CHOREA IN CHILDHOOD,
an etiological investigation with an analysis of 300 cases.

Mark S. Fraser, M.B., Ch.B., 1906.
In spite of the vast accumulation of literature and statistics on the subject of Chorea within recent years, so little is yet definitely known as to the real cause of the disease, and as to its relationship to other affections of childhood that I think no excuse is necessary for bringing forward a further series of statistics and analysis of cases.

It is my object in this thesis to discuss clinical evidence with regard to the etiology of Sydenham's Chorea. All the cases included in the statistics are examples of true Sydenham's Chorea and have been in-patients ofPaddington Green Children's Hospital, London during the last twenty years.

The argument is divided into three sections:-

Section I.
Discussion on the influence of rheumatism in the production of Chorea.

Section II.
Discussion on the influence of a neuropathic tendency in the patient.

Section III.
Discussion on the pan-rheumatic origin of chorea.

In each section statistics are brought forward in proof of the various views advanced and finally a number of selected cases are set forth to exemplify different clinical points in the origin of Chorea.
SECTION I.
The influence of rheumatism in the production of Chorea.

The connection between rheumatism and Chorea was first advanced by G. Sée so long ago as 1850 (Taylor - Nervous Diseases in Childhood and Early Life, p.242). Since then the association has been traced in various ways which may be tabulated as follows:-

(1) The occurrence of rheumatism before an attack of Chorea.
(2) The occurrence of rheumatism during an attack of Chorea.
(3) The large percentage of organic cardiac affections in Chorea. (endocarditis; myocarditis; pericarditis)
(4) The occurrence of rheumatism after an attack of Chorea.
(5) Family history of rheumatism and Chorea in patients suffering from Chorea.
(6) The correspondence between the age and seasonal incidence of rheumatism and Chorea respectively.
(7) The finding of micrococci in the brain.
(8) Experimental production of Chorea.
(9) Latent Chorea.
In the thesis presented I have endeavoured to put forward statistics confirming the first six of these sub-sections. The sub-sections 7, 8, and 9 have not been investigated and no further reference will be made to them. The subject of Latent Chorea has been fully dealt with by Reginald Miller in the Lancet, December 18th, 1909.

(1) Occurrence of rheumatism previous to the attack of Chorea.

Out of 300 cases investigated definite evidence of rheumatism before an attack of Chorea was found in 150 cases = 50%. By this is meant that 50% of all cases had previously suffered from rheumatic fever, articular rheumatism (acute or subacute), or well marked rheumatic pains.

Cases with a history of sore throats, slight "growing pains", and previous organic heart lesions alone have not been included.

As a very large number of the patients admitted with Chorea had either been previously in the wards suffering from rheumatism or had been attending the out-patient department where there is an elaborate system of note-taking, it has been possible to a very large extent to confirm the history of a previous attack of rheumatism.

(2) Occurrence of rheumatism during an attack of Chorea

In the 300 cases treated 24 showed during the attack of Chorea, while under observation in hospital,
definite evidence of rheumatism, i.e. 8% of all cases. Of these 24 cases, 9 (i.e. 3% of all cases) had no history of a previous attack of rheumatism.

Therefore 53% of all cases of Chorea investigated showed either previous to or concurrent with the attack of Chorea definite evidence of rheumatism. As has been already stated heart conditions alone were not taken as evidence of rheumatism.

(3) **Cardiac affections after investigation produced the following statistics:**

<table>
<thead>
<tr>
<th>Cardiac Affections during attack of Chorea.</th>
<th>189 cases = 63%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart enlarged, no murmur</td>
<td>12 cases = 4%</td>
</tr>
<tr>
<td>Heart enlarged with mitral systolic murmur</td>
<td>27 &quot; = 9%</td>
</tr>
<tr>
<td>Mitral systolic murmur alone</td>
<td>115 &quot; = 39.3%</td>
</tr>
<tr>
<td>Double mitral murmurs</td>
<td>24 &quot; = 8%</td>
</tr>
<tr>
<td>Other cardiac affections</td>
<td>11 &quot; = 3.7%</td>
</tr>
</tbody>
</table>

N.B. Six cases showed pericarditis with or without other heart affections and are included in the above table, i.e. 2% of all cases had pericarditis.

Of heart affections in Chorea I consider that a double mitral murmur may be fairly taken as evidence of rheumatic endocarditis. This granted, I find that 9 cases or 3% having this condition had no other evidence of rheumatism previous to or concurrent with the attack of Chorea. Already 53% of all cases of Chorea have been shown to have definite evidence of
When these 9 cases are added the percentage is raised to 56%.

Still, in his "Common Disorders and Diseases of Childhood", p.457, says

"It may be admitted that a systolic apical bruit alone may not be evidence of endocarditis and therefore objection might be taken to including the last groups of cases" (referring to systolic apical bruits only) "as evidence of rheumatism; ....... there was however no reasonable doubt that in a large proportion of cases where there was only a systolic bruit this was due to endocarditis and probably the error would be smaller if we included them in our estimate of rheumatism than if we omitted them."

This opinion applies in a marked degree to the series of cases under consideration as many of the cases with a mitral murmur only had undoubtedly endocarditis. If cardiac mitral murmurs where the heart was not enlarged be taken as evidence of rheumatism then there would be 63 more cases without other evidence of rheumatism to add bringing up the percentage to 74%. As seen from Table 1. mitral systolic murmurs with enlargement of the heart have not been included.

It is very interesting here to note that in the series the percentage of cases with evidence of rheumatism was found to be much larger in the last 100 cases than in the first. This no doubt is due to the fact that the relationship of rheumatism to Chorea
has within recent years been much more generally recognized and consequently in making notes of the cases a more thorough investigation is carried out.

(4) **The occurrence of rheumatism after an attack of Chorea.**

In looking over the hospital records it has been found that a large number of choreic patients subsequently developed rheumatism, but it has not been possible to make any accurate statistics on the subject.

F. E. Batten in the "Lancet", November 5th, 1898 who followed up the history of 115 cases of Chorea treated at Great Ormond Street Children's Hospital, found that at least 20% of those who at the time showed no signs of rheumatism developed it within 6 years.

(5) **Family history of rheumatism and Chorea in patients suffering from Chorea.**

<table>
<thead>
<tr>
<th>Family history of rheumatism and Chorea.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite family history of rheumatism</td>
</tr>
<tr>
<td>&quot; &quot; &quot; &quot; Chorea</td>
</tr>
<tr>
<td>&quot; &quot; &quot; &quot; both rheumatism &amp; Chorea</td>
</tr>
</tbody>
</table>

N.B. By definite family history is meant evidence of rheumatism or Chorea in parents or in brothers or sisters.

In many of the cases investigated no mention was made of the family history. Therefore the percentages
given are probably much smaller than they ought to be.

A strong family history of rheumatism is so often obtained in Chorea cases that it must surely at least have some bearing on the relationship of rheumatism and Chorea. In the analysis 216 patients or 72% of all cases had either a personal or strong family history of rheumatism. If cardiac systolic mitral murmurs alone, be taken as evidence of rheumatism then out of the 300 cases of Chorea 270 or 90% had either personal or strong family history of rheumatism.

(6) Correspondence between the age and seasonal incidence of Chorea and rheumatism respectively.

Table 3.

Age incidence of Chorea.

