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THESIS

ON

SYDENHAM'S CHOREA

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APRIL, 1909.
SYDENHAM'S CHOREA.

Derivation.

The word chorea is derived from the Greek \( \chi\omicron\omicron\omicron\omicron\omicron \omicron \alpha \), which means dancing and Sydenham's name was applied to it because he was the first to describe chorea in such a way as to enable it to be recognised as a distinctive disease.

Synonyms.

The various synonyms are St. Vitus' Dance, Chorea Minor, and Chorea Angiorum.

The use of the term chorea as applied by Sydenham to a particular disease, chiefly affecting children from five to fifteen years of age, is rather unfortunate, as the same appellation had already been given to another and totally different affection which was purely hysterical in character and which is known as the Dancing Mania, or as Osler has called it; Chorea Major.

History of the Dancing Mania.

Von Hecker in his work "Epidemics of the Middle Ages" published by the Sydenham Society gives a very graphic account of the Dancing Mania as it was seen on the Continent in the fourteenth century.

He states that the sufferer, by his wild bacchantic leaping, screaming and foaming at the mouth, presented all the appearances of a person possessed.

It was not confined to one particular locality, but was /
was propagated by the sight of the sufferers like a
demoniacal epidemic over the whole of Germany and the
neighbouring counties of the north west, which were
already prepared for its reception by the prevailing
opinions of the time."

Strasburg was visited by the Plague in the year
1418 and the same infatuation developed among the people
there as in the towns of Belgium and the Lower Rhine.

Swarms of dancers were to be seen passing
through the streets day and night, accompanied by
musicians playing bagpipes and by crowds of spectators
among whom were the anxious parents and relatives of
some of the dancers. The Town Council benevolently
took an interest in the afflicted, divided them up
into separate parties with attendants and had them
conveyed to the Chapels of St. Vitus in Zabern and
Rotostein where priests were in attendance to work
upon their misguided minds by religious ceremonies.
After divine worship they were led to the altar where
they deposited some small offering of alms, and where
it is possible, through the tranquillising influence
of the devotions and the sanctity of the place, many
were cured of their hysterical affection.

St. Vitus was a Sicilian youth who suffered
martyrdom at the time of the persecution of the
Christians under Diocletian in the year 303.
In the year 856 his body finally found a resting place
in Corvey and he became a Saint of great importance,
and the miracles which were manifested at his shrine
were of considerable service in confirming the Roman
faith. His altars were multiplied and many afflicted
persons came to them in the hope of gaining relief.
At /
At the beginning of the fifteenth century, or perhaps even so early as the fourteenth, a legend was invented, "that St. Vitus just before he died, prayed to God that he might protect from the dancing mania all those who should solemnise the day of his commemoration and fast upon its eve, and that thereupon a voice from heaven was heard saying, "Vitus thy prayer is accepted." Thus St. Vitus became the patron Saint of those afflicted with the dancing mania." As St. Martin of Tours was at one time the succourer of persons in smallpox, St. Antonious of those suffering under hellish fire and St. Margaret was the Juno Lucina of puerperal women.

It was not till the beginning of the sixteenth century that St. Vitus Dance was made the subject of Medical study by Paracelsus, who speaks of it as follows:—"We will not however admit that the saints have power to inflict disease and that these ought to be named after them, although many there are who in their theology lay great stress on this supposition ascribing them rather to God than to nature, which is but idle talk. We dislike such nonsensical gossip as is not supported by symptoms, but only by faith, a thing which is not human whereon the Gods themselves set no value."

He divides the dance into three varieties.

(1) That which arises from the imagination (Vitista Chorea, imaginative, aestimativa), by which the original dancing plague is to be understood.

(2) That which arises from sexual desires, depending on the will (Chorea Lasciva).

(3) That which arises from corporeal causes. (naturalis, coacta).
This last he explains in the following way—"that in certain vessels which are susceptible to an internal
thrance and produce laughter, the blood is set in motion in consequence of an alteration of the
vital spirits whereby involuntary fits of intoxicating
joy and a propensity to dance are engendered."

On the communication of the St. Vitus Dance by
sympathy, Paracelsus speaks of sensual impressions
which find their way to the heart—the seat of joys
and emotions—which overpower the opposition of
reason and whilst all other qualities and natures are
subdued, incessantly impel the patient, in consequence
of his original complaint and his all-conquering im-
agination to imitate what he has seen."

It will be seen that in all these varieties of
the disease which Paracelsus describes, the
disease is of hysterical character.

His treatment for the first variety was that the
patient should make a wax image of himself, and con-
centrate all his blasphemies and sins on it and when
he succeeded in this he was to burn the image so that
not a particle of it should remain.

For the second variety which chiefly affected
women he recommended solitary confinement and other
hardships till the misery brought them to their senses.
They were then permitted gradually to return to their
usual occupation. Severe corporal punishment was
also permitted and when necessary immersion in cold
water was to be used to calm excitement.

The third variety he treated with wonderful
remedies, quintessences etc.

About this time the disease began to decline, and
at the same time became milder in its manifestations. While it attacked people in all stations of life, it was especially prevalent among those who led a sedentary life, e.g. tailors, shoemakers, etc. but it also attacked robust country labourers.

In the severer forms, the patient might be so badly affected as to dash his brains out against a wall or building and to avoid such tragic results the bystanders used to place benches and chairs in front of them so that by high leaps they were thus tempted to take, they became all the sooner exhausted.

One attack might be sufficient to effect a cure. Women advanced in pregnancy were capable of going through an attack of the disease without the slightest injury to their offspring which they protected merely by a bandage round the waist.

As certain kinds of music exaggerated the condition the magistrates used to provide musicians so as to make the attacks all the more severe and get the patients all the quicker through them. At the same time the wearing of red garments was prohibited, because at the sight of the colour the affected persons became so furious that they flew at the person who wore them and were with difficulty restrained from doing them violence. They often tore their own clothes, and were guilty of other improprieties, so that the richer people employed attendants to look after them.

Soft harmony was employed to calm their excitement and it is mentioned as a character of the tunes played with this view, that they contained transitions from a quick to a slow measure and passed gradually /
gradually from a high to a low key.

Dancing Mania was very rare at the beginning of the seventeenth century and wars carried on in Western Europe for thirty years with varying success served to dispel superstition in its old form and along with it the belief in the dominion of spirits which existed in the Middle Ages.

Classification of Chorea and Choreiform affections

Esler in his work "Chorea and Choreiform Affections" makes the following classification of the various affections:

(1) **Chorea Minor.** Sydenham's Chorea.

(2) **Chorea Major.** Under this head he included Dancing Mania and the various rhythmical or hysterical disorders of motion. (This has also been designated Chorea Germanorum, because it occurred, as already shown, largely in Germany as opposed to Chorea Anglorum by which is meant Sydenham's Chorea.

(3) **Choreiform Affections and Pseudo Choreas.** This includes the various forms of Habit Spasm or Tic, local or generalised, which are perhaps best grouped under the latter term, in the more extended use as employed by French Writers.

(4) **Secondary or Symptomatic Chereas.** Chronic disorders of motion which depend upon degenerative and irritative lesions of the motor cortex -- Pre-Hemiplegic and Post Hemiplegic disorders of movement; the so called Spastic Chereas and many of the cases of Chronic and Congenital Chereas, one malady alone being separated as an independent affection -- Huntingdon's Chorea.

Various /
Various other types of Chorea have been described such as — Endemic Chorea, Electric Chorea, Hysterical Chorea, Saltatory Spasm, Oscillatory Spasm, etc. These last would probably be included under the head of Chorea Major.

Taylor in his book ("Nervous Disease in Childhood and Early Life" 1905) gives the following classification:

(1) Chorea Proper
   (a) Rheumatic Chorea — Sydenham's Chorea.
   (b) Huntington's Chorea.
   (c) Hysterical Chorea.
   (d) Myoclonus.

(2) Diseases in which Choreic or Myoclonic movements, or movements closely simulating these sometimes occur as symptoms but which are widely separated from the true Choroeas by their etiology and pathology.
   (a) Cerebral Diplegia.
   (b) Friedrich's Ataxia.
   (c) Gross organic disease of the cerebrum (vascular lesions, tumours and abscesses.)

(3) Diseases to which the term Chorea has been applied but which in the nature of the occurring movements are essentially distinct from those of Chorea.
   (a) Habit Spasm.
   (b) The several varieties of Tic.
   (c) Dancing Mania.

Chorea Minor — Acute Chorea — 'Sydenham's Chorea.'
While an ordinary case of Sydenham's Chorea presents manifest appearances which render the recognition of the disease a very simple matter, still it is difficult to give a definition which, while being complete and accurate, will present a good picture of the disease.

Sturges in his work on "Chorea" 1881, defines Chorea as a state of "exaggerated fidgetiness". This is a very apt definition as, the parent of a child suffering from Chorea, often considers and states that her child is just more fidgety than usual.

Osler defines it as follows — "An acute disease of childhood, rarely of adults and the aged, characterised by irregular voluntary movements, muscular weakness and a variable amount of psychical disturbance and often associated with endocarditis and arthritis."

With advantage, the endocarditis and arthritis might be omitted from the definition and rheumatic complications substituted. They would include endocarditis, arthritis — rheumatic nodules, rheumatic rashes etc. By many, chorea itself is now considered to be a rheumatic manifestation and is called cerebral rheumatism.

Sydenham in his "Schedule Monitoria" 1686 (Latham's translation Vol II p. 198) gives the following description of the disease.

"St. Vitus Dance is a sort of convulsion which attacks boys and girls from the tenth year until they have done growing. At first it shows itself by a halting or rather unsteady movement of one of the legs which the patient drags. Then it is seen in the hand of the same side. The patient cannot keep it a moment in /
in its place whether he lay it on his breast or on
any other part of his body. Do what he may, it will
be jerked elsewhere convulsively. If any vessel
filled with drink be put into his hand before it
reaches his mouth he will exhibit a thousand gesticula-
tions like a mountebank".

**GENERAL ETIOLOGY.**

**Sex.**

Cador in a series of 554 cases states that 161 were males and 390 were females. The proportion
being therefore about 2 boys for 5 girls.

In the Report of the Collective Investigation
Committee of the British Medical Association (British
Medical Journal, 1887, Vol 1) out of 459 cases there
were 114 males and 322 females and 3 cases in which
the sex was not specified. This gives a proportion
of nearly 3 females to 1 male.

Krafft Ebing (Weiner Klin. Woch. No.43 1899) in an
analysis of 200 cases had 136 females to 64 males.
This gives a proportion of rather more than 2 females
to 1 male.

Allen Starr (Philadelphia Medical Journal 1900)
in 1400 cases at the Vanderbilt clinic gives the pro-
portion as nearly 2 females to 1 male.

Pye Smith (Textbook of Medicine) in 1610 collect-
ed cases gives the proportion of about 2 males to 5
females.

Wood (British Journal of Children's Diseases, June
1907 p. 266) gives the same proportion as Pye Smith.
From an examination of these statistics it may be said
that for every 2 boys there are 5 girls affected with Chorea.

**AGE**

It is essentially a disease of childhood and early adolescence. It rarely occurs in adult life. Cases before 4 years of age are very rare, only a few authentic cases being described.

Koplick in his work "Disease of Children" describes a case as occurring in a boy at 21 years and Mackenzie (British Journal of Diseases of Children July 1906 p. 319) records a case which occurred in a girl of 2 years after a slight rheumatic attack.

In the Collective Investigation Report of the British Medical Association (B. M. J. 1887 I.) the youngest case recorded was a girl of 3 years, and there was another case in a boy of 3½ years which last proved fatal.

In Allan Starr's collection of 1400 cases, 8 of them occurred before the 5th year.

The disease is therefore rare under four years of age. Congenital cases are probably in all instances caused by definite cerebral changes and belong to entirely different diseases e.g. meningeal haemorrhage and the changes subsequent to this. They are in fact symptomatic Choreas.

**Age Incidence of Chorea in the 439 cases of the Collective Investigation Committee of the B.M.A.**

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<tr>
<th></th>
<th>4</th>
<th>5</th>
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<th>15</th>
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<th>20</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>40</th>
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</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td>114</td>
<td>15</td>
<td>44</td>
<td>6</td>
<td>5</td>
<td>13</td>
<td>16</td>
<td>9</td>
<td>14</td>
<td>11</td>
<td>11</td>
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<td>15</td>
<td>2</td>
<td>7</td>
<td><strong>1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td>322</td>
<td>1</td>
<td>13</td>
<td>11</td>
<td>17</td>
<td>15</td>
<td>29</td>
<td>30</td>
<td>39</td>
<td>28</td>
<td>28</td>
<td>16</td>
<td>56</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td><strong>15</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Sex not Specified</strong></td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
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<tr>
<td><strong>Total</strong></td>
<td>439</td>
<td>2</td>
<td>13</td>
<td>15</td>
<td>25</td>
<td>20</td>
<td>43</td>
<td>46</td>
<td>49</td>
<td>42</td>
<td>41</td>
<td>39</td>
<td>20</td>
<td>71</td>
<td>10</td>
<td>2</td>
<td>1</td>
<td><strong>16</strong></td>
</tr>
</tbody>
</table>
Arranged in decades.

First decade 155 cases.
Second decade 262 "
Third decade 12 "
Fourth decade 2 "
Above Fourth decade, 6 "

Arranged in hemi-decades.

First hemidecade (under 5) 6 cases 1.36 %
Second hemidecade (6 -- 10) 149 " 33.96 %
Third hemidecade (11 -- 15) 191 " 42.5 %
Fourth hemidecade (16 -- 20) 71 " 16.15 %
Fifth hemidecade (21 -- 25) 10 " 2.29 %
Sixth hemidecade (26 -- 30) 2 " 0.45 %
Seventh hemidecade (31 -- 35) 1 " 0.22 %
Eighth hemidecade (36 -- 40) 1 " 0.22 %
Above Eighth hemidecade (over 40) 6 " 1.36 %

The greatest incidence of disease in this series is from 11 -- 15 inclusive and next to this 6 -- 10.

A great falling off is noticed in hemidecade 16 -- 20. Over 93% occurred in the 2nd, 3rd and 4th hemidecade 6 -- 20 years and above 77% occurred in 2nd, and 3rd hemidecade 6 -- 15 years.

Incidence in the two sexes at different ages.

It is very nearly equal.

In the three most important hemi-decades taken together the proportional incidence in males is very slightly in excess which is entirely due to the preponderance of males in the second hemi-decade.
In the second hemi-decade
- males 46  . 40.34%
- females 102 . 31.67%
In the third hemi-decade
- males 49  . 42.98%
- females 140 . 43.47%
In the fourth hemi-decade
- males 15  . 13.15%
- females 56  . 17.39%

**Age Incidence.** Osler's Cases.

|                      | Total | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 20 | 21 | 25 | 30 | 35 | 40 | OVER |
|----------------------|-------|---|---|---|---|---|---|----|----|----|----|----|----|----|----|----|----|----|----|----|----|      |
| Males                | 137   | 7 | 5 | 10| 11| 9 | 20| 11 | 21 | 2  | 14 | 5  | 11| 11 | 1  | 4  | 1  | 1  |     |     |     |
| Females              | 285   | 11| 12| 25| 28| 22| 45| 39 | 37 | 20| 19 | 23 | 25 | 51 | 5  | 4  | 1  | 1  |     |     |     |
|                      | 422   | 18| 15| 35| 51| 63| 50| 58 | 31 | 33| 28 | 36 | 62 | 6  | 4  | 1  | 2  |     |     |     |

**Arranged in decades.**
First decade  261 cases
Second decade  248 "
Third decade  10 "
Fourth decade  1 "
Above Fourth decade  2 "

**Arranged in hemi-decades.**
First hemidecade  33 cases
Second  228 "
Third  212 "
Fourth  62 "

It is thus shown by this table that three-fourths of the cases occur in the second and third hemi-decades. The second hemi-decade contains the greatest number of./
of cases in males and the third hemi-decade in females.
Of Allen Starr's 1400 cases -- 75% occurred between
the age of 5 and 15.

Sir William Gowers (Text-Book of Nervous Diseases
1891) states that not more than 5% of the cases occur
after 20 years of age.

Eve Smith states that among cases after 17 years
of age the preponderance of females over males is even
greater than is the case in childhood.

For 27 women affected above 15 years there were
only 4 men.


tate Chorea.

Chorea is extremely rare after 25 years of age
except in pregnant women.

Eve Smith records a case in a woman 38.
Among the Collective Investigation Report there
were only 10 cases above 25 years of age and 5 of
these were in old women between 63 and 86.

Greaves reports a case of an apothecary in Dublin who
was attacked when 70 years old.

Rongberg saw a case in a woman of 76 which had how-
ever began when she was six years old.

Trousseau records a case in a lady of 83 who re-
covered from the disease in 5 weeks.

Charcot, in his "Lectures on the Nervous System"
has described Senile Chorea as a variety of the disease
but it does not seem necessary to recognise this as a
special class by itself.

Social Status.

Statistics on this subject are given in the
Collective Investigation Report.

Of the 439 cases these were:

The /
The upper classes ... 12 (males 3, females 9)
The middle classes ... 115 (" 41, " 74)
The lower classes ... 203 (" 71, " 232)

9 cases were not classified.

Preponderance falls very heavily on the lower classes, to which of these tabulated cases, 70.46% belong, whilst 26.74% to the middle classes and 2.79% to the upper classes.

Osler states that nearly all his clinical information of the disease has been drawn from hospital work.

Sturge in his work on ("Chorea, 1881") considers that the crowding of the children of the poorer classes into schools and keeping them there for the livelong day explains the frequent occurrence of this disease in these classes. From the Collective Investigation Report we also see that the type of child apt to be affected was one rather bad to moderately-well nourished and of moderate strength.

The Previous Mental Condition.

This, in the great majority of cases, is well up to or even beyond the average. In very few cases is it below the normal.

Bodily Growth.

Not infrequently we find that the subject of Chorea especially in the third decade exhibit rapid growth. In the Collective Investigation Reports, growth is put down as rapid in 36.21% of the collected cases.

Sexual Functions in the Female.

There is nothing special to note under this heading.
heading. A tendency to Menorrhagia has been noted.

Pregnancy

McCann's (Transactions of the Obstetrical Society London vol. XXXIII) gives a report on 34 collected cases of Chorea in Pregnancy.

Of this number eleven previously had had Chorea.

The ages of the patient are given:

Three occurred at 17 years of age.
Four " " 18 " " "
Six " " 19 " " "
Eleven " " 20 " " "
Two " " 21 " " "
Three " " 23 " " "
One " " 24 " " "
One " " 25 " " "
Three " " 26 " " "

Primiparae are most frequently attacked.

<table>
<thead>
<tr>
<th>Period of occurrence during Pregnancy</th>
<th>36 cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>One month</td>
<td>2</td>
</tr>
<tr>
<td>Two months</td>
<td>6</td>
</tr>
<tr>
<td>Two and a half</td>
<td>1</td>
</tr>
<tr>
<td>Three</td>
<td>6</td>
</tr>
<tr>
<td>Three and a half</td>
<td>2</td>
</tr>
<tr>
<td>Four</td>
<td>7</td>
</tr>
<tr>
<td>Five</td>
<td>4</td>
</tr>
<tr>
<td>Five and a half</td>
<td>2</td>
</tr>
<tr>
<td>Six</td>
<td>3</td>
</tr>
<tr>
<td>Six and a half</td>
<td>1</td>
</tr>
<tr>
<td>Seventh</td>
<td>1</td>
</tr>
<tr>
<td>Eighth</td>
<td>0</td>
</tr>
<tr>
<td>Ninth month</td>
<td>1</td>
</tr>
</tbody>
</table>

36
The following details as to causation are given in 34 cases.

Chorea occurred previously in 11 cases.
Rheumatic Fever and Scarlet Fever in 2 cases.
Rheumatic Fever alone in 2 cases.
Fright only cause stated in 2 cases.
No cause stated in 4 cases.

(in one case one sister had had rheumatic fever and in another Chorea and rheumatism existed in the family.)

Rheumatic Fever and Fright 1 case.
Rheumatic Fever and Chorea 7 cases.
Scarlet Fever, Rheumatic Fever & Chorea 1 case.
Chorea and Fright 3 cases.
Mental Disturbance due to Pregnancy 1 case.

Out of 32 cases in which there was no artificial interference the patient was delivered at term in 26.
In 1 case accidental haemorrhage and miscarriage occurred at the 4th month; in 1 case miscarriage at the 5th month, and in 4 cases miscarriage at the 6th month.

Out of 39 cases death occurred in 7: 3 from Mania, 3 from exhaustion and 1 from cerebral peritonitis. This gave a death rate of 18%.

Barnes and Sir William Gowers give statistics as regards age of occurrence of Chorea in Pregnancy in 23 cases.

8 occurred at 20 years of age.
3 occurred at 17, 18 and 19 years respectively.
2 occurred at 21 and 22.
6 occurred at 23 years of age.
1 /
1 occurred at 24 years of age.

Ruist of Dundee (Obstetric Transactions Edinburgh vol. XX p. 141) gives an analysis of 225 collected cases of Chorea occurring in Pregnancy.

Chorea was present at the onset of pregnancy in 6 cases and the variety of its behaviour in relation to the gestation is both interesting and curious. In 1 case, a Chorea of seventeen years standing ceased suddenly at the onset of pregnancy and in another it ceased suddenly after labour. In another case it continued unchanged and in the others after being exaggerated during the pregnancy disappeared after delivery.

Chorea is stated to have occurred previously in 66 cases, 42 being primiparae.

Hysteria noted in 14 cases.
Shock, fright or violent emotional disturbance immediately or at a short interval preceded the Chorea in 23 cases.
Rheumatic History was present in 45 cases.
Insanity previously is recorded in 1 case while it occurred in 8 other cases after Chorea.

As regards age it is evident from his statistics that the disease is increasingly less common after 24 years of age.

He also states that it seems from his statistics that illegitimacy is not an exciting cause.

