediately after an attack of Sc.F.

In 533 cases of Sc.F. reported by Carslaw, Chorea developed in three. Osler obtained a previous history in 141 cases, but in none of them was there a direct sequence. In 112 of our cases of chorea there was a history of 18 having had Sc.F. and of those 4 were directly followed by Chorea.

All the epidemics of so called chorea, have not been true chorea minor, but hysteria. One of course comes across cases where two children in one family are affected, the one after the other, such as case 102 chorea in group (2) mentioned below. And in those cases it is much more probable that the chorea was due to the family predisposition than to imitation.

(Case 102 Chorea).

Male, aet. 9, admitted January 1910, duration of illness 6 weeks. M.R. S.R. One brother has had chorea 5 times. Second brother chorea 5 times. Third brother chorea. Second brother has had rheumatism. Patients comes in complaining of S.T., G.P., M.S., $2P^{x}$, and chorea.

(Case 103 Chorea).

Male, aet. 4, admitted February 1910, duration 5 weeks. This patient is the brother of the preceding case and took ill one week before the brother was

removed to hospital. He complained of S.T. and Hx.

Those cases also show good examples of family predisposition.

Of the other infectious diseases not much need be said. We found that in 112 cases Chorea, 77 had previously had Mor. and there was a direct sequence between that illness and chorea in only one. Diphtheria and Influenza were said to account for one case each. Henoch found that in his cases there was one Diphtheria and two Mor. Sturges found that of 394 cases 91 had suffered from Wh.C. Leslie gives 47% Wh.C. between 5 and 12 years old. We find that there is a previous history of Wh.C. in 48 out of 112 cases and in none is there any relationship to the attack of chorea.

Neurotic history. Holt says that chorea is more frequent in children of neurotic parents. We have found that in 26 cases was there a history of one or other of the parents being insane or very nervous people. In 16 cases there is a history of convulsions during early years of life. Amongst the cases of group (3) out of 204 there are only 5 which have previous history of convulsions, i.e. 2.5% as compared with 6% of Chorea.

Family History.

Chorea cases are often found in families and there is distinct proof that hereditary tendency occurs. Out of 554 cases looked at by Osler, there were 80 in which there was a history of chorea in

either parent or in another member of the family. A history of rheumatism in the family is also very commonly found. Grabois in an investigation of 136 cases in Paris found that 8% had a history of chorea being present in one parent, and 15% had a rheumatic history in one of the parents. Sturges says that there is a history of 20% of his cases with rheumatic family history.

Cheadle has pointed out that rheumatism is transmitted from parents to children as much as gout, and he found that out of 173 cases 20% of these with a rheumatic family history had developed undoubted rheumatism, while of those without a family history only 4% had developed rheumatism. Voelcker says that rheumatism is a family disease. The liability to it may be inherited from either or both parents, and when both parents are affected this liability is distinctly increased. According to Leonard Guthrie the per centage of family predisposition to rheumatism in chorea cases compares with that of acute rheumatism itself. Syres estimated rheumatic inheritance in chorea cases at 32.2%.

We find from our cases that in 93 out of our total 263 is there a rheumatic family history and in 26 is there a family history of chorea, i.e. 34.1% with rheumatic history in family and 9.8% with chorea. There was a history of both rheumatism and chorea in 14 cases; and of either chorea or rheumatism in 105 or almost 40%.

In the series of cases in Group (3) we find that there is a family history of rheumatism in 95 out of 203. i.e. 46.5%, so that in a series of 466 cases of rheumatism or chorea there is a family history of rheumatism in 40.3%.

No attention was paid to rheumatic history of grandparents or uncles or aunts, some being mentioned fully in the reports, while others were omitted. In every case the family history as regards M.F.B. and S. was fully taken so that our statistics as regards those are quite accurate.

Family histories with Rheumatism.

M.R.	F.R.	FM.	X.R.	Total.
33	26	13	21	93 = 34.1%

Family histories showing chorea.

M.C.	F.C.	X.C.	Total.
8	1	17	26 = 9.8%

Total number of cases with both rheumatic and choreic family histories = 14.

Total number of cases with either rheumatic or choreic history, 105 = 40%.

In Group (1). History of rheumatism in family.

M.R.	F.R.	FM.	X.R.	Total.
10	7	4	2	23

History of chorea amongst X.C. in 4 cases which had history of rheumatism as well.

In Group (3). History of rheumatism in family.

M.R.	F.R.	FM.	X.R.	Total.
41	25	9	20	95

Percentage of cases with history 46.5%.

Rheumatic Family Histories Percentage.

	M.R.	F.R.	FM.	XR.
Group (1)	12.3%	8.6%	4.9%	6.15%
Group (2)	12.6%	10. %	4.9%	10. %
Group (3)	20. %	12.2%	4.4%	9.8%

Total number of cases of chorea 263.

Cases in Group (2) with definite rheumatic history 182 or 69.2%.

If those with family history of rheumatism are also added the percentage = 77.9%.

Recurrences.

From the earliest time recurrence was a feature of the disease. Sydenham believed that the affection recurred each year at about the same time. In order to try to prevent the onset of the recurrences he was in the habit of ordering the patient to be well purged a short time previous to the expected time of onset. Holt is of the opinion that there is a strong tendency for chorea to recur especially in the spring months. Osler agrees with this and noted that in 410 cases investigated from this point of view, 240 had a single attack, in 110 was there a second, in 35 a third, in 10 a fourth, in 12 a fifth, while in 3 there were six attacks. Gowers mentions a case in which there were 9 attacks. Other authors have worked out this feature of chorea i.e. recurrence with reference to the time of year, and they all seem to agree with Holt and Osler in their statement that chorea is more apt to recur in the spring months.

RECURRENCES.

There were 263 cases examined,

	Group (1)	Group (2)	Total.
One	67	111	178
Two	9	46	55
Three	4	14	18
Four	1	4	5
Five	0	5	5
Seven	0	1	1

One case has history of being in the Royal In-

firmary four times and in the Sick Children's Hospital, Edinburgh, eight times.

(Case 151 Chorea).

Male, aet. 9½, admitted April, suffering for one month. X.C. Shows M.S. Was five times in R.I.E. and four times in R.H.S.C. before this. Readmitted June, and once more in May next year.

Recurrences in Group (1) = 17.3%

Recurrences in Group (2) = 39.%

Here, a point is brought out which has not been mentioned by authors like Cheadle or Osler. Namely the greater proportion of recurrences in cases of chorea and rheumatism than in cases of pure chorea. We see from the above statistics that the percentage of recurrences in Group (2) is more than twice as large as that of Group (1).

Statistics of cases with definite signs of Rheumatism.

Chorea.

Rh.F.	33	=	12.1%
G.P.	16		6%
Nod.	6		2.2%
Ery.	6		2.2%
G.P.^{x}Nod.	3		1.1%
G.P.^{x}S.T.	9		3.3%
S.T.	12		4.5%
S.T.^{x}Jts.	13		4.9%
S.T.^{x}Ery.	1		.4%
S.T.^{x}Ery.^{x}Epi.	1		.4%
S.T.^{x}Nod.^{x}Jts.	1		.4%
Epi.	1		.4%
Nod.^{x}Ery.	1		.4%
Jts.	44		16.7%
Jts.^{x}Nod.	3		1.1%
Jts.^{x}Ery.	4		1.5%
Nod.^{x}Ery.^{x}Jts.	1		.4%
Total	155		

Heart affections with no other signs of rheumatism.

M.S.^{x}2P^{x}	8	3.1%
M.P.^{x}2P^{x}	3	1.1%
M.S.^{x}H^{x}	3	1.1%
Definite Rh.	13	4.9%
Total	27	

The Association of Chorea with Rheumatism.

Since the beginning of the eighteenth century chorea has been recognised as a disease which was in some way associated with rheumatism, though the definite points of association were not very well understood. Every now and then some authority comes forward producing statistics to prove that the connection between those two affections is not so close as was supposed. On the other side, that is on the side which tries to prove the rheumatic connection to be stronger than was thought, we have numerous writers and each one has prepared statistics. Some of these results are of value to us and in our comparison we have to note certain differences. The majority of authors count as cases of rheumatism only cases which have suffered from arthritis of some description. Our statistics are drawn from cases which have shown many varied symptoms of rheumatic infection, so our results are bound to be much higher. Sturges recognises the connection there is between chorea and rheumatism, but he thinks that writers make too much of this. He says that the presence of rheumatism in the previous histories of children really shows a percentage of 15% to 20%, and that a percentage which is so small should not be made so much of. We feel that if the presence of rheumatism in the histories of the cases really showed so small a percentage Sturges would be right in his view. However if all the manifestations

of the rheumatic infection are studied, the number of cases with a rheumatic history is a large one, and certainly justifies the important place it has been given in the origin of chorea.

The Collective Investigation Committee bases their results entirely on cases of articular rheumatism, and found that 32% had rheumatism either previously or else at the same time as chorea.

Osler in 554 cases says that 15.8% had a history of either previous present or subsequent articular rheumatism and if, to those, were added the cases in which there was a history of pains of a vague nature, the per centage is found to be 21%.

It is a very difficult thing to trace cases after they leave hospital. However this was done by Batten, with 115 of his cases. While in hospital 32.2% had histories of rheumatism. Three years later 43.5% had a history of rheumatism, while six years later the percentage was increased to 52.2%. There were 38 cases which could not be followed and if these are allowed for the result of his work shows that after six years 65% showed history of rheumatism.

Townsend reported 148 cases with a rheumatic history in 21%. Crandall found "88 cases with rheumatism of some form either before or after the chorea in 54%."

The arthritis or other sign of rheumatism, in a very large proportion of the cases, precedes the

chorea, which develops after their subsidence. In other instances the chorea is followed by the rheumatism. This was seen to be the case in 2% of the patients reported on by the Collective Investigation Committee of the B.M.A.