<table>
<thead>
<tr>
<th>Between 2 and 3 years</th>
<th>Females</th>
<th>Males</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot; 3 &quot; &quot; 4 &quot;</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>&quot; 4 &quot; &quot; 5 &quot;</td>
<td>7</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>&quot; 5 &quot; &quot; 6 &quot;</td>
<td>3</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>&quot; 6 &quot; &quot; 7 &quot;</td>
<td>14</td>
<td>10</td>
<td>24</td>
</tr>
<tr>
<td>&quot; 7 &quot; &quot; 8 &quot;</td>
<td>26</td>
<td>12</td>
<td>38</td>
</tr>
<tr>
<td>&quot; 8 &quot; &quot; 9 &quot;</td>
<td>27</td>
<td>20</td>
<td>47</td>
</tr>
<tr>
<td>&quot; 9 &quot; &quot; 10 &quot;</td>
<td>42</td>
<td>18</td>
<td>60</td>
</tr>
<tr>
<td>&quot; 10 &quot; &quot; 11 &quot;</td>
<td>37</td>
<td>5</td>
<td>42</td>
</tr>
<tr>
<td>&quot; 11 &quot; &quot; 12 &quot;</td>
<td>38</td>
<td>11</td>
<td>49</td>
</tr>
<tr>
<td>&quot; 12 &quot; &quot; 13 &quot;</td>
<td>13</td>
<td>0</td>
<td>13</td>
</tr>
<tr>
<td>&quot; 13 &quot; &quot; 14 &quot;</td>
<td>10</td>
<td>0</td>
<td>10</td>
</tr>
</tbody>
</table>
N.B. The ages given in the above table are the ages at which the patients were admitted to hospital, but as most of the cases were admitted shortly after the disease commenced, these figures give a very accurate idea of actual age incidence.

Leonard G. Guthrie (Proceedings of the Royal Soc. of Med., May 1908) gives the following table showing the age incidence of acute articular rheumatism in childhood, the statistics being compiled from cases treated at Paddington Green Children's Hospital.

<table>
<thead>
<tr>
<th>Age</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between 2 and 3 years</td>
<td></td>
</tr>
<tr>
<td>&quot; 3 &quot; 4 &quot;</td>
<td>1</td>
</tr>
<tr>
<td>&quot; 4 &quot; 5 &quot;</td>
<td>2</td>
</tr>
<tr>
<td>&quot; 5 &quot; 6 &quot;</td>
<td>2</td>
</tr>
<tr>
<td>&quot; 6 &quot; 7 &quot;</td>
<td>7</td>
</tr>
<tr>
<td>&quot; 7 &quot; 8 &quot;</td>
<td>7</td>
</tr>
<tr>
<td>&quot; 8 &quot; 9 &quot;</td>
<td>13</td>
</tr>
<tr>
<td>&quot; 9 &quot; 10 &quot;</td>
<td>10</td>
</tr>
<tr>
<td>&quot; 10 &quot; 11 &quot;</td>
<td>11</td>
</tr>
<tr>
<td>&quot; 11 &quot; 12 &quot;</td>
<td>15</td>
</tr>
<tr>
<td>&quot; 12 &quot; 13 &quot;</td>
<td>19</td>
</tr>
</tbody>
</table>

A comparison of the above table with Table 3 shows that both Chorea and rheumatism are rare under 5 years of age. It will be seen that there is a close resemblance between the two tables, there being, in Chorea, a marked increase in the number of cases between the ages of 6 and 12, and in acute rheumatism between the ages of 7 and 12.
CHART I

Age Incidence in Chorea and Rheumatism.

Black = Chorea (300 cases)
Red = Rheumatism (96 cases, Guthrie)
N.B. At Paddington Green Children's Hospital, where both series of cases were collected boys are admitted up to 12 years and girls up to 14 years of age.

From Table 3 it will also be seen that in Chorea the highest numbers of cases were admitted in females between the ages of 9 and 10 and in males between the ages of 8 and 9.

Table 4.
Seasonal incidence of Chorea.

<table>
<thead>
<tr>
<th>Month</th>
<th>Cases admitted</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>38</td>
</tr>
<tr>
<td>February</td>
<td>20</td>
</tr>
<tr>
<td>March</td>
<td>22</td>
</tr>
<tr>
<td>April</td>
<td>16</td>
</tr>
<tr>
<td>May</td>
<td>27</td>
</tr>
<tr>
<td>June</td>
<td>17</td>
</tr>
<tr>
<td>July</td>
<td>24</td>
</tr>
<tr>
<td>August</td>
<td>16</td>
</tr>
<tr>
<td>September</td>
<td>27</td>
</tr>
<tr>
<td>October</td>
<td>25</td>
</tr>
<tr>
<td>November</td>
<td>28</td>
</tr>
<tr>
<td>December</td>
<td>40</td>
</tr>
</tbody>
</table>

From this table it will be seen that by far the largest number of cases was admitted in the months of November, December, and January.

<table>
<thead>
<tr>
<th>Month</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nov.</td>
<td>106</td>
</tr>
<tr>
<td>Dec.</td>
<td>58</td>
</tr>
<tr>
<td>Jan.</td>
<td>68</td>
</tr>
<tr>
<td>Feb.</td>
<td>68</td>
</tr>
<tr>
<td>May</td>
<td>68</td>
</tr>
<tr>
<td>Aug.</td>
<td>68</td>
</tr>
<tr>
<td>Mar.</td>
<td>68</td>
</tr>
<tr>
<td>June</td>
<td>68</td>
</tr>
<tr>
<td>Sep.</td>
<td>68</td>
</tr>
<tr>
<td>Jul.</td>
<td>68</td>
</tr>
<tr>
<td>Oct.</td>
<td>68</td>
</tr>
</tbody>
</table>
CHART II.

Seasonal Incidence in Chorea.
(300 cases)
With regard to the seasonal incidence of rheumatism W. S. Church in Albutt's System of Medicine, Ed.2. Vol.II. quotes Dr Gabbett's figures from 2,000 cases of rheumatism treated at the London Hospital which shows that the largest number of cases was admitted during the months of October, November, and December, the maximum being in November.

In considering what has already been said regarding the large percentage of cases of Chorea following an attack of rheumatism and from the fact that a great many of these cases commenced immediately after the attack of rheumatism one would expect the highest seasonal incidence of Chorea closely to follow that of rheumatism. From a comparison of Table 4 with Gabbett's results it will be seen that this is in fact the case, the maximum seasonal incidence of Chorea being one month later than that of rheumatism.

For convenience sake the selected cases exemplifying the various clinical points in the origin of Chorea have been placed together at the end of the thesis.

The cases illustrating points in Section I may for reference be enumerated here.

**CASE I.**

An illustration of a case of chorea with associated endocarditis and Graves' Disease.

**CASE II.**

An illustration of an attack of rheumatism followed immediately by Chorea.
CASE III.

An illustration of an attack of rheumatism followed after an interval by Chorea.

CASE IV.

An illustration of rheumatism and Chorea occurring at the same time.

CASE V.

An illustration of an attack of Chorea followed by rheumatism.

CASE VI.

An illustration of a case of Chorea with an associated pericarditis.

CASE VII.

An illustration of case of Chorea with marked rheumatic family history.
SECTION II.

Discussion on the influence of a neuropathic tendency on the patient.

A neuropathic tendency undoubtedly plays an important rôle in the production of Chorea in rheumatic patients. The question as to whether it is ever the only factor in the etiology of Chorea will be considered in Section III.

That a neuropathic tendency is certainly a very important factor as an exciting cause of an attack of Chorea is shown by

(1) Sex incidence.
(2) Neuropathic family history.
(3) The occurrence of fright and other emotional disturbances preceding the onset of Chorea.