<table>
<thead>
<tr>
<th>Onset of Chorea</th>
<th></th>
</tr>
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<tbody>
<tr>
<td>1st month</td>
<td>29</td>
</tr>
<tr>
<td>2nd &quot;</td>
<td>30</td>
</tr>
<tr>
<td>3rd &quot;</td>
<td>42</td>
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<tr>
<td>4th &quot;</td>
<td>22</td>
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<tr>
<td>5th &quot;</td>
<td>25</td>
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<tr>
<td>Post-nuptial</td>
<td></td>
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<tr>
<td>6th month</td>
<td>19</td>
</tr>
<tr>
<td>7th &quot;</td>
<td>13</td>
</tr>
<tr>
<td>8th &quot;</td>
<td>5</td>
</tr>
<tr>
<td>9th &quot;</td>
<td>7</td>
</tr>
<tr>
<td>Post-partum</td>
<td>16</td>
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</table>
In the first three months 138 cases.
  "  second  "    70 "
  "  last  "    25 "

Termination of Pregnancy:

<table>
<thead>
<tr>
<th>Month</th>
<th>Cases</th>
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<tr>
<td>3rd</td>
<td>5 times</td>
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<tr>
<td>4th</td>
<td>4 &quot;</td>
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<tr>
<td>5th</td>
<td>6 &quot;</td>
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<tr>
<td>6th</td>
<td>8 &quot;</td>
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<tr>
<td>7th</td>
<td>8 times</td>
</tr>
<tr>
<td>8th</td>
<td>7 &quot;</td>
</tr>
<tr>
<td>9th or term</td>
<td>106 &quot;</td>
</tr>
</tbody>
</table>

Effect of Delivery.

In most uncomplicated cases of Chorea, the Chorea symptoms gradually improved after delivery, even without treatment.

A-Choreic Pregnancies preceded the choreic in 31 patients, the number of such pregnancies being 52. Of these patients only 5 are recorded to have had previous Chorea.

Recurrences.

Recurrent Chorea in pregnancy in 31 cases, the number varying from 2 - 5 recurrences.

Maternal Mortality.

In 255 pregnancies 45 were fatal. A mortality of less than 1 in 5, and this must be in excess of the actual mortality, inasmuch as so large a number of the milder cases are not reported.

Recovery from the Chorea was stated to have occurred in 63 cases before the termination of the pregnancy.

Sixteen cases died after spontaneous delivery, but at least in eight of these the Chorea was not the cause.
cause of death.

**Induced Delivery.**

This took place in 20 cases: of these 8 died, but in several the complications were so grave that the results can scarcely be attributed to the Chorea.

**Rheumatism.**

The association of Rheumatism with Chorea is very common. This was first conclusively shown by Sée ( Mémoires de L' académie de Medicine Tome XV, 1850) while Roger, in 1867 put forward the opinion that Acute Rheumatism, Cardiopathy and Chorea were several manifestations of the same disease. (Arch. Gén. de Medicine, Dec. 1866 and Jan. 1867.)

There is often a history of Rheumatism in the family and acute articular Rheumatism, Growing Pains, Cardiac disease is often got in the individual affected.

In the absence of these other signs of rheumatism there may be a history of Rheumatic Erythema, Purpura, Erythema Nodosum, Rheumatic nodules etc.

Signs of Rheumatism are not unfrequently present in a Choreic attack, e.g. Endocarditis, pericarditis, nodules Erythema, purpura. The occurrence of articular rheumatism during an attack is rare.

German investigators with the exception of Meyer (Jahrb. of Kinderh. 1895 XL p. 144) who attributes 90 % of his Chorea cases to Rheumatic infection, have not attached much importance to the Rheumatic causation.

**Bright of Guy's Hospital** (Syllabus on outlines of Lectures of Practice of Medicine, published at Guy's /
Guy's Hospital (1802 and 1820) recognises the relationship.

Babinbton (Guy's Hospital Report 1841 VI. p. 411) noted also the occasional association of Chorea with Rheumatic affections of the heart and pericardium.

Pritchard of Bristol (London Medical Repository Vol XXI. 1824) reports several severe and fatal cases of Chorea and in speaking of one where pericarditis was present, says the disease seems to have arisen from the metastases of Rheumatism.

Tylden found that the proportion of 72% of the patients suffering from Chorea had a personal or family history of rheumatism.

Collective Investigation Report gives the percentage of those cases in which acute or sub-acute Rheumatism preceded the Chorea as 26.

Besides these definite cases other 14% were said to have had vague pains, the term suggested in the enquiry, rheumatic or "growing pains." In those cases of vague pains 49% showed a family history of rheumatism.

Rheumatism also occurred during or after the attack in 26 cases, and this added to the other cases of definite rheumatism raises the percentage to 32.

Horsic Donkin (Westminster Hospital Report Vol I p. 5) in 104 consecutive cases of Chorea in children found antecedent rheumatism in 27 or in nearly 26% and of these, in 15 the Chorea seemed to be immediately caused or excited by Rheumatism.

Osier in his monograph on "Chorea and Choreiform affections" gives 15.5% as having a history in the family of rheumatism, while 15.3% had had at some time /
time or other either prior to, with, or subsequent to
the attack of Chorea, acute or sub-acute articular
swellings.

In other 33 cases there was a definite history of
pains in various parts, sometimes described as rheumatic
but not associated with joint trouble. If these are
regarded as rheumatic and added to the manifest ar-
thritis, the percentage is raised to 21.

Towseend. (Transactions of American Pediatric Soc-
ociety) vol IV 1892) reported 143 cases with rheumatic
history in 31 or 21%.

Grandeii reported 33 cases with rheumatism either
before or after, in 54%.

Allen Starr gives 335 cases with definite
rheumatism in 19%.

Kraft-Ewing (Weiner Klin. Woch. No.43 1899) gives
200 cases with 50 as having had acute articular
rheumatism -- 25%.

Ség (La Méd. Mod. Oct. 15. XXII 1891) states
that 134 out of 196 cases of Chorea showed rheumatism
and he states that in the majority of cases Chorea is
the result of Rheumatic diathesis, although cases oc-
cur which must be considered true neuroses. -- 65.4%.

Allen Starr (Phil. Med. Journal May 26th 1900) in
1400 collected cases states that 290 had a distinct
history of rheumatism. 20.7%.

Osler (Pacific Medical Journal August 1395) gives
an account of 140 cases where in 51 the heart was normal
in 72 symptoms of organic lesions were present and in
17 cardiac disturbances. There was no rheumatic
History in 66% of these cases. He considers the
cause to be an infection allied to Rheumatism but
differing /
Mfisr (Glasgow Medical Journal 1897) tells of six Chorea cases, all in young persons, seventeen to twenty-one years of age in whom the disease was very grave, and proved fatal in two. The previous association of Scarlatina or Rheumatism (articular, endocardial, and pericardial) was noted in every case and also the occurrence of the Chorea on the same side as had been formerly affected with rheumatism.

Purves-Stewart (Med. Brief June 1893)
Out of 29 Choreic patients personally examined, in 7 there was a history of previous rheumatic fever in the patient; in 4 there was a strong family history of rheumatic fever; in the remaining 9 there was no history of rheumatic fever but out of those 9, 2 had mitral stenosis, 5 had mitral regurgitation and only 2 had no valvular lesion.

Williams (Memphis Lancet August 99)
He states that about 21% of all Choreic cases give a rheumatic history, either in the parents or in themselves previous to the disease.

Batten (Lancet vol II p. 1195 1898) states that in 115 children suffering from Chorea 32% suffered from rheumatism previously. Three years later he followed up these cases that had not suffered from rheumatism previously, and notwithstanding the fact that he was unable to trace some of the cases, 11.3% of the total number had developed rheumatism in these three years. On making further observations three years later he found that a further 9.7% of the total had developed rheumatism within six years of the Chorea, giving a total percentage of 53.

Goodhart /
Goodhart and Still (Diseases of Children 1900) suggest that Chorea itself is the first manifestation of rheumatism in some cases and that rheumatism occurs later. They also state that about 30% of families taken indiscriminately there is a history of rheumatism while in Chorea cases the percentage is about 60.

Gilles de la Tourette (Rev. Neurol. June 30th 1900) states that Chorea is not associated with rheumatism and gives a statement of 17 consecutive cases where only 1 had rheumatism before or during an attack, and of several who returned after recovery none had shown any sign of rheumatism.

Farre and Pye Smith (Text Book of Medicine Vol I 392) quote the following statistics:

<table>
<thead>
<tr>
<th>Author</th>
<th>Number of Cases</th>
<th>Had Rheumatism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sturges</td>
<td>100</td>
<td>20</td>
</tr>
<tr>
<td>Donkin</td>
<td>104</td>
<td>27</td>
</tr>
<tr>
<td>Goodhart</td>
<td>130 collected</td>
<td>89</td>
</tr>
<tr>
<td>Angel Money</td>
<td>214</td>
<td>56</td>
</tr>
<tr>
<td>Stephen</td>
<td>172</td>
<td>47</td>
</tr>
<tr>
<td>MacKenzie</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hallstead</td>
<td>163</td>
<td>53</td>
</tr>
</tbody>
</table>

(In 35 of these 53 cases of Hallstead, the rheumatism only preceded the Chorea by 6 months.)

Wood (British Journal of Children's Diseases June 1907) in a report on 223 cases only gives 13% as having had a definite history of rheumatism. In only 1 case did he get rheumatic nodules.

Butler (Pediatrics vol. XX no. 10 p. 613) inquired into the history of 59 consecutive cases of Chorea presenting themselves at the Cork County Hospital /
Hospital Chicago.

In 71% he obtained a history of tonsillitis and in 61% he obtained a history of rheumatism, while in 28% endocarditis was present. He further states that with a few exceptions a close relationship in point of time was evident between the attacks of tonsillitis rheumatism and Chorea.

From what has been written on the relationship between Chorea and Rheumatism, it is evident that the majority of the observers recognise that there is a close relationship between the two diseases. Rogers as early as 1867 expressed the belief that Chorea and Rheumatism were manifestations of one and the same disease. The researches of Batten are also very interesting, indicating that Chorea itself may be the first manifestation of Rheumatism and in this opinion he is supported by several other observers such as Still, Garrod etc. It is moreover very probable that, if the wide conception which Chedle held regarding rheumatic manifestations in childhood, had been taken into consideration in all cases in the search for either a personal or family history of rheumatism the percentages of Chorea patients who could have been labelled as rheumatic would have been in many instances, much larger.
INFECTIONOUS DISEASES.

Various infectious diseases have been stated to have occurred previously to Chorea and possibly to have had an influence in the production of the disease. Carslow (Glasgow Medical Journal 1891) reports that Chorea developed in 3 of 533 cases of Scarlet Fever.

Osler states that a previous history of Scarlet Fever was obtainable in 141 of his 522 cases but in not one was the sequence immediate.

Priestley (British Medical Journal Sept. 25th 1897) asks the question — "Should Chorea be considered a sequel to Scarlet Fever?" He goes on to say that Cheadle recognises it as such, but qualifies the opinion by adding that in 1894 and 1896, 8360 Scarlet Fever cases were under treatment in the North Eastern Hospital and of these 5355 were completed there.

Thirteen cases of Chorea were observed: 1 - 142 completed cases.

Osler found 1 case of Chorea to every 130 patients of all classes. Hence it would appear that Chorea is less frequent among Scarlet Fever patients than among patients in general.

Of Osler's 13 cases, 5 had rheumatic manifestations which in each instance immediately preceded or appeared simultaneously with the Chorea.

Rheumatism or Joint Affection which occurs as a complication of Scarlet Fever sets in towards the end of the first week: but in these cases it was considerably later, indicating a difference in the nature of the joint affection.

Williams /
Williams (Memphis Lancet August 1899) states that Chorea follows an attack of Scarlet Fever in about 25% of cases.

Collective Investigation Report.
Scarlet Fever was present in 129 cases of the 439 cases. In 27 it was the sole antecedent and in 10 of these cases there was some heart affection. In 7 of the 10 cases the heart was known to be normal before the attack and only 1 of these suffered from rheumatism. The tendency of the fact is to show that Scarlet Fever predisposes to the occurrence of heart affection in Chorea. Rarely, however, are any cases associated with Chorea and we must regard them as rather an indirect exciting cause of Chorea.

Ross is of the opinion that Chorea follows Scarlet Fever frequently, and explained the association by the fact that rheumatism frequently follows Scarlet Fever.

Measles, Variola, Diphtheria, Enteric Fever, Cholera, Pneumonia and Influenza have all been known to be followed by Chorea.

Sturges emphasises the fact that whooping cough is an important antecedent to Chorea.

Pyaemia, Gonorrhoea and Malaria are also said to predispose, while Féré maintains the same in regard to secondary syphilis. Matthew (American Journal of Medical Science Sept. 1903) considers that congenital Syphilis may be a cause. It cannot be said that any infectious disease specially predisposes to Chorea. The occurrence of any one, such as Scarlet Fever before the onset of Chorea, is no proof of any causal connection. Some few cases are recorded where the Chorea /
Chorea seemed to develop immediately after Scarlet Fever but the question of whether rheumatism is not really the cause cannot definitely be settled.

Anaemia.

This seems to be less often an antecedent than a sequel of Chorea, and though cases develop in children who are anaemic and in poor health, this is not the rule.

Osler considers that it may develop after chlorosis in girls about puberty.

Litten (Charité Anaemie Bl XI) quotes a case of Roesor in which Chorea developed six days after a copious epistaxis and proved fatal. He also tells of two cases which occurred in the course of pernicious anaemia, both ending fatally; showing at the post mortem, definite signs of fresh endocarditis.

Barr (Pediatrics Feb. 1st 1897) states his results from examination of the blood in 40 cases.

There is usually a diminution in the haemoglobin and a relatively slighter decrease in the number of red corpuscles, in other words the anaemia is chlorotic in type. There is no relation between the severity of the anaemia and the severity of the attack. Anaemia is not an immediate direct exciting cause but frequently a predisposing one.

In the Collective Investigation Report, anaemia is recorded as an antecedent in 92 cases (males 26, females 66) or in 20% of all the cases.

Rashford (Medical News, April 22nd 1893) strongly supports the idea that anaemia is a very important predisposing cause.

Imitation /
Imitation.

This is often mentioned as a predisposing cause of Chorea.

Osler in his cases cannot find one single instance of this.

Goodhart and Still (Diseases of Children) state that they have never seen a case of this.

West (Diseases of Children) records instances but the rate of occurrence cannot be very great, and when they do arise they probably do so in patients who are choreically disposed.

Steiner (Jahrbuck fur Kinderh. 1870) has described an outbreak at Prague, but he could see nothing to support the imitation theory and thought that atmospheric conditions had more to do with it.

Weir Mitchell in his (Lectures on Nervous Diseases) describes an epidemic at a church home for children in Philadelphia in 1880 but in this as in the majority of such outbreaks, hysteria was the real condition.

Vichmann (Deutsche Medicinishe Wochenschrift 1890) describes an outbreak at Wildbad and in this instance some of the cases seem to have been true Chorea but the majority were examples of hysteria.

In the Collective Investigation Report imitation is stated to be the exciting cause in 13 cases.

The action of imitation in producing Chorea has been compared to the occurrence of yawning which often makes itself apparent in several people in a group when one starts it.

Goodhart and Still discuss this and state their opinion as follows: “There is no parallelism between /
between the two conditions. Yawning is an orderly sensori-motor action while Chorea is an irregular combination of involuntary movements on the part of the muscles which are for the most part habituated to perform movements under the control of will. One cannot conceive of the Choreic movements being elicited by any more sensori-motor disturbances such as starts a yawn."

Psychical Influences. Emotion. Fright.

Osler states that a history of fright was obtainable in 33 of his collection of cases (15.5%). In the majority of cases no very close connection existed between the fright and the onset of the disease, although in a few cases the attacks came on at once.

Risien Russell (Allbutts System of Medicine vol. VII Article Chorea) says that many histories of fright have to be discredited as mothers realize that this is a recognised cause of the disease and in many of such cases there is a considerable interval between the fright and the onset of Chorea. There are however some well authenticated cases where the disease followed immediately after the fright so as to leave no reasonable doubt as to the relationship of cause and effect:—

Romberg's case. A little girl was attacked the same day with Chorea after a severe fright from a dog jumping on her.

Von Ziemssen refers to a case where the disease became very severe a few hours after a fright (Cyclopedia of Medicine Art. Chorea.)

Trousseau (Clinical Lectures on Medicine) reports two such cases occurring both in boys. In one it /
it was due to an unexpected pistol shot close to his ear and in the other boy it was due to him falling from an apple tree (in his rapid descent) on being detected.

It is however unusual to find the disease occurring so soon after the exciting cause. Usually several days to a week elapses and where there is an interval of several weeks it is difficult to see any casual relationship.

Collective Investigation Report.
Fright is given as the exciting cause in 93 out of the 439 cases.

School Life.

Sturges in particular mentions the strain of school life as being an important etiological factor in the causation of disease.

We often find that Chorea develops in children who are bright and active at school and very anxious to do well. Sturges points out how that the majority of the cases occur in poor children who are compelled to "painful stillness during school hours and suffered to run wild during the rest of the day".

Mental overwork at school does seem to be a fairly common predisposing cause of Chorea.

As regards fright, Still, Duckworth, and others have shown that rheumatic children, especially nervous timid children are much more subject therefore to be frightened.

It is also possible that the child may have been suffering from mild unnoticed Chorea before the fright which so aggravates the condition as to render the Chorea quite manifest.

Poisons.
Poisons.

There are a few observations pointing to the occurrence of Chorea due to poisoning by gases, medicines etc.

Denny (Abstract in Schmidt's Jahrb Bl. 3CXXIV) reports a case of a boy, aged 6 years with caries of the Cervical Vertebrae who had an Iodoform bougie placed in the fistula on August 1st. He did not feel very well after it for a week or so. On the 13th a second was inserted and on the following morning Choreic movements began in the arms and legs. He gradually improved and by September 15th was well. About the end of October the fistula was injected with Iodoform again and 4 times subsequently. The Choreic movements returned.

Eye Conditions.

Stevens (Transactions of the New York Academy of Medicine 1874 - 76 and New York Medical Journal 1876) has claimed that ocular defects lie at the basis of many cases of Chorea and that if these be corrected the Choreic movements will cease.

De Schweinitz has examined this statement and makes the following report:-

"Hypermetropia and Hypermetropic astigmatism are vastly the preponderating condition in eyes of Choreic children, being found in about 77% of cases, exactly as hypermetropic refraction is the preponderating condition in childhood, being found in 76% of the eyes of children in elementary schools.

The evidence, however, seems quite as lacking that hypermetropic refraction is the basal cause of Chorea, as it is that Chorea is the cause of Hyper-
hypermecroopia."

De Schweinitz further writes to Osler (Osler on Chorea and Choreiform affections) and states that he has examined over 100 cases suffering from Chorea, and adds the following observations:

"That ordinary Chorea and many of the forms of facial spasm, habit spasm etc. are materially benefited by correcting refractive errors and anomalies of actions of the ocular muscles just as they are helped by a variety of other treatments but I do not believe there is any proof to show that eye strain of itself is responsible for its origin, with perhaps the single exception of the so called habit-spasm affecting the orbicularis and immediate facial area. Certainly many of these will disappear promptly after the refractive error is corrected without any treatment whatsoever and they will not disappear unless, if you do not relieve the eye strain."

"In a constitution predisposed to Chorea I presume eye strain is a very important factor in fostering or perhaps provoking attacks but that is all."

Stevens (Journal of Mental and Nervous Diseases 1333 p. 543) showed 5 cases to the New York Neurological Society but not one of them was true Chorea Minor.

Ramsey (Text-Book of Nervous Diseases 1391) strongly upholds the view that Chorea is due to refractive errors and he give the history of many cases which he calls Chorea which were immediately relieved and cured by correcting eye defects. It is probably however that as his cases were chiefly all in adults.
adults, that they were really examples of habit-spasm e.g. a clergyman who suffered unceasingly from facial contortions when in the pulpit and who was cured immediately when some eye defects were corrected.

We learn from this the importance of looking for and remedying eye defects if they exist in cases of habit-spasm which affect especially the face.

Climatic and Seasonal Relationships.

Morris J. Lewis (A Study of the Seasonal Relations of Chorea and Rheumatism for a period of fifteen years) analyses 717 separate attacks of Chorea as regards the month in which they occur. We learn that the occurrence is not influenced by climate but there is a distinct relation as to the time of the year as regards the onset of Chorea.

The smallest number occurred in November 24 or 3.3%. In December there was a rapid rise 55 or 7.3% The number then remained fairly stationary during January and February and rose to its highest point in March, 101 or 14.3%. It fell somewhat in April, 63 or 3.7% and rose again in May to 80 or 11.1% and after this there was a gradual fall in its monthly coincidence till the November minimum was reached.

Sir William Gowers found that in 100 of his cases, 33 occurred in the first 3 months of the year, 25 in the second, 20 in the third and 27 in the fourth. He however states that there was a distinct relation to the time of the year as regards recurrences.

He records one case where the recurrence occurred at /
at the same season for several years. But the time of recurrence was not constant for all the recurrences even in a given case, nor was there any particular time of the year for recurrence in all cases.

Lewis states the following conclusions:

1. Seasonal relationship between Chorea and Rheumatism is proven.

2. While over-study assuredly plays a most important role in predisposing children to Chorea, the months of greatest study and therefore presumably of greatest depression of bodily vigour do not coincide with or even precede with any regularity the months of greatest frequency of Chorea.

3. The weather is probably an important factor in Chorea, though precisely which meteorological fact is the baneful one does not clearly appear. No one element of "weather" explains fully the fluctuations of these tracings of Chorea although in the barometer and the storm statistics their relationship appears to be closer than to any other etiological factor or factors, that have as yet been advanced.

4. Either this apparently close relationship must be acknowledged to have an important place in the etiology of these diseases or else the resemblance must be considered to be purely accidental which seems most unlikely from a study of the tables shown.

Geographical Distribution.

Hirsch (Historical and Geographical Pathology) says:- /
No part of the world is altogether free from Chorea, but it would seem to occur more frequently in some places than in others. There are on the whole no considerable differences noticeable in the diffusion of the disease within the temperate zone of Europe and America. In the Countries of Southern Europe also, the malady would appear to be not altogether rare, according to Faure's information from Spain and Rigler's from Turkey.

Palgrave saw several cases in Arabia and Barch-grevink says it is very common in Madagascar. In Egypt, Pruner only saw a few cases and only in strangers.

It has also been seen in Algiers among the Arab population; in Sanagambia also among the natives; among the same class in the Western Sudan and on the Gold Coast.

Rubesof only saw one case in Pondicherry in India during a somewhat long residence there.

In China it is said (doubtfully) to be quite unknown. It appears to be very rare in the West Indies according to Rubz in Martinique where he only saw one case during twenty years and Direste in the same island never saw a case in 30 years, and Rouchoux gives the same account for Gandeloupe.

There is no information about Chorea from the equatorial regions of the western hemisphere: but it is spoken of as occurring somewhat frequently in the table land of Mexico.

No climate would appear to be immune to the disease.
Locality

The disease prevails more in towns than in the country, but it is extremely widespread as shown by the report of Isaac W. Owen (Transactions of the International Medical Congress Washington Vol. V page 157).

Race.