Charles believes that a rheumatic diathesis is conducive to chorea. Cramer states that in most cases there is a slight rheumatic involvement of joints or slight rheumatic pains, and if there is added on to this some additional cause, chorea may develop.. According to Ibrahim chorea very often follows an attack of rheumatism and many times rheumatism follows after an attack of chorea. Barlow says "I do not know of any acute fever of childhood of which it is a sequel, except occasionally of Scarlet Fever, with which, significantly enough, rheumatism is apt to occur. Duckworth says that all observers are now prepared to agree that rheumatism holds at least the first place in the etiology of chorea. We have seen from our cases that there are 182 in group (2) and 81 cases which have no history of rheumatism at all. In 23 of those cases in Group (1) there is a definite family history of rheumatism.

We will examine the cases in Group (2) more in detail. It is difficult to define exactly what is meant by a history of rheumatism. Among our cases of chorea 33 or 12.1% gave a history of having suffered from rheumatic fever, with pains in the joints

and swelling and in many cases showing on admission marked rheumatic heart lesions. We have drawn out statistics on a previous page which show the number of cases which have been put down as rheumatic, and they are arranged so that heart symptoms are not taken into account if there is any other sign or symptom of rheumatism. There is a second list of cases shown, in which those cases, which have only heart symptoms are noted.

Erythema Nodosum.

This is one of the Erythemata which are found often affecting rheumatic cases. Poynton describes it as "either a symptom of many infections, including the rheumatic, or the rash of some peculiar infection, closely allied to, and associated with rheumatism." Norman Walker states that this form of erythema "frequently occurs in rheumatic patients, and even more frequently in those who have suffered from some of the other diseases which are associated with that poison such as chorea".

Osler considers that though Erythema Nodosum has not been proved to be closely related to rheumatism still Erythema Marginatum and Papulatum are practically certain signs of the rheumatic state.

In the cases we have looked into, there are 6 which have some form of erythema as the only rheumatic sign. Of the total number of cases examined in Group (2) there were 18 with a history of erythema. Among those in Group (3) there were 21, and those

occurred mostly in females, in the proportion of 1 to 2.

One cannot definitely say that this is a sure sign of a rheumatic history, but if combined with this affection one finds heart mischief then one is justified in considering the cases as definitely rheumatic.

In all the 6 cases mentioned above there were certain signs of heart affection being present. Because of this they have been numbered amongst those of Group (2).

In other cases we find that Herpes was present twice and Lichen and Purpura each once. Psoriasis was present in two of the cases.

Epistaxis as a sign or symptom of rheumatism.

This symptom is very seldom found mentioned in the literature. It is quite clear that it is a common occurrence with many children. According to Langwill haemorrhage occasionally occurs in connection with pre-existing valvular disease of the heart of an organic nature, though epistaxis is not one of the forms it commonly assumes. He however shows cases in which he brings out this as a sign that an attack of rheumatism has occurred.

In our cases Epistaxis occurred once as the only definite symptom of a rheumatic history, and in this case it was associated with a rheumatic endocarditis. In Group (2) this symptom was found to have occurred 4 times and each time there was definite cardiac

trouble. In Group (3) epistaxis was present in 17 cases, and in all those cases it was accompanied by a cardiac lesion.

No explanation of epistaxis as one of the symptoms of rheumatism is to be found in any of the literature. Osler, Cheadle and Fagge do not mention it at all.

From our cases however, and the results they show, it seems to us that there is some definite relationship between epistaxis and rheumatism. If it is not related to the rheumatism itself, it would appear to occur as a symptom of heart affection. More investigation is needed on this point but the fact that all our cases which showed epistaxis also suffered from some heart lesion is very suggestive. If the explanation of the epistaxis is to be found in the fact that there are old standing mitral lesions present, one would have expected it to have occurred more frequently than was the case. It would not besides only occur as in case (114) each time just before an attack of chorea, or before any other sign of illness appeared as in cases (30) Rheumatism and (61) Rheumatism.

Case 61 Rheumatism.

F. aet. 8, admitted October 1908, duration 6 months. MR. GP. Sc.F. aet 7 GP. appeared in April and next month severe Epi. This recurred. Child very nervous. On admission Nod. were seen on auricles and arms. MS. H^{x}. Readmission April 1909. Six weeks

ago Epi. now breathless on exertion. MS. H^X. $2P^X$.

Case 30 Rheumatism.

F. aet. 9, admitted September 1912, duration 2 months. Epi. was first noticed. Patient then complained of pains in the legs, which were said to be "growing pains". F.R. There is an irregular pulse and MS. MP.

Case 114 Chorea.

F. aet. 9, admitted January 1905, duration since March 1904. Onset was by Epi. then developed Chorea. She got better but had a relapse in April which began after Epi. Before admission she had epistaxis once more. On examination MS. H^X.

It would be difficult to ascribe the epistaxis in those and other similar cases to any heart lesion. In those cases it is seen as a symptom before there were any other signs of illness. Matthew has shown a case where epistaxis occurred coincident with an attack of endo- and peri-carditis.

This point, however, would need more investigation.

Arthritis.

This arthritis found in children, differs in many notable features from the acute articular Rheumatism of later life. As is well known the joint symptoms in a child are often so slight that the parents take no notice of them, often believing these pains to be what are called "growing pains". Even a very careful inquiry is necessary in many cases before one can be sure that there has been an

attack of pain and swelling of a joint. It is of course not uncommon to find cases of Rheumatism where there would seem to be no history of joint affection at all. In the child the arthritis is not so extreme as that of the adult; there is certainly less swelling and tenderness and pain. Cheadle says "there is often merely a little pain and a little stiffness and tenderness, limited perhaps to a single joint or set of joints, hardly attracting notice, soon forgotten, often overlooked altogether, and constantly not severe enough for the doctor's aid to be called in. It is subsequent heart disease which sends these cases to hospital." In most of the attacks the onset does not show a very high temperature, usually from 100 to 101.5.

In our cases articular lesions were found either present on admission or in the previous history, in 74 in Group (2) and in 176 in Group (3). This shows a percentage of 28.1 of the Chorea cases, while among the rheumatism cases in Group (3) the percentage is 86.2.

Regarding these as symptoms of rheumatic infection very little need be said, as there is no doubt that the joint trouble which one so frequently finds in chorea cases is rheumatic in origin, though it cannot be compared in severity with the articular rheumatism of the adult.

"The small joints of the hands and feet are often

affected, and the larger joints, particularly the knee joints, show the same transitory swellings, though to a lesser degree than in adult life". (Poynton).

In children the differential diagnosis of joint trouble is not all plain sailing, care being needed to eliminate specific disease and tuberculosis of the joint as well as infection from the gonococcus. Two of our cases are of interest.

Case (190) Rh.

Female aet. 2, admitted 6/10/00, with swollen joints and pain and tenderness over the affected parts. M.R.

She was found to be suffering from a vaginal discharge, and this was found to contain Gonococci in numbers.

Case (49) Chorea.

Female aet. 11, admitted November having been ill for 14 days. Has been complaining of pains in the joints.

She had on admission definite signs of tubercular disease of the right knee joint, as well as her rheumatic condition.

Those serve to show that care is needed lest we include other affections under the name of rheumatism.

Arthritis

Group (2)

Arthritis was present as only symptom in 44 cases or 16.7%.

Arthritis associated with other symptoms in 30 cases.

Total number of cases with arthritis 74 or 28.1%.

Group (3)

Arthritis present as only symptom in 77 cases or 37.7%.

Arthritis associated with other symptoms in 99.

Total number of cases with arthritis 176 or 86.2%.

Subcutaneous Tendinous Nodules.

These nodules were first described by Hillier, in 1868. Since then work has been done on this subject by Meynet, and others. The credit of pointing out their frequency and their great importance as clinical signs in the various manifestations of rheumatism belongs to Barlow and to Warner. These nodes are very seldom seen in adults. Cheadle mentions that he had only seen two cases where they were present in adults.

According to the reports of the Collective Investigation Committee in 655 cases of articular rheumatism only 36 showed nodules, while among the cases of chorea mentioned in those reports there were only 12 cases which showed nodules.

Barclay Ness states that "for diagnosis the presence of these nodules is worth a great deal, as they may be taken as undoubted evidence of the rheumatic nature of the chorea". We have used this as our authority and counted those cases in our chorea cases as having a definite rheumatic history, though there are no other evidences present besides nodules. This occurred in 6 cases and they have been classed in Group (2) accordingly.

Nodules were found in our cases situated in all the places noted as common sites. They also were seen over the intercostals and in one case nodules were found in the auricles.

According to Voelcker not only are they pathognomonic of rheumatism, but they also indicate a grave form of it, since they are found almost invariably, associated with some form of peri- or endocarditis.

The great point of interest is the relationship of these nodules to cardiac disease. As Ness, Osler and Cheadle all say, they are connected with the graver forms of rheumatism. There are authorities like Cheadle who consider them as a sentence of death. He says "they mean persistent cardiac disease, generally uncontrollable, and marching almost infallibly to a fatal ending." Money found nodules in half the cases of rheumatism in which well marked heart disease occurred.

From the examination of the cases in Groups (2) and (3), we find that though there were 278 cases with definite signs of heart disease, still there were 55 cases only which showed a complication of nodules and Heart disease.

In the cases we have examined there were 27 with histories of pericarditis and of these 11 showed the presence of nodules. Death terminated 23 of our cases and nodules were present in 8 only. This is a much smaller number than one would have expected from reading the literature on the subject.

The pathology of rheumatic nodules is still not very definite and although much has been written, there is still a good deal of work to be done.

Poynton says that the pathological lesion found in pericarditis and in these nodules are very similar. "They show necrosis and exudation, round celled infiltration, swelling of the affected tissues that are not destroyed, and dilatation of the blood vessels in the neighbourhood." He also states that he has seen diplococci Rheumatici present in the nodules.