Evidence and statistics will be brought forward bearing upon sub-sections (1) and (3).

(1) Sex incidence.

In the 300 cases the proportion of males and females was as follows:-

Females - 219 cases = 73%
Males - 81 " = 27%

That is a proportion of 2.7 females to 1 male.

It is important to again note that boys were admitted up to the age of 12 years and girls up to the age of 14 years.

By reference to Chart III (p. 15) the age incidence
CHART III.

Sex and Age Incidence in Chorea.
(300 cases.)

Black = males.
Red = females

<table>
<thead>
<tr>
<th>Age Group</th>
<th>2-3</th>
<th>3-4</th>
<th>4-5</th>
<th>5-6</th>
<th>6-7</th>
<th>7-8</th>
<th>8-9</th>
<th>9-10</th>
<th>10-11</th>
<th>11-12</th>
<th>12-13</th>
<th>13-14</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
of both males and females can be seen at a glance. The highest numbers of cases were admitted in females between the ages of 9 and 10, and in males between the ages of 8 and 9.

I shall here quote from Still (Common Disorders and Diseases of Childhood, 1909, p.453)

"The sex incidence of Chorea is interesting. As is well known, girls are much more liable to this affection than boys: My own statistics showed in 150 consecutive cases 108 girls and 42 boys, a proportion of about 2.5 to 1. This difference of incidence does not correspond so closely to the sex incidence of other manifestations of rheumatism as might be expected; in 150 as far as possible consecutive cases of rheumatic arthritis or endocarditis, with no history of previous or concurrent chorea, there were 82 girls and 68 boys. If Chorea were dependent upon no other factor beside the invasion of the brain by a special toxin or a specific microorganism there does not seem to be any particular reason why girls should be so much more often affected by Chorea than boys. It seems reasonable to suppose that there may be a physical factor also in determining its occurrence, and that the excitability of the cortex induced by the micro-organism or toxin of rheumatism is more likely to produce Chorea when the cortex acted upon is that of an unstable excitable child, hence the special liability of the girl."
As is well known after puberty the occurrence of Chorea is very much more frequent in female than in male patients and the fact that in adult women during pregnancy, a time when they are in a more or less nervous and emotional state, attacks of Chorea are common also goes to prove that the neuropathic tendency as shown by sex has a distinct bearing on the etiology of Chorea.

It is interesting here to note that in one of the cases investigated both the mother and grandmother of the child had suffered from Chorea gravidarum.

(2) Neurpathic family history.

Although a careful investigation was made into this subject, in the 300 cases taken there was not sufficient evidence to make it possible to tabulate any accurate statistics regarding a family history of insanity, epilepsy or other nervous disorders; in a large number of cases however there was a history of nervous diseases occurring in other members of the family.

(3) The occurrence of fright and other emotional disturbances preceding the onset of Chorea.

That fright and other emotional disturbances play a part in the production of attacks of Chorea in certain cases in the investigation made is evident. Probably the part played is only that of an exciting factor.
Guthrie (Functional Nervous Disorders in Childhood, 1907. p.214) says:-

"It has often been stated as an argument against the rheumatic origin of Chorea that rheumatism will not account for a large number of cases in which the disease follows fright or emotional disturbance, over-pressure at school and traumatism, nor for cases of so-called reflex Chorea dependent on refraction errors, adenoids, worms, phimosis and other kinds of distal irritation.

"The difficulty may be overcome by considering what is meant by fright and its results.

"Fright is only shock under another name. The results of physical or mental shock are the same. They are extreme nervous prostration, lowered vitality, enfeebled circulation, disordered metabolism, and probably defective elimination. They may be due to physical injury or to emotion. The ill effects of chill or exposure to wet and cold are identical with those of shock.

"Now the symptoms of Chorea at once suggest a history of fright, and such a history is often provided because it is suspected, whereas acute rheumatism suggests exposure to chill or damp. Yet were the connection established, it is probable that a history of fright or emotional disturbance in cases of acute rheumatism, and of chill in cases of Chorea would be equally forthcoming in children."
Table V.
"Fright" followed by Chorea.

<table>
<thead>
<tr>
<th>Description</th>
<th>Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite history of fright preceding attack of chorea</td>
<td>36</td>
<td>12%</td>
</tr>
<tr>
<td>Definite history of fright preceding attack of chorea without any evidence of rheumatism in personal or family history</td>
<td>16</td>
<td>5.3%</td>
</tr>
<tr>
<td>Definite history of fright preceding attack of chorea with personal evidence of rheumatism</td>
<td>9</td>
<td>3%</td>
</tr>
<tr>
<td>Definite history of fright preceding attack of chorea with family history of rheumatism</td>
<td>11</td>
<td>3.6%</td>
</tr>
</tbody>
</table>

From this table it is seen that only 12% of all cases investigated had a definite history of fright. Of these only 5.3% had a history of fright without having any evidence of rheumatism either personal or in the family.

Mention may here be made that under the term "fright" was included:-

- Fright accompanied by physical violence.
- Mental shock alone without physical violence.
- Emotional disturbances.

For example, a girl, age 11, with no personal or family history of rheumatism, was admitted with the following history given by the mother.

"On December 3rd a woman on the floor below made a great noise at night after they had gone to bed. The child got up frightened and was found out
Choreic movements were noticed on December 5th. Another girl, age 7½, having both family and personal history of rheumatism was frightened by some boys telling her that there was a policeman after her. She went home trembling all over and choreic movements followed at once.

A boy, age 11, having no history of rheumatism was severely caned at school and an attack of chorea followed next day.

A girl, age 9, having a family history of rheumatism developed a second attack of chorea immediately on the death of her sister.

A boy, age 6, with family history of rheumatism was knocked down by a bicycle and an attack of chorea followed soon after.

A girl, age 11, without history of rheumatism who had had a previous attack of chorea, but was quite cured developed a second attack shortly after having received a severe burn.

A boy, age 9, developed rheumatic pains and chorea three days after playing truant from school and being wet through by the rain. It was doubtful whether the attack was precipitated by the emotional disturbance caused through fear of punishment or whether it was the result of exposure and chill.

A boy, age 6, with a strong family history of
rheumatism was almost run over by a carriage and two days afterwards developed an attack of acute rheumatism followed in a week's time by an attack of chorea.

From these examples it will be notice that attacks of both rheumatism and chorea have been attributed to fright.

The cases illustrating points in Section II are for reference enumerated here.

CASE VIII.

An illustration of an attack of chorea following on emotional disturbance.

CASE IX.

An illustration of an attack of acute rheumatism and chorea following a fright.

CASE X.

An illustration of an attack of chorea following a fright.
SECTION III.
Discussion on the pan-rheumatic origin of chorea.

This may be taken up in two sub-sections:—

(1) Is chorea ever non-rheumatic?
(2) Is chorea ever a pure neurosis?

Before entering upon a discussion on the pan-rheumatic origin of chorea it would be well to give a definition of what is meant by Sydenham's Chorea.

Definition:—

Chorea is a disease characterised by involuntary spontaneous movements, weakness and want of precision in voluntary movements, emotional instability, with, at times, more serious psychical disturbance: it has a marked tendency to recurrence, a great liability to association with acute endocarditis, and a very slight case mortality.

Statistics bearing on this definition may here be inserted.

Relapsing cases of chorea.