According to W. S. Mitchell Chorea is rare among the negroes. Sinkler has only met with one case in a full blooded negro. Osler met with no case of the disease in a negro child of full blood.

Disease is said to be unknown in the coloured children of Cuba although not uncommon on the whole among the whites.

Osler's enquiries elicited no cases among full-blooded Indians.

It is of common occurrence among Hebrews (Risien Russell).

Hereditary Tendencies.

Osler reports that Chorea occurred in other members of the family in 30 of his 554 cases. 14.4%. The proportion of cases where Chorea is present in the parent is small.

Sir William Gowers (Manual of Nervous Disease 1893 p. 547) states that some neuropathic heredity can be traced in one sixth of the cases -- epilepsy, insanity or Chorea itself. Although the total proportion is not great, the family tendencies in some cases are very marked e.g. Three sisters suffered from true Chorea.

A sister also had Chorea and the mother suffered from epilepsy. The mother of one patient was insane and her sister's child had also Chorea. The father of another had Chorea in early life and so had two
two children of his brother. Five relatives of another had been insane.

"Many other similar facts have been met with in the investigation of the clinical history of Epilepsy e.g. A man was epileptic, his sister insane and two of her children had Chorea. Two sisters of an epileptic girl had had Chorea. Two children suffered from Chorea, their mother's sister being epileptic and insane."

Reflex Agencies.

Under this heading are to be mentioned worms, phimosis, adenoids, eye conditions etc.

Qatar states that in his 554 cases he could not discover history of intestinal parasites as being casual factors in the occurrence of the disease. It is however not improbable that they may act to a certain extent in determining the onset of attack of Chorea in a choreically disposed child, when we recognise that convulsions may in a child be caused from phimosis.

In a case reported at the end of this thesis one girl who was affected with Chorea improved rapidly to a very considerable extent after treatment with antonin for intestinal worms.

Burnet (British Journal of Children's Diseases April 1904) considers that even in rheumatic subjects Chorea may have its origin in causes other than the rheumatic toxin and gives two cases in which tapeworm was present and the Chorea rapidly subsided when the tapeworm was expelled.

Operations and accidents have also been said to be immediate causes of an attack, but probably the concomitant /
concomitant emotion is the cause.

Hysteria.

As Chorea is so much more commonly found in girls than in boys, it is not surprising that the two conditions should be sometimes present at the same time in one individual.

Duchateau (Thése de Paris 1893) has collected 34 such cases.

On the other hand a few cases have been recorded where hysteria has reproduced the clinical picture of Chorea with wonderful exactness but the hysterical stigmata should be enough to distinguish these from the genuine cases.

Dahove (Sociét Medicales des Hopitaux 1890) reported such a case. It was a man aged 21 years who had all the features of Chorea (Sydenham's). He also however presented well marked hysterogenetic zones and the strongest possible compression over the lower part of his abdomen corresponding to the ovarian region stopped the attack. The patient had also anaesthesia of the pharynx. The movements began the day after he had had a serious trouble with his fiancée and an attempt to commit suicide in which however the rope broke.

Temperament.

Chorea very frequently occurs in bright happy active-minded children.

Morbid Anatomy.

Brown-Sequard and Godfellow (Chorea -- Transactions of the Pathological Society vol XIII 1892 p. 19) /
19) observed softening of the Spinal Cord.

Tuckwell (British and Foreign Medico-Chirurgical Review Vol. XL 1867 p. 506) found a branch of the middle cerebral and another of the posterior cerebral artery occluded by emboli and foci of softening in the cortex of the brain corresponding to the distribution of the occluded vessels.

Ogle (Remarks on Chorea Sancti Viti, British and Foreign Medico-Chirurgical Review Vol. XLI 1883 p. 203 and 365) publishes the result of postmortem examinations on 16 Choreic cases. In six, congestion of the nervous centres was noted. In a girl of 17 years of age who died from maniacal Chorea during pregnancy, hyperaemia of the surface and softening of other parts of the brain were observed. The anterior columns of the spinal cord in the lower dorsal region was swollen and softened which consisted on microscopical examination, of extravasation of blood and granular exudation.

Maynert (Ueber die geweblichen Veränderungen in den Central-Organen des Nervensystems bei einem Falle von Chorea Minor. All Weiner Med. Zeitung Bd. XIII 1873 p. 67 and 73) describes hyaline swelling with molecular degeneration of the protoplasm of the cells and the cortex of the brain, partial sclerosis of the cells and the cortex of the Island of Reil and of the basal ganglia and multiplication of the nuclei of the nerve cells. He also got multiplication of the nuclei of the neuroglia cells and swelling of Deiters cells in the Spinal Cord.

Elischer (Virchow's Archives Bd. LXI 1874 p. 489 and Bd. LXIII 1875 p. 104) in a pregnant woman who died /
died of Chorea discovered nuclear proliferation, hyperplasia of connective tissue and thickening of the tunica adventita of the small vessels of the Corpus Striatum and division of the nuclei of the nerve cells in the claustrum. The Spinal Cord presented thickening and nuclear proliferation in the walls of the vessels, thickening of the ependyma of the Central Canal and nuclear proliferation of the connective tissue round the nerve cells in the grey matter. The cells themselves presented a dull appearance, were destitute of nuclei and filled with pigment. The white substance was hyperemic and the fibres in the peripheral nerves diminished in number. 

Boven Lewis (Medical Times and Gazette 1876 II p. 283) records a fatal case where large haemorrhage occurred in the cerebellum.

Baxter relates a case where the haemorrhage was in the cerebrum (Brain April 1871).

Discrepant results have been described on examination of the motor neurons of the cerebral cortex.

Turner in 5 cases found some of the large pyramidal cells swollen and their protoplasm cloudy and dense looking (Path. Soc. Transactions London, 1392 XLIII.

Berkelz (John Hopkins Hospital Reports 1891 II p. 137) in a case of Osler's which he examined found no special changes in these motor neurons.

Dana found the pyramidal cells in a state of hyaline degeneration, but his case was complicated by chronic lepto-meningitis.

Eligerer and Jakowska (Wieszlock Psichiatr i nervopatol 1883) have described peculiar round hyaline /
hyaline bodies concentrically laminated and strongly refractile, to which they have given the name Chorea corpuscles. They were discovered in the perimuscular sheaths of the vessels of the corpora striata and the internal capsule but Wollenberg (Arch. of Psych. and Nervenkrank 1892 XXIII p. 167) who carried out control investigations in 46 persons not affected with Chorea found the brains of six persons so affected. He concluded that although these bodies are present in some cases of Chorea, they have no special significance as they are also to be found in persons who have never had the disease.

Changes found in the pons, medulla and Spinal Cord are not very characteristic. Hypaeremia punctiform haemorrhages and perivascular round cell exudation. The supposed degenerative changes described by Elisher in the neurons of the Spinal Cord are not characteristic. Elisher has described the following changes in the peripheral nerves:- Hyaline swelling of the axis cylinders with increase of interstitial tissue but this is of questionable significance.

Reichardt of Zehnitz (Deutsche Archive fur klinische Medizin 1902 p. 506) in an investigation of two fatal cases of Chorea found small haemorrhages irregularly scattered with collections of leucocytes chiefly mononuclear and dilatation of vessels with peri-vascular small celled infiltration in many parts of the brain in varying amount. No changes were detected in the ganglion cells but there were areas of fatty degeneration of nerve fibres. In the Spinal Cord the parts most affected were the root fibres and the anterior and lateral horns of the posterior /
posterior columns, the anterior and lateral tracts were free.

Foxton and Holmes (Lancet 1906 vol. II p. 132) in an article on the Pathology of Chorea gives the results of post mortem examinations in three fatal cases of Chorea.

The study of the Central Nervous System has shown that the pathological changes which form the morbid anatomy of the disease are composed of (1) vascular and inflammatory changes in the central nervous system and its membranes and (2) changes in the nervous tissue itself consisting of lesions secondary to the vascular changes and of alteration of the morphological characters of the nerve cells.

(1) The most prominent vascular changes in all the three cases was the great hyperaemia of all the parts of the brain and the presence of thrombosed vessels whether the thrombi were primary or secondary to embolism it is impossible to say, but no emboli were seen in sections which were studied. Patches of softening were seen in one case and were evidently the result of vascular occlusion.

There was however, more constantly in all the three cases evidence of inflammatory reaction by the presence of perivascular small round cellular infiltration and serous exudation around the vessels.

Changes in the Nerve Cells.
The changes are those described by the term chromatolysis, they consist of partial solution of the stainable substance of the cell and slight alteration in the appearance and position of the nucleii. In some of the cortical cells in case III the changes
changes had even advanced further and both the bodies and the nuclei of the cells stained deeply and homogenously as if they had undergone coagulation necrosis. These changes represent a vital action of the cells to some abnormal influence.

They go on to say that these changes are also observable in over-activity of the cells and they allow that in the motor area, part of this condition may be so explained but the changes were so widespread and occurred in areas where there were no grounds for supposing that excessive activity was present.

While it is important to carefully study fatal cases of Chorea, post-mortem, in order if possible to discover the cause of the disease it has to be recognised that as Chorea in regard to the manifestations of cerebral disturbances is usually quickly recovered from, the cerebral lesions cannot be of a lasting or very deep character.

**Bacteriology.**

Dana (New York Medical Journal August 19, 1883) describes a diplococcus which he observed only in the deep layer of the pia and the superficial part of the cortex.

Previous to this Donkin (Medical Times and Gazette 1884 II) had described rod-like bodies.

Pianese (Nata infetiva della corte del Napoli Sydenham 1893) obtained from the nervous system of the subjects of chorea a bacillus which he has cultivated on artificial media and which when inoculated into animals produced convulsions. He has further obtained pure cultures of the same organism from /
from the central nervous system of animals inoculated.

Apart (Société de Biologie Jan. 20th 1893. vol V n. 123) described a diplococcus which resembles the organism of Dana and which he distinctly states was, he thought, identical with the diplococcus of Triboulet and Theroloy in aggravated Scarlet Fever.

Mori (Gaz. dagli Osp. et delle clin. August 22nd 1897) reported the discovery of a Lanceolate encapsulated diplococcus extremely pathogenic to guinea pigs.

Maraglino (Centralb. f. innere Med. XX n. 439 1899) considers that the microbe of Pianese is probably the bacillus coli and considers that a coccus also found by Pianese was probably the more potent agent as he considers the staphylococcus the chief source of infection in Chorea. Staphylococci he states have been found twice as often as all other organisms put together.

Fornaco (Riforma Medica No 74 1901) states that while the importance of pyogenic micro-organisms in relation to Chorea is generally recognised, recent bacteriological examinations of the spinal fluid of Chorea patients go to show that the relationship is closer than is usually supposed.

He states he discovered staphylococci in the cerebro-spinal fluid of his own cases and in a third case of erysipelas which was followed by Chorea of a severe type, streptococci were demonstrated in the fluid and also in the blood and urine.

P.A. Pobobrazhensky (Medizinskoe Obozreniiti vol LVIII Nov. 21 1902) states that Chorea not unusually is an infective disease. Not rarely either is it a streptococcal
streptococcal infection. He considers that polyvalent anti-streptococcal serum is a rational remedy for the treatment of the disease. He also urges that a bacteriological examination of the blood should be made in all cases of Chorea.

Westphall and Wasserman and Walkoff in 1899 published their important case of a child who died from rheumatic pericarditis and Chorea from whose pericardial and cerebro-spinal fluid was isolated a diplococcus which produced arthritis in a series of 30 rabbits. These observers maintained that this diplococcus was the cause of acute articular rheumatism. Cultures were made from the heart's blood, the pericardial fluid, mitral valves, spleen and brain. The diplococcus resembles that described by Von Leyden in Rheumatic Vulvitis. The organism appeared as a diplococcus in the tissues but grew in culture as a staphylococcus. The number of micro-organisms in the tissue of the patient was very small. They would not grow on ordinary media but on a highly alkaline one. Incubation period was from 3 - 10 days. All the tissues of the joint were inflamed and in the fluid there was a considerable number of leucocytes. In addition there was exudation in the tendon sheaths and bursae. The micro-organism was found in the arthritic exudation and cultures from the animals reproduced the disease in other animals.

Poynton and Peine (Lancet Sept. 1900) recorded the occurrence of choreiform movements in a rabbit as the result of the intravenous inoculation of the diplococcus and in 1901 they verified the occurrence of a diplococcus in the cerebro-spinal fluid of a child.
child who had died from severe rheumatism with Chorea.

Fritz Meyer in 1901 met with a diplococcus which produced twitching movements in six rabbits.

Beaton and Walker (British Medical Journal Vol I. 1903 p. 257) gives a description of Poynton and Paine's Diplococcus rheumaticus. They discovered it in 15 cases: in 8 cases of acute rheumatism, in 3 cases of Chorea and in 4 cases of Acute Endocarditis. They consider it the organism described by Triboulet, Wasserman and Poynton and Paine.

In the majority of cases the organism was got in pure culture.

Microscopical and Staining Characters.
It is a small micrococcus arranged in pairs and short chains in films from ordinary cultures.
(1) It retains the stain by Gram. -- in the Weigert modifications of the method.
(2) It stains well with ordinary dyes.
(3) It is not encapsulated.
(4) It is not agglutinated either by the serum of a convalescent rabbit or man.

Poynton and Paine call it a diplococcus but German workers call it a streptococcus.

The pairing is usually very marked in recent cultures and more especially in original cultivations and in body fluids and agar-agar and chains may be quite infrequent in the films. Under cultivation the chains become more marked and cultures grown on milk or in highly alkaline bouillon frequently present long chains which in some cases may extend across one or more fields of the microscope with one twelfth oil immersion objective. When growing in the tissues they /
they usually appear in pairs, but sections not infrequently show chains of half a dozen individuals or more.

There is nothing in the ordinary cultural characters of the organism which distinguishes it from any other streptococcus and for its differentiation, animal experiments are therefore necessary.

Varmorek has found that if a fluid medium in which streptococci of human origin have been grown be filtered free from micro-organisms and again employed as a culture medium that streptococci of human origin are entirely unable to multiply on it.

Beaton and Walker using such a media in two cases got luxuriant growths of the micrococcus rheumaticus. They state that if this is confirmed it will go far to prove that it is specifically different from streptococci.

(Beatte (Journal of Exp. Medicine 1905 and Journal of Medical Research 1905 – 1906) does not support this.)

Beaton and Walker could not report the occurrence of Chorea from inoculations into the blood stream of young rabbits. Their cultures from Chorea in man only gave them acute rheumatism in rabbits.

In one occasion they observed violent spasmodic movements of the head upon the second day after inoculation in an animal which had developed a mono-arthritis within 24 hours of its inoculation and died on the third day with beady vegetations on the mitral valve, and in another animal which suffered from poly-arthritis and eventually recovered completely, there was for several days paralysis of both hind limbs.
Beattie (Journal of Medical Research XIV 1905 - 1906 page 533) gives the result of his experiments to differentiate between the micrococcus rheumaticus and other streptococci as regards the differences in the morbid anatomy of rabbits into which he inoculated these organisms.

He states that the injection of the micrococcus rheumaticus produces polyarthritis and synovitis, valvitis, and pericarditis without any suppurative change, and in this he considers is the essential difference between it and streptococci.

In one rabbit into which he intravenously inoculated four blood agar tubes Chorea developed without arthritis. In speaking of this case he says "Few observers have described this condition. In my own case the choreiform movements were very definite though the movements were all in one direction. They were quite different from the convulsive movements which I have described in one animal before death, and which Cole has also described (Journal of Infectious Diseases 1904)."

He also states that in uncomplicated cases of acute Rheumatism the organism is not usually found in the blood or joint exudates. In most of the ordinary cases the organisms appear to be localised and probably produce their toxin and unless these localised areas are examined there is no possibility of getting cultivations.

Muir and Ritchie (Bacteriology 1907 p. 193 and 194) give an excellent resume of this subject. The state that the organism of rheumatism and Chorea is sometimes spoken of as a diplococcus but it is best described as a streptococcus growing in short chains.
chains; in the tissues however, it usually occurs in pairs. It is rather smaller than the streptococcus pyogenes, although it can be stained by Gram's method, it loses the colour more readily than the streptococcus. In the various media it produces a large amount of acid and usually clots milk after incubation for two days; on blood agar it alters the haemoglobin to a brownish colour. Its growth on media generally is more luxuriant than that of the streptococcus and it grows well on gelatin at 30°C.

Intravenous injection of pure cultures in rabbits often produced polyarthritis and synovitis, vulvitis and pericarditis without any suppurative change, (lesions which it is stated are not produced by ordinary streptococci — Beattie). In one or two instances chorioform movements have been observed after injection. The organism is most easily obtained from the substance of inflamed synovial membrane where it is invading the tissues, a part where there is special congestion should be selected as being most likely to give positive results. It is only occasionally to be obtained from fluid in joints. It has also been cultivated from the blood in rheumatic fever, from the vegetations on heart valves and other acute lesions. In many cases however cultures from the blood give negative results. Poynton and Paine cultivated it from the cerebro-spinal fluid in three cases where Chorea was present and also detected it in the membranes of the brain. They consider that this disease is probably a slight meningo-myelitis produced by the organism.

Andrews finds that the organism has the same cultural
cultural characters and fermentative effects as the streptococcus faecalis, a common inhabitant of the intestine. Even however if the two organisms were the same it might well be possible that rheumatic fever is due to an infection of the tissues by this variety of streptococcus. The clinical data, in fact, rather point to Rheumatic Fever being due to an infection by some organism frequently present in the body, brought about by some state of predisposition or acquired susceptibility.
Embolic Theory.

Kirke in 1852 (New Sydenham Society Textbook 1863) noticed the constant presence of vegetations on the valves of the heart in fatal Chorea and suggested that the disease might be caused by the irritation produced on the nerve centres by fine molecular parts of fibrin which were set free from the inflamed endocardium and carried by the blood currents into the capillaries of these cavities. "The frequent existence of a cardiac murmur during Chorea and the presence of warty vegetations on the valves of the heart, so commonly found in fatal cases of the disease, are in favour of such views."

Hughlings Jackson 1864 (London Hospital Reports vol I p. 459) wrote as follows -- "I think from many circumstances that embolism is a frequent cause of Chorea. I do not say plugging of the trunk of the middle cerebral but probably of some of its rami as which supply convolutions near to the corpus striatum."

Sir William Broadbent in 1885 (British Medical Journal 1889) arrived at much the same conclusion as Jackson and also laid stress upon the importance of alterations in the blood stream as a predisposing cause.

M. W. Tuckwell (British and Foreign Medical and Chirurgical Review p. 506) in 1887 published a case of Chorea where the post-mortem showed an extensive superficial softening of the convolutions due to embolism and later in the St. Bartholomew's Hospital Reports vol. V. 1889) recorded another fatal case in which there was embolism of the right posterior cerebral...
cerebral artery.

Hughlings Jackson (British Medical Journal 1876 Dec. 23rd) says — "I suppose that the excessive movements recurring either in Chorea or Epilepsy, or epileptiform seizures are produced by discharges of the grey matter which, except for great instability from over-nutrition, (not better nutrition) is healthy. We cannot expect to discover with our present means of research the alterations in grey matter on which excessive discharges depend." He goes on further to say "that in his opinion the direct pathological state leading to instability of grey matter producing Chorea movements, is increased quantity of blood in the periphery of the capillary district embolised."

He also asserts that the frequent one-sided nature of the movements of the limbs and their often dying out into definite hemiplegia, as pointing to disease at or near the corpus striatum (London Hospital Reports 1866).

The elaborateness of the movements is a strong warrant for the inference that the changes causing them must be seated in the brain and not in the spinal cord and he also thinks it most probable that the convolutions are the parts diseased.

Dr Dickinson in 1876 (Medical Chirurgical Transactions vol LIX p. 15) says "We see in Chorea a widely distributed hyperaemia of the nervous system, not due to any mechanical mishap but produced by causes mainly of two kinds — one a morbid probably humeral influence which may affect the nervous centres as it affects other organs and tissues; the other irritation in some mode usually mental but sometimes what is called reflex which especially belongs /
belongs to and disturbs the nervous centres and affects persons differently according to the inherent mobility of their nature.

The course of this disease is sufficiently traced in hyperaemia and its results."

He failed to discover emboli and in fact stated that the changes repeated themselves in certain portions and the equality with which they affected both sides of the body is conclusive evidence against this hypothesis. In noticing these observations Hughlings Jackson suggests that the local parietal softenings account for the Choreal paresis while the cerebral changes are not very unlike those producible by emboli, regard being had to size and shape of the arteries plugged.

Dr. Bastian (Remarks on the Pathology of Chorea B.M.J. Jan. 20th 1877) supports in some respects the conclusions of Dickinson but agrees with Hughlings Jackson in attributing chorea to a disturbed nutrition in the Corpora Striata and adjacent parts. In place of embolism however he puts thrombosis. He states his case as follows:— "I look (certain rare cases excepted) to an altered and often anaemic blood state as its predisposing cause in individuals of a certain age and nervous temperament. Secondly I look to the irritation in such individuals of a disturbed nutrition in the corpora striata and adjacent parts of the brain, tending to issue and often actually issuing in what, for want of any more appropriate term may be called sub-acute inflammation of these centres, often characterised in part by the production of multiple minute thromboses."

Hughlings /
Hughlings Jackson in referring to the work of Bastian who found plugging due to thrombosis writes:—

"It may be, I would admit here that the hypothesis of embolism will be displaced by the hypothesis of thrombosis as an explanation of many cases of Chorea.

Further he says "Having regard to the great elaborateness of the movements of Chorea I still think it is most probable that the convolutions are the parts diseased."

Ogle (British and Foreign Medical Chirurgical Review Jan. 1869) also supports the view that there is an altered blood condition and in this way he accounts for the fibrinous deposits on the heart valves in Chorea.

In favour of the embolic theory it has been stated that the disease is often one-sided, most often right-sided, as in the case of hemiplegia due to embolism and due it is thought the straighter course the arterial passage offers to the transit of emboli to the left side of the brain than to the right.

In capillary embolism also lies a rational explanation of psychical changes which occur so often in the disease. In several cases, the smaller vessels of the brain have been actually found plugged by competent observers.

Against this theory however it has been argued that the preponderance of a right-sided affection has been denied by many. Sturges gives the seat of onset as 36 for each side, and Pye Smith (Guy's Hospital Reports series III vol XIX) gives 15 right and 13 left, of laterally limited hemichoreas in 33 cases.

Moreover /
Moreover a strict limitation of the disease is undoubtedly rare and even if one side is more markedly affected, still other parts are usually found also affected to a lesser extent. In the majority of cases any affection of the heart does not as a rule develop for several weeks after the onset of the disease and thus, since the endocardial changes on the heart valves are the seat of origin of the emboli, the embolic theory is difficult of acceptance.