As the result of our investigations in this subject, we find that the statements of some authors as to the extreme gravity of cases of Rheumatism and chorea which show the presence of nodules, is exaggerated. Though there are subcutaneous nodules found in 20% of the cases of rheumatism we have examined still death occurred in only 7 cases showing nodules or 3.4%. This is a very small percentage. If we consider the cases of a serious nature which showed nodules and in which recovery took place, the same small percentage is seen. Though there were recurrences in 86 cases of chorea or 32.7% yet in only 7 of them or 2.6% was there a history of the presence of nodules. From these facts we think that the presence of nodules is not of so grave import as is commonly supposed. Most authors on this point are quite definite in their opinion that nodules point to a very bad prognosis; but it is interesting to note that this is not borne out by our cases.

Statistics regarding presence of nodules.

Groups (1 and 2).

20 cases showed nodules, or 7.7%.

In 3 cases of death, 1 showed nodules.

In 6 cases of Peri. 3 showed nodules.

In 123 cases of Heart trouble 15 showed nodules.

Group (3).

41 cases showed nodules, or 20%.

In 20 cases of death, 7 showed nodules.

In 21 cases of Peri. 8 showed nodules.

In 155 cases of heart trouble 40 showed nodules.

"Growing Pains."

These slight pains are often complained of by children, and are in many cases all the history obtainable of an attack of rheumatism. After an attack of pain of this description one may find distinct signs of the heart being affected. These pains are considered by Guthrie to be signs of rheumatism thought not of primary importance. Others again think that one cannot lay any stress on these as signs of rheumatic infection, as the pains are so indefinite. We, however, have considered any cases where growing pains were present as symptoms, to be cases of rheumatic nature, and we find that 6% of our cases show this as the only sign. Growing pains are present in 37 cases in Groups (1 and 2), and in 20 cases in Group (3). In the majority of cases we get the

history of "growing pains" and sore throats, and in these we can very safely take the cases as rheumatic.

Sore Throats.

Tonsilitis and sore throat are frequent in the rheumatic. In some children with acute rheumatism we find a diffuse redness and swelling of the tonsils, and this may be followed in three to five days by arthritis, and within a week by carditis.

Poynton points out that in 1,000 observations made by him, the symptoms of rheumatism were "sore throat, arthritis, pains and carditis". Trousseau recognised a rheumatic sore throat, and showed how this often alternated with torticollis and joint pain. Fowler estimated that sore throats occurred in 80% of the cases of rheumatism. According to the reports of the Collective Investigation Committee there were 27.1% of their cases with a history of tonsilitis. Cheadle states that "Tonsilitis may occur at any period of the rheumatic series, although most often it comes first - immediately preceding the arthritis."

It is probable that tonsilitis may occur as a solitary expression of the rheumatic state, according to Cheadle.

In the chorea cases we have examined, we find that 37 or 14% show a history of sore throats. In 12 of these tonsilitis was the only symptom of rheumatic infection. Our rheumatic cases in Group (3) gave a history of tonsilitis in 56 or in 27.4%.

Pleurisy.

Though, this is not a symptom of rheumatism in itself, yet its occurrence in cases of rheumatism is common. It is specially common in cases where there is carditis. Poynton states that he has hardly ever "seen a case of fatal carditis without rheumatic pleurisy." This pleurisy may occur either in association with bad heart disease or as a separate infection. In our rheumatic cases there was a history of pleurisy in 7 or 3.4% while it was only present in 2 of our chorea cases.

Heart Affections in Chorea.

In chorea the only serious conditions which may arise are in association with the heart. Death often occurs in chorea cases with heart symptoms and hardly ever in those without. Osler says that "anomalies in the cardiac action may depend upon faulty inervation, upon changes in the muscular walls, or in the condition of the circulating blood, or upon inflammation of the valve segments". Marked irregularity of the heart was seen in most of our cases, there being various reasons for this. Some of the patients were very excited and so had very fast beating hearts, others had a slow beat or an irregular beat from drugs or from jerky choreic movements. We have given no notes of the heart rates as nothing could be definitely said about them. The difficulty of getting good records in all the cases was also a hindrance.

Dilatation of Heart.

One of the most common symptoms, found on examination, was that of dilatation of the heart. This was found most often as a dilatation of the left ventricle. According to Lees in a study of rheumatic fever, even in the most subacute attacks, acute dilatation of the heart is invariably found. When the attack is over the dilatation lessens and the cardiac dullness may again become of normal extent.

In children the dilatation is of much greater importance than valve lesions, and "it is remarkable how small may be the superficial evidence of a rheumatic toxaemia, which yet is able to produce a perceptible enlargement of the heart". (Lees). Garrod, Osler, Lees and Poynton all agree that dilatation of the heart is common in cases of chorea even in cases in which there is no indication of rheumatism other than the chorea, and no history of any previous rheumatic attack. Poynton and Lees found that in 33 cases without history of previous rheumatism there was dilatation of the left heart in 29.

This enlargement of the heart in cases of chorea and rheumatism is difficult to explain. It is not merely caused by debility, for it is often much more distinct in mild attacks of rheumatism than in more severe diseases. It is evidently in some way a special result of the rheumatic process. Pericarditis does not form the only cause of dilatation. Poynton

puts forward an explanation of the occurrence of the symptom. He says "that in rheumatism there is some toxic action exerted, on the cardiac muscle, enfeebling it and causing it to give way, before the normal blood pressure. This explains why the first sound becomes short, the area of dullness increased, and the impulse diffused. The feebler diastolic rebound, causing a weaker suction action in diastole, explains why the pulmonary second becomes accentuated." This we believe to be the case in the patients we have examined.

Dilatation occurred in -

Group (1) 3.

Group (2) 50. Total chorea cases 53 or 20%.

Group (3) 80. Total Rheumatism cases 39.2%.

Cardiac dilatation was associated with accentuation of the second pulmonary in 25 cases of chorea and in 47 cases of rheumatism.

Cardiac Murmurs.

Murmurs are frequently found in cases of chorea. Some of these are anaemic in origin, while some are due to chorea of the cardiac muscle itself. This point is not quite settled. Holt believes that this chorea of the cardiac muscle is possibly a cause. Osler on the other hand does not. The large majority of cases are due however to concurrent endocarditis.

There are some cases where the murmurs arise

from functional causes purely. There may be a soft systolic murmur heard at the base or at the apex which has its origin in the anaemia and debility which so often are accompaniments of chorea. This murmur according to Russell is propagated from the Pulmonary area, but occasionally in bad cases may be caused at the apex.

Though some authors hold that a murmur may be caused by "disordered action of the muscular apparatus connected with the valve," (Walshe.) others however among whom is Kirkes, are very much opposed to this theory.

In those cases, where there are definite signs of rheumatic infection we have no doubt, if cardiac trouble appears but that it is due to rheumatic endocarditis. Of our cases these form the majority. There are few causes of organic valvular disease and of these rheumatism is by far the most commonly found. West gives statistics which are taken from the examination of 135 cases of valvular disease, and he found 60% caused by rheumatism. Roger gives the percentage as 78% which Goodhart gives his results as showing 62%. Cheadle finds that 79% of his cases have rheumatic histories of articular rheumatism.

In those cases where no history of rheumatism is found there are often murmurs present. It is difficult to say whether these are examples of

rheumatic endocarditis or not. According to Cheadle "the cardiac affection of chorea is as a rule organic and not merely functional". He brings forward as proof of his statement some reasons which are well worth repeating. Functional murmurs usually are found in the Pulmonary areas, while in chorea the murmur as a rule is mitral. Also the murmur appears quite early in the disease before there is time for the anaemia and debility to develop. The view that the murmur is the result of spasm of the involuntary muscle of the heart has been practically disproved by Osler, who shows that all involuntary muscle was immune to choreic disturbance.

Cheadle goes on to show from post mortem examinations that the lesions are identical with endocarditis from other causes.

Osler states that there is no disease in which, post mortem, endocarditis has been found so frequently.

The majority of choreic murmurs persist or reappear later in life. Mackenzie found that 60% to 80% murmurs were found from one to five years afterwards. Osler found that chorea cases examined two years after dismissal, showed heart trouble in 54%. Grabois states that in his cases there was organic heart disease in 40% and of those 35% had a previous history of rheumatism. The Collective Investigation Committee results show that in chorea cases associated

with rheumatism there was a murmur in 50%, while in cases without rheumatic history, murmurs were present in only 35%.

On examining the reports of our cases we find that there was a mitral systolic murmur present in 112 or 42.2%. In 10 of those the mitral systolic murmur was not accompanied by any other sign of rheumatism or heart trouble. In the examination of the patients the heart murmur was found to be conducted well round into the axilla in 51 of the 112 cases.

In 32 cases or 12.1% there was present a mitral systolic murmur accompanied by cardiac dilatation, and in 19 of these was the murmur conducted round into the axilla. A persisting murmur at the mitral area conducted round into the axilla, is good evidence of some regurgitation through the mitral orifice. This regurgitation by the increase of tension caused tends to produce some dilatation of the ventricle. This we consider to be the case in the 19 mentioned above. Of the other cases where dilatation was present along with a mitral systolic murmur, which was not conducted, we might say that the dilatation was not caused by the valve lesion but that both dilatation and regurgitation were the results of an acute attack of rheumatism. The dilatation was there due to the toxic influences acting on the heart muscle while the regurgitation was due to endocarditis. In many cases according to Poynton

the damage to the valve is very slight, and does not account for the cases of cardiac failure found amongst this class of choreic patients. Lees is sure that the dilatation has much more to do with these cases of failure than the valvular lesion.

In our series of chorea cases there were three deaths, and each of these showed dilatation of the heart accompanied by mitral regurgitation. One of these was associated with an aortic systolic murmur and another with pericarditis.

Presystolic murmurs.

The presence of murmurs of this description is common in chorea and more often met with than is generally supposed. This lesion is according to Cheadle one of early life, and in children is especially rheumatic in origin. Of 273 cases of organic heart lesions Cheadle found that 33 showed presystolic murmurs at the mitral area. Sansom found 50% of his cases with presystolic murmurs were rheumatic and Duckworth gives his at 60%. There is a gradual development of this murmur in cases of chorea. The second sound at the mitral area becomes impure. This is then followed by a reduplication, and Sansom states that "reduplication of the first or of the second sound is an early sign of stenosis." There are many theories to explain this reduplication but none of them is yet known to be the correct one. Ludwig and Hesse have done some good work on this point. They

have tried to explain this symptom, by the fact that the mitral flaps, being thickened, cannot stretch so quickly as those of the other valves. So the sounds caused by their tension do not coincide with the sounds of the other valves. This reduplication of the second sound at the mitral area is the first sign of early mitral stenosis.