<table>
<thead>
<tr>
<th>Attack</th>
<th>Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>214</td>
<td>71.3%</td>
</tr>
<tr>
<td>2nd</td>
<td>68</td>
<td>22.7%</td>
</tr>
<tr>
<td>3rd</td>
<td>17</td>
<td>5.7%</td>
</tr>
<tr>
<td>4th</td>
<td>1</td>
<td>0.3%</td>
</tr>
</tbody>
</table>
### Table VII.

**Showing age incidence of cases admitted for a 1st attack.**

<table>
<thead>
<tr>
<th>Age Interval</th>
<th>Number of Cases Admitted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between 2 and 3 yrs</td>
<td>1 case was admitted</td>
</tr>
<tr>
<td>3-4</td>
<td>2 cases were</td>
</tr>
<tr>
<td>4-5</td>
<td></td>
</tr>
<tr>
<td>5-6</td>
<td></td>
</tr>
<tr>
<td>6-7</td>
<td>20</td>
</tr>
<tr>
<td>7-8</td>
<td>32</td>
</tr>
<tr>
<td>8-9</td>
<td>31</td>
</tr>
<tr>
<td>9-10</td>
<td>41</td>
</tr>
<tr>
<td>10-11</td>
<td>29</td>
</tr>
<tr>
<td>11-12</td>
<td>33</td>
</tr>
<tr>
<td>12-13</td>
<td>7</td>
</tr>
<tr>
<td>13-14</td>
<td>5</td>
</tr>
</tbody>
</table>

### Table VIII.

**Showing age incidence of cases admitted for a 2nd attack.**

<table>
<thead>
<tr>
<th>Age Interval</th>
<th>Number of Cases Admitted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between 4 and 5 yrs</td>
<td>1 case was admitted</td>
</tr>
<tr>
<td>5-6</td>
<td>No cases were</td>
</tr>
<tr>
<td>6-7</td>
<td>2</td>
</tr>
<tr>
<td>7-8</td>
<td>5</td>
</tr>
<tr>
<td>8-9</td>
<td>7</td>
</tr>
<tr>
<td>9-10</td>
<td>14</td>
</tr>
<tr>
<td>10-11</td>
<td>15</td>
</tr>
<tr>
<td>11-12</td>
<td>15</td>
</tr>
<tr>
<td>12-13</td>
<td>5</td>
</tr>
<tr>
<td>13-14</td>
<td>4</td>
</tr>
</tbody>
</table>
Table IX.
Age incidence of cases admitted for a 3rd attack.

<table>
<thead>
<tr>
<th>Age Range</th>
<th>Cases</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 to 7</td>
<td>1</td>
<td>1 case was admitted.</td>
</tr>
<tr>
<td>7 to 8</td>
<td>1</td>
<td>1 case was admitted.</td>
</tr>
<tr>
<td>8 to 9</td>
<td>5</td>
<td>1 case was admitted.</td>
</tr>
<tr>
<td>9 to 10</td>
<td>4</td>
<td>1 case was admitted.</td>
</tr>
<tr>
<td>10 to 11</td>
<td>1</td>
<td>1 case was admitted.</td>
</tr>
<tr>
<td>11 to 12</td>
<td>3</td>
<td>1 case was admitted.</td>
</tr>
<tr>
<td>12 to 13</td>
<td>1</td>
<td>1 case was admitted.</td>
</tr>
<tr>
<td>13 to 14</td>
<td>1</td>
<td>1 case was admitted.</td>
</tr>
</tbody>
</table>

For convenience a chart has been made to show the age incidence of cases admitted for a first, second and third attack (Chart IV. p. 25), and from it several interesting observations may be made.

The youngest patient admitted between 2 and 3 years (to be exact 2 years and 6 months).

Of the patients admitted for a first attack there was a slight increase in the numbers up to the age of 6 years. From 6 to 10 there was a rapid increase, the maximum (41 cases) being attained between the ages of 9 and 10. From 10 to 12 there was a slight decrease and from 12 years and upwards the numbers rapidly diminished.

For a second attack the youngest patient was admitted between the ages of 4 and 5 years and the maximum (15 cases) was attained during the two age
CHART IV.

Relapsing cases of Chorea.

Black = 1st. attack
Red = 2nd attack
Blue = 3rd. attack.
age periods 10 to 11, and 11 to 12.

The youngest patient was admitted for a third attack between the ages of 6 and 7 and the maximum was reached between the ages of 8 and 9.

Table X.

Case mortality in Chorea.

Deaths from chorea alone ... 0 = 0%
" " " with complications 5 = 1.7%

The causes of death in the five fatal cases were:-
1. Acute rheumatism, Broncho-pneumonia, and Chorea.
2. Septicaemia and Chorea.
3. Exophthalmic Goitre, Endocarditis, and Chorea.
5. Pericarditis, Endocarditis, and Chorea.

Osler (Principles and Practice of Medicine, 6th Edition 1905, p.1047) states that he had collected from the literature the records of 73 autopsies in Sydenham's Chorea and of these 62 had endocarditis.

Sub-section (1).

Is chorea ever non-rheumatic?

In trying to give a definite answer to this question the difficulties which arise are very great.

In the first place, the case mortality in chorea being so exceedingly low, post-mortem examinations are very seldom made.
Doubtless many post-mortem examinations are made on adults who in childhood suffered from chorea but the fact that they have had chorea is often forgotten by the patient and his friends and is therefore not known by the pathologist. Consequently when old standing endocarditis in an adult is found, the fact that it may have originated in an attack of chorea during childhood is not recognised.

Another difficulty is that a slight attack of rheumatism previous to the onset of chorea is frequently not recognised by the parents, the child may not be so ill that a doctor is consulted or the medical attendant possibly may not recognise the condition as one of rheumatism.

Another difficulty lies in the fact that frequently children are brought to the hospital by people other than the parents and in consequence a correct statement of the previous health is not obtained.

There is no reasonable doubt that many of the so-called cases of non-rheumatic chorea are not cases of true Sydenham's chorea at all. Choreiform movements have been seen in:-

(a) Cerebral diplegia with lesion in Optic Thalamus.

(b) Tuberculous meningitis.
   In a few very rare cases of this disease choreiform movements appear for one or two days before coma supervenes.
(c) Iodoform poisoning (R. Demme, Ein Fall von Chorea Minor mit Glykosurie. Schweiz. Ztschr f. Heilth., Bern, 1862, 1, 345-350)

These conditions may be distinguished from true Sydenham's chorea by the course of the disease.

The strongest evidence that can be produced from the investigation and statistics of the 300 cases in favour of the non-rheumatic origin of chorea is the fact that a number of the cases analysed showed no evidence of rheumatism either in their personal or family history. Even when all the cases of chorea, where a mitral systolic murmur alone is taken as evidence of rheumatism, are added to the cases showing definite personal or family evidence of rheumatism there still remain 30 cases or 10% of all cases apparently non-rheumatic. As already stated Batten found that 20% of patients suffering from chorea of an apparently non-rheumatic type developed rheumatism within 6 years. Batten only investigated 115 cases and it is probable that, had a larger number of cases been followed up for a longer period, a much higher percentage of patients would have been found to develop rheumatism.

Granting that chorea is a cerebral manifestation of rheumatism there is no reason why it should not be the only manifestation in a certain number of cases. Still aptly remarks:-

"If rheumatism be, as clinical and bacteriological evidence suggests, an infective disease which is not
"limited to joints but may affect the heart, the brain, and possible the lung and the pleura, it would be as reasonable to deny that a tuberculous meningitis was tuberculous because there was no pulmonary tubercle as to deny that the brain affection, chorea, is rheumatic because there is no arthritis." (Com. Disorders and Diseases of Childhood, 1909 p.455.)