Again Chorea is very uncommon in adults while embolism is common enough.

It cannot be doubted that acute endocarditis from whatever cause arising, leads not infrequently to capillary embolism, though not it would appear to Chorea.

In very many cases no signs of embolism were discovered on post mortem examination.

Mention may also be made here of the experiments of Angel Money (British Medical Journal vol. III p.99) in his paper "Capillary embolism of the Brain and Spinal Cord experimentally studied." He conducted experiments on thirteen monkeys. In all he injected artificial emboli into the common carotid artery in the direction of the blood stream.

The post-mortem examinations showed that chronic inflammatory changes were set up by the starch granules, lodged in the capillaries and arterioles, and that the starch granules became broken up and ingested by the large inflammatory corpuscles. His conclusions regarding the embolic theory are as follows:- My general impression after a study of Chorea clinically and as well as of the facts of experimentation, is not in favour of the views that embolism is the /
the cause of Chorea. On the other hand I do not agree with Dr Sturges in thinking that Chorea is a purely functional disease. My tendency is to regard a large number of cases of Chorea as due to a rheumatic localisation in the motor nervous centres."

He finishes up with the following propositions.

"(1) Human Chorea is generally and mainly cerebral in origin, though a few cases may be purely spinal source.

(2) The commonest lesion causing Chorea is a rheumatic affection of the circumvascular connective tissue of the motor apparatus of the brain and cord.

(3) Embolism may be a cause of Chorea, in rare instances.

(4) Chorea may be set up by fright when the lesion is probably a molecular disturbance of the cerebro-spinal motor apparatus."

While Angel Money's experimental research does not by any means disprove the embolic theory, still the arguments for this theory seem to be altogether outweighed by the arguments against.
Chorea -- A Functional Disease.

Sturges is the exponent of this view and gives his reasons for supporting it in a very able and clear manner in his monograph on "Chorea" published in 1331. He urges that the movements of Chorea in their method and mode of distribution over the body together with the rarity of any spasm or paralysis give no grounds whatever for assuming the existence of any definite morbid change.

In their variety and half voluntary character the movements would seem to have their origin in the cerebral convolutions but not as he says in "convolutions diseased or disturbed in any way that anatomy can discern."

He points out how the limbs become subject to Chorea in the order of their use as intelligent instruments and not as they would do upon any assumption of injury or irritation of a motor centre. Thus untaught muscles, and such as never have been employed as the agents of intelligence, never suffer from Chorea while further, the "more complex the muscular employment, the higher the place so to speak in the intellectual scale of any particular limb or group of muscles, the greater the liability in this respect." The withdrawal or weakness of the will power over the specially educated muscles will produce Chorea. The muscles from some cause or other are thrown back in their education, which renders them restless and unruly and the will power no longer is sufficient to act as an efficient controlling power.

In further support of this he states that the older the patient is the more are the muscles educated and the greater therefore is the distortion and spasm, and
and he also calls attention to the fact that the disease very often commences in the hand of a child which is most obviously taxed in the modern requirements of education.

This is also seen when the particular event or catastrophe which caused the Chorea happens to concern a certain group of muscles. A boy of 8 years of age gets Chorea in the hand in which he was holding the reins of a runaway horse. A girl of 11 years stooping to pick up, as she thought, her brother’s cap, found to her great alarm that she had seized a dead rat. She became Chorea in the same hand.

The muscular over-movement except for its exaggeration and disregard of will is the same as the natural movement.

The mental symptoms of Chorea become more marked as the patient approaches puberty and these symptoms are usually absent in young children and this he says is explained by the development of the nervous centres and is comparable to Hysteria.

He also compares this to the expressions of grief in a child who endeavouring to restrain the usual outward manifestations of grief suffers acutely mentally until "the violent convulsion which we call sobbing together with free movements of the limbs occur and then comes mental serenity."

Considering next the course of the disease he finds further support to his contention in the variability of intensity of the disease and the continual ups and downs which are so marked a feature of the disease (for which he gave no active medical treatment) and the fact that any mental shock may at once interrupt what looked like a rapid recovery and reduce /
reduce him to a condition even worse than his previous one. Recovery depends on the gradual strengthening of the will power.

As regards the occurrence of Chorea after a fright he considers that the interval which is stated to exist, is more apparent than real as the disease is usually of gradual onset and is not noticed until it has progressed to a certain extent.

He explains the absence of the disease in adult life by saying that the will power has become so strengthened and the muscular actions so defined by habit, that the patient is able to withstand any such mental shock or fright.

"Again what hypothesis" he says "except a functional one can be made to provide for a disorder which is recurrent as Chorea is, recovering perfectly time after time and at last when adult life is reached finally disappearing: the signal for its departure being nothing else in the world but the departure of childish ways and the substitution of new modes of response to nervous impressions."

The Chorea of pregnancy he attributes to elevation of the emotional element which pregnancy produces and the will power once more becomes inadequate and Chorea in old age is apt to persist because the childish mind persists and the extra will power necessary for a natural recovery never comes.

Brain lesions of whatever kind do not as a rule exhibit Chorea at any part of their course and conversely Chorea implies no appreciable liability to cerebral disease. He admits however that there are reasons to believe that this functional disorder may of itself in some rare instances so affect the nerve centres /
centres as eventually to produce definite structural
changes in other parts of the body or in the nervous
centre itself.

He finishes by saying:—
"The condition of the living patient supplies all the
material for analysis we are ever likely to possess,
and this in so absolute a sense that the event of
death, which alone could give access to more, would
at the same time impart such an element of exception
as to forbid our drawing conclusions from this par-
ticular case.

The symptoms are not those of disease but of
exalted function and their recurrence many times in
the same subject and the same limbs with no ultimate
harm gives conclusive proof that those repeated
visitations are not destructive.

Chorea in a word, for its full development re-
quires the service of a brain and cord structurally
uninjured and so soon as textural injury intrudes, it
will alter its proper form and give room to some one
or other of the recognised symptoms of brain disease."

While the general appearance of the patient and
the character of the movements in respect of their
half voluntariness and the fact that the disease in
some cases at least does develop immediately after a
severe fright lends some support to the theory that
the disease is a neurosis and in those cases where
there are no evidences of rheumatic infection or
endocarditis it is difficult to state definitely
that it is not a neurosis. The great stumbling block
to the acceptance of this theory is the frequent close
association of this disease with rheumatism in a
 specially /
specially intimate manner and the occurrence of endocarditis which as Osler has shown may be recovered from at the time but the inflammation in the heart valves often leads to subsequent shrinkage and consequent insufficiency. The theory that it is a neurosis fails altogether to account for the definite organic heart lesions and joint complications which, from very early in last century were noted as common complications or even signs of the disease. The occurrence also at the beginning of the attack in some cases of an elevated temperature leads one also away from the theory that it is a neurosis to the theory that it is in reality a blood infection.

**Infected Theory.**

While many writers both from a clinical and bacteriological point of view have stated their belief that Chorea was due to a blood infection, it was left mainly to Payton and Paine in their researches in the Lancet September 1900 to place the infective theory of both Rheumatic Fever and Chorea on a definite basis and to show that one and the same bacillus could be obtained in pure culture in both diseases and could produce, when cultures of it were inoculated into rabbits, manifestations of rheumatic disease, — arthritis, pericarditis and endocarditis, and choreiform movements. We have seen how since 1310 in this country the close association of Chorea and Rheumatism has been noted by many clinical observers and how Chorea in its onset and characters is comparable to other infective diseases. Thus from the assumption that Chorea and Acute Rheumatism were closely /
closely associated and from analogy Chorea has for some considerable time before the researches of Poynton and Paine been considered as very likely to be explained by an infective theory which would include both diseases.

Barkley (John Hopkins Hospital Reports August 1931) hints at the possibility of an infectious origin for Chorea.

He reports a case of Chorea insaniens in a woman aged 27 who had had two attacks of rheumatism and with the second had had delirium and irregular movements of the limbs. The autopsy showed an acute endocarditis, abscess of the parotid, and catarrhal pneumonia of both lungs. No special germ could however be discovered. He states that Chorea is a general systemic infection acting with greatest intensity upon the vascular system and the leptomeninges; its cause is to be sought for in a special bacillus.

Dane (New York Medical Journal August 19th 1933) records the autopsy of a case in which microscopical examination showed a conspicuous chronic lepto-meningitis involving the vertex of the brain: a proliferating process without exudation or much cell infiltration. In the superficial layer of the cortex he found cellular infiltration with degenerative changes. At this point a diplococcus was found. The microorganisms were observed only in the deep layer of the pia and the superficial part of the cortex.

Churtin (Medical News Dec. 4th 1937) gives a study of 100 cases. He considers that the toxin may be a glycosin for which reasons micro-organisms will
will not be found in the blood.

Mai (Gaz. Dayli. Osra. et delle Clin. August 22nd 1917) reports the discovery of a lanceolate encapsulated diplococcus extremely pathogenic to guinea pigs in which it determines a haemorrhagic haemorrhagia hyperaemia with diminished fibrin and no oedema. The histological examination showed that the effects were more toxic than septic and also showed an elective action on the vessels. This he says supports the theory that Chorea is a syndrome determined by some infective or toxic agent on a soil prepared by an inheritance of neurotic and arthritic tendencies.

Maragliano (Centralb. d'inner Med. XX 1917) states the conclusions he has come to as follows:

"(1) Rheumatic Chorea is infective and depends on the action of toxins of micro-organisms on the nervous system.

(2) Staphylococci are the chief source of infection in that they have been found twice as often as all other organisms put together.

Fornacq (Riforma Medica No 74 1901) found staphylococci in the cerebro-spinal fluid of two of his own cases and in a third case where there was previous erysipelas he got streptococci in the cerebro-spinal fluid, in the blood and in the urine.

The statistics of Triboulet he mentions, which show that a third of all the cases of Chorea give a history of antecedent febrile attack of which the most common are Scarlatina, Measles and Erysipelas. He considers that the cerebro-spinal fluid should be examined in all cases of Chorea.

P. A. Preobrazhensky (Medizinskoje Obozreni)
vol. LVIII Nov. 21st 1902) states that Chorea not unusually is an infective disease, not rarely either is it a streptococcal infection.

Avert (Societe de Biologie Jan. 20th 1393 vol. 5 p. 128) described a diplococcus which he states was identical with that of Triboulet and Théroloix in aggravated rheumatic fever.

The diplococcus was obtained from the blood of a Choreic patient but (apart from local induration) inoculation with the diplococcus into a guinea pig was negative.

Westphall, Wasserman and Walkoff in 1393 ("Über den Infektiosen character und den Zusammenhang von Acuten Gelenkrheumatismus und Chorea") give a report on a severe case of Chorea following acute rheumatism with violent delirium, hyperpyrexia collapse and death.

They isolated a diplococcus resembling morphologically that found by Von Leyden in Rheumatic Vulvitis and in 80 rabbits it produced fever and multiple arthritis. They go on to state that the organism appeared as a diplococcus in the tissues but grew as a streptococcus in culture.

Paynton and Paine (Lancet Vol II 1900) after reviewing the work of Dana, Avert, Westphall and Wasserman and Walkoff, and Triboulet, give the results of their experiments with what they consider the same diplococcus. They record a case which they called Chorea in a rabbit due to the intra-venous injection of diplococci into it. This rabbit was extremely nervous and in addition manifested sudden twitching movements of the limbs and face. These movements were
were definite but not violent and there was about them that peculiar sudden involuntary character which is characteristic of slight Chorea. In the Lancet 1901 vol. I p. 1261 they give notes on the examination of the brain of the rabbit so affected. They discovered diplococci in the pia mater and endothelial cells of the blood capillaries.

The pia mater in places showed some slight swelling of its connective tissue and cell exudation. The organisms were also present in the walls of some of the blood vessels of the pia mater.

On three occasions they isolated diplococci in the blood stream in acute rheumatic pericarditis. This is a proof that they circulate in the blood stream and therefore presumably in the cerebral vessels and possibly escape into the cerebral tissues in acute rheumatism with pericarditis. They thus are led to the conclusion that the onset of Chorea is associated with the presence of diplococci in the brain and perhaps the pia mater, as well as with the presence of toxins produced by these diplococci.

They then describe the changes locally seen in acute rheumatism in the heart valves, the myocardium, pericardium and nodules, and state from this with due consideration for the special anatomical characteristics of the brain, the changes they would expect to find in the brain in Chorea. There is proliferation of connective tissue cells; where there are blood vessels there is hyperaemia and perhaps small haemorrhages and fibrino-cellular exudation. Cardial muscle fibres lose their striation and there may be well marked fatty changes. In more chronic
and less severe cases there is sclerosis. The diplococci in the valves, pericardium and nodules are rapidly destroyed at the sites of the lesions, but probably their toxins may remain after the diplococci disappear. Thus, they state, the lesions we would expect to find in Rheumatic Chorea would be as follows:

In very acute cases we should expect hyperaemia and possibly small haemorrhages and foci of necrosis in the cerebral tissues, also minute emboli or thrombosis in the terminal capillaries. In less acute cases, in the region of minute blood capillaries we should expect cell exudation and proliferation and also degeneration of some cells. In chronic cases we should expect perivascular fibrosis and small areas of sclerosis. There would probably be considerable difficulty in demonstrating the organisms especially in the more chronic cases. In other cases they consider that the organisms may be plentiful enough to block the most minute blood capillaries or having got into the tissues by setting up an inflammatory action there to occlude these vessels by pressure from without.

Dr. B. Lass (British Medical Journal August 29th 1903 p. 443) writes an article on the pathology of Chorea supporting the infective rheumatic theory. "The most obvious fact in Chorea" he says, "is dis-orderly muscular movement, spasmodic, clonic, ir-regular, involuntary, with imperfect control and coordination." He goes on to show that the muscles over which we have most voluntary power are most affected: those of the face, tongue, and those of the hands and arms; respiration over which we have more /
more control than over the action of the heart is more affected than the heart action. The heart is sometimes irregular in Chorea but it may act quite steadily while the respiration is quite irregular. Chorea movements usually subside during sleep and reappear when the patient awakes; and Chorea movements are often worse on one side than on the other. From these facts he argues that it is the cerebral cortex that is affected and especially the cerebral cortex of the Rolandic area from which area originate most of the muscular movements which are especially under control.

He writes that when a child is asked to speak, the choreic movements in the face and limbs become exaggerated before the child does so. This is due to the overflow of the outgoing impulse into the immediately adjacent areas. He points out how one side of the cortex is often more affected than the other, and then speaks of paresis which is apt to supervene when the irritability subsides.

These symptoms do not indicate in his opinion the total brain affection. Speech may be lost for several weeks or months and this he considers must be due to an affection of the speech centre itself, quite apart from the muscular articulating mechanism. He next discusses the psychical condition; the crying without any obvious cause, the excitement, hallucinations of vision, and even mania, or if these be absent, the dull vacant listlessness in a patient who previously was bright and intelligent. Sensibility is also probably lowered.

Thus he says:— "it is clear that in Chorea much more
more than the Rolandic area is affected and it is probable that the whole brain suffers more or less. If there is any cord affection it is overshadowed by the cerebral condition, unless increased knee jerks and a tendency to prolonged extension when the patellar tendon is struck point to this.

In this connection he also states that Sir William Gowers has described optic neuritis.

He states his conclusion as follows:

"In Chorea then there seems to be a disorder of the whole cerebral cortex, probably the whole brain, possibly the nervous system in general. The disorder is not a destructive one: it usually ends in complete recovery. The pathological changes therefore, if organic, are of slight intensity and it seems probable that the morbid state may be largely due to a toxæmia. Yet the symptoms may be definitely localised and this fact suggests that the cause cannot simply be a toxæmia. There must be something local as well."

He next describes the pathological changes reported by Reicherdt of Chemnitz (Deutsches Archiv für klinische Medicin 1902 p. 504) which have already been quoted on page 7.1.

He calls attention to the clinical evidence of the association of Chorea with a morbid blood state such as rheumatism, in the form of tonsillitis, arthritis, erythema, nodules, cardiac dilatation, endocarditis, pericarditis and pleurisy. The most frequent of these is cardiac dilatation for in Chorea as in rheumatism the left ventricle is usually enlarged, extending beyond the left nipple line often as much as a finger breadth, and the first sound being feeble.
feeble.

In almost all fatal Choreas there is endocarditis of the mitral valve and careful examination of the patient often discloses nodules over joints, on hands or feet, in association with tendons, on the occiput and vertebral spinous processes. He records how a nodule aseptically excised 2 hours after death and immersed in a suitable medium and cultivated for 43 hours gave an exuberant growth of diplococci without showing any other organism.

A rabbit inoculated with a culture of diplococcus developed symptoms remarkably like Chorea, the rabbit was killed and diplococci were demonstrated in the lymphatic sheath of the vessels of the pia mater and in the endothelial cells of the blood capillaries, and even penetrating the cortex of the brain.

Dana, Apert, Wasserman etc. have isolated a diplococcus from the brain in fatal cases of Chorea.

He finishes by saying: "Are we then to say that Chorea is cerebral rheumatism? — Yes, if we add in the majority of cases."

Beaton and Walker (British Medical Journal Vol I p. 237 1903) give 15 cases where they discovered the diplococcus of Poynton and Paine.

They observed it in the following cases:—

In 3 cases of Acute Rheumatism, in 3 cases of Chorea, and in 4 cases of Acute Endocarditis in rheumatic subjects. They support the observations of Triboulet, Wassermann and Poynton and Paine. In the majority of cases the organism was obtained in pure culture. Their description of organism and its cultural
cultural characters have already been given on page 46.

They cannot describe a true Chorea as resulting from their inoculations even although young rabbits were used. Their cultures even from Chorea in man have only so far given acute rheumatism in animals. On one occasion they got violent spasmodic movements of the head upon the second day after inoculation in an animal which had developed mono-arthritis within twenty-four hours and which died on the third day with beady vegetations on the mitral valve, and in another animal which suffered from polyarthritis and eventually recovered completely, there was for several days paralysis of both hind limbs.

Beattie (Journal of Medical Research XIV 1905 and 1906 p. 399) gives the results of his experiments whereby he holds he has differentiated between the micrococcus rheumaticus and streptococci from the different actions after inoculation into rabbits, of these organisms.

In one rabbit into which he intravenously inoculated four blood agar tubes, Chorea developed without arthritis.

In speaking of this case he says:— "Few observers have described the condition. In my own case the choreiform movements were very definite though the movements were all in one direction. They were quite different from the convulsive movements which I have described in one animal before death, and which Cole has also described."

He also states that in uncomplicated cases of acute Rheuma asis the organism is not usually found in the blood and joint exudates. In most of the ordinary cases /
cases, the organisms appear to be localised and probably produce their results by a toxin and unless these localised areas are examined there is no possibility of getting cultivations.

Poynton and Paine. (Lancet Dec. 1905 p. 1752)
They refer to their view stated in May 1901 that Rheumatic Chorea was the result of a diplococcus infection and state that they use the term "rheumatic" advisedly, not being prepared to maintain that all Chorea is rheumatic, because it may be probable that other organisms especially of the streptococcal group can sometimes produce the disease.

As regards the etiology of Fright, they state they have examined the history of 250 cases under twelve years of age and support D. B. Lees' opinion that fright as an exciting cause has been greatly exaggerated, but they believe that as a predisposing cause it is one of some importance. They state "that they believe implicitly in the teaching of those who have held that the great majority of cases of Chorea are rheumatic and rheumatic in the sense that the Chorea is an actual manifestation of the disease. Here again we would submit that the clinical evidence in favour of this view is overwhelming."

To prove this, it is essential in the explanation of rheumatic chorea to bring forward evidence of the presence of the diplococcus in the nervous system or general circulation where rheumatism is active and has been the cause of death. Such evidence they state is slowly accumulating and they give a review of the work of Dana, Anart, Triboulet, Wasserman, Poynton and Paine. In 1901 Fritz Meyer with a diplococcus.
diplococcus obtained from Rheumatic Angina faecium produced irregular twitching in six rabbits. They record also Beaton and Walker's work. In 1903 at the International Congress (Transactions of the International Congress of Medicine Section of Pediatrics 1903) Poynton and Paine record a fatal case of Rheumatism with Chorea in which they had isolated the diplococcus from the pericardial fluid and produced not only arthritis as had Wasserman, but also pericarditis and endocarditis.

Beattie in 1904 produced choreiform movements in a rabbit from inoculation with the diplococcus from an arthritic exudation in fatal rheumatism.

They themselves again in two cases of Rheumatic Fever dying with symptoms of Chorea have isolated the diplococcus from the cerebro-spinal fluid. In the second of these cases not only was arthritis but pericarditis and endocarditis were produced when cultures were inoculated into a rabbit.

They next report on a case of Dr. D. B. Lees—"A boy aged 9 years who had suffered from acute Rheumatism at three years of age was admitted to St. Mary's Hospital on Nov. 22nd 1904 suffering from a second attack. Previously to admission he had been ill for 7 weeks with sore throat, multiple arthritis and general Chorea and he only lived two days in Hospital, dying with general pericarditis and no violent Chorea. The post-mortem examination showed a pericardium bound down by recent and old adhesions but there was no endocarditis. The brain was Hyperaemic." Cultures were made. One tube from the heart's blood was negative, the other contained
a pure growth of diplococci; the tube with the
cerebro-spinal fluid also contained a pure growth: a
tube from the pericardial fluid contained the dip-
lococcus and staphylococcus albus which was separated
by plate culture.

One rabbit into which a large dose was intravenous-
cus injected died in 24 hours and diplococci were
present in the blood. The diplococci were again
isolated and cultivated.

The contents of three blood agar tubes from the
original strain were intravenously injected into one
rabbit which developed arthritis on the 3rd day and
died on the 5th from general pericarditis, cardiac
dilatation and mitral endocarditis.

They then ask that, for the moment, the view that
rheumatic chorea is due to the diplococcus should be
accepted and go on to discuss how the results are
produced.

They state that there are two possibilities:
(1) "Chorea may be a result of local lesions compara-
ble to other local rheumatic lesions or
(2) It may be that the delicate nervous tissues are
exceptionally sensitive to the general rheumatic
poisoning."

They then state that they incline to the view that
Chorea is due to many slight local lesions caused by
the escape into the tissue of the diplococci external
to the vessels. The local changes might cause haemor-
rhages, thrombosis, perivascular exudation and in the
chronic cases perivascular fibrosis. The toxins
elaborated would cause the degenerative changes in the
brain tissue. The Pia mater, they consider, be-
cause /
because of its analogy with other serous membranes would especially suffer.