The next change we meet with is the occurrence of a definite presystolic murmur. This murmur is usually blowing in character.

This murmur is very often associated with a mitral systolic murmur, and in those cases it usually comes on a short time after the other has developed.

The reports of the cases we have examined show that a presystolic murmur was present in 26 of them. It was accompanied by a systolic murmur in all but eight. That is almost 10% of all our chorea cases had presystolic murmurs 20.3% of the heart cases belonged to this series. Osler found that 17.1% of his chorea patients showed presystolic murmurs even two years after they had been dismissed from Hospital.

Examining the cases in Group (3) we find that 49 showed mitral stenosis or 24% of all the cases and 31% of the heart cases.

We see from this that mitral stenosis is a common accompaniment of rheumatism in children (24%), and also of chorea where there is some rheumatic history (10%). It is uncommon in cases of chorea without rheumatic history. We found a presystolic

murmur present only in three cases out of 81 in Group (1), and in two of these there was a history of rheumatism in the mother of the patient.

Diastolic murmurs.

Marked murmurs were heard, diastolic in time, in 21 cases, that is 7.8% in Groups (1 and 2), and in 32 cases or 15.6% in Group (3). In each case this murmur was accompanied by a mitral systolic murmur. These murmurs did not develop till late in the history of the illness. In some of the chorea cases it was only a passing condition and the murmur disappeared entirely or was replaced by a presystolic.

Examination of the Base of the Heart.

This examination revealed the presence of murmurs over the Aortic area in six cases in Group (2), where systolic murmurs were made out, and in Group (3) in eighteen cases where systolic murmurs were heard and in four others where the murmur was diastolic in time. These murmurs were accompanied by mitral murmurs in Group (2) in four and in Group (3) in all the cases. They were usually associated with arthritis either present on admission or previous to admission.

Accentuation of the second pulmonary sound was found in 95 cases in Groups (1 and 2), eighteen being in Group (1). In Group (3) 100 cases are found to have this condition. Its presence is explained on a

previous page.

One of our cases is worthy of note in that there is found along with a rheumatic condition a congenital pulmonary bruit.

Case (49 Rheumatism).

F. aet 4¾, admitted Oct. 09., duration of attack one week. MR. There were pains in arms and legs during the last week and she has been troubled with sore throats. Is now complaining of breathlessness and joint pain. On auscultation there is a loud systolic murmur heard over the 4th interspace. It was diagnosed as a case of congenital pulmonary stenosis.

According to some authors, as Leslie, previous chorea cases are more likely to have heart trouble than other cases which have not had an attack of chorea before. We find that in our reports there are 86 cases which have been treated previously for chorea, and of those 46 show symptoms of cardiac murmurs, or 54.6% of the recurrent cases.

In those cases in which no cardiac murmur is heard, Barlow points out that there is still a possibility of their having cardiac disease. He reports a case where, though there was no murmur heard during life there were at the autopsy vegetations along the mitral valve. Kirkes pointed out that the apical murmurs of chorea were variable and Osler mentions two cases in which vegetations were

found post mortem, although no murmur had been detected on auscultation. One finds that if a patient leaves hospital after an attack of chorea, where there has been a valvular lesion which has disappeared, and is readmitted for a recurrence, the old lesion will be found to have returned, as in the following case.

Case 21 Chorea.

F. aet. 10 Dec. duration 2 weeks. FR. BR. GP. one year before admission. Chorea one year ago, was ill for 6 weeks. Two weeks ago developed "growing pains". Nod. on right elbow and both knees. Accentuated second pulmonary. Developed MS. Patient improved and was dismissed with no choreic movements and only faint MS. Readmitted April showing MS. now conducted round to the axilla. Readmitted March 1907 and April 1908.

Pericarditis.

As a definite sign of rheumatism this has been long recognised. Bright in 1836 in his Lumleian lectures of that year pointed this symptom out as one occurring in cases of chorea. Sibson states that out of 180 cases of acute rheumatism with affections of the nervous system 21 had chorea, while 15 of those patients with chorea had pericarditis. Osler found that pericarditis was present in 19 of his autopsies in chorea cases, or in 26% of deaths. There was a history of rheumatism in 8 of these cases that is in

42.1%.

Pericarditis according to Sturges is invariably present in cases of rheumatic carditis. He gives statistics of 100 cases of heart disease examined post mortem, and in only one did he find recent endocarditis without the pericardium being involved. This was a case of acute chorea which had no history of rheumatic infection. Lees found that in 150 cases the pericardium was affected in 141, and of these 75% showed adhesions of a more or less grave nature. These are cases of rheumatism however. Statistics of chorea cases are not to be found. These figures show that in fatal cases of Rheumatism the endocarditis is usually complicated with pericarditis.

In Osler's statistics of cases examined clinically, there were 20 out of 330 cases of rheumatic fever, that is 6% with pericarditis. Of our rheumatism cases in Group (3) 22 showed pericarditis, 10.7% and in Group (2) 6, 2.5%. Of these 50% recovered and were dismissed from hospital as well. There were 20 cases which ended fatally in Group (3) and 11 cases of these showed pericarditis, 55%. In our cases of Chorea there were 3 deaths, 2 showing pericarditis, 66.6%.

Summary of results of examination of hearts.

	Chorea.		Rheumatism.
MS. present in	112 cases or	42.2%	143 or 70%.
MS. & H^X	32	12.1%	66 or 32.3%
MP.	26	9.9%	49 or 24%
MD.	21	5.8%	32 or 15.6%
Total hearts affected	128 or	48.2%	158 or 77.4%
Definitely rheumatic	83 or	31.5%	or 64.8% of heart cases.
Cases which in themselves are rheumatic Heart affections			13
Cases of MS. and $2P^X$ and of MP. and $2P^X$ which have been counted as rheumatic			11
Cases of MS. and H^X with rheumatic family histories			3
Heart cases not counted as rheumatic			13 or 10%.
Cases of Pericarditis	6 or	2.5%	22 or 10.7%
Cases of death with peri.	2 or	66.6% of deaths	11 or 55% of deaths.

Fatal cases.

These cases are very few in number, and in the literature cases are very seldom mentioned of chorea patients who have died of chorea unassociated with some grave heart condition. In London Hospitals Sturges collected the reports of 80 fatal cases, and of these only five were found in which the heart was unaffected. Osler collected 73 cases of which 62 had endocarditis, and states that he has personally seen five cases end fatally in four of which there was endocarditis present. In the 73 cases collected by

Osler from the literature there were 43 or 58.9% in which the mitral valve was affected, in 13 the mitral and aortic valves were affected, and in one case was the aortic valve involved alone.

Death occurred in only three of our chorea cases and the reports are interesting.

Case (126) Chorea.

F. aet. 8½, admitted February 1906, duration of present illness three weeks. Had pains in the joints in December 1905. Now admitted with pain and swelling of the joints; the wrist and ankle joints are swollen. Erythema is present on the legs. There are nodules to be found in numbers especially over the ankles and behind the elbows. On examination MS. is found and the murmur is conducted well round into the axilla. The left and right borders of the heart are found to be displaced outwards. Pericarditis is present and for a time there is a friction rub heard at the base. This disappeared however. Death supervened after a fairly long illness. P.M. showed the presence of an adherent pericardium and both mitral and aortic endocarditis.

Case (206) Chorea.

F. aet. 6, admitted December 1906, duration 6 days. In previous illness we find she had Sc.F. and M. Six days before admission patient complained of pains in wrists and legs. On examination the pulse was found to be very irregular, and over the apex a mitral murmur systolic in time was heard. On the 8th

day after admission patient suddenly became very breathless. Death occurred from acute cardiac failure. Heart showed dilatation and myocarditis.

Case (253) Chorea.

F. aet. 11, admitted May 12, duration of illness 12 days. FR. father also insane. Patient became sick, and developed a temperature. Was very fevered on admission. Became delirious. On examination MS. was found which had murmur well conducted round to the axilla. Pericardial friction sounds then were heard. After this, child showed choreic movements for the first time. The second pulmonary sound became accentuated. On the second day after admission the patient died. No P.M. was granted.

In each of cases (126) and (253) there was definite sign of fluid in the pericardium and of myocardiac change as well.

Cardiac dilatation was found in all three associated with a murmur of the mitral area, systolic in time.

Percentage of deaths in chorea is 1.1% in our cases.

Conclusion.

We have now completed our inspection of the cases and it remains for us to look back over our results and to try to draw together the main points found. The inclusion of the cases of rheumatism in this examination is made for one main reason, though

there are some minor ones which are not in themselves so important. The reason is that one cannot go thoroughly into the origin of chorea without having to compare the results found with rheumatic cases. To do this well one must find cases which compare closely, as regards age, social conditions and natural conditions, (such as weather and climate). This is best done by taking notes of those cases occurring in the same hospital, during the same period of years, and treated by the same physicians. In our cases of rheumatism in Group (3) we have a series of cases which fulfil those requirements. We now turn to the theory of the rheumatic origin of chorea, and try to look at it in the light of our results.

From our results with regard to the sex of the majority of chorea patients, we find that females are affected far more frequently than males, 72.8% of the cases being female. If we compare the results obtained from Group (3) we see that females were affected only in 56% of the cases. This fact leads us to believe that chorea affects females more often than males because of the naturally more excitable condition of the brain cortex in the female.

The age incidence chart shows us that males are affected earlier in life than females. This is not a very definite conclusion but there is a marked rise in the number of males aet. 6 while the rise in number of females is more noticeable aet. 7.

In the comparison of the age incidences of chorea and rheumatism there is a very definite relationship seen. By the chart we see that the ages at which those two affections arise are practically similar. The percentage of cases of rheumatism occurring at the ages of 7 and 8 are rather less than the per centages of the chorea cases. Otherwise the two percentages are similar.