The occurrence of chorea after scarlet fever has been put forward as evidence of its non-rheumatic nature. Statistics have been prepared to show the relationship between chorea and the following infectious diseases, namely scarlet fever, diphtheria, measles, and whooping cough.

Table XI.
Relation of certain infectious diseases to chorea.
From analysis of 300 cases.

Previous history of Scarlet fever in 39 cases = 13%
" " Diphtheria " 15 " = 5%
" " Measles "192 " = 64%
" " Whooping Cough "118 " = 37.3%

A further investigation of 200 of the above cases showed that 26 of these had previously suffered from scarlet fever of which 2 developed chorea within one month of the attack, 2 within 6 months, and 1 within 12 months.

Diphtheria was treated in a similar way with the result that out of 12 cases of diphtheria in the 200
only one developed chorea within a year.

Measles and whooping cough as far as could be seen had no connection whatever with the onset of chorea. Neither scarlet fever nor diphtheria as seen from what has been said above seem to be of any appreciable importance in the etiology of chorea.

Scarlet fever may in a few very rare cases, on account of its destructive action on the tonsils, make the patient more liable to attacks of rheumatism and chorea. The same might be said of Diphtheria to a lesser extent.

The fact that almost all cases of chorea examined post-mortem show endocarditis, apparently of a rheumatic type may be fairly taken as very strong evidence in favour of the rheumatic theory of chorea.

In answer, then, to the question placed at the heading of this section we may say definitely that the majority of cases of chorea are closely associated with rheumatism, that probably chorea is the cerebral manifestation of rheumatism, and that possibly all cases of true chorea are rheumatic in origin.

Sub-section (2)

Is chorea ever a pure neurosis?

Confusion has frequently arisen between chorea and habit-spasms. A careful observation of doubtful cases usually makes the diagnosis quite certain.

It may be stated with a fair degree of certainty
that chorea is seldom, if ever, a pure neurosis.

The strongest argument which I can give against the theory of chorea ever being a pure neurosis is by quoting the following rare and interesting case which I had the opportunity of observing during the last month of her life.

There was no evidence of rheumatism during life and no history of rheumatism either personal or in her family. The mitral murmur which was present was thought to be functional. At the autopsy endocarditis was found similar to that seen in rheumatism.

CASE I. Exophthalmic Goitre; Chorea; Endocarditis.

Ellen W. Age 12 years.

Admitted 5th February 1910.

Died 5th March 1910.

Complaint. St. Vitus Dance and goitre.

History. Since October 1909 patient has been treated at Golden Square Throat Hospital for goitre. Treated with medicine and goitre now much smaller.

A fortnight ago patient began to jerk her right hand and arm and swing them about. Difficulty in speaking also began a fortnight ago. She speaks as "if she had a plum in her mouth". She has been getting worse for the last few days and has been away from school for one week.

Previous Health. Patient has had, measles, pneumonia, pleurisy, whooping cough, diphtheria,
scarlet fever, mumps, chickenpox and tonsillitis.

No rheumatic fever and no other evidence of rheumatism if the tonsillitis can be excluded.

Family History. Father and Mother both alive and well; two other children died of pneumonia following measles; no other children. No evidence of rheumatic family history.

On admission. Temperature 100°, Pulse 112, Respiration 32.

Patient is very tall for her age and markedly emaciated. While being examined she threw her arms and legs about. All the muscles of the body seemed to be involved. She is very excitable and emotional, laughing and crying alternately. Face is flushed and pupils dilated. Speech is jerky and difficult to understand. Slight grimacing with facial muscles. Tongue cannot be protruded for more than a second or two and is jerked out and in.

Thyroid Gland. The thyroid is considerably enlarged and soft. Swelling is equal on each side of the neck.

On auscultation over the gland there is a fairly loud humming bruit. Pulsation marked in thyroid and vessels of neck.

Circulatory System. Pulse rapid, 136 per minute, regular, fairly strong.

Heart. Apex beat one inch external to nipple line in 5th intercostal space.
Visible pulsation in 4th and 5th left intercostal spaces. Distinct thrill on palpation. At apex there is a soft blowing systolic murmur propagated well into axilla. There is also a pulmonary systolic murmur.

**Respiratory System.** Nothing abnormal to note.

**Nervous System.** Reflexes. Knee jerks both markedly exaggerated. Ankle clonus present to a slight degree in both legs.

Planta reflexes show flexion in both legs. Abdominal reflexes active.

**Eyes.** Slight prominence of eyes, but no marked exophthalmos. Eyes have a peculiar staring expression. Von Graefe's sign not present.

**Skin.** The skin is moist and pigmented. (Patient had had no arsenic, as learned from her medical attendant at Golden Square.) a brownish colour with sharply defined white areas here and there over trunk and both legs. This pigmentation is specially marked on front of knees. Pigmentation on body is symmetrical in arrangement especially on back and pelvis.

**Alimentary System.** Tongue slightly furrowed.

Abdomen. Abdominal walls firmly resist palpation. Liver and Spleen not enlarged.

Marked pulsation (aortic) above umbilicus.

**Urine.** Deposits urates, sp.grav.1028, acid, no sugar, pus, blood or acetone.
Chlorides not increased.

Slight trace of albumin and nucleo-albumin.

Progress.

7.2.10. Urine. Deposit urates, sp.grav.1029, acid, no sugar, pus, blood, acetone, albumin or nucleo-albumin.

8.2.10. There is to-day a fine tremor present in hands and slightly in feet also.

Von.Graefe's sign present.

9.2.10. Became very violent and then quite delirious.

10.2.10. Incontinence of both urine and faeces occurred once to-day.


16.2.10. Much quieter to-day, heart same, thyroid same.

19.2.10. Apex beat is now internal to nipple line. Choreic movements very slight. Right hand and arm much weaker than left.

23.2.10. Choreic movements markedly increased. Apex beat ½ in. external to nipple line. Appetite not so good.

28.2.10. Wasting is very marked and is rapidly
progressing. Incontinence of both urine and faeces to-day.

2.3.10. It is very difficult to get patient to take any food.

3.3.10. Very restless. Great difficulty in swallowing.

Pulse very rapid, 200 per minute.

4.3.10. Very restless, and excited in morning. Unconscious in evening.

5.3.10. Death at 1.30 a.m.

I have made no mention of treatment as that does not come into the scope of the thesis.

Post-mortem examination.

External appearances. Emaciated to last degree. Brownish pigmentation general, with white patches on trunk and limbs. Decomposition beginning on abdomen.

Thorax. Adhesions all over right lower lobe. Both lungs congested, no consolidation and no tubercle.

Heart. Left-ventricle full of blood, wall hypertrophied not dilated, pulmonary, tricuspid and aortic valves nothing abnormal. Vegetations on Mitral valve similar to those found in cases of Acute Rheumatism.

Thyroid gland enlarged, weight 2 ounces 1½ drachms, congested and firm. The right lobe was about twice the size of the left; the isthmus was enlarged. The trachea showed distinct signs of lateral pressure from lobes of thyroid.
Thymus enlarged, weight 5 drachms 50 grains.

Abdomen. Mesenteric glands enlarged and some of them calcified.

Intestines, stomach, kidneys, liver and spleen showed nothing abnormal.

Adrenals enlarged, right weighed 1½ drachms, left weighed 2 drachms.

No signs of puberty.

Brain. Vessels injected, Sulci bridged by slightly opaque arachnoid, over vertex sub-arachnoid fluid glairy, no adhesions in Sylvian fissures, no meningitis at base.