They consider that the changes described by Reichart (already quoted on page 41) support to some extent the view that Chorea is due to such local conditions rather than to a general cerebral toxæmia.

They discovered the diplococci in the neighborhood of the capillary blood vessels. The destruction of these diplococci must be rapid for Chorea is a lesion comparable to rheumatic arthritis in the tendency to complete recovery and it is not easy to find them in acute arthritis.

They lay stress on the fact that they discovered the diplococci in the Pia mater near capillary blood vessels in the rabbit which showed twitching movements. They record that Mayer and Beattie independently confirmed the twitching choreiform movements which they had described in an inoculated rabbit. The search was most tedious and difficult. They summarise their conclusion as follows:

"We believe that eventually rheumatic Chorea will prove to be a local infection of the nervous system, and that most of the symptoms are the result of a slight meningo-encephalitis, possibly meningo-myelitis. Our reasons for this belief are as follows.

(1) We have isolated and cultivated the diplococcus from the cerebro-spinal fluid in a few cases of fatal rheumatism, in three of which there was Chorea at the time of death.

(2) We have produced twitching movements, arthritis, endocarditis and pericarditis by intravenous injection /
injection of the diplococcus into rabbits.

3. We have demonstrated the presence of diplococci three times in the cerebral pia mater and once in the brain from cases of Chorea.

4. We have demonstrated them in the brain and pia mater of the rabbit that had shown the twitching movements.

Poynton and Holmes (Lancet Vol. II p. 182) in an article on the pathology of Chorea give the results of post-mortem examinations in three cases of fatal Chorea. The morbid anatomy has already been described on page 42. They discuss the question whether it is necessary for the production of the disease that there should be an infection of the cerebral cortex and its membranes by the diplococci or whether the disease may be caused by toxins generated at some distance from the seat of the cerebral disturbance. Diplococci were found in the brain and the occurrence of hemichorea suggests such a local infection with diplococci, but they state that Leignot-Levasstine has shown that such changes may be produced in the brain cell by a tubercular toxemia without the presence of the bacteria in the brain. They consider that the palsy of Chorea is due to a toxic action and not due to the exhaustion of the nerve cells. It is quite conceivable that a toxin in a certain dose may produce exaltation while in other cases in a different dose or acting more rapidly may paralyse the cell function.

The changes in the brain and spinal cord are generalised and the fact that the symptoms are mainly referable /
referable to the motor area may indicate that the motor area is specially excitable, but the symptoms may be such as to indicate a universal cerebral affection.

They maintain that Chorea may be the first symptom of rheumatism and consider that the bulk of the evidence goes to show that Chorea is mainly rheumatic. They explain relapses by either a fresh infection or the outburst of a quiescent infection.

Fright they consider acts by lowering the vitality of the nervous centres and thus gives the toxins a chance to act on the nerve cells.

They also call attention to the fact that rheumatic children are very emotional and that a child's nature may be greatly changed after an attack of rheumatism and suggest that these changes may be due to organic changes of even a more delicate nature than those found in Chorea.

In discussing the Chorea of pregnancy they quote Buist's cases, where 45 out of 228 cases gave a history of previous rheumatism. In their own cases 23 out of 37 had a previous history of Chorea; in 16 of the 37 there was a previous history of rheumatism.

They also quote French and Hicks (Practitioner August 1906) who report 23 consecutive cases of Chorea Gravidarum from the records of Guy's Hospital and in 19 of these 23 cases there was a previous history of rheumatism or chorea and 15 of these 19 had suffered from Chorea before pregnancy.

They are convinced that the pathology of Chorea Gravidarum is the same as for Infantile Chorea.

Nettler (American Journal of Medical Science Sept. 1903) takes the view that Chorea is merely a symptom /
p. 73.

symptom and considers that it may be due to a variety of infections such as rheumatism, pneumonia, scarlet fever, gonorrhoea and typhoid fever etc. He records a case in which he considers Chorea was due to congenital syphilis in a boy aged 11 years. This patient however was supposed to have had an attack of rheumatism with some heart affection some years previous.

He quotes other cases in which Chorea was associated with congenital syphilis and considers in such cases that energetic anti-syphilitic treatment is called for.

Stiller (Journal of American Medical Association Feb. 11th 1905) considers that the relation of Chorea to rheumatism has been greatly exaggerated. In most cases he could not find any relationship.

The advocates of the infective theory have established a very strong case and it is the only theory which explains all the manifestations of the disease. They have brought forward very conclusive proof that Chorea is not only an infective disease but that it is caused in at least the great majority of cases by the same infective agent as causes acute rheumatism. On clinical grounds alone the association is very marked not only in the frequent occurrence in the personal history of a previous rheumatism but from the alternation of Chorea and rheumatism in its fullest sense that occurs in some patients. A girl suffers from rheumatism acute or sub-acute for 4 or 5 weeks and then develops Chorea. Then again the organic lesions which accompany Chorea, the pericarditis, tonsillitis...
tonsillitis, endocarditis, arthritis etc. are exactly comparable with the conditions one finds in acute rheumatism.

While it is necessary to admit that many cases of Chorea may occur without having exhibited a previous rheumatism it does not seem difficult from the researches of those who wish to prove the infective theory that this is itself the first symptom of rheumatism. Dr Batten's researches are of special importance showing how many of the cases who did not suffer from rheumatism previous to Chorea were found to suffer therefrom within 6 years after. page 22.

Neither the embolic theory nor the theory that chorea is functional give any explanation of the endocarditis and pericarditis which occur with such frequency in chorea, and which as Osier has shown may produce such serious permanent lesions in the heart.

Osier has shown that the prognosis of Chorea is really like rheumatism dependent largely on the endocardial myocardial and pericardial affections which supervene in the disease. The question of Fright as being a cause of the disease does not really affect the infective theory, at least in the direction of disproving it.

One often finds in enquiring of the mother of the patient who is suffering from chorea as to the possible cause, that fright is not often mentioned, unless the occurrence of it is made a leading question by the doctor, when the mother anxious to relegate the cause to some condition quite outside the child and knowing the importance that has been given to the matter in the causation of the disease will perhaps manage /
manage to remember that some fright did occur although one can often make out further that the child has often suffered before from even greater frights than the one to which the onset of the disease is attributed. It does seem however that a few cases, and only very few are quoted, commence so soon after a fright as to leave no reasonable doubt that it is a factor in the production of the disease. Its action however can be perfectly well explained as producing a profound lowering of the vitality of the nervous centres and giving the diplococci or their toxins if present a chance to act and produce the disease. All clinical observation thus points to Chorea as being an infective disease closely associated with rheumatism.

When the subject is considered from the bacteriological point of view there seems to be ample facts to confirm the opinion resting on long clinical experience.

Poynton and Paine have discovered a diplococcus — micrococcus rheumaticus, which they have shown to be the cause of acute rheumatism. They have discovered the same organism in the brain of patients who have died with chorea. They have been able to cultivate pure growths of the diplococcus from the blood and cerebro-spinal fluid of such patients. The inoculation intravenously into young rabbits has produced arthritis, pericarditis, endocarditis and choreiform movements and the diplococcus has been discovered in the brain of such rabbits as showed the choreiform movements.

They have not proved that chorea may not be caused /
caused by other infective agents but there seems to be every reason for their claiming that in the majority of cases Chorea is a rheumatic manifestation.

Poynton and Paine and later Poynton and Holmes have discussed the mode of action of the diplococcus. As to whether a toxin developed away from the cerebral centres could produce the disease or whether it is necessary that organisms should be present in the brain or pia mater to act locally and at the same time produce their toxin to act over practically all the central nervous system.

They have themselves discovered the diplococcus in the brain and pia mater and when we consider a case such as Risien Russell reports in (Allbutt's System of Medicine vol VII Article on Chorea) where in a boy there was no symptom for several weeks except paresis of the forefinger of the left hand experienced on holding the reins of his pony, there seems to be strong grounds for considering that a small local infection of the brain is necessary. The fact also that Chorea may be, for at least a great part of its course mainly confined to definite parts of the body, affecting other parts only slightly if at all, seems to uphold this.

Poynton and Holmes on the other hand record the observation of Laignet-Levastine who has shown that changes like these in Chorea can be produced in the brain cell by a tubercular toxæmia without the presence of the bacteria in the brain. They explain the difference of intensity of the symptoms in different parts to the greater excitability of certain parts such as the Rolandic area.

However, the diplococcus may act from the morbid anatomy /
anatomy and the symptoms it would seem that the action is very widespread in the brain and perhaps also the spinal cord. They consider that the diplococci live only for a very short time when they get to the brain and the recovery, which usually takes place completely from the cerebral manifestations of Chorea, uphold this contention.

The occurrence of paralytic chorea they explain by either an excessive dose of the diplococcus or its toxin or by rapidity of its action and probably the rapidity of action would best explain these mild cases of paralytic Chorea.

Symptoms of Chorea.

In considering this subject it is well to recognize that except in those few cases where definite choreic symptoms have occurred almost immediately after some exciting cause such as a fright, on careful enquiry, one could elicitate that for a week or so before the onset of the choreic movements or paralytic, the patient showed a departure from ordinary health in various directions. It might be that the patient was sleepless, very irritable, capricious as regards food or on the other hand dull and listless, and in a very considerable number of cases vague pains about the joints, tonsillitis or other such rheumatic condition might be complained of.

The recognition of this stage is important in view of the possibility of aborting an attack especially if the symptoms complained of are of a rheumatic nature.
The most noticeable feature of a case of Chorea of moderate severity is the incessant muscular movement. Usually this is first noticed in either the hand or the face and in some cases it does not extend, at least to any marked extent, beyond one side of the body, and we have what is called a hemi-chorea. It is noticed that the patient is always fidgeting, that he cannot sit still for any length of time. What often first attracts attention is that the patient spills his tea, he has difficulty in using his fork and knife and a girl cannot arrange her hair satisfactorily in the morning before going to school. The movements from being of small compass at first and affecting only one part, gradually increase in range and diversity and often become general all over the body. The facial contortions and grimaces are constantly present, the arms are continually moving, and if the legs are also affected, walking becomes clumsy and difficult. Respiration may also be affected and become spasmodic. The child becomes changed in nature, he is either very emotional, weeps without cause and is very irritable and capricious, or on the other hand is dull and listless. Prof. Wyllie describes the movements as twisting, turning, squirming and grimacing.

The symptoms of Chorea may be classed under the following headings. The first three divisions referring to the muscular movements:

(1) Involuntary spontaneous movements.
(2) Muscular Paresis.
(3) Incoordination (loss of precision in muscular movements).
(4) /
(4) Psychical disturbances.

(1) **Involuntary spontaneous movements.**

They occur irregularly and are of constantly varying nature.

**Face.**

The eyebrows may be drawn up, the eye closed and then rapidly opened. The forehead is thrown into wrinkles, the angles of the mouth drawn downwards and outwards and the lips are pursed. The mouth is opened and rapidly closed. The movements may cease for an instant, and then a purposeless smile or a grimace shows on the face. The tongue is thrust into one cheek and the patient when asked to protrude his tongue does so, perhaps after some delay, with a sharp jerk outwards and then it is suddenly withdrawn with a jack-in-the-box like rapidity. If he keeps his tongue out he does so by putting his teeth on it.

**Speech.**

It may be very little affected, it may be difficult or it may be completely impossible.

In mild cases it is little affected but in the severer cases words are often spoken rapidly and spasmodically, and if a quiet interval in the muscular movements occurs, the child starts out a sentence as quickly as possible. A sentence is often cut short rapidly by a sharp sudden inspiration due to spasm of the respiratory muscles, a word may only be uttered spasmodically in parts. The articulation may /
may become so defective as to make speech quite unintelligible. In some of the paretic cases speech may be absent for weeks. D. B. Lees calls attention to this and argues it is due to affection of the speech centre itself. The speech defect may remain after the symptoms have disappeared, in the form of stuttering. In a case reported at end of thesis where a boy, who previous to Chorea had no impediment in his speech, suffered from it after the muscular movements had disappeared.

Von Ziemssen (Cyclopaedia of Medicine, article Chorea) has observed irregular movements of the vocal cords on laryngoscopic examination.

**Swallowing.**

It is very difficult or impossible in severe cases on account of the lack of the usual sequence of voluntary movements essential to the accomplishment of this act. Not only because of the lack of orderly movements of the jaw and tongue but also of the palate and pharynx.

Tuckwell records a case where the spasm of the muscles of the lower jaw was so powerful as to break several teeth.

**Eye-muscles.**

Squint may be present in severe cases when the degree of spasm of the two eyes is unequal or the two eyes may turn concomitantly with the head preserving the parallelism.

The pupils are frequently dilated and this has been
been observed to be most frequent on the side on which the spasmodic movements have been most severe. The optic discs are usually normal but Sir William Gowers reports optic neuritis as occurring in some cases which supports the infective theory of the disease, as the neuritis disappears without the correction of any error of refraction which may be present.

**Eye Symptoms.**

Langmead (Lancet Jan 13th 1903) mentions several pupillary symptoms present in chorea: hippus — rhythmical oscillatory movements of the iris — is sometimes present: the pupils may not react synchronously, contraction of the pupils to light is ill-sustained, and one may begin to dilate, as if tired of contracting, before the other: the pupils may show varying inequality, now one, now the other, being the larger: the pupil may become eccentric especially when it is contracted. He states that he has not found the hippus, the altered accommodation, or the eccentricity of the pupils in any other general condition except in articular or cardiac rheumatism, a point of interest in view of the common etiology of rheumatism and chorea.

**Optic Neuritis.**

Carpenter (Lancet Nov. 30th 1907) records a case in a girl of 32 years of double optic neuritis: "the temperature rose to 106.4°. The child died and..."
and the post-mortem showed nothing abnormal in the brain.

Upper Limbs.

They are always affected in greater degree than the lower limbs and often the disease is first noticed in the arm or hand; either because of some defect in coordination or because of the involuntary twitching movements. Hughlings Jackson in cases of hemi-chorea, is of the opinion that the right side is more often affected than the left, but according to Pye Smith and Sir William Gowers the left is more often affected than the right. When the upper limbs are affected the movements appear first in the thumb which is more restless than the fingers which are spread out, then pressed together and flexed and extended alternately. The wrists twist and turn about in an irregular fashion: at one moment, the patient may be sitting with his hand on his lap with his palm upwards and the next moment he rapidly pronates it and then as quickly supinates it.

Flexion and extension of the elbow joint also occur and practically every possible movement of the shoulder joint may occur in any order. When the patient endeavours to use a fork and knife or to raise a glass to the lips, these involuntary movements are well seen associated with the lack of coordination which /
which causes him difficulty to reach his mouth. His hand is rapidly jerked from one side to the other and he may spill the most of the contents of the glass before it reaches his mouth, when he may grasp it with his teeth and gulp over the contents as rapidly as possible. This is very well described by Sydenham in his description of the disease.

Lower Limbs.

They are never so much affected as the upper limbs. The movements in the lower limbs are best seen when the child is lying down. The contractions of the leg muscles causing the body to be jerked to one side and then to the other.

There may be considerable difficulty in walking and in severe cases it may become quite impossible. There is no peculiar gait; it is very clumsily executed the legs being thrown about in quite unusual ways, sometimes away far out or the one leg thrown across the other or the leg may be rotated first outwards and then inwards.

The element of paresis perhaps gives the most characteristic gait in Chorea, the dragging of the affected limb.

Trunk Muscles.

Hughlings Jackson states that even in hemi-choreas the muscles of both sides of the trunk are affected. There is twisting and turning of the trunk and shrugging of the shoulders. If the contractions are very severe they may even throw the patient out of bed; violent flexion, extension and lateral
lateral flexion occurring.

Respiration.

William Graves (Journal of the American Medical Association January 30th 1919) writes an article on this subject.

His observations he states as follows:

(1) "Irregular breathing -- This is probably due to two factors.

(a) Lessened amplitude in some respiratory movements and increased amplitude in others giving one the impression that no two respiratory excursions are equal.

(b) Sudden halts -- cogs occurring either during the inspiratory or expiratory phases and most frequently during the latter, but rarely more than one cog occurring in any phase. These do not occur in every respiratory movement but are of frequent occurrence and appear to be due to a sudden arrest of the movement in one phase by the development of an incomplete movement of the opposite phase, but the original movement usually goes on to completion. These cogs render the respiratory movements distinctly jerky and may well be called choreic inspiratory and expiratory cogs.

(2) Now and then a deep sudden inspiration occurs far exceeding in amplitude the usual unequal respiratory movements and is succeeded by a sudden explosive expiratory movement (forced) the whole differing essentially from the sighing respiration by its rapidity in its every phase and by the absence /
absence of a pause at the end of expiration.

(3) Relative decrease in the length of the normal expiratory phase and relative increase in the length of the so-called expiratory pauses; so that the time of an expiratory phase may become equal to or less than the time of an inspiratory phase and distinct pauses are of frequent occurrence at the end of expiration.

(4) Sudden changes from abdominal to costal or from costal to abdominal breathing occur frequently both being absorbed at times in a single respiratory excursion giving one the impression for the moment of a combination — a mixture of abdominal and costal breathing and the frequent impression of incoordination — a dissociation in the respiratory movements. When "pure abdominal breathing" occurs in chorea, movements of the whole chest are reduced to a minimum and when "pure costal breathing" occurs, the upper abdomen remains practically motionless.

(5) The respiratory rate is frequently accelerated but occasionally this may be slower than normal.

Not only may these phenomena be seen in the mildest cases of Chorea Minor, but one may also become aware of them by palpation and auscultation. When these occur there seems to be a sudden reversal in the direction of the onrushing air; at any rate at such moments the respiratory sounds cease abruptly and then begin again."

He mentions that the trunk muscles are involved more or less in all choreas, the degree depending on the severity of the case.
The conclusion which he comes to is that the most of these respiratory phenomena cannot otherwise be explained than by assuming an involvement of the respiratory centre by the agent causing Chorea Minor.

The face most commonly is the first part to show the movements and it is always affected bilaterally (Hughlings Jackson) and the hand is next to follow, the right hand more commonly than the left according to Jackson but the opposite according to Gowers. The movement may then spread to the leg of the same side first, or to the opposite arm. So far as the limbs are concerned we may have a hemi-chorea.

Choreic movements after having ceased on one side and having been confined to that side, may start and persist on the other side — double hemi-chorea — or general chorea may occur.

In severe cases the movements are generally of a wide extent. The muscles contracting so powerfully as to cause injury to various parts of the body, the skin over the bony prominences becoming ulcerated from friction with the bed clothes and the limbs and head may be badly bruised and cut from contact with adjacent objects. Wounds may occur which are liable to infection e.g. abscess, erysipelas and pyaemia. The patient may be thrown out of bed, teeth may be broken and the tongue badly bitten.

Choreic movements cease during sleep except in the most severe cases when in fact sleep is often difficult to obtain. After a good sleep the movements seem quieter for a time but any mental excitement or attempt to perform any definite movements aggravate the spasm but in many of the less severe cases the spasms may /
may be controlled by getting the patient to perform some special acts which require coordination and their attention e.g. writing.

J. W. Russell (Lancet 1399 vol I p. 394) has shown that the patient can for a time inhibit choreic movements even of a very pronounced nature and write it may be even a word of six or seven letters with perfect steadiness. He divides chorea into groups according to the character of the handwriting:

1. The writing gives evidence of the power of control over the movements, incoordination being probably present in many cases.
2. The power of control is practically complete and there is no incoordination.
3. Choreic movements are either absent or very slight and yet much incoordination is revealed by the handwriting.
4. Control over choreic movements is perfect but the handwriting betrays great incoordination.
5. Choreic movements are moderate, but writing is impossible from mental defect.

Character of the Involuntary Movements.

They are very irregular as regards time and nature. No two movements in succession seem to be the same. They begin suddenly and end so. According to Von Ziemssen all the movements are not involuntary, some being voluntary and quickly added to cover the fault. The majority of the movements are complicated movements involving several muscles and often more than one joint. Sturge points this out and states that the older the patient, the more elaborate are the movements.
movements depending upon the further education of the motor coordinating centres in older people. All the movements more closely resemble volitional movements than other involuntary movements do.

Sturgis states that they are exaggerated volitional movements which become so because the will power is lessened. They have also been called quasi-purposive movements.

**Paralysis.**

The muscular power as measured by the dynamometer may be nearly normal in slight cases, usually a decrease of power is noticeable on both sides and this is most marked in those limbs in which we have the greatest choreic movement. More severe paresis not infrequently accompanies or succeeds the appearance of the spasm and such paresis is usually localised in order of frequency as follows brachial paresis, hemi-paresis, para-paresis (Taylor). The paresis begins to be noticeable soon after the choreic movements appear. It may be observed in one limb or upon one side of the body that the choreic movements are becoming less marked and the paresis more noticeable. The arm hangs loose and cannot be raised up to the head, and the patient drags the leg when walking. It is important to note that the paresis never becomes complete paralysis and in almost all cases one can discover slight choreic movements.

Often the mono-paresis or hemi-paresis corresponds with a hemi-chorea, but they are met with equally in general chorea and in some cases the paresis is the most marked feature of the disease. It is also apt to occur in successive attacks but not always affecting /
affecting the same part.

Todd (Medical Times and Gazette 1852) fully describes this type of the disease to which the term Chorea Mollis (limb Chorea) has been applied. Sir William Gowers suggests the same paralytic Chorea.

The paralysis sometimes appear late after the Chorea movements have disappeared and it may persist for a considerable time e.g. Osler's case where wrist drop persisted for two years after the disappearance of Chorea movements in the right arm.

In 23 of Osler's cases in which muscular enfeeblement was noted it was monoplegic in 16, hemiplegic in 4, paraplegic in 6, in 1 case all the four extremities were affected and in 1 both arms without the legs.

Chorea Mollis.

It may be preceded with the usual Chorea movements. More often the paralysis is the first noticeable phenomenon, and it may develop rapidly in a day or two.

There is complete flaccidity of the limbs, the child lies motionless on his back and the head rolls to one side and rests on the ear. It is usual however in a severe case like this on careful examination to find some few small choreic movements either in the face or fingers. The deep reflexes are not consistently absent. Some involuntary movements of a kind is usually present in every muscle though no effective act can be performed.

(1) The paralysis may come on gradually during an attack of Chorea with the usual choreic movements.

(2) It may come on suddenly before any of the choreic movements.
movements have been noticed.

(3) It may develop after the choreic movements have subsided and in this class it may persist for a considerable time.

When the choreic movements and paresis exist together they usually disappear together also.