With regard to our results from the study of the seasonal incidence of chorea, we find that the cases of chorea are more plentiful in the months of December, January and February while the cases of rheumatism are more plentiful in the months of November, December and January. We consider that as we find such a large number of cases of chorea following after attacks of rheumatism, the above results are naturally to be expected. The seasonal incidence of chorea seems to be one or two months later than that of rheumatism.

We have found that exciting causes were present in many cases. Thread worms, imitation and fright have been considered. There is, however, in those cases probably some rheumatic infection. Two of the thread worm cases out of three, the imitation case and 64.8% of the cases of fright had definite rheumatic histories. These causes are only superimposed on other conditions which are more truly the cause of the chorea.

Certain forms of arthritis may develop after

Scarlet Fever and cases may show heart symptoms as well without the case being one of rheumatism. However there is no means of differential diagnosis between the two conditions.

Neurotic history has been considered a point which is against the rheumatic theory. We found that 6% of our cases gave evidence of some neurotic condition either in their previous histories or in the lives of the parents. In the rheumatic cases there was only 2.5% with a neurotic history.

When we note this point and consider along with it the sex incidence and the history of onset of the illness being so often associated with fright or shock, as the result of injury, operation or emotional disturbance, we see that there must be some neuropathic condition necessary before the rheumatic infection can bring on chorea, if it is this infection which is the cause of the illness. The shock or fright in chorea seems to act in the same way as a chill acts in acute rheumatism, by lowering the vitality of the patients and making them more susceptible to the invading organismal infection.

This whole factor of the neuropathic history, in chorea cases, is in our minds in favour of the theory that chorea is a cerebral form of rheumatism. Also we find that the toxins or micro organisms of rheumatism act on the brain cortex of unstable and excitable children in such a way as to bring on an

attack of chorea minor.

In considering the family history of our chorea cases, we see 34.1% had some definite history of rheumatism having occurred in the family, while there was a percentage of 40% of our cases which showed either rheumatic or chorea in the family history. Chorea itself occurred in 9.8% in the family histories. We have dealt fairly fully with this question before, but would just bring it up now to show the close relationship there is between the two affections. It is pretty certain that if a child suffers from "growing pains" and there is some family history of rheumatism then the child is rheumatic as well. Because of this we might count as rheumatic those cases in Group (1) which have a definite rheumatic family history.

Recurrences appeared in 32.7% of our cases of chorea. The number occurring in cases in Group (1) is about half in percentage as compared with those in Group (2). This point may be explained by the fact that during first stay in hospital there may be no symptoms other than the chorea; but when a case is admitted for the second time we may find that some complication has set in. It is well known that on first attack of chorea there is a tendency for the heart to dilate. On second attack this dilatation may be accompanied by a murmur.

The definite signs of rheumatism found in chorea

cases have been gone into before and there are only one or two points which we would like to bring out now. There is the question of the relationship of epistaxis to rheumatic infection, associated with heart disease. We would also like to mention here the fact that nodules were not common accompaniments of chorea, but when they did appear, the case was a serious one though not necessarily associated with pericarditis or terminating in death. Though those cases were dangerous still they did not turn out to be so hopeless as some authors would have us believe such cases are. Heart cases were very common among our chorea patients and the majority of these were rheumatic in nature. Mitral systolic murmurs were found in 42.2%. Dilatation was also very common. Enough, however, has been said about the heart troubles in previous pages. We have only mentioned the point now to emphasise the fact that rheumatism is at the origin of the majority of these.

So far we find that 155 cases of chorea have definite symptoms of rheumatic infection. To these we have added those cases which have been found with heart lesions originating from some rheumatic infection. This brings our number up to 182, showing a percentage of 69.2%. Now to those we might add the cases in Group (1) which have rheumatic family histories. This would give us a percentage of 77.9%. This result shows that there is certainly ground for the theory that rheumatic infection is the origin of

the majority of the cases of chorea. We look at the 22.1% of cases in which there is not found any rheumatic history and find that in some cases the child was brought to hospital by a neighbour who did not know any thing about the previous health of the child. Other cases may have had slight attacks of rheumatism without being treated for them or without any rheumatic affection being suspected. Then the family history is only a note of definite rheumatism in the parents or other members of the family and does not touch minor affections which are in themselves rheumatic; nor does it include mention of heart lesions. These unavoidable deficiencies in the evidence prevent us from arriving at a clear answer to the question whether <u>all</u> choreas are rheumatic in origin. Certainly they would seem to account for at least some of the cases included in the 22.1% which have no rheumatic history recorded.

What is clear from the study of our cases is that there is a very close relationship between the age and seasonal incidences of chorea and the age and seasonal incidences of rheumatism, and also that a very large percentage of cases of chorea show rheumatic histories. When we take these facts along with the very definite Bacteriological results of Poynton, Paine, and others to which we have alluded early in our paper there can be little doubt that the origin of all true cases of chorea minor is rheumatism.

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Lists of Abbreviations used.

M. Male. F. Female.

F.R.	Father with rheumatic history.
M.R.	Mother with rheumatic history.
X.R.	Brother or sister rheumatic.
F.M.	Father and mother rheumatic.
F.C.	Father with chorea.
M.C.	Mother with chorea.
X.C.	Brother or sister with history of chorea.
N.	History of fits in patient or insanity or nervousness.
G.P.	Growing pains.
S.T.	Subject to sore throats.
Rh.F.	History of attack of rheumatic fever.
Jts.	History of pains in joints with swelling.
Nod.	Rheumatic nodules.
Ery.	Erythema nodosum.
Pr.	Previous attacks of chorea.
F.	Fright.
M.S.	Mitral systolic murmur.
M.P.	Presystolic Mitral murmur.
$2P^X$.	Second sound at Pulmonary area accentuated.
H^X.	Dilated heart.
Sc.F.	Scarlet Fever.
Wh.C.	Whooping cough.
Mor.	Measles.
Peri.	Pericarditis.
D.	Death.
T.W.	Thread worms.

Reports of cases of Chorea in Group (1).

2. 8½. F. Mar.

4. 4. M. Dec. MR.SCh. 2nd.

5. 7½. F. Feb.

13. 6. F. Aug.

25. 11. F. Feb. N.

33. 9¾. F. Feb. N.

38. 7. F. Sep.

42. 6. M. Sep.

57. 5½. F. Sep. Mor.

58. 9½. F. May, FR.SR.

65. 10. F. Feb. MCh.

69. 6½. F. Mar. FM.R. SR. 2nd.

86. 7¾. F. Oct.

92. 7. F. Mar.

106. 10. F. Mar. FR. Sch.

107. 10. F. Apr.

111. 10. M. Aug.

112. 7. F. Jul. 3rd. Accident.

118. 7. M. Apr.

119. 9. F. Aug. Mother epileptic.

121. 6. F. Oct. FR. FM. nervous. F.

125. 5. F. Jul.

127. 7. F. Mar.

138. 7½. F. Jan. FR. N.

158. 5. M. Nov. MR. Sc.F.

164. 11. M. Jun. FR. Mor. Wh.C.

168. 8¾. F. Oct. Wh.C. Mor. $2P^{X}$, imitation.

Group (1) continued.

176. 11½. F. Oct. Wh.C. Sc.F., 2nd.

177. 9. F. Jun. Mor. Sc.F.

197. 10. F. Nov. Mor. Sc.F. 2nd.

198. 10. M. Dec. Wh.C. Mor.

202. 8¼. F. Feb.

205. 9. F. Nov. Wh.C. Mor.

207. 10. F. Dec.

214. 5. F. Nov. N.

216. 6½. F. Oct. MR.

217. 8. F. Feb. Wh.C. Mor. F.

218. 10. M. May. N. Mor. Accident.

220. 8. M. Jul. Wh.C. Mor.

224. 11. M. Sep. Wh.C. Mor.

227. 7. F. May. FM.R. Mor.

251. 11. F. Apr. Wh.C. Mor.F.

252. 7. F. May. Mor.

256. 7. F. Jun. Mor.

263. 11. M. May. Mor. F.

103. 4. M. Jan. BR. 2BCh.

231. 7. F. Oct.

Cases of Chorea associated with heart trouble not definitely rheumatic.

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10. 6½. F. Jan. MS.

11. 8¾. F. Jul. FM.R. MS.

72. 9. M. Jun. MS.

Group (1) continued.

115. 6. M. Dec. MS.

201. 9. F. Feb. MS. T.W.

211. 5. F. Apr. MR. Wh.C. Mor. MS.

228. 11½. F. May. Wh.C. Mor. Sc.F. MS. Caused by Sc.F.

235. 10. F. Feb. N. Mor. MS. F.

248. 6¼. M. Jul. N. Mor. MS. Caused by Diphtheria.

241. 6. F. Sep. MS.

12. 6½. F. Jan. MR. MP. F.

41. 8. F. Aug. MR. MP.

225. 7½. M. Jan. Mor. MP.

The following cases have signs of chorea and dilated Heart.

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113. 7. F. Jan. MR. H^X.

120. 7. F. Sep. XR. XCh. H^X.

236. 5½. M. Dec. FR. H^X.

Cases where there is history of chorea with reduplication of second pulmonary sound.

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8. 8½. F. Dec. $2P^X$.

9. 4. F. Mar. $2P^X$.

39. 11. F. Jan. $2P^X$. 2nd. Readmitted for 3rd. aet. 12.

93. 7. F. Apr. MR. N. $2P^X$.

122. 9½. F. Nov. $2P^X$.

132. 8. F. Jun. $2P^X$.

Group (1) continued.

148. 8. F. Mar. 2P^{X}. F.

159. 12½. F. Oct. 2P^{X}. F. 3rd.

160. 8. F. Dec. MR. 2P^{X}. Mor. Wh.C. Sc.F.

171. 10. M. Sep. FR. N. 2P^{X}. T.W. 3rd.

189. 9. M. Oct. 2SCh. Mor. Sc.F. Wh.C. 2nd. 2P^{X}.

213. 7¼. F. Aug. Mor. 2P^{X}. F. Readmitted for 2nd. aet. 8.