The cases illustrating points in Section III are for reference enumerated here.

CASE XI.

An illustration of an attack of chorea following an attack of scarlet fever.

CASE XII.

An illustration of an attack of chorea following
CONCLUSIONS.

From the investigation carried out and statistics produced it can with certainty be said that a very large proportion of the cases of chorea were closely associated with rheumatism.

As has already been said 56% of all cases showed either previous to the attack of chorea or concurrent with it definite evidence of rheumatism. This percentage is probably much lower than it should be because previous histories of sore throats, many of which were probably rheumatic, and slight growing pains were not taken as evidence of rheumatism.

It has been shown that if cases having no other evidence of rheumatism than a mitral systolic murmur were added the percentage would be increased to 74% of all cases. Many of the cases with that heart condition alone were undoubtedly suffering from endocarditis and, taking this into account along with the fact that in almost all post-mortem examinations made on choreic patients endocarditis is found, it is probable that the addition of these cases would make the statistics more accurate than if they were not included.

CASE I may be taken as very strong evidence in support of this view.

44% of cases were found to have a strong family history of rheumatism. From the fact that information
regarding the family history was lacking in many cases this percentage is probably much smaller than it ought to be.

Taking all cases showing evidence of rheumatism, either personal or in the family history, a percentage of 72 was obtained, and if the cases with mitral systolic murmurs are added the percentage is raised to 90.

Although no statistics were prepared to show the proportion of cases who subsequently developed rheumatism from an examination of the hospital records it was seen that a considerable number did so.

The age incidences of rheumatism and chorea as seen in Chart II are closely associated. A very large number of cases of rheumatism were found to develop chorea very soon after the attack. Hence we would expect that the maximum seasonal incidence of chorea would be slightly later than that of rheumatism and this has been shown to be the case, the maximum for rheumatism being in November, that for chorea in December.

The chief and most frequent infectious diseases of childhood, namely, scarlet fever, diphtheria, whooping cough and measles were not found to be important factors in the etiology of chorea although it seems possible that scarlet fever and diphtheria,
on account of their injurious influence on the tonsils, may open up a channel through which the rheumatic injection may enter the system and hence indirectly predispose to chorea.

The neuropathic tendency as shown chiefly by the sex incidence (2.7 girls to 1 boy) is undoubtedly a predisposing factor to chorea.

The importance formerly given to fright and other emotional disturbances as direct causes of chorea seems to have been greatly overestimated. Only 12% of all cases had a definite history of fright and of these, only 5.3% (less than half) had no evidence of rheumatism. Fright and emotional disturbances, by lowering the vitality of the nervous system, seem rather to act merely as exciting causes of attacks of both rheumatism and chorea.

Notice must be taken of the fact that in a great many instances histories of fright were obtained in patients who were suffering from a second or third attack of chorea.

That chorea is a disease which is very liable to recur is amply shown from the tables given on relapsing cases.

In the series no deaths occurred in uncomplicated cases. The case mortality was only 1.7%.

The theory that chorea is sometimes a pure neurosis has probably originated in the confusion of diagnosis between chorea and other conditions which
in some respects resemble it clinically. Case I, in which during life rheumatism was not suspected, but in which endocarditis was found after death, is strong evidence against this theory.

It has frequently been observed in that patients suffering from exophthalmic goitre have in earlier life been the subjects of chorea, and there is apparently some relation between the two diseases. The instance here given in Case I of the two diseases occurring simultaneously is extremely rare.

All the above facts taken together give very strong support to the belief that chorea is a cerebral manifestation of rheumatism.
CASES

illustrating some important points in the etiology of Chorea.
CASE II.

Rheumatic attack followed at once by an attack of chorea.

D.B. male, age 9 years.
Admitted September 5th, 1901.
Discharged September 25th, 1901.
Result, almost cured.

**Complaint.** St. Vitus’s Dance.

**History.** Patient had rheumatic fever about six weeks ago. A fortnight after the onset of the acute rheumatism attack, as he was recovering from the pains in the joints (knees and wrists) his mother noticed that he dropped spoons and cups and that his mouth was twitching. When he sat in a chair he kept moving his legs. He was allowed to sit up about 9 or 10 days after the onset of the first symptoms. He has been under treatment some time; when first seen his speech was unaffected, but a fortnight ago he had considerable difficulty in speaking; this condition has since improved. Since his illness the boy has been very emotional, previously he was not so.

**Previous Health.** The patient has had measles as a baby and whooping cough, but not scarlet fever.

**Family History.** Father has chronic rheumatism; otherwise there is no family history of rheumatism. No neuropathic history.
There are six other children alive and well. Four are dead. Causes - pneumonia, convulsions, diarrhoea and vomiting, and bronchitis.

State on admission. The child is fairly well nourished; is very emotional, laughing and crying readily and indiscriminately. The grasp is weak and is accompanied by exaggerated facial contortions. The heart is not enlarged. There is a rough pre-systolic murmur followed by a high pitched systolic mitral murmur. The lungs are healthy. There is some rigidity of the limbs; knee jerks are active but not exaggerated. The movements chiefly affect the muscles of the face and hands.

Progress. During his stay in hospital the movements nearly disappeared, and also the rigidity of the limbs. The mitral murmurs remained unchanged and when patient was sent to a convalescent home on September 25th he was almost cured.
CASE III.

Rheumatism followed after an interval by chorea.

S.N. Female, age 7½ years.
Admitted December 5th, 1908.
Discharged January 4th, 1909.
Result - Cured.
Complaint - St.Vitus's Dance.

History. Patient was quite well till a month ago when she complained of slight pains in thighs and fingers and also seemed lifeless and began to lose weight. This condition continued for a fortnight when she was brought up to Paddington Green Children's Hospital as an out-patient. It was noticed that there were jerking movement of the right arm and leg and twitchings of the right side of the face.

Since that time the slight pains have entirely disappeared but the movements and twitchings have continued. She has a good appetite but the bowels are very constipated. Her mother says that she is not an emotional child, but that she is very intent on her lessons and is considered to be a very clever child.

Previous health. Full time child, breast fed, always a healthy infant. She has had measles and whooping cough, and a year and a half ago had an attack of rheumatic fever. She is not specially
addicted to headaches or sore throats. For a considerable period she has been very easily fatigued.

**Family history.** Father and mother both alive and well; two other children alive and both quite healthy, none dead. No history of miscarriages and no rheumatic family history.

**State on admission.** Patient looks a healthy well-developed child, but is very nervous and highly strung. Choreiform movements of all the limbs as well as of the trunk are well marked and there are twitchings of the face. All these movements are more manifest on the right side. There is also some difficulty of speaking. The tone of the muscles is good all over the body; the grasp of both hands is strong, but not sustained.

There is well marked lateral nystagmus present; the pupils are equal in size, regular in outline and moderately dilated. All the deep reflexes are exaggerated but there is no clonus. The heart is slightly dilated, the apex beat being the fifth intercostal space one inch external to the nipple line. In the mitral area there is a loud and long blowing systolic murmur propagated outwards into the axilla and carried upwards into the aortic and pulmonary areas. In the pulmonary area the second sound is accentuated and reduplicated. There is no irregularity of the heart's action.
The lungs are both healthy and there is no enlargement of the liver or spleen.

On December 8th the twitchings and movements were less evident. There was no pyrexia. The patient complained of hunger.

On December 12th the movements were still present but to a much less degree. The condition of the heart remained unchanged.

On December 19th the movements had quite disappeared but a mitral systolic murmur was still present and along with it accentuation and reduplication of the pulmonary 2nd sound.