Risein Russell (Allbutt's System of Medicine vol VII article Chorea) tells of a case of Allbutts where the first symptom of chorea in a boy was weakness in his left forefinger which became noticeable on holding the reins of his pony. The paresis came on gradually. This was absolutely the only symptom for weeks until choreic movements and a transient systolic mitral murmur clinched the diagnosis.

He also tells of another case, admitted to Leeds Hospital under Prof. Allbutt which was deeply paralysed in all four limbs. The state was said to have come on quickly in a few days. The negative features of the case led to a provisional diagnosis of Chorea and within a week twitching appeared and chorea became unmistakably evident.

Incoordination.

This may be the first symptom to attract attention and it may precede the appearance of the spasmodic movements. It is very marked when the spasm is slight and it is most noticeable in the movements of the hand and forearm which lack precision.

The motor centres do not obey the dictates of will and the relaxation of some of the muscles necessary to the completion of some act does not occur e.g. Sir William Gowers' case of a boy when bowling at
at cricket, he held on to the ball too long so that instead of it going towards the wicket it fell at his feet. On the other hand the muscles may relax when not intended to do so and the patient will let fall some object he is carrying, or again when the patient endeavours to pick up some object he will stretch out his hand beyond the object but the involuntary action of the muscles may also have some effect in producing this result.

It is difficult to say whether a certain combination of erratic movements in the attempt to accomplish any act are due to this lack of coordination or to the addition of the usual involuntary movements. This incoordination as has already been shown is also well seen in the case of Respiratory movements.

Muscles.

They may undergo some diminution in size as a part of the general wasting of muscular tissue due to malnutrition and there is a considerable degree of hypotonus in all cases where paresis is a marked sign.

Electrical reactions.

These have been studied more especially by Benedikt and Rosenthal (A Clinical Treatise on the diseases of the Nervous System New York 1879 p. 3) and by Sir William Gowers (Text Book on Diseases of the Nervous System). All these observers agree that there may be some increase of electrical irritability to both faradism and galvanism. Sir William Gowers has observed this increase of irritability to be absent at the commencement, to come on during the course /
course of the disease and to subside with recovery. An altered qualitative mode of response to galvanism has also been described, when instead of cathodal closing contraction occurring with a weaker current than the anodal closing contraction, the latter may occur as readily as the former. Thus we have \[ KOC = ACC \text{ instead of } KOC > ACC. \]

**Sensory Symptoms.**

Sensibility as a rule is unaffected. Headache is common (see B. W. J. Collective Investigation Report). It may precede the appearance of the movements or appear later and is either more or less continuous or paroxysmal. It is usually worse where a considerable degree of anaemia is present.

Pain in the muscles and limbs is rarely complained of but fatigue because of the muscular spasm is common. In severe cases pains like those of neurasthenia may be complained of.

Most of the cases where pain has been described in the limbs have been hemi-choreas and have been called by Wair Mitchell "painful choreas".

Sir William Gowers records a case where there was intermittent neuralgic pains in face, arms and legs for some weeks before the onset of right-sided chorea. It was accompanied by hysterical hemi-anesthesia. Instances have been recorded where there was tenderness along the nerve trunks and tender spots have been described along the spine at the points of emergence of the spinal nerves from the deeper structures (Cartier «Quelques considérations sur la Symptomatologie de la Chorée»). Osler.
Osler however, very rarely met with this in a large series of cases.

Trousseau insisted on the occurrence of tingling and formication in the affected parts.

Pare not uncommonly found numbness noted in the cases at Guy's Hospital.

Blunting of the sensibility exists in a considerable proportion of all serious cases and is found chiefly on the hand and arm.

Gowers maintains that if there is any impairment of sensibility that it is hysterical and not a symptom of ordinary Chorea.

Pare asserts that it is common and has been found in nearly all the cases specially examined and especially in those parts where choreic movements are present.

Purves Stewart detected it in 10 out of 41 cases. The defect is usually one of slight diminution of perception and of localising faculty and is usually most marked in the limb showing the most spasm.

Reliable results are however very difficult to obtain in a disease with the psychical manifestations of chorea.

Hysterical manifestations of disturbed sensation are not rare in patients who have reached puberty and occur especially towards the end of the illness and sometimes after the movements have ceased. Hemi-anesthesia occurs and is usually accompanied by contraction of the visual fields and sometimes also by hemi-anosmia and diminution of hearing on the affected side (Taylor's Nervous Disease in Childhood and Early Life). Analgesia and hyperesthesia are occasionally present.
Reflexes.

Skin Reflexes.
They are generally normal, sometimes diminished and where hemi-anesthesia exists they may not be obtained on the anesthetic side.

Deep Reflexes.
They are usually normal in slight cases but in some severe cases there may be slight increased activity of the tendon jerks.

Sinkler (Osler on Chorea) in 50 cases found the knee jerk normal in 25, diminished in 15 and absent in 9.

W. Gordon (British Medical Journal March 30th 1901) describes an alteration in the character of the knee jerk.

"If the patient be in the recumbent position, one raises the knee, allowing the heel to rest on the couch, making sure that all the muscles of the limb are relaxed for the time being and if one then tests the knee jerk in the usual way, the foot is found to rise more or less smartly, but instead of falling back immediately it remains suspended for a variable time, hung up as it were, and then slowly sinks back into its normal position."

In paretic Chorea the reflexes may be considerably diminished and also in severe cases of ordinary chorea. Loss of knee jerk in moderate cases where paresis is not an outstanding feature has been ascribed to arsenical treatment but it is unlikely that this would be the only manifestation of the drug in the production of /
of interstitial neuritis.

Taylor reports a case where in each of several recurrences of a moderate chorea, the knee jerk was absent, returning each time as the convalescence was established.

The sphincters are not affected but in severe cases of Chorea Mollis and where there is great mental dullness the calls of nature are neglected. The evacuations may be expelled involuntarily.

Deep Reflexes in Chorea.

Gordon Sharp (Practitioner February 1908 p. 270) points out that the deep reflexes vary much in Chorea. "Chorea" he says, "is a complex combination of pathological conditions:—(1) the whole motor tract from the cortex above down to the motor nerve endings in the muscles may suffer. (2) All the various portions of the tract may not suffer in an equal degree. Thus the condition of the reflexes will depend on the extent of the tract affected, as well as on the severity with which the various portions are attacked. One portion may be stimulated while another may be depressed or inhibited while yet, in another instance, the whole tract may suffer inhibition and so in this way one may account for the cases of paralytic chorea. If the upper motor neuron is severely affected the restraining influence on the lower neuron is removed, and hence the reflexes are excited. Experience testifies to the correctness of this assertion, for in those patients
in whom speech is affected (hence attack on the upper neuron) the knee jerks are generally exaggerated. On the other hand, if the lower neuron is specially marked out for attack, the knee jerks may be depressed or be even in abeyance.

Mental Condition Psychical Disturbances.

This has been specially studied by Marcé (De L’État Mental dans la Chorea Paris 1863).

At the outset the patient may be bright and intelligent but in the majority of the cases there are psychical disturbances though usually of a mild kind and perhaps especially marked in older children. As a rule it is in proportion to the severity of the disease. It may take the form of dullness, lack of power to concentrate the mind on any subject and loss of memory. It is difficult to arouse the child’s interest even in what before would only have awakened pleasurable sensations. He not only is unable to learn his lessons, but story books, toys etc. don’t attract him and he lies with a dull listless look on his pale expressionless face.

There may be a marked change in the child’s behaviour and this is very common. He laughs or weeps without cause. He is very irritable and very capricious, wont take the food he used to be fond of; asks for something and when it is obtained for him, refuses.
refuses to take it. He may fly up in a temper and
storm a bit and then burst out into a flood of tears.
He may be very obstinate.

Severe mental disorders may complicate the
Chorea which, it has been argued, are not dependent
directly on the Chorea but are the results of some in-
toxication or the outcome of the neuropathic heredity
which is so often present. The severity of these
symptoms in slight cases and their absence in severe
cases supports the latter view.

Delirium.

It may occur in acute and grave cases. It is
usually very violent and talkative and resembles other
toxic deliriums. After its appearance the Chorea be-
comes rapidly worse and convulsions, followed by
coma and death, may ensue.

Hallucinations.

(Axenfeld and Huchard, Traité de Nervoses 1333)
They are most commonly of sight, though sometimes of
the other senses. They may even be of common sensi-
bility or in rarer cases of the genital sense.

Such hallucinations may be originated in
dreams and perpetuated during the waking hours. They
are only as a rule met with in children from the age
of 12 and onwards and usually disappear with the
Chorea.

Insanity.

The forms of insanity which may occur during or
after chorea may be mania, melancholia and some may
even /
even terminate in dementia.

Chorea Insanious.

This was described by Bernt (Monograph of Chorea). The subjects of this disease are usually young females between the ages of fifteen and twenty but children are also attacked and one of the most rapidly fatal cases on record, in which death occurred in 130 hours, was in a girl aged 9 years. (Cooke and Beale B.M.I. 1333 vol I p. 795).

The disease may be very severe from the onset but more commonly it begins as an ordinary chorea and the maniacal symptoms develop gradually. In one of Osler’s cases there was a succession of frights and the mental worry and excitement which provoked the attack were very considerable.

At the beginning there may be hallucinations which may give place to incessant incoherent babbling and in some cases to a furious mania.

Before death as in Osler’s second case, the movements considerably lessen.

Fever is almost invariably present and there may be hyperpyrexia (e.g. Hutchison Lancet 1899 vol I) where the temperature rose to 107° and also Donkin’s case (Medical Times and Gazette) where temperature rose to the same height.

Duration of fatal cases is usually no more than two weeks. Mortality is high but Gee reports he only lost one out of seven cases, one of his cases with high fever and parotitis recovering.

Convulsive attacks.

Gowers
Gowers (British Medical Journal 1876) has shown the relation of Chorea to convulsive seizures in 10 cases.

Fry (Journal of Nervous Diseases of Childhood 1892) has reported cases of athetoid movements, of rhythmical spasms, probably hysterical and of tremors in cases of Sydenham's Chorea.

Epilontiform seizures may occur. In Osler's Infirmary series there are recorded 5 cases where there were convulsive attacks and periods of sudden unconsciousness.

Circulatory System.

The pulse is usually increased in frequency and may be very irregular both in force and frequency and intermittent. While the frequency is usually greater in the severe cases, such is not so with the irregularity, as has been pointed out by Sturges. The irregularity has been differently accounted for.

Sturges, Goodhart and Still (Diseases of Children 1905, page 681) and others believe that it is due to some choreic disturbance of the heart muscles. It is of little moment whether the effect be paresis of the papillary muscle alone as some have contended, or a more general affection. The younger the child and the more recent the case, the more likely is it to be present. (Sturges).

Others again attribute the irregularity to the disturbance of the respiratory movements of the thorax.

Sir William Gowers has observed several instances in which posture had less effect on the heart's action than in health, there being little difference if any between the pulse rate in the upright and recumbent postures.
postures.

**Condition of the Heart Itself.**

Apart from the rapidity and possible irregularity of the action of the heart, murmurs are often heard over the pericardial region, the significance of which has been much discussed. Various explanations have been offered:

1. That they are haemic dependent on the associated anemia.
2. That they are the result of regurgitation at the mitral orifice due to irregular contraction of the papillary muscles not closing the mitral valves properly.
3. That the murmurs are not due to anemia and yet cannot be called organic, but are probably due to some toxic condition acting on the heart muscle.
4. The fourth class comprise those which are of organic origin. Thus there are two distinct classes of murmurs namely the haemic and the organic. Those murmurs included under headings 2 and 3 are not so definite.

As regards the theory which is put forward under heading No. 2 we can neither prove nor disprove it. It finds an able advocate in Sturgis.

That the haemic and organic do exist there can be no doubt, although it may be very difficult to differentiate between the two. From the point of view of prognosis it is only of importance to consider these two classes.

In arriving at an opinion as regards this question we have to take into consideration the following /
following points which will help us to get a proper understanding regarding the significance of the murmurs:—

1. **The time of the murmur.**
   A systolic murmur may be organic or functional, but a presystolic or diastolic is always organic.

2. **Valve Area.**
   It is comparatively rare to get Aortic endocarditis in Chorea, the mitral valves being affected in the large majority of cases.

3. **Seat of Maximum Intensity and Propagation.**
   In anaemia the seat of maximum intensity is usually at the base of the heart, or it may be in the fourth intercostal space to the left side of the sternum when it is heard up to but not beyond the apex of the heart.

   In organic disease, by far the most common murmur is a systolic murmur of maximum intensity at the apex, conducted into the axilla and it may be round to the angle of the scapula. It may occur in anaemia without the presence of a valvular organic lesion and thus it is necessary to look for other indications of anaemia:—venous hum in the neck and haemis murmur at the base having its maximum intensity in the 2nd left intercostal space. Sometimes organic and haemic murmurs exist together.

   Osler (Practice of Medicine page 1034) makes the following statements regarding the heart murmurs in Chorea:—

   "(1) In thin nervous children a systolic murmur of soft Quality at the base is extremely common, with accentuation of the second sound particularly at /
at the second left costal cartilage and is probably of no moment.

(2) A systolic murmur of maximum intensity at the apex and heard also along the left sternal margin is not uncommon in anaemic and enfeebled states and does not necessarily indicate either endocarditis or insufficiency.

(3) A murmur of maximum intensity at the apex, of rough quality and transmitted to the axilla or angle of the scapula indicates an organic lesion of the mitral valve and is usually associated with signs of enlargement of the heart.

(4) When in doubt it is much safer to trust to the evidence of the eye and hand than to the ear. If the apex beat is in normal position and the area of dullness not increased vertically or to the right of the sternum there is probably no serious valvular lesion.

(5) The endocarditis of Chorea is almost invariably of the simple or warty form and in itself is not dangerous but it is apt to lead to those sclerotic changes in the valve which produce incompetency.

(3) Pericarditis is an occasional complication of Chorea, usually in cases with well-marked Rheumatism.

In the majority of cases terminating fatally either in the course of Chorea or shortly afterwards, a fibrinous bead-like fringe is found edging the mitral valve and occasionally the aortic valves as well (Sturges). He collected 31 cases of fatal Chorea and only 5 of these had neither endocarditis nor pericarditis.

Osler /
Osler has collected 73 fatal cases of which 62 showed endocarditis.
In 43 cases the mitral valve was alone affected.
In 13 " " and aortic segments were affected.
In 3 " " tricuspid segments were involved with the aortic.
In 2 " " tricuspid, mitral and aortic were all involved.
In 1 " " aortic segments were alone involved.
Acute or subacute arthritis had occurred in 31 of the cases, in 4 it was doubtful and in 37 it was specially stated not to be present.

Of the fatal cases where endocarditis was not present, in 2 there was pericarditis, in 2 chronic mitral valvular disease and in 1 the heart was fatty.

The endocarditis was almost invariably of the simple variety as is shown by the presence of a few small bead-like vegetations just within the margins of the auricular surface of the mitral cusp.

Osler states that they present the usual characters of such structures and differ in no respect from the endocarditis met with in rheumatism and in the secondary affections in various febrile disorders.

Sir William Gowers (Text Book of Nervous Diseases) states that valvular disease is found in 30% of all fatal cases.

Sturges (in his work on Chorea 1831) gives the following statistics:
In Dr. Dickinson's 60 cases (Medico-Chirurgical Transactions vol LIX) 43 had heart affection of some kind.
Of the 21 cases that were 3 years or under, 17 had cardiac affection and of the 43 above 3 years, 31 were /
were so affected.

As regards irregularity of action the 53 cases give 23 such examples, 12 of irregularity alone and 3 of irregularity with murmur.

Sturge's 132 cases (seen at a later stage of the disease) give 47 (more than one third) that have heart murmur or irregularity.

59 of 3 years or under have 24 cases of heart disorder.

72 over 3 years have 23 cases of heart disorder.

From these statistics Sturge argues that the heart affection of Chorea is not less but more marked in early than in later childhood, and secondly that observation of the disorder at a later period discovers less heart disturbance than at an earlier, and especially that cardiac irregularity, a distinguishing feature of the Chorea of early childhood is found at the beginning rather than the end of the attack.

Thus he states the greatest liability to Chorea heart disturbance nearly corresponds with the age of greatest liability to Chorea.


There was some heart affection of more or less definite character in 141 cases (males 40 females 101). This gives a percentage of 32. In addition to those there were 73 cases (males 21 females 51 and sex not recorded in 1) or 16% there was some derangement of function or change of an indefinite character and probably not organic.

Mitral disease was the most common (116 cases to 6 of pure aortic disease).

Heart /
Heart affected in first attacks in 95 cases.
  "  "  second  "  "  27  
  "  "  third  "  "  7  
  "  "  several  "  "  10  

Of cases suffering from a first attack of chorea, the heart was affected in 93 cases and in 2 others the heart affection was doubtful during the attack.

This gives the proportion of heart affected as 33% in first attacks against 32% affected in all cases tabulated irrespective of the particular attack.

Of the first attacks it is also stated that the condition of the heart before the attack was not known in 41 cases. It was normal in 30 cases and was doubtful in 3 cases, while a murmur was present before the attack in 19 cases.

Of primary attacks a murmur was present after the attacks in 63 cases and in 5 cases a doubtful murmur existed. In primary attacks heart enlargement is noted in 10 cases.

Association of Heart affection with Rheumatism.

In the 141 cases where there was definite heart affection, it was found to be associated with acute or sub-acute rheumatism in 71 cases 50% and it was associated with pains in 13 cases, 13%.

There was no antecedent or consequent rheumatism in 50 cases 35%.

Osler states that the heart sounds in some cases have a foetal rhythm. The heart’s action in bad cases may be very rapid but in cases where there is mental enfeeblement /
enfeeblement it may be abnormally slow.

He states he has never seen a case in which the disordered movement was of such a kind that it might be attributed to a special choreic action of the heart muscle.

Of the 554 Infirmary cases 170 or 30.7% presented heat murmurs, in 149 apical in maximum intensity and in 21 basic.

In 141 cases of Chorea minor examined at the Dispensary for Nervous diseases at the John Hopkins Hospital there were 42 with a cardiac murmur nearly 30%.

Walsh brings forward a theory to explain the frequency of heart murmurs in chorea. He considers that irregular and occasional reflux takes place at the mitral orifice through disordered action of the muscular apparatus connected with the valve. Against this view Kirkes urged that there was no proof of the involuntary muscular organs partaking in the choreic disturbance, nor is there such inconstancy and variability in the apical heart murmurs of Chorea as would be inevitable did the condition result from valvular insufficiency in consequence of want of correspondence between the fibres of the ventricle which obliterate the cavity and those which close the valve.

Sturges also points out that the degree of heart affection does not correspond with the intensity of the choreic disorders of the voluntary muscles.

Galili (II. Polici. Nov. 21st 1903) however considers there is a true chorea of the heart muscle and that this is proved especially by the rapid variations /
variations in the cardiac dullness.

Organic Murmurs.

It is generally admitted that acute endocarditis is of frequent occurrence in Chorea.

The apical systolic murmur heard in many cases of Chorea is doubtless due to endocarditis.

It has also to be remembered that cases are on record which during life presented no murmur but which showed on post-mortem examination endocarditis of the mitral valve. (Baxter Brain vol II and Frank Allg. Weiner Med. Zeitung).

It seems probable that as Sturges says the murmurs due to endocarditis do for a time clear up but that they do not afterwards injuriously affect the heart, as he states, has been amply disproved by Stephen Mackenzie and Osler.

The apparent temporary improvement no doubt is due to the vegetations becoming smoother and smaller and firmer, only later by contraction to result in incompetency.

Wood (British Journal of Childrens Diseases June 1907 p. 263) states that 46% of his 333 cases were left with some heart affection.

Thayer (Journal of American Medical Association) in 308 cases of Chorea got cardiac murmurs in 41% and more than half of these cases with bruits, were thought to have organic heart disease.

Goodhart and Still (Diseases of Children page 662) give an analysis of their cases with regard to heart affection.

Of 133 cases they supposed 41 to have had valvular
valvular disease and all but one mitral disease. 22 had apical systolic bruits which suggested a doubt notwithstanding that they were persistent.

Stephen MacKenzie (Transactions International Medical Congress in London) estimates the murmurs to be present in 31% of his cases of Chorea with heart affection.

Murmurs indicating the presence of organic heart disease before the attack may be present and may be due to a previous attack of this disease or to rheumatism. On the other hand primary attacks of Chorea are met with in which there had been no antecedent rheumatism and in which no murmur is present.

In some such cases a murmur may appear during the attacks and may persist after convalescence while in other cases as the patient recovers from Chorea the murmur disappears.

Another class of cases is met with where there is no murmur during the attack but in which the murmur shows itself at a later period.

Condition of the Heart After an Attack of Chorea.

Stephen MacKenzie (Lancet Jan. 13th 1833) has shown that in an examination of 33 cases at periods varying from one to five years after the attack, definite heart disease persisted in 53.6% of the Chorea cases examined.

Donkin (Diseases of Children, London 1833 p. 302) gives notes on the heart condition of 44 patients who had from two to twelve years previously suffered from the disease and in 13% of them (40%) there were signs of organic heart disease.

Osler
Osler (Monograph on Chorea 1894) gives the results of his investigations regarding the condition of the heart in 140 cases, the length of time after the attack varying from 2 - 16 years.

In 51 cases the heart was normal.

In 17 cases there was disturbance of action which might be reasonably regarded as functional.

In 72 cases there were signs of organic heart lesion.

Normal Cases.

15 had had 3 or more attacks.
8 " " 2 attacks.
27 " " 1 attack.

There was a history of rheumatism in 9 cases and in 7 of these the attack was of the acute articular type.

Functional Cases.

There were 17 such cases. They were cases of localised or variable murmurs with no enlargement of the heart.

11 presented soft apical systolic bruits not propagated to the axilla and in 4 varying with the position of the patient. In most of these cases also there was accentuation of the second sound in the second left interspace but this is by no means uncommon in perfectly normal hearts of the young. It was also found in 10 of the normal cases that the pulmonary second sound was distinctly louder than the aortic second sound and in several instances reduplicated. No stress was laid on cardiac respiratory murmurs.
murmurs.

He considers that some of the cases may have been examples of organic heart disease.

Cases with Organic Heart Disease.

Of the 72 cases 30 had 3 attacks or more. The question of rheumatism was carefully enquired into in each case.

In 25 or 34.13% there was a history of acute arthritis which in 7 of the cases had followed Chorea in from 1 - 5 years.

Articular affection occurred in only 24.2 of his total series of cases and only in 17.11% in the group of 51 normal cases.

This he points out shows the greater liability to serious heart mischief in the cases with joint complications.

In 63% of the cases however no history of arthritis could be obtained although carefully enquired for.