223. 7. F. Sep. 2P^{X}. F.

234. 11. F. Dec. MF.R. Mor. Sc.F. Wh.C. 2P^{X}. F.

240. 4. F. Aug. 2P^{X}. Mor. Wh.C., 2nd.

246. 10. F. Sep. 2P^{X}. 2nd. Readmitted for 3rd aet.12.

255. 7½. F. Mar. 2P^{X}. Mor.

233. 8¼. F. Aug. 2P^{X}. Mor. F.

Statistics of GROUP (2) Chorea and Rheumatism.

1. 9. F. GP. ST. $2P^X$. 3rd.

3. 6. F. Nov. Sc.F. ST. 2nd.

6. 8. M. Apr. N. MS.MP. $2P^X$.

7. 8½. M. Aug. Mor. Sc.F. Wh.C. GP. caused by Sc.F.

14. 10. F. Jun. Jts. readmitted aet. 11.

15. 6¾. F. Aug. MF.R., ST. Jts. $2P^X$.

16. 11. F. Feb. ST.

17. 7. M. Aug. Jts. Nod.

18. 9. F. Jan. ST. Jts. AS. $2P^X$.

19. 5¾. F. Nov. MP. $2P^X$. readmitted aet. 6½.

20. 6. F. Jan. MR. N. Eryth.

21. 10. F. Dec. XR. GP. Nod. MS. $2P^X$. 5th.

22. 11. F. Nov. GP. MS. F.

23. 6. F. Jun. Mr. N. ST. Jts.

24. 6. F. Sep. MP. $2P^X$. Ery. 2nd.

26. 7. F. Sep. Jts.

27. 8. M. Aug. Mor. ST. Jts. MS.

28. 9. F. Jul. FR. MS. MD. $2P^X$. Peri. 3rd readmitted aet. 10 and 11.

29. 10¾. F. Nov. MP. H^X. Peri. Nod.

30. 9½. F. May. MS. MP.

31. 7. F. Jan. FM.R. Jts. MS. $2P^X$.

32. 11. F. Jun. Jts. MS. $2P^X$.

34. 8. F. Jan. Sc.F. MS. MP. H^X. caused by Sc.F.

35. 7. F. Mar. MR. N. Sc.F. MS. $2P^X$. H^X. caused by Sc.F.

36. 9. F. Feb. GP. ST. 2nd.

37. 6½. M. Sep. MR. Jts. MS. H^X.

Group (2) continued.

40. 11. F. Feb. MS. MP. $2P^{X}$. 2nd.

43. 9½. F. Jan. Jts. N. 2nd.

44. 5. F. Feb. Jts.

45. 11. M. Jan. G.P.

46. 8. M. Feb. FR. MS. 5th.

47. 9. M. Feb. Rh.F.

48. 5. F. Dec. MS. $2P^{X}$.

49. 11. F. Nov. Jts.

50. 6. F. Oct. Mor. H^{X}. $2P^{X}$. 2nd.

51. 8. M. Dec. Mor. GP. ST. Nod. caused by Mor. 2nd. Readm. aet. 9.

52. 11½. F. Dec. FR. Jts. Ery. MS. F. 2nd.

53. 10. F. Apr. Jts. MS. F.

54. 7. M. Apr. FR. Jts. Ery. N. MS. $2P^{X}$.

55. 11. M. Feb. SR. SCh. ST. 2nd.

56. 4¾. M. Feb. MS. H^{X}.

59. 9. F. May. MS. $2P^{X}$. 5th. readm. aet. 9½ and 10.

60. 8. F. Aug. ST.

61. 7. M. Jan. GP.

62. 10. F. Jul. Rh.F. MS. 4th.

63. 9. M. Aug. Jts. ST. 2nd.

64. 7½. F. Jan. Sc.F. GP. caused by Sc.F.

66. 8½. M. May. MS. $2P^{X}$.

67. 12. F. Nov. Nod. Peri. MS. MP. MD. 2nd.

68. 10. M. Jan. Jts. Nod. $2P^{X}$. 2nd.

70. 7. F. May. MR. $2P^{X}$. AS. 2nd.

71. 6. F. Feb. MR. Jts. Ery. F. Rh.F.

Group (2) continued.

73. 6½. F. Jun. MR. MS. $2P^X$.

74. 10. F. Jun. N. Jts.

75. 10½. F. May. FM.R. S&B.Ch. Rh.F. Ery. MS. MP. $2P^X$. 2nd.

76. 6½. M. Aug. MS. MD.

77. 11. M. Jul. Rh.F. MS. $2P^X$. H^X. F.

78. 9¾. F. Jul. FM.R. XCh. ST. MS. MD. H^X.

79. 9. F. Sep. Nod. MS. $2P^X$.

80. 7½. F. Sep. MS. H^X.

81. 6. F. Oct. ST. Jts. Sc.F. caused by Sc.F.

82. 8¾. F. Oct. FR. Jts.

83. 10. M. Apr. Jts. AS. 2nd.

84. 8¼. F. Nov. BR. BCh. GP. ST. H^X. Jts.

85. 9¾. M. Nov. MR. Jts. $2P^X$. F.

87. 11. F. Nov. MR. Jts. 2nd.

88. 7½. F. Nov. MR. ST. Jts. MS. MP. MD. H^X. $2P^X$. 2nd.

89. 9. F. Dec. MR. Jts. 2nd.

90. 9½. F. Oct. XR. Jts. Nod. Ery. MS. $2P^X$.

91. 7½. F. Sep. Jts.

94. 8. F. Dec. MR. N. ST. Jts. $2P^X$. F.

95. 8½. M. Apr. N. GP. Jts.

96. 11. M. Apr. XR. MCh. GP. ST. Nod. MS. $2P^X$. readm. aet. 12½.

97. 7¼. F. Mar. F. MP. $2P^X$. 3rd. readm. aet. 8½.

98. 3½. F. Feb. GP. MS. $2P^X$. 2nd.

99. 9. F. Aug. FR. MS. MD. H^X.

100. 7¾. M. Nov. Nod.

101. 10. F. Apr. Rh.F. MS.

Group (2) continued.

102. 9. M. Nov. BR. XCh. ST.

104. 7¾. M. Feb. ST. H^X. 4th.

105. 7½. F. Jul. Ery. Epi.

108. 8½. M. Jan. BR. GP. MS. MD. $2P^X$.

109. 8¼. M. Sep. FR. Jts. MS. MD.

110. 9. F. Jul. Jts. MS. MD.

114. 9. F. Mar. Epi. MS. H^X. 3rd.

116. 8. F. Feb. MR. Rh.F.

117. 9. F. Apr. MR. Jts. MS. $2P^X$.

123. 8¼. M. Apr. Jts. MS. $2P^X$. F.

124. 8. M. Jan. FR. MS. $2P^X$.

126. 8½. F. Feb. Jts. Nod. Ery. Peri. MS. AS. H^X. D.

128. 6. F. Mar. N. Jts. Ery. MS.

129. 9¾. M. Feb. N. GP. MS. MP. F.

130. 8½. F. May. MR. N. Jts. MS.

131. 10½. F. May. Rh.F. $2P^X$.

133. 6. M. May. FM.R. N. GP. ST. MS. $2P^X$. 4th.

134. 10. F. Aug. FR. GP.

135. 10. F. Oct. Jts.

136. 9¾. M. Oct. N. Ery. MS.

137. 8½. F. Nov. Nod.

140. 10¾. M. Jan. FR.MCh. GP. MS. MD. $2P^X$.

141. 7¾. F. Mar. MCh. MS. MP. $2P^X$. 2nd.

142. 7. F. Sep. ST. Jts. Nod. Rh.F. MS. MP. MD. readm. aet. 9.

143. 11. F. Apr. FM.R. MS. H^X. $2P^X$. Psoriasis. 2nd.

144. 5¾. M. Jul. XR. MS. H^X. $2P^X$.

145. 6. M. Oct. FR. N. Ery. MS. $2P^X$. H^X. readm. aet. 7.

Group (2) continued.

146. 9. F. Sep. XR. GP. Rh.F. $2P^X$.

147. $11\frac{3}{4}$. F. Oct. MCh. Nod. H^X. $2P^X$.

149. $7\frac{1}{2}$. F. Oct. SR. Rh.F.

150. $7\frac{1}{2}$. F. Dec. XR. Mor. Jts. Epi. MS. MP. MD. AS. $2P^X$. H^X. 2nd.

151. $9\frac{1}{2}$. F. Mar. SR. Jts. F. MS. $2P^X$. H^X.

152. 10. F. Mar. N. Jts. MS. 11th time in hospital.

153. 7. F. Apr. Mor. Jts. MS. H^X.

154. 9. M. May. N. ST. MS. F.

155. 11. F. Jun. MS. MP. $2P^X$. F. 2nd.

156. $8\frac{3}{4}$. M. Aug. Mor. Sc.F. Jts. MS. readm. aet. $9\frac{1}{2}$.

157. 9. F. Mar. Mor. ST. Nod. MS. MP.

161. 12. F. Dec. MR. Mor. Wh.C. GP. F. 2nd.

162. 5. M. Jan. SCh. N. Mor. ST.

163. 8. F. May. FM.R. N. GP. Nod. Rh.F. $2P^X$. H^X. F.

165. $8\frac{1}{2}$. F. May FM.R. N. Mor. Jts.

166. $10\frac{1}{2}$. F. Jun. FM.R. BCh. Mor. Sc.F. Wh.C. GP. ST. H^X. readm. aet. $11\frac{1}{2}$.

167. 9. F. Oct. Jts. MS.

169. $9\frac{1}{2}$. F. Dec. XR. Jts. Nod. MS. $2P^X$.

170. $10\frac{1}{2}$. F. Nov. Mor. Wh.C. Jts. $2P^X$. H^X.

172. $9\frac{1}{4}$. F. Jan. BR.N. Mor. Wh.C. ST. Jts.

173. 4. F. Mar. MS. H^X.