On January 4th, patient was sent home cured.
CASE IV.

Rheumatism and chorea occurring simultaneously.

N.G. Female. Age 4½ years.

Admitted - 22nd September 1909.

Died 9th October, 1909.

Complaint - Acute rheumatism, chorea, and broncho-pneumonia.

History. Three weeks ago patient suddenly was awakened during the night by pains in the legs and arms. Next day there were well marked choreic movements and the speech became affected at the same time. Since then the movements have become less marked.

Previous health. Full time child, always healthy, except for an attack of measles.

Family History. Father is healthy; mother suffers from chronic rheumatism; five other children all more or less rheumatic; one has had rheumatic fever and another has suffered from chorea.

State on admission. Temperature 100.6°, pulse 120, respirations 36 per minute. She is a well nourished child. There are rheumatic nodules over elbows, knees, ankles and heads of metacarpal bones; there is marked swelling of both wrist joints.

Heart. The apex beat is in the 5th intercostal space slightly external to the mammary line, the right border is half an inch to the right of the
sternum, and there is some pulsation in the vessels in the neck. There is a distinct thrill over the praecordia. On auscultation there is a rough mitral presystolic murmur followed by a blowing mitral systolic murmur which is propagated into the axilla.

Nervous system. Patient is very nervous and cries on the slightest pretext. There are facial grimaces, the tongue when protruded shows irregular jerking movements and the speech is affected. There are choreiform movements of the upper and lower extremities but only to a slight degree.

Lungs healthy. Liver and Spleen not enlarged.

Urine. Reaction acid; Sp.gr. 1025, deposit of urates; no blood, albumin, or sugar; slight trace of acetone.

On September 25th some new nodules appeared on one of the tendons on the front of the wrist. The heart's action was still rapid.

On September 29th the choreic movements were practically gone. The heart murmurs were unchanged.

On October 1st the temperature began to rise and patient began to cough. Coarse crepitations were heard over the bases of both lungs but there was no dulness.

On October 2nd there was impairment of resonance at the left apex and coarse crepitations were still marked at both bases.
On October 4th the cough was not so troublesome and looser. On the left side posteriorly there were both coarse and fine crepitations and on the right side posteriorly there were rhonchi and coarse crepitations.

On October 6th the physical signs were more marked in both lungs; on October 8th there was some dulness over left apex in front and in that region vocal fremitus and vocal resonance were both increased while posteriorly in both lungs the accompaniments were less marked.

On October 9th there was a patch of bronchial breathing at left apex in front. The patient looked very ill and the pulse was feeble. On the evening of the same day she died.
CASE V.

Chorea, rheumatism and second attack of chorea.

F.T. Female, age 10 years.

Admitted - July 22nd, 1902.

Discharged - August 13th, 1902.

Result - Almost cured.

Complaint - St. Vitus's Dance.

History. Choreic movements appeared about three weeks ago in the legs and arms and speech was also affected. The movements have become worse during the last fortnight. Patient is said to be a very excitable child and at times very sulky.

Previous health. Slight attack of chorea two years ago. Nine months ago patient was treated in St. Mary's Hospital for five weeks for an attack of rheumatic fever. As a baby she had measles, whooping cough, and chickenpox. Four years ago she had scarlet fever.

Family history. Both mother and father have suffered from rheumatic fever. Four other children, all healthy.

State on admission. Patient seems very nervous and excitable. There are choreic movements in both arms and legs, more marked however on the right side; there are also twitchings of the face.
Heart - not enlarged. At apex there is a double mitral murmur. The pulmonary second sound is accentuated. Lungs both healthy. Liver and spleen not enlarged. The deep reflexes are all present but not exaggerated.

On July 25th in the mitral area the systolic murmur alone was present.

On August 13th patient was discharged almost cured, the movements now being very slight.

CASE VI.

Chorea, acute rheumatism and pericarditis.

A.M. Female. Age 7 years.

Admitted - April 9th, 1909.

Discharged - May 17th 1909.

Result - cured.
Complaint - St. Vitus's Dance and rheumatic fever.

**History.** A month ago the parents noticed that the child seemed out of sorts. She complained of headaches and general aching in the body which flitted from place to place. This went on for a fortnight, the child not being confined to bed. At the end of that time a doctor was called in and ordered the patient to bed explaining that she had rheumatic fever. A week later choreic movements started in the hands and face. Since then she has become very dull and her speech has become more and more affected and indistinct till at the present time she can only say a few almost unrecognisable words. During the last few days the knees and hands have become greatly swollen but not painful. For the last two days she has had attacks of profuse sweating with occasional pains in her left elbow joint.

**Previous health.** Health has always been good except for attacks of measles and chickenpox in infancy.

**Family History.** Father, mother and two other children are all alive and healthy. No history of rheumatism.

**State on admission.** Patient is extremely restless and fidgety and has no control over her movements. She is greatly emaciated and the skin all over the body is very moist. There is a small septic spot
over the left external malleolus and over the buttocks there is a septic rush. The face on the right side is constantly twitching while on the left it remains expressionless and placid. There is no apparent weakness of the left levator palpebrae superior. She is unable to speak but occasionally utters a few inarticulate sounds. She is unable to protrude her tongue although she apparently tries to do so when asked.

There is no nystagmus; pupils are equal, moderately dilated, and react to light.

She seems unable to move her left arm and she frequently attempts to lift it with the right hand. The power of the left leg has been almost completely lost. The right leg is rigid and extended. The knee jerk on the right side is much exaggerated, on the left it is diminished. There is no ankle or knee clonus and no Babinski sign. The left knee is swollen, hot and contains fluid.

Heart - somewhat enlarged. Apex is in 5th interspace \( \frac{4}{6} \) inch external to left nipple line. There is a long and loud mitral systolic murmur which is conducted well into the left axilla. A fine double friction rub is heard all over praecordia.

Lungs - both apparently healthy.

On April 4th there was swelling of and effusion into both wrist joints and both knees. Pericardial
friction was most evident over the base of the heart.

On April 12th the pericardial friction was less marked. Only a very small quantity of urine was being passed.

On April 13th movements were still marked and patient was still unable to speak or protrude her tongue. All the large joints were now swollen. Pericardial friction is now only heard at the base.

On April 14th no pericardial friction was heard and there was no increase in pericardial dulness.

On April 15th the swelling had disappeared from all the joints and the patient was much quieter.

On April 19th the temperature which had been normal for 4 days again rose and pain was complained of in the left shoulder. Pericardial friction was again heard all over praecordia.

On April 22nd coarse crepitations was heard in both lungs. The temperature again appeared to be settling. Pericardial friction as before.

From April 24th till May 17th, the date of discharge pericardial friction was not again heard and the patient made a gradual but complete recovery so far as the choreic movements were concerned, the mitral systolic murmur still being heard.
CASE VII.

Case of chorea with rheumatic family history.
P.T. Female. Age 8½ years.
Admitted - December 12th, 1908.
Discharged - January 6th, 1909.
Result - Cured.
Complaint - St. Vitus's Dance.
History. Patient was quite well till three weeks ago when suddenly she started to have choreic movements in her right arm and leg.

Previous Health. Full-time child, healthy as an infant. She is an adopted child and her early history is not very clear. She has had chickenpox, mumps, measles and whooping cough. She has never had any rheumatic pains nor sore throats. She is always a very bright child and is said to be very clever. She has always been very excitable and emotional, though not nervous. She is very fond of her school work and is at the top of all her classes. Her work is said to come easily to her and not to worry her or cause her any effort.