In the large majority of cases the signs were those of mitral disease, usually incompetency indicated by the systolic murmur of maximum intensity at the apex and propagated to the left with signs of enlargement of the heart and sometimes also by a thrill and marked accentuation of the second sound in the pulmonary area.

He states his conclusions as follows.

"(1) Endocarditis is a very common complication of Chorea Minor.

(2) In the majority of such cases the endocarditis is independent of and is not associated with acute /
acute arthritis unless indeed we regard the valvular lesion as itself a manifestation of rheumatism holding with Bouilland that "chez les jeunes sujets la coeur se comporte comme une articulation".

(3) In a considerable proportion of cases much larger indeed than has hitherto been supposed, the complicating endocarditis lays the foundation of organic disease."

Stephen Mackenzie (Lancet Jan. 12th 1839) and Donkin (Diseases of Children 1893 p. 302) maintain that the endocarditis is really rheumatic.

Pericarditis in Chorea.

This was first described by Bright who states that it had long been recognised by the physicians at Guy's Hospital (Medical Chirurgical Society Transactions vol XXII p. 10).

He gives in his paper notes on 5 cases of pericarditis with chorea and rheumatism and in one of these cases there was also endocarditis. He thought the connection between Chorea and Pericarditis was through the phrenic nerve which communicated the irritation from the inflamed pericardium to the Spinal Cord.

Sibson (Reynolds System of Medicine) states that of 21 cases of Chorea with acute rheumatism 15 had pericarditis, 6 had no pericarditis, while 14 had endocarditis, 3 had no endocarditis and 3 were doubtful as regards endocarditis.

Of the 73 post-mortem cases collected by Osler pericarditis occurred in 10 cases and was associated with endocarditis.
In 31 of these cases there was a history of acute articular rheumatism, 1 case had sub-acute rheumatism, 1 rheumatic pains while 9 had not had any rheumatism.

In 3 the pericarditis was old; in 3 the death was stated to have been caused directly by the pericarditis, pleurisy occurred in 5 cases, pneumonia in 4, acute phlebitis in 1 and acute nephritis in 1.

Wood (British Journal of Childrens Diseases June 1907) states that in each of his two fatal cases in 226 cases there was pericarditis.

Garrod (Clinical Journal Feb. 3th 1905) says that pericarditis is specially apt to develop in those cases where rheumatic nodules are present.

Myocarditis.

Poynton and Leas have shown this to be present in more or less intensity in almost all cases of endocarditis.

From what has been written on this subject it will be seen that endocarditis is of very frequent occurrence in Chorea, and that it very often lays the foundation of permanent cardiac disease and when we learn from Osler's observations that in 140 cases of Chorea which were examined 2 - 15 years after the attack over 50% showed permanent organic heart disease, we are led to suppose that many of the murmurs heard during the attack and supposed to be haemic or functional are in reality due to endocarditis.

Osler also, from the post-mortem examinations of fatal cases states his opinion that the endocardial vegetations are in all respects comparable to those seen in acute rheumatism and when it is also shown that in those cases of the Chorea where organic heart affection existed that a much higher percentage of rheumatic...
rheumatic history could be obtained than in the total series of cases, we get further and striking proof of the close association of Chorea and rheumatism.

Stephen Mackenzie and Donkin, as already has been shown, consider the endocarditis of Chorea to be rheumatic.

Regarding the Choreic heart affection which has been stated by some observers to exist, when we consider that there is no proof that any hollow muscular organ is affected in Chorea, when we know that the alimentary canal is not affected, and that the irregularity of the heart's action does not in any way correspond with the severity of the disease, we are led to consider any Choreic heart affection to be very unlikely.

Cutaneous Affections.

The more important of the skin affections met with in Chorea serve to support the close relationship claimed to exist between Chorea and Rheumatism. The majority of them are really manifestations of rheumatism; Erythema Nodosum, Purpura, purpuric urticaria and subcutaneous fibrous nodules; Herpes Zoster sometimes described as occurring in connection with Chorea is probably due to the administration of arsenic and so probably is excessive pigmentation of the skin. In some cases however the skin shows absence of pigmentation in patches.

Alopecia Areata has also been described but it must be carefully differentiated from the patches of baldness produced by the excessive friction between the head and the pillow.

Urine /
Urine.

Walsh and others showed that there is an excess of urea in some cases and uric acid may be deposited in considerable quantities.

Handfield Jones has described an increased deposit of phosphates. These conditions are proportional to the severity of the muscular movements.

A pigment, urohaematoporphyrin discovered by McMinn in the urine of rheumatic subjects was found by Garrod in the urine of 14 out of 20 cases of chorea: this he regards as an additional proof of the close relationship between Chorea and rheumatism (Garrod, Medical Chirurgical Society Transactions, London 1839 LXXII p. 145).

Albumen when present usually indicated nephritis; according to some observers it may be produced by renal embolism but no proof of this exists.

Glycosuria has been described as occurring in a choreic patient.

De Marchis (La Reforma Medica July 5th 1902) makes the following statement regarding the urine in Chorea:

"There is diminution in the daily quantity: specific gravity is relatively high: total acidity is increased: diminution during the disease of the quantity of nitrogen which is not eliminated as urea: increased elimination of uric acid: decrease in elimination of chlorides: increase of phosphates: total quantity of sulphuric acid and allied substances unchanged."

Other rare complications may occur.

Simon (Pediatrics July 1906, p. 484) records the occurrence /
occurrence of hemiplegia and aphasia in a chorea case. The paralysis was still present five months later as was also the speech affection. He regarded the condition as the result of embolism from latent endocarditis.

Leone and Gaudone (Ann de Med. et Chir. July 15th 1905) reported a case of chorea where death was said to have resulted from meningitis. Rheumatism is occasionally associated with meningitis and it might be expected that chorea would show a similar tendency to this complication especially when the diplococcus has been discovered in the meninges. In the case however, reported which was of a very serious character there were several very extensively ulcerated bed sores and a staphylococcus was found in the turbid cerebro-spinal fluid which was obtained by lumbar puncture. It is probable that the meningitis was a result of the septic absorption from the bed sores. They did not seem to have made a post-mortem.

Barjon in this connection also reports a case of pseudo-meningitis in the course of a case of chorea (Archives de Neurologie July 1904 page 53).

Sir William Gowers (Text Book of Nervous Diseases-Chorea and the Borderland of Epilesy) reports several cases in which the epilepsy dated from an attack of chorea. In one case he says that convulsive attacks limited to one side occurred during the fortnight after the cessation of chorea and similar attacks persisted as chronic epilepsy. There was no cardiac murmur.

He also states that persistent spasm is occasionally /
occasionally not with. In a young child there was continuous spasm in the arm during an attack which was one of true chorea. The elbow and wrist joints were flexed and the aspect of the limb was similar to that seen after infantile hemiplegia. The choreic movements were present in the arms although in much less degree than in the other limbs. As the child recovered the persistent spasm lessened and the movements in the limbs became greater.

Similar spasm had been present in a previous attack.
There is no difficulty in recognising chorea in its usual form. The appearance of the patient, and the very characteristic movements at once suggest the correct diagnosis.

In some cases occurring in early infancy the spasmodic movements may be mistaken for those which occur in cerebral disease and probably some of the cases of chorea recorded in infancy were examples of such cerebral disease. The history would serve to give a correct diagnosis. There will also be difficulty in diagnosing those cases where the choreic movements are inconspicuous and where they are dominated by other symptoms such as paresis or acute mania.

Paralytic Chorea.

Spontaneous movements may be almost entirely absent and the case come to be regarded as one of some form of paralysis. As a rule the history would help to clear up the diagnosis. The paresis usually comes on gradually in chorea, taking perhaps several weeks to be fully developed. It is never as a rule absolute paralysis. The arm is usually affected more than the leg and the hand more than the shoulder. The face generally escapes. There is no muscular wasting and no pain and while signs of spasticity are absent, reflexes are usually present. In some cases however there may be considerable paresis of sudden onset, and where this is hemiplegic in distribution and /
and associated with heart disease, it is more difficult to make the differential diagnosis. In such cases, however, there is no history of loss of consciousness or convulsions and the paralysis is never so well marked as in embolism and in chorea, or even when the arm and leg on one side are affected the face escapes.

Where there is monoplegic paresis e.g. one arm, the slow onset distinguishes the condition from organic affections; or if the onset happens to be sudden the escape of both leg and face and the absence of convulsions and other grave symptoms at the onset exclude a cerebral lesion.

The absence of atrophy and reaction of degeneration exclude Infantile Paralysis and they also exclude peripheral neuritis; the exclusion may possibly be aided by the absence of any defect of cutaneous sensibility which is common in this type of chorea. In the majority of cases also of paretic chorea, a careful enquiry will often give the history of spasmotic movements prior to the onset of the paresis.

Very often also on careful observation of a paretic case slight spasmotic movements can be detected in the face or fingers especially when the patient attempts some sustained act, e.g. keeping the mouth open and the tongue protruded or holding his arms above his head with the fingers spread out. Again if such a child be asked to hold an object firmly in one hand and keep it tightly clasped it may be noticed he will fail to do so, the muscles involuntarily relaxing.

Delay may also be noticed when a patient attempts /
attempts to relax a certain group of muscles.

Where cerebral lesions exist the resulting paralysis is a spastic one and other signs such as headache, vomiting and optic neuritis, would be present.

Gradual paralysis of Spinal origin is associated either with pain and spasticity or with muscular atrophy and loss of the deep reflexes.

Sir William Gowers' observation should be borne in mind that "when a child between the age of 7 and 12 years is said to have gradually lost the power of one arm or one side the disease is most often chorea."

Maniacal Chorea.

This may be mistaken for acute mania.

Characteristic movements are however present in most cases and the subjects are younger than most of the subjects of acute mania. There is less continuous talkativeness than in acute mania and there may be absolute loss of speech. The previous history would serve to identify those cases where the mental symptoms supervened when the choreic movements stopped.

Chorea and Hysteria.

Duchateau has collected 34 cases where Hysteria was present at the same time as chorea (Thése de Paris 1893).

Debove (Soc. Médic. des Hopitaux 1890) reported a case where hysteria produced all the features of typical chorea.

A history of imitation can often be got in the hysterical affection and the subjects are generally girls.
girls about the age of puberty or adolescence.
The movements are more sudden and single muscles may contract and again in hysteria a rhythmical character can often be distinguished in the movements.

Hysterical paralysis also closely simulates choreic parasis but if this is flaccid it is usually complete and both this condition and hysterical chorea are apt to have the stigmata of hysteria associated with them, such as hemi-anesthesia, "stocking and glove" anesthesia, amblyopia, etc.

Senile Chorea.
This is distinguished by reason of the patient's age. The condition is never so acute as in younger people and is slowly progressive, unattended by any relation to rheumatism or endocarditis.

Huntington's Chorea.
This chorea is like Senile Chorea.
In addition there is a history of it attacking previous generations and often many of them. Such cases are very slowly progressive as a rule and are associated with gradual mental deterioration, a condition of smiling and complacent dementia.

Electrical Chorea. (Dubini's disease).
The spasms are much more sudden and shock like. Associated with them are rapid muscular atrophy and pyrexia in acute cases.
The course is progressive and a large proportion of cases end in death. Epileptiform convulsions are common.

Para-myoclonus /
Para-myoclonus.

The spasms are more sudden and shock-like and are of a more simple character and as a rule they affect similar muscles on the two sides of the body and are commonly restricted to the muscles of the trunks and proximal segments of the limbs, though in some cases they may be more generalised. There is no relation to endocarditis or rheumatism and the condition is apt to persist longer than true chorea.

Athetosis.

The movements are slow and writhing or undulating in character and are attended with permanent stiffness of the limbs. In some cases also there is an increase in the size of the muscles. They often suffer from convulsive attacks and the intellect is defective. Bilateral Athetosis, diplegia with choreic movements, diplegia with myoclonic movements and diplegia with intention tremors are the clinical varieties which have been included in congenital chorea. They are chronic maladies and usually progressive and have associated with them signs of grave organic disease, contractures, paralysis and recurring convulsions, spasticity etc. Such signs are also present when choreic movements manifest themselves in the subjects of cerebral tremors and vascular brain lesions.

Thus the greatest difficulty in making a diagnosis is in paralytic cases where either the choreic movements are very slight or where there has been no history of them or where the paralytic has come on very suddenly.

Risien Russell (Allbutt’s System of Medicine
article Chorea in vol VII gives instances of two
difficult cases.

One a boy in whom there was no other symptoms for
some weeks except paresis of the forefinger of the
left hand experienced in holding the reins of his
pony. Slight choreic movements and a systolic mitral
murmur showed the real nature of the case when they
occurred.

The other case was one of Chorea Vollis: a girl
deeply if not completely paralysed in the four limbs.
She lay motionless but the movements of organic life
continued. The condition was said to have come on
in a few days. Within a week choreic twitchings
appeared.

There would also be considerable difficulty in the
differential diagnosis of acute mania especially when
the choreic movements had disappeared.
TREATMENT.

General Considerations.

There is wide diversity of opinion regarding the most suitable treatment for Chorea. Numerous drugs have been used, great success from the use of some particular drug in the hands of one person, being conspicuously absent when used by another.

There are however certain essentials in the treatment of the disease which all agree should be carefully attended to.

All sources of peripheral irritation, which in a child of unstable nervous system, must at least be a predisposing cause of some considerable importance, should if possible be removed. If there is any suspicion that the patient is suffering from round worms or thread worms, steps should be taken at once to get rid of them. That this in itself has a beneficial influence on the disease is undoubted.

The same applies to constipation, eye defects—which can be remedied by spectacles—and dental irritation.

As regards adenoids, phimosis, enlarged tonsils etc. no attempt should be made to remedy these after the onset of the chorea. The adenoids and enlarged tonsils may also be the place of entrance of the rheumatic micrococcus. It is also generally agreed that at first in all cases rest in bed and seclusion is desirable. The child at once should be removed from school, lessons completely stopped, no books allowed. No other children should be allowed near the /
the patient, who should be put under the charge of one person who is not neurotic. The improvement which is often noted on removal of a case of chorea from the home of a working man to an hospital is due to the fact that rest in bed and isolation can be procured there, in a way which at home is impossible. While it is true that rest is an important factor, all through the course of the disease, not only as Risien Russell points out, to allay irritation of the motor neurons, but also as a prophylactic or curative measure in the treatment of the cardiac manifestations of the disease, still this should never be obtained at the cost of making the child fretful. If bed proves irksome it is well to let the child up and secure rest by setting him on to a couch at intervals during the day and trying to keep him amused during those intervals.

In all severe cases absolute rest in bed should be enjoined until the disease has become greatly lessened.

Isolation also is very important as one can easily imagine in patients who are highly strung. If the child is allowed to play with other children who consequently make fun of and call attention to the peculiarities he possesses, his grinsces, etc, the duration of the disease is considerably lengthened. It is however important that the child should be nursed by a cheery person who will exert herself to keep the child in a quiet placid contented frame of mind and guard him from all excitement while at the same time she does not allow the patient to become depressed.
depressed or melancholic. A bright and cheery room should be obtained and if possible a trained nurse. For an adult the trained nurse can be introduced at once and allowed to take charge of the case, but for a child who in this condition is fretful and shy and acutely self-conscious, the nurse ought to be left gradually in full charge till the little patient becomes accustomed to her presence and instead of disliking her, comes to look forward to her coming. Still it is interesting to note that rarely is there any aggravation of the disease when a child is removed from home and placed in the general ward of an hospital. The adaptability and pleasure derived from new sensations, with the inquisitiveness of childhood being probably the reason.

In many cases the mother is very neurotic and therefore quite unfit to act as efficient nurse to the child and it is difficult unless by the help of a trained nurse to keep the child quiet and contented.

Bartz (Arch. Ped. Jan. 1905 p. 34) recommends that the child should be kept in a darkened room in bed for several hours in the afternoon. School is to be absolutely forbidden even in mild cases.

Bruel (British Journal of Childrens Diseases Jan. 1907 p. 33) considers that the degree of isolation of rest may be varied according to the severity of the case and divides the treatment into four degrees of completeness.

"(1) Going to bed early and rising late so as to spend fourteen hours in bed.
(2) Going to bed also for two hours in the middle of the day.
(3) /
(3) Absolute rest in bed for a fortnight with very little visiting by relatives.

(4) Darkening of the room as well except at meal times."

He considers also that some advantage may be obtained from blue light.

In addition to rest in bed and isolation it is well to see that the patient is well clad in woollen garments as the spasmodic movements during the night often shake off the bed clothes and expose the patient to the chance of a chill.

A plentiful nutritious diet should also be provided. This is of course especially important in those cases which occur among the poorer classes where no attempts are made to entice the child to eat any other food than "what is going," whereas it should be coaxed to take as much milk and light food as possible.

Massage should be employed in the milder cases, when the spasmodic movements permit of it being employed, to keep up the nutrition of the muscles. This should especially be used in the paretic form of the disease.

Scrupulous cleanliness must be observed because of the liability to bed sores and especially in those patients who pass their evacuations in the bed and who are often in such a condition of mental apathy as to be unaware of what has taken place. The nurse must therefore in these cases always be on the watch.

**Hydro-therapy.**

In very mild cases douching with cold water may be employed and in all but the most severe cases tepid and warm baths may be used at night to encourage sleep.
sleep, care being taken not to let the child injure itself. In the very severe forms of chorea, with high temperature, the cold pack may be used, or the cold bath may be used where the spasmodic movements do not prevent this.

Charpy (Rêve de Thérapeutique Medico Chir. Vol 1, 1395) considers the wet pack the best method. A sheet dipped in water at 51° to 54° F. then lightly rung out, spread over mattress with oil cloth then closely wrapped round the patient: latter rubbed from head to foot within sheet in woollen blanket and returned to bed.

Hollopeter (Medical Record June 25th 1904) strongly recommends hydropathic measures. He places the child in a bath at a temperature of 90° - 100° F. and keeps him there for one to two hours twice daily. During ten minutes while the child is in the bath gentle superficial massage is to be applied to the arms and legs and trunk. The child meanwhile is to be allowed to amuse himself with playthings in the water. He treated forty or fifty patients in this way with excellent results. The duration of the attacks he says were shortened from three months to six weeks.

In several cases the ordinary type of bed is not very suitable even when it is padded all round. A very large bed (with a water mattress) and which also is very carefully padded all round is necessary in the severe cases. An excellent bed can be made by placing a mattress on the floor in a corner of the room, the walls being protected by two halves of a mattress.
mattress placed upright and the other two sides protected by pillows.

If the ordinary cot is used it must also be carefully padded, cotton wool wrapped carefully round all the iron parts and then kept in position by bandages. It may also in some cases be a wise precautionary measure to bandage up the child's elbows and knees etc., which because of the bony prominences are likely to be injured. If the limbs do become bruised or cut they must be carefully covered with an abundance of cotton wool over which a bandage should be lightly placed.

Administration of Nourishment.

In severe cases the difficulty of getting the child to take sufficient nourishment may be very great. When the spasms are very marked it may be only possible to administer food in a liquid form and that through a feeding bottle. A china feeding cup should not be used in case the end should be bitten off and swallowed.

When swallowing becomes very difficult it is perhaps better at once to resort to nasal feeding which rarely causes as much disturbance as the exhausting and often ineffectual efforts to swallow food. It is advisable, since vomiting may occur after nasal feeding, not to give large quantities at a time but small quantities frequently and not more than three quarters of a pint should be given at one time to a child of 3 years. (Taylor). The nostrils should be used alternatively and olive oil is useful as a lubricant with perhaps if necessary some /
some cocaine. Weak antiseptic lotions should be applied to the nasal mucous membrane if any signs of nasal irritation present themselves.

Nutrient enema may also be used in severe cases.

The treatment so far described is pretty well followed out by most physicians, who all agree as to the advisability of rest and isolation and nutritious diet and cleanliness with the other steps necessary in the severer cases, to prevent the infliction of self injury when the spasmodic movements are excessive.

Sturges, who has many supporters, submits that this is all that is necessary in the treatment of ordinary uncomplicated chorea. He holds that by this treatment the child is given time to regain that control of will power, the temporary loss of which, over the ordinary muscular movements being in his opinion, the cause of the disease. Others who do not necessarily agree with him as the causation of the disease, consider with him that in all mild cases and even in cases of ordinary severity that this treatment is quite sufficient. They may allow the administration of Cod Liver Oil where the stomach permits of it being taken, or of Iron where there is anaemia but they do not consider it wise or beneficial to administer any drug which has been recommended as exercising a definite controlling action and finally removing the spasmodic movements.

While in many mild cases, this may undoubtedly be sufficient especially where there has been some source of reflex irritation which was removable, and while it is a consistent and rational treatment in the hands of those who maintain that the disease is /
is a functional one; still it is undoubtedly that certain drugs given in the proper way and in the proper dosage will lessen the duration of the disease. When it is also remembered that, as has been shown, that those persons who have been the subject of chorea do in very many cases, subsequent to the attack, show definite signs of serious and permanent cardiac disease, it is surely right that active means should be taken in the hope that when controlling the outward manifest muscular spasms the tendency to those endocardial changes may also be modified or perhaps removed.

Of all the drugs that have been used in the treatment of this disease probably arsenic still holds the field. It has been shown to be beneficial and often markedly so. It has however certain disadvantages and certain precautions are necessary in its administration and dosage both to get satisfactory results and to obviate any toxic after effects.

The general condition of the patient is usually but improved by it. Whether or not it acts directly, and especially on the motor neurons improving their mitrit, cannot definitely be determined.

Graham Brown (Treatment of Nervous Diseases) says: "There is indeed some reason to believe that it excites phagocytosis, exerting a positive chemiotaxis in relation especially to the large mono-nuclear leucocytes. Be that as it may there is no doubt that it benefits the motor neurons and favourably affects the prognosis of the case. Some observations go to show that arsenic replaces phosphorus in the cortical neurotic cells and if this is so, it may explain /
explain that diminution of the excitability of the neuron which follows its administration in chorea."

It is customary to commence with small doses well diluted and given along with food, three times a day and then gradually increase it till perhaps 15 minims are given three times a day. After a short trial if arsenic does not produce the desired effect, it has to be discontinued, especially if the dosage has been large for fear of arsenical neuritis which is apt to follow and produce a condition which is very slowly recovered from. Some patients also from the very start are unable to tolerate the drug and in them of course it is necessary that some other drug should be employed. At all times the effects of this drug has to be very carefully watched and discontinued immediately any sign of its toxic effects are seen.