174. $8\frac{1}{4}$. F. Apr. SCh. Sc.F. Jts. MS. $2P^X$. caused by Sc.F. readm. 4 times.

175. 6. M. Jun. MR. N. Mor. Sc.F. GP. ST. $2P^X$.

178. 9. F. Aug. Wh.C. Jts. 2nd.

179. 9. M. Aug. MR. Mor. Wh.C. Jts. MS. 2nd.

180. 8. F. Aug. FR. Mor. Wh.C. ST. MS. MP. MD. $2P^X$. T.W. readm. aet. 9.

Group (2) continued.

181. 6. F. Oct. MR. Jts. $2P^X$.

182. 6. M. Nov. XR. Mor. Wh.C. ST. Jts. MS. H^X.

183. 11. F. Dec. Mor. Wh.C. GP. H^X. readm. aet. 12.

184. 9½. F. Feb. GP. MS. H^X.

185. 11¾. F. Apr. MS. $2P^X$.

186. 8½. F. Jun. MS. MD. $2P^X$. H^X.

187. 10. F. Jul. Mor. ST. Rh.F. Jts. H^X. $2P^X$.

188. 11. F. Sep. Mor. Wh.C. GP. MS. $2P^X$. 2nd.

190. 4½. F. Dec. SCh. Mor. Sc.F. Wh.C. Jts. MS. H^X. $2P^X$. readm. aet. 5.

191. 10. F. Feb. N. Mor. Wh.C. GP. H^X.

192. 10. F. Feb. SCh. Mor. MS. H^X. F.

193. 9. M. Mar. Mor. Wh.C. Jts.

194. 8. F. May. SR. N. Mor. Jts. MS. F. 2nd.

195. 9. M. Aug. MR. N. Mor. Sc.F. Wh.C. MD. $2P^X$. F. 3rd.

196. 5. F. Aug. Mor. Wh.C. Jts. Peri. MS. MD. $2P^X$. H^X. readm. aet. 5½.

199. 10½. F. Dec. Mor. Jts. F. 2nd. readm. aet. 11.

200. 11¼. F. Apr. MR. GP. MS.

203. 10. M. Sep. Mor. ST. F.

204. 9. F. Nov. Mor. Wh.C. ST. Ery. MS. accident. 2nd.

206. 6. F. Nov. Mor. Sc.F. Jts. MS. H^X. D.

208. 10. F. Feb. FR. Mor. Wh.C. GP. ST. MS.

209. 11. F. Mar. Wh.C. GP. ST.

210. 8. F. Jan. ST. MS. $2P^X$. 2nd.

212. 10. F. Aug. FR. Mor. Wh.C. ST. Jts. F.

215. 5½. F. May. FR. Jts. MS. MP. H^X. $2P^X$. 2nd.

Group (2) continued.

219. 10. M. Jun. N. Mor. Wh.C. Rh.F. Jts.

221. 10. F. Jun. Jts. 3rd.

222. 7¼. F. Jun. Mor. Sc.F. MS. caused by Sc.F.

226. 5. F. Dec. MCh. Wh.C. MS. $2P^X$. 3rd.

229. 5. M. Feb. XR. Mor. Wh.C. Ery. MS. $2P^X$. H^X.

230. 8. M. Oct. Wh.C. MS. MD. $2P^X$. H^X. 3rd.

232. 9. M. Jun. FR. FCh. Mor. Wh.C. ST. Jts. Nod. MS. MP. MD. H^X. $2P^X$. readm. four times.

237. 11. M. Feb. FM.R. Mor. Sc.F. Wh.C. ST. Rh.F. 3rd.

238. 9. F. Mar. MR. Mor. Sc.F. Rh.F. MS. H^X. caused by Sc.F. 2nd.

239. 7. M. Apr. MR. Mor. Wh.C. MS. $2P^X$. F. 3rd.

242. 11¼. F. Oct. FR. Mor. Wh.C. Nod.

243. 8. F. Dec. Mor. Wh.C. Jts. Herpes.

244. 11. F. Feb. N. GP. ST. Rh.F. Epi. MS. MP. $2P^X$. 2nd.

245. 8. F. Sep. Mor. Wh.C. Ery.

247. 6½. F. Jul. N. Mor. ST. Jts. Nod. MS. MP. MD. H^X. Lichen.

249. 7¼. F. Feb. FR. Mor. Wh.C. Rh.F. Jts. MS. AS. MD.

250. 9½. F. Mar. MCh. N. Mor. Wh.C. Rh.F. MS. $2P^X$.

253. 11. F. May. FR. N. Rh.F. Peri. MS. $2P^X$. D.

254. 9. M. May. XR. SCh. Mor. Wh.C. Jts. $2P^X$.

257. 9. M. Nov. Mor. Wh.C. GP. ST. Ery. MS. $2P^X$. H^X.

258. 11½. F. Dec. Mor. Jts. MS. MD. $2P^X$. H^X. 3rd.

259. 9. F. Dec. Mor. Wh.C. GP, ST.

260. 5¼. F. Jan. MCh. Mor. ST. Jts. MS. $2P^X$.

262. 6½. M. Jan. BR. Mor. GP. ST. $2P^X$. H^X.

Reports of cases of Rheumatism. Group 3.

1. 9½. F. Mar. ST. Jts. Ery. MS. H^X.
2. 11 mths. F. Nov. MS. AS. MP. Peri. H^X. D.
3. 10. F. Feb. MR. Jts. ST.
4. 10. F. Jan. MR. MS. H^X.
5. 8. F. Mar. MR. Jts.
6. 7. F. Sep. MS. MP. H^X. D.
7. 10. M. Sep. ST. Jts. PS. H^X. $2P^X$.
8. 8. M. May. Jts. Nod. MS. MP. MD. $2P^X$. H^X.
9. 10. M. Dec. JTS.
10. 8. M. Nov. JTS. MS. H^X. $2P^X$.
11. 8. M. Jun. JTS. ST.
12. 5¼. M. Jul. JTS. MS.
13. 8½. M. Aug. JTS. MS. MD. MP. AS. H^X. $2P^X$. D.
14. 10. F. Feb. BR. MS. H^X.
15. 7. F. Jul. JTS. MS.
16. 8½. M. Mar. FR. FCh. JTS. NOD. ST. Peri. GP. MS. $2P^X$. H^X.
17. 6. F. Sep. JTS. ERY. MS. $2P^X$. D.
18. 8. M. Sep. JTS. ST. GP.
19. 6½. F. Dec. Peri. MS. MP. H^X. D.
20. 9¼. M. Dec. MR. MP. MD.
21. 9. M. Dec. JTS. EPI. MS. AD.
22. 3½. M. Mar. FR. ST. MS. Peri. H^X.
23. 4¾. M. Jan. FR. JTS. NOD. EPI. ERY. MS. MD. $2P^X$.
24. 9. M. Oct. JTS. MS.
25. 2¾. F. Dec. MR. JTS. NOD. ST. MS.
26. 5½. F. Dec. FR. ST. JTS. ERY.
27. 9. M. Dec. JTS. ST.

Group (3) continued.

28. 7. M. Oct. JTS. MS.

29. 5. F. Jun. FR. NOD. MS. H^X.

30. 9. F. Jul. FR. EPI. JTS. MS. MP.

31. 5. M. Dec. JTS.

32. 11. F. Sep. JTS. ST. MP.

33. 9. F. Mar. FM.R. JTS.

34. 8. F. Feb. GP. JTS.

35. 8½. M. Jan. FM.R. JTS. NOD. MS. MP. $2P^X$.

36. 5. M. Feb. FR. JTS. NOD. ST. MS.

37. 8. F. Jan. NOD. MP. MS. $2P^X$.

38. 6. F. Mar. MR. JTS. MS. $2P^X$. H^X.

39. 9. M. Apr. MR. GP. NOD. MS. MD. $2P^X$.

40. 11. M. Mar. MS. H^X.

41. 7. F. Dec. JTS. MS.

42. 7½. F. Jan. FM.R. JTS. MS. $2P^X$.

43. 4. M. Dec. GP. JTS. MS. MD. AS.

44. 5. F. Nov. JTS. MS. $2P^X$.

45. 9. F. Jul. BR. ST. JTS. MS. MD.

46. 10½. F. Aug. GP. ST. MS. $2P^X$.

47. 9. F. Jun. JTS. ST. MS. AS. $2P^X$.

48. 11. M. May. JTS. ST. NOD. MS. $2P^X$.

49. 4¾. F. Oct. MR. JTS. ST.

50. 11. F. Jul. FM.R. GP. ST. NOD. MS. MD. $2P^X$.

51. 9. M. Oct. GP. ERY. JTS. MS. MP. $2P^X$.

52. 9. F. Nov. FR. GP. JTS. MS. MP. MD. AS. Peri. $2P^X$. H^X. D.

53. 10. M. Jan. MS. JTS. Peri. MP. MD. $2P^X$. H^X.

54. 4. F. Jan. JTS. MS. $2P^X$. H^X. D.

Group (3) continued.

55. 6½. F. Sep. JTS. MS. $2P^X$. H^X.

56. 3. F. Jan. MF.R.BR. ST. JTS. ERY.

57. 5. F. Feb. MR. JTS. ERY.

58. 9. M. Feb. JTS. MS. $2P^X$.

59. 10½. F. Jul. ST. JTS. MS. $2P^X$. H^X.

60. 7. M. Aug. FR. FCh. MR. JTS. ST. MS.

61. 7½. F. Mar. MR. EPI. GP. NOD. ST. $2P^X$. H^X.

62. 3½. M. Sep. JTS.

63. 9¾. F. Dec. MR. NOD. JTS. Peri. MS. $2P^X$. D.

64. 7. F. Dec. JTS. MS. $2P^X$. H^X.

65. 10. M. Dec. ST. MS. NOD. MP. H^X. $2P^X$.

66. 8½. M. Jan. JTS. MS. AS. Peri. H^X. $2P^X$. D.

67. 11. F. Jan. MR. ST. JTS. EPI. MS. $2P^X$.

68. 7. F. Nov. JTS. ST.

69. 9½. F. Dec. MF.R.ST. JTS. H^X.