Family History. Father alive and well. Mother died from heart-disease following an attack of rheumatic fever. There are seven other children all alive and well; no history of miscarriages.

State on Admission. Patient is a healthy looking, well nourished child. There are well marked choreic movements of the right arm and leg and of the right side of the body. There is no cyanosis, nor undue pallor. The tone of the muscles is very good all over the body, and she has a strong grasp with both hands, though rather irregular on the right side.

The deep reflexes are much increased, there is no clonus and no Babinski. The abdominal reflex is only present on the left side.
Pupils moderately dilated, equal and regular, react to both light and accommodation. No nystagmus present.

Heart. It is not enlarged; its action is very irregular, and about one beat in every eight is missed. In the mitral area there is a long rough presystolic murmur, which is best heard at apex and is not propagated any distance.

Lungs healthy, liver and spleen not enlarged.

On December 30th a mitral systolic murmur appeared along with presystolic. During her stay in hospital the heart action improved, becoming stronger and more regular. The choreic movements also improved and when patient was sent home on January 6th the choreic movements had quite gone, but there still was a double mitral murmur present.
CASE VIII.

Chorea following emotional disturbance.

Q.B. Female age 11½ years.

Admitted May 5th, 1909.

Discharged May 28th, 1909.

Result - Cured.

Complaint - St. Vitus's Dance.

History. Quite well till seven weeks ago; patient then became very nervous, was unable to sleep and started to worry very much about her school work. She also, at this time, complained of slight pains in her limbs.

About a week later, she developed choreic movements. During the last fortnight her speech has become affected and she has been frequently letting things drop.

Previous Health. Always a healthy child, no complaint of pains and no previous attacks of chorea. She has had measles, chickenpox and whooping cough.

Family History. Father, Mother and four other children all alive and well, six other children all died in infancy and there were four miscarriages.

State on Admission. Patient is a healthy looking child. She is very emotional and nervous. There are well marked choreic movements of both upper and lower limbs; the tongue shows also irregular movements.
There is no twitching of the facial muscles, no grimacing and no nystagmus present. There is considerable wasting all over the body, the tone of the muscles is very poor and the grasp is irregular and jerky.

Deep reflexes present, but not increased, no clonus and no Babinski.

Heart. Not enlarged, no murmurs.

Lungs healthy, liver and spleen not enlarged. No rheumatic nodules found.

Patient was sent home on May 28th cured.

Note.

The exciting cause in this case seems to have been disappointment. The patient along with the other girls in her class was being taught a song which was to have been sung before the Princess of Wales. For some reason she was not allowed to be present and after that the attack of chorea soon came on.
CASE IX.

Rheumatic fever and chorea following fright.

H.W. Male, age 6 years.

Admitted May 6th, 1901.

Discharged June 29th, 1901.

Result - Cured.

Complaint - St. Vitus’s Dance.

History. About five weeks ago patient was almost run over by a carriage; two days after this patient had an attack of rheumatic fever, with high temperature, and pain and swellings of joints. A week after this choreic movements came on. His speech was affected and he was unable to feed himself.

Previous Health. Good, no history of rheumatism or chorea; patient has had scarlet fever, whooping cough and measles.

Family History. There was a definite family history of rheumatism.

State on Admission. Patient looks a healthy, well-nourished child. Choreic movements were general; speech very indistinct.

Heart. It is slightly enlarged; there is a double mitral murmur; aortic 2nd sound reduplicated.

Pupils. Small, equal, regular and react to light. Deep reflexes present, but not increased.

Lungs healthy, liver and spleen not enlarged.

Patient improved very quickly, and when he was
sent home on June 29th the choreic movement had gone but the double mitral murmur was still present.
CASE X.

Chorea following fright.

W.M. Age 7 years.

Admitted December 21st, 1903.

Discharged February 13th, 1904.

Result - Cured.

Complaint - St.Vitus's Dance.

History. Patient was quite well till nine weeks ago when a dog flew at him and frightened him. He "went white" and "trembled all over". The next day his mother noticed that his left arm was twitching and that he could not stand still. Soon after this the left side of his face began to twitch and he was unable to speak.

Previous Health. Patient has had measles, chickenpox and whooping cough, and for some time he has had a discharge from both ears.

Family history. Father has had rheumatic fever; he has two sisters alive and well.

State on Admission. Patient is a thin but healthy looking child. The left arm and leg are held more or less rigid, but every now and then there are slow spasmodic movements of both. The right arm and leg were only slightly affected.

Heart. There is a late diastolic murmur heard in the mitral area and the pulmonary second sound is reduplicated. The heart is not enlarged.
Lungs healthy; liver and spleen not enlarged.

The pupils are equal and there is no squint or nystagmus.

Mental condition good, although patient is rather dull and sleepy.

Choreic movements are not general.

On December 27th the movements were less.

On January 10th movements still present; all rigidity gone.

On February 13th patient was sent home quite free from choreic movements.
CASE XI.

Rheumatic fever and chorea following scarlet fever, (very rare).

F.S. Female, age 9 years.

Admitted June 8th 1903.
Discharged July 30th 1903.

Result - Relieved.

Complaint - St. Vitus's Dance.

History. Last December 1902, the patient had an attack of scarlet fever and was taken to a fever hospital. She remained in the hospital seventeen weeks as soon after the attack of scarlet fever, she developed rheumatic fever which was soon followed by chorea. During that period she also suffered from acute nephritis and otorrhoea.

After patient left hospital for some time the choreic symptoms were very slight, but before very long they became very marked again. Just a short time before patient was admitted to Paddington Green Children's Hospital her speech had become very much affected.

Previous Health. Good escape from an attack of measles.

Family History. No history of rheumatism or chorea.

State on admission. All the four limbs as well as the body are involved in the choreic movements.
Speech is markedly affected, but there are no twitchings of the face.

Heart. Not enlarged; there is a soft mitral systolic murmur, no other murmurs.

Lungs healthy, liver and spleen not enlarged.

On June 20th the systolic murmur had almost disappeared, the choreic movements are not so violent as when patient was admitted.

On June 25th patient was not so well, and had an attack of diarrhoea with blood in the stools.

On the morning of June 26th the temperature rose to 102°, but fell again in the evening.

On July 30th when the patient was discharged the movements had almost ceased.
CASE XII.  
Chorea following history of sore throats.
W.A. Female, age 10 years.
Admitted March 18th, 1903.
Discharged April 17th, 1903.
Result - Cured.

Complaint - St.Vitus's Dance.

History. Four weeks ago patient's mother first noticed that the child "could not hold things". Soon after jerking movements affected all the limbs; the speech became indistinct.

Previous Health. Patient has had no previous attacks of either rheumatism or chorea. She has had no growing pains but has from time to time suffered from sore throats. No history of fright. She has had measles.

Family history. There is no history of rheumatism in other members of the family; the only other child constantly suffers from sore throats.

State on admission. The lower extremities and the hands show choreiform movements but the upper extremities as a whole are fairly steady.

Heart. Not enlarged. No murmurs.

Lungs are healthy; liver and spleen are not enlarged. The tonsils are both enlarged and the throat is injected. The knee jerks and triceps jerks on the left side are more marked than those on the right.
Up till March 30th the patient made a rapid recovery and the heart condition remained unchanged.

On April 1st sore throat was complained of and the temperature rose to 101°, but fell to normal again on the following morning. The tonsils were enlarged and the throat slightly injected. No rash appeared.

This condition subsided in a day or two and on April 17th, the patient was discharged cured.