It has also been recommended that arsenic should be administered hypodermically because much larger doses can be administered after this method than by the mouth without producing intolerance. This treatment may be feasible in adults but has obvious disadvantages in children. It can be carried out after it has had to be discontinued by mouth on account of gastro-intestinal disturbance. There is however very considerable danger of arsenical neuritis, several irrecoverable cases, where this form of treatment has been used, having been reported. It does not seem to possess any special advantages.

Arsenic has also been administered in the form of cacodylates both by mouth and also by hypodermic injection. Hypodermically the dose used was one third of a grain of cacodylate of soda, which was gradually /
gradually increased to two thirds of a grain daily. It has also been given by rectal injection. No advantage however can be claimed over the liquor arsenicalis of the Pharmacopoeia. Arsenic for the treatment of chorea was first recommended by *Martin of Reigate* (Medico-Chirurgical Transactions vol IV 1813). He says that "he began with 5 drop doses and increased 1 drop every day until it might begin to disagree with the stomach and bowels" which usually occurred when a dose of 14 drops was reached. The dose was then diminished and continued at 10 drops for six weeks.

*Murray* (Rough notes on Remedies 1902, p. 17) strongly advocates the use of very large doses from the very beginning of the treatment. He was advised to this treatment by Linton who stated that "Fowler's solution in 15 - 20 drop doses might be given to children for a few days without disturbing the stomach and that so given it was an almost infallible cure for chorea within a week. Murray states that 5 drop doses or less are of no use and he relates a case of violent chorea bordering on mania where this treatment was rapidly successful. In twenty years experience of it he only saw one case which resisted this treatment by large doses of arsenic. "A minimum dose of 15 minims in every case is necessary, 10 minims will not do." The remedy should be taken with food in the middle of /
of a meal and if it does not act in a week it must be dropped as after that time the toxic action of the remedy would come into play and the patient would be injured.

Levy (These de Lyon 1900) recommends the use of butter along with arsenic. He showed that the butter should invariably be fixed at 10 grammes whatever the quantity of the active principle incorporated with it. To prepare the mixture, a known quantity of arsenious acid is taken, according to the dose to be administered. To this is added sodium chloride in such proportion that 0.1 gram corresponds to 0.005 gram of arsenious acid. This mixture of sodium chloride and arsenic is titrated with 10 gms. of butter and this amount is given on bread, a form of medication extremely palatable to children. The whole dose is given at one time, but two doses a day seem to be sufficient. Under this method of treatment it is not necessary to confine the patient to bed or to put him on a milk diet. A more liberal diet gives better results.

Allen Starr (Phil. Medical Journal May 20th 1900) makes the following remarks. Arsenic pushed to its physiological limits and then reduced slightly is the best drug in the treatment and antipyrine is second. Exalgin, Phenacetin, bromide and chloral and paraldehyde produced little effect. He, however, considers that better than medicine is change of air.

W. Von Bechterew (Centr&blatt f. Nervenheilk. u. Psychiatrix August 1900) states that sedatives are of value combined with arsenic, the latter being given in the form of Fowler's solution or as a solution of arsenious acid in doses which are rapidly raised to twice
twice or three times what is usually accepted as the maximum. At the same time sodium or potassium bromide and antipyrine are given in large doses.

While the relationship between rheumatism, endocarditis and chorea is a sufficient indication for the routine use of the salicylates in conjunction with other remedies.

Railton (Med. Chronicle Feb. 1900) reports several cases of neuritis which supervened after the cure of chorea by arsenic. In these cases 10 drops of liquor arsenicalis had been given thrice daily for three or four weeks by which time the patients had taken an equivalent of from six to eight grams of arsenious acid. None of the cases gave any warning of the advent of neuritis during the administration of the arsenic but the symptoms developed after an interval of from a week to a fortnight subsequent to its discontinuance. He considers that no dose amounting in aggregate to 4 grains of arsenious acid should be administered to a child suffering from an attack of chorea.

Lanoois (Revue de Therap. Med. Chir. LXVII Nov. 5th 1901) tells of three cases of Chorea treated with sodium cacodylate instead of arsenic. The cacodylate was given hypodermically first in doses of from one-third grain to two-third gr. The patients recovered in from one to three weeks. In all the ordinary treatment had been previously tried without success.

Tscherno-Schwartz (Archives f. Kinderb. vol XXXV p. 454 1903) records 86 cases treated with arsenic and which were of shortest duration: 29 cases which were treated with rest in bed in new surroundings and without
without any medicines, were of a little longer duration. 20 cases treated with antipyrine showed this drug to be less useful. Bromide was used in 47 cases and Quinine in 31 cases and were found to be quite useless.

The average time of recovery with arsenical treatment was 65 days.

Pope (British Medical Journal Oct. 18, 1902) writes an excellent article on the treatment of Chorea by arsenic. He gives first the cases unsuited for arsenical treatment.

(1) Cases obviously unsuitable.
(2) Those intolerant of the drug.
(3) The small minority who resist it.

Class (1) Consists of the following types of cases.
(a) very acute cases, with coma or paralysis.
(b) those treated for some time with small doses of arsenic.
(c) where there is reason to believe that the rheumatic process is going on in acute form where salicylate treatment is to be used till the temperature is normal and then arsenic.
(d) cases of advanced cardiac disease.

Class (2) Those intolerant of the drug. These do exist notwithstanding careful and proper administration of the drug.

Class (3) Personal Idiosyncrasy may be made to account for almost anything. Some of this class are those which have been running on for some time and where the movements have become /
become as much a habit as a product of pathological nervous discharge. It is certain that there are fewer among recent and fairly acute cases. The majority of the cases in this class are however those referred to under Class (1) D. When in those cases there are no definite signs of poisoning, wait for a few days and recommence with larger doses but of course if these signs are present arsenic must be withheld altogether.

He gives arsenic in all suitable cases after the following plan:

(1) See that the tongue is clear before commencing treatment.

(2) Put the patient on a bland and easily digestible diet (principally milk in children) and, but not necessarily, so in adults.

(3) Give the drug in a much diluted form and in the same dilution throughout.

He commences with 21 m of liquor arsenicis in 1 oz. of water as the first dose to a young child, and when increasing the dose he gives a larger quantity of the same mixture instead of increasing its strength. The smaller and earlier doses are usually given after meals, the larger ones taken during meals. He states that there is no difficulty in getting a child to take 6 or even 7 ozs. of fluid.

The initial dose to children is equivalent to .0018 grs. of arsenious amydris three times daily.
daily; the dilution being 1 : 50,000 or 0.002%.

(4) Do not discontinue on the first attack of vomiting. This is often due to accidental causes and the patient may be able to go on for two or three days without a recurrence.

(5) Increase the dose daily. A daily increase of 2 dram. is usually well borne.

(6) Keep the patient in bed during the treatment.

(7) If the vomiting persists discontinue the drug for 24 hours and then give the same dose as the last.

(8) Examine the patient very carefully daily for any signs of toxic symptoms.

He points out that by this method less arsenic is given than by repeated small doses over a long interval, which is of considerable importance as arsenic is a cumulative poison.

"Patients treated in this way usually show much improvement in 3 or 4 days. The movements are more easily controlled and if they have not entirely disappeared in 7 or 8 days the disease will almost always have become a trifling one, the patient being allowed to feed himself, take ordinary diet and may be allowed to get up and take outdoor exercise. In some cases a return of the movements may take place when the patient should be returned to bed and the treatment resumed, commencing with 3 or 4 oz. of the mixture. On discontinuing the arsenic, I usually give a mixture containing iron for a few days."

Shaikovitch (Vratch Sept. 1903) after using arsenic in large doses for chorea concludes that no beneficial /
beneficial influence has been proved, and that in the view of diarrhoea and vomiting or even more serious neuritis which has resulted in some cases it should not be used for children in large doses.

Chapman (Brooklyn Medical Journal July 1904) condemns large doses of arsenic and says that while they may reduce the choreic movements, they are likely to influence the patient's general condition unfavourably by disturbing digestion.

The use of arsenic by the hypodermic method where it disagrees with the stomach, he strongly condemns. Where Fowler's solution is not taken well he recommends Dunovan's solution (Liq. arsenici et hydragyri).

Berg (Arch. Ped. Jan. 1905, p. 34) gives arsenic in increasing doses of 1 drop "until slight swelling of the eyelids is present in the morning." He gives it in water or after meals and any signs of gastric disturbance necessitate its omission as does also albumenuria.

Kobak (Arch. f. Kinderb. 1903) considers that arsenic is the best treatment for chorea when there is no antecedent rheumatism discoverable.

Inglis (Pediatrics June 1906) recommends a combination of arsenic Iron and Strychmne as giving better results than pervic doses of arsenic alone. He also advocates forced feeding and plenty oils and fats.
Arsenic.

Korlik (Medical Record May 18th 1903) states that in some children arsenic acts as a direct poison and in others again it is well borne. The effect of arsenic on the kidney is to be especially remembered. In some cases, before the appearance of other toxic signs a trace of albumen was found in the urine, and if the administration of the drug was continued, casts would appear and sometimes blood cells: the urine became normal on stopping the arsenic. As little as 5 drops of the liquor arsenicalis, three times a day, was sufficient to cause such symptoms in a child of 5 or 6 years. He also considers that the liquor arsenicalis may become concentrated by keeping as the solution evaporates, and unwittingly a much larger dose was given than intended.

Gordon Sharp (Practitioner, Feb. 1908) gives arsenic in 10 or 12½ minim doses.

For all cases between 3 - 15 years of age he gives the following prescription:-

Liquor Arsenicalis 240 (or sometimes 300) minims.
Tincture of Capsicum 25 minims.
Liquid Extract of Liquorice 240 minims.
Chloroform Water 6 fl oz.
Water to 12 fl oz. mix.
Take one tablespoonful three times a day immediately after meals.

The liquorice is a good vehicle for the arsenic and pleasant in taste to children. Chloroform and capsesicm are anaesthetic to the mucous membrane of the stomach and the chloroform helps to preserve the mixture in warm weather.

If /
If at the end of a week the patient is no better he then increases the dose to 121 minims. This latter procedure he states is not often necessary. If arsenic is going to do good in chorea it will show its beneficial action in the first fortnight. When the remedy is doing good he continues it till the patient can walk along a straight line and stand on the leg of the affected side with steadiness. This usually represents a period of 3 or 4, or at the outside six weeks.

For after treatment he gives the following.

Sodium Bicarbonate 120 grains.
Tincture of Capsicum 25 minims.
Liquid Extract of Liquorice 240 minims.
Chloroform water 6 fl. oz.
Water to

Mix and dissolve.
Take a tablespoonful three times a day after food.

He considers that the bicarbonate "washes the arsenic out of the tissues" but says that this statement may not be a scientific one.

It will thus be seen that the majority of those who have had experience in the use of arsenic in the treatment of this disease are satisfied as to its real advantages, and while some have recorded very serious cases of neuritis in some cases incurable due to the use of this drug, others have again shown that these toxic signs can be avoided by the use of the drug in larger doses for a shorter period.

Murray of Newcastle in his work "Rough Notes on Remedies" is very emphatic that if large doses of arsenic /
arsenic are used the drug must be stopped within 8 days or so at the outside. Some of the cases of neuritis reported where the drug was continued for 5 or 6 weeks in very considerable doses need not occasion any surprise.

While arsenic continued over a long period in small doses is of use, more perhaps as a general tonic, in alleviating this disease still as has been shown, the striking effects this drug can produce are only got when it is used in doses considerably larger than what one is accustomed to.

Pope has given excellent advice as to the preparation of the patient before arsenic is to be employed and as to the length of time the treatment has to be continued. The majority of children seem to tolerate arsenic very well if it is given well diluted and after meals. A dose of 5 m. may be started with and rapidly increased, always keeping it well diluted and giving it with food, up to 15 minims or even more. If this does not produce the desired result within a week then the drug should be stopped and other means adopted. Such a treatment does not seem likely to be succeeded by any development of peripheral neuritis.

Zinc.

The sulphate, the valerionate, the phosphide and the oxide of zinc have all been used.

The oxide or the sulphate was usually given in doses of 3 to 5 grains three times a day after meals. The dose was gradually increased up to 8 - 10 grains or even more.

The phosphide was only used where there was considerable /
considerable nervous exhaustion and was given in doses of one-twentith to one-tenth grain three times a day in a pill with 1 or 2 grains of hyoscyamus extract.

The valerianate and the bromo-valerianate in 1 - 3 grain doses in the form of a pill with extract of hyoscyamus 1 - 2 grains is specially recommended by Burney Yeo (Manual of Medical Treatment vol II p. 393) who states that he often found this drug more useful than arsenic in "florid, well-nourished children whose chorea has been apparently of rheumatic origin, as well as in some of the hysterical forms.

Fagge (Textbook of Medicine 1901 vol I p. 394). "For many years sulphate of zinc has been largely used in Guy's Hospital, one grain doses used to be given at first which were gradually increased till the patient took a scruple. It seems more adyisable to begin with a larger dose say 5 grains. This drug seldom causes nausea or sickness more than once or twice. Carbonate of Iron also used to have a great reputation."

Iron.

In anaemic subjects, and as we have seen chorea is often associated with anaemia, which is probably due, as D. B. Leas points, to Rheumatism, iron may advantageously be combined with arsenic and if from any cause the administration of arsenic has to be stopped iron should be continued. In weakly children Syrupi Ferri Phosphatis either alone or combined with cod-liver oil is very useful. Phosphate of Lime: Cod-liver oil, either itself as an emulsion, malt preparations etc. are all useful in certain cases either along with arsenic or given when the arsenical treatment /
treatment has been stopped.

Strychnine.

Trousseau (Lectures on Clinical Medicine) recommends the use of this drug. It is difficult however to see how it could be useful in the early and acute manifestations of the disease. It is useful in cases of nervous exhaustion and debility in the later stages and when the element of paralysis is marked.

Quinine.

This drug has been largely used with good results by H. C. Wood and it has been well spoken of by many American Physicians but does not find favour in this country.
D. B. Lees (British Medical Journal 1903 vol II. p. 451) strongly recommends the use of sodium salicylate in the treatment of Chorea. He gives large doses, saying that the disease should be treated so, as cerebral syphilis is treated with large doses of Potassium Iodide. The dose for a child of 6 - 10 years should be at first 10 grains with 20 grains of Sodium Bicarbonate. After two or three days the dose should be increased to 15 and 30 grains respectively. After two or three days more the dose may if necessary be increased to 20 - 40 grains. The doses should be given every two hours during the day and every three hours during the night, ten doses in the twenty-four hours. Thus the total amount of sodium salicylate given daily is at first 100 grains, then 150 and later 200 grains.

A careful watch is necessary for salicylate poisoning and especially for a condition resembling air-hunger of diabetes. If this occurs the medicine must be at once stopped.

He considers it very important that each dose of the salicylate should be accompanied by twice as much of the bicarbonate. Occasionally vomiting is troublesome but is usually overcome by stopping the treatment for several hours and then beginning with a smaller dose which should gradually be increased. He states also that pulse failure occurring in children and adults is not generally due to the remedy but is caused by acute rheumatic dilatation of the left ventricle. Children, he says, often become much brighter /
brighter with this treatment. Albumuriae does not occur. He recommends a diet of milk with complete rest in bed.

Langmead (Salicylate Poisoning, Lancet vol II 1906 p. 1822) calls attention to certain dangers which attend the administration of very large doses of salicylic acid.

He first states that D. B. Lees in the Harvein Lecture of 1903 records one case who had large doses of salicylate and in whom symptoms of diabetic coma developed.

These he looked upon as evidence of acid poisoning and gave double the dose of sodium Bicarbonate with each dose of Sodium Salicylate as a prophylactic.

Langmead then describes the symptoms of acid poisoning:- "Drowsiness deepening into coma and if untreated ending in death, air hunger of the Kassmall type increasing with the drowsiness. The child is flushed, the eyes are bright and there is usually great thirst, vomiting usually but not always precedes these symptoms. The drowsiness may be replaced or be associated with delirium."

The urine in all these cases where these symptoms showed themselves was examined by Langmead and showed acetone and he did not get Legal's test for acetone in any of the cases treated with salicylic acid which did not exhibit signs of the poisoning. The sweet odour of acetone was present in the breath of all the severe cases.

He states that there is great variation in the amount required to give toxic symptoms and that therefore the drug should be given cautiously at the commencement so that the personal factor may be estimated and any idiosyncracy detected.

Another /
150.

Another noteworthy feature in all the 8 cases recorded was that constipation was a very marked feature so that greater accumulation of the drug in the system may have taken place, pointing to the necessity of maintaining a free action of the bowels during salicylate treatment.

Langnerd states his conclusions as follows:—

"(1) Salicylate of Soda sometimes causes in children symptoms resembling the acid-poisoning symptom of diabetes.

(2) The toxic dose is variable depending upon the idiosyncrasy of the patient and the presence or absence of constipation.

(3) Acetone may be detected in the urine and the breath, its presence constituting one of the first symptoms of the poisoning and affording a valuable danger signal.

(4) Treatment should be directed to keeping the aciidity of the urine low and the bowels opened in cases of patients taking this drug.

(5) If acetone found in the urine sets more and more strongly acid, the salicylate should be omitted and the alkali given alone."

He carefully examined the salicylate as regards purity and was quite satisfied in that respect and he mentions a case where the same symptoms occurred with aspirin.

He considers it possible in these cases that two factors are at work which in some way produce auto-intoxication (a) salicylic acid (b) the diplococcus of Poyton and Paine which is known to be an acid forming organism.

Berg (loc. at) considers Sodium Salicylate the most useful drug in the disease and says it should be given /
given in moderate doses every five hours until slight ringing of the ears is produced and then he stops the drug till this ceases.

Zanssiroff (Wraschnaja Gaz. 1904 no. 6) says that he obtained striking results with salicylates in cases in which the rheumatic diathesis was thought to be entirely absent.

Williamson (Lancet vol II p. 526 1903) records 35 consecutive cases treated with aspirin.

He commenced with 10 grains as a powder twice a day in children over seven years of age and rapidly increased up to 10 - 15 grains (according to age) four times a day. In children under seven years of age he gave smaller doses.

It is important to look for toxic symptoms and to discontinue or diminish the drug if these disappear.

The poisoning symptoms resemble those of the salicylate group but they do not often occur and most children take these large doses with no ill effects. Sometimes the drug causes gastric irritation and vomiting even in doses of 5 grains. This is probably due to some impurity in the drug or idiosyncracy of the patient. It is important that no alkali should be given just after the aspirin as it is then liable to be decomposed in the stomach and produce gastric symptoms. He usually gave the drug in water to which a few drops of lemon juice were added. It was not necessary in any of the 35 cases mentioned to discontinue the drug because of any untoward symptoms. He believes also that it is only when the doses mentioned can be taken without discomfort that the action of the drug can be satisfactorily attained.

Amongst /
Amongst the 35 cases treated with aspirin, these were cases of very long duration in which the symptoms persisted unchanged until this drug was given; decided improvement first occurred under treatment with aspirin and complete recovery followed, after other methods of treatment had been tried without effect. Also in some cases seen soon after the onset of the disease, improvement rapidly occurred when the patient got aspirin in large doses and complete recovery soon followed.

He also states that while all the patients were kept from school none of them were confined to bed and he holds that the favourable results were therefore not attributable to the influence of rest. Though the drug does not immediately check the choreic movements its administration is usually followed by distinct improvement in six or seven days after a dose of 10 - 15 grains four times a day has been reached. This improvement steadily continues until complete recovery occurs.

Besançon and Paulasgeo obtained recovery in 10 cases in three or four weeks from aspirin given in 15 grain doses twice daily for five days and then omitted for three then given again for five days.

Garmangig (Phar. w. Ther. Rund's. March 1906) gives even larger doses increasing the aspirin gradually up to 46 grains daily for short periods. He used as much as 77 grains in one day in a case of pregnancy chorea with good results.

Burnet (Lancet vol. I p. 1194 1905) in a paper on the therapeutics of Aspirin and Mesoten speaks of aspirin in the treatment of chorea which he says may be taken as a rheumatic affection in practically every /
The first case of Chorea I treated with aspirin was that of a girl aged 13 years. She had been under treatment previously for the same condition. She was small for her age and had a definite history of rheumatism. There were marked mitral presystolic and systolic murmurs and the heart was considerably enlarged. She was moreover very anaemic. It was considered advisable to keep her in bed and put her on iron and digitalis as she suffered considerably from breathlessness. Under this treatment she improved slightly but at the end of a month I could not pronounce her condition as satisfactory. Accordingly the iron and digitalis were stopped and instead I ordered 10 grains of aspirin to be given every four hours. She began almost immediately to improve so that in three weeks time she was comparatively well and in two months was completely restored to health. Even the anaemia was scarcely perceptible although the cardiac murmurs remained."

He further states "I have yet to meet a case of chorea in which aspirin given in suitable doses failed to bring about alleviation of the patient's condition. The dose must be 10 up to 15 grains. Children take it well and usually show no toxic symptoms!"

Wall (Medical Press, May 20, 1938) states that he has found aspirin of more value than either arsenic or sodium salicylate. The danger of producing severe vomiting by aspirin can be avoided by giving it only when there is food in the stomach.

Aspirin should never be given in tablet form but always as a powder. In large doses it may produce haematuria.
haematuria.

From what has been said regarding the treatment of chorea by salicylates and aspirin, it is evident that with this class of drug as with arsenic, only large doses produce any striking action in the treatment of the disease. The dose must at least be 10 grains and gradually increased up to 15 and at the same time it is to be given more frequently day by day. The onset of acid poisoning can be determined with sufficient quickness where the case is carefully watched to avoid any serious poisoning effects and if they do occur the cessation of salicylate treatment and the replacing of it with alkaline treatment is usually sufficient to clear up those symptoms.

It has also been pointed out that as indeed is the case in the treatment of rheumatic fever, the bowels must be carefully regulated and the often attendant constipation will in many cases require the use of powerful laxatives.

**Ergot.**

Eustace Smith (British Medical Journal July 18th, 1903 p. 133) gives the Liquid Extract of Ergot in one drachm doses every three or four hours for many weeks together to children of 7 or 8 years without seeing any toxic effects.

He also gives 20 minim doses or more for months at a stretch without seeing any ill effects and in the majority of cases he states the beneficial action has been very decided.

"While taking ergot the pulse falls in frequency perhaps /
perhaps as much as 15 - 20 beats but I have not noticed
dilatation of the pupil or heard complaints of head-
sache, nausea or abdominal pains."

Ergot he considers acts more quickly than
arsenic, never as far as he has seen, has it disagreed
it and has been successful where arsenic has failed.
He gives to children of all ages, one drachm doses
diluted every three or four hours and enforces com-
plete rest in bed.

He advises that the exunctorary organs of the body
should first be examined to