70. 10¼. M. Feb. JTS. MS.

71. 8¾. M. Jan. FR. JTS. $2P^X$.

72. 11¼. F. Jul. XR. JTS. ST. MS. $2P^X$.

73. 11. F. Aug. FR. JTS. EPI. NOD. MS. MP. AS. $2P^X$. H^X.

74. 5. M. Mar. NOD. JTS. Peri. MS. PS. H^X. $2P^X$.

75. 6½. M. Feb. FR. JTS. H^X.

76. 3¾. M. Feb. JTS. ERY.

77. 10¾. F. Apr. XR. JTS. MS. MP. H^X.

78. 9. F. Apr. NOD. MS. MP. AS. AD. $2P^X$.

79. 6. F. Jun. JTS. NOD. Peri. MS. AS. H^X.

80. 11. F. Jul. MS. MD.

81. 11. F. Sep. XR. JTS. MS. MD. MP. $2P^X$.

Group (3) continued.

82. 7¼. M. Nov. JTS. NOD. $2P^X$.

83. 10. F. Dec. MR. SCh. GP. ST. $2P^X$.

84. 6. M. Dec. JTS. MS. H^X.

85. 9. F. Jan. JTS. MS. MD. $2P^X$.

86. 10. M. Jan. MR. JTS. $2P^X$.

87. 12. M. Jan. MR. JTS. MS. $2P^X$.

88. 6. M. Feb. FR. JTS. MS. $2P^X$. H^X.

89. 11. M. Dec. JTS. NOD. $2P^X$.

90. 9½. M. Sep. FM.R.ST. JTS. MS. MD. MP. H^X. $2P^X$.

91. 10. M. Mar. SR. JTS. EPI.

92. 7. F. Mar. JTS.

93. 8. M. Apr. ST. JTS. NOD. MS.

94. 6¾. F. May. SR. JTS.

95. 8. F. May. JTS. ERY. NOD. MS. MD. $2P^X$. H^X.

96. 8. M. Jun. XR. SCh. JTS. MS. MP. $2P^X$.

97. 6. M. Oct. GP. JTS. NOD. MS. MP. $2P^X$. H^X.

98. 9. M. Feb. GP. ST. H^X.

99. 6. F. Sep. JTS. Peri. MS. MD. H^X.

100. 6½. M. Jan. JTS. MS. $2P^X$.

101. 6. M. Feb. JTS. H^X. $2P^X$.

102. 5¾. M. Apr. JTS. ERY.

103. 11. M. May. MR. JTS. ST. ERY. MS. H^X. $2P^X$.

104. 9. F. May. ST. JTS. ERY. MS. MP.

105. 3½. M. Jun. JTS.

106. 9. M. Jan. NOD. Peri. MS. MD. MP. PS. PD. H^X. $2P^X$. D.

107. 2¾. M. Dec. ERY. NOD. EPI. Peri. MS. MD. H^X. D.

108. 7. M. Sep. JTS. MS. MD.

Group (3) continued.

109. 7¾. F. Nov. JTS. NOD. Peri. MS. MD. AS. H^X. 2Px. D.

110. 9. M. Dec. JTS. H^X.

111. 3. F. Aug. SR. JTS. NOD. EPI. Peri. MS. MD. D.

112. 10½. M. Sep. JTS. NOD. MS. AS. MD. H^X. $2P^X$.

113. 11. M. Dec. FR. JTS. NOD. MS. PS. H^X. $2P^X$.

114. 4½. F. Jul. JTS. ST.

115. 9½. F. Feb. XR. JTS. EPI. $2P^X$.

116. 11. M. Oct. JTS. MP. H^X.

117. 8¼. M. Apr. NOD. ST. MS. MD. H^X.

118. 10. F. Apr. XR. JTS. ST. GP. H^X. [illegible].

119. 11. M. Apr. FR. JTS. NOD. MP. $2P^X$. H^X.

120. 11. M. Mar. GP. NOD. JTS. $2P^X$.

121. 8. F. Aug. XR. SCh. EPI. JTS. MS. H^X.

122. 5½. F. Jul. ST. NOD. MS. MD. $2P^X$. Psoriasis.

123. 5. F. Aug. MR. MP. MS. H^X.

124. 11¾. F. Oct. MR. JTS. ERY. Peri. MS. H^X. D.

125. 6. F. Feb. JTS. MS. $2P^X$.

126. 12. F. Mar. JTS. XR. MS. NOD. $2P^X$. H^X.

127. 13. F. Mar. XR. JTS. ST. MS. Peri. H^X.

128. 10. F. Apr. MR. JTS. EPIS. MP. MS.

129. 4. F. Apr. FR. ERY. JTS. MS. MP. ST. Peri. $2P^X$. H^X.

130. 11. M. May. MR. JTS. MS. MP. ST.

131. 10½. F. Aug. MR. GP. JTS. MS. Peri. $2P^X$. H^X.

132. 8. F. Sep. FR. JTS. MS.

133. 9. F. Mar. MR. JTS. MS. MP. AS. $2P^X$.

134. 6½. F. Apr. MR. JTS. MS. H^X. $2P^X$. D.

135. 4. M. Jan. ST. JTS. MP. MS. H^X. $2P^X$.

Group (3) continued.

136. 9. F. Jun. MR. GP. MS. H^X.

137. 10½. F. Apr. MR. GP. ERY. EPI. ST. JTS. MS. MP. MD. AS. H^X. $2P^X$.

138. 8. F. Jun. FR. JTS. NOD. MS. MP. AS. AD. $2P^X$.

139. 10. F. XR. ST. JTS. MS. AS. MP. H^X. $2P^X$. Peri.

140. 6½. M. Nov. FR. JTS. MS. $2P^X$.

141. 10. M. Oct. MR. ST. MS. MP. AS. Congenital heart.

142. 7. F. Jan. MR. JTS. EPI. MS. H^X.

143. 10. F. Feb. JTS. MS. MP. MD. H^X. $2P^X$.

144. 7. F. Aug. MS. MP. MD. H^X.

145. 7. F. Sep. JTS. ERY. EPI. MS. MD. H^X. $2P^X$.

146. 6. M. Aug. JTS. MS. Hx. $2P^X$.

147. 9. F. Oct. BR. MS. AS. H^X. $2P^X$. D.

148. 10. F. Dec. JTS. MS. $2P^X$.

149. 9. M. Apr. MR. NOD. MS. MD. MP. H^X. $2P^X$. D.

150. 7. M. Jun. ST. JTS. MS. MP. $2P^X$.

151. 11. M. Nov. JTS. ST. MS. $2P^X$.

152. 10. F. Jan. GP. H^X. $2P^X$.

153. 7. M. Mar. FR. MS. MP. D.

154. 9. F. May. JTS. MS. MD.

155. 7. F. May. JTS. ST. MS. Peri. H^X. $2P^X$.

156. 4¼. F. Nov. FM.R. ERY. JTS. MS.

157. 9. M. Sep. ST. GP. MS.

158. 5. M. Oct. MR. NOD. MP. $2P^X$.

159. 6. M. Dec. XR. JTS. MS. $2P^X$.

160. 4¼. M. July. JTS. MS. $2P^X$.

161. 10½. F. Feb. JTS. ERY. NOD. MS. AS. H^X. $2P^X$.

162. 10½. F. Feb. ST. MS. MD. MP. AS. AD. H^X. $2P^X$.

Group (3) continued.

163. 6. F. Nov. MR. JTS. MS. MP. H^X. $2P^X$.

164. 6½. F. Jun. MR. ST. MP. $2P^X$.

165. 5½. M. Oct. JTS. ERY. MD. PS. MS. H^X. $2P^X$.

166. 7. F. Jan. JTS. MS.

167. 11. F. Nov. GP. ST. JTS.

168. 9. F. Jan. MR. JTS. MS. H^X.

169. 10½. M. Jul. ST. JTS. GP. MS. $2P^X$.

170. 11. M. Jul. GP. JTS. MS. $2P^X$.

171. 4. F. Nov. MR. JTS. MS. MP. H^X. $2P^X$. D.

172. 10¼. F. Dec. XR. JTS. MS.

173. 3. M. Jan. JTS.

174. 8¼. F. Feb. XR. JTS. ST. H^X. $2P^X$.

175. 9¾. F. Nov. JTS. MP. MS. $2P^X$.

176. 8½. F. Jan. FR. JTS. ST. MS. MP. $2P^X$.

177. 7. F. Apr. FR. JTS.

178. 6. F. Jan. MR. JTS. MS. $2P^X$.

179. 7. F. Jul. ST. $2P^X$.

180. 10¾. F. Nov. JTS. EPI. MS. MD. MP.

181. 11½. M. Mar. JTS. EPI.

182. 4½. F. Jul. MS. $2P^X$.

183. 8. F. Aug. JTS.

184. 10. F. Dec. MR. JTS. MS. $2P^X$.

185. 9. F. Dec. MR. JTS. MS.

186. 6. M. Apr. JTS.

187. 10. F. Apr. JTS. NOD.

188. 5. F. Sep. MR. JTS.

189. 6¾. M. Sep. MR. ST. JTS. MS.

190. 2. F. Oct. MR. JTS.

Group (3) continued.

191. 10. F. Oct. JTS. MS. MP.

192. 10. M. Dec. BR. JTS. MS.

193. 9½. M. Mar. FR. JTS. ST.

194. 7. F. Jul. FR. ST. JTS. MS. MD. $2P^X$.

195. 11. F. Oct. XR. GP. NOD. MP. MS.

196. 8. M. Dec. JTS.

197. 11. F. Jan. ST. GP. MP. $2P^X$.

198. 7¾. M. Mar. SR. JTS. EPI. ST. MS. $2P^X$.

199. 11. F. Nov. FR. JTS. EPI.

200. 8. F. May. BR. JTS. MS. $2P^X$. H^X.

201. 6½. F. Oct. JTS.

202. 11¼. F. Oct. JTS.

203. 10. F. Dec. JTS. NOD. MS.

204. 9. F. Jul. JTS. $2P^X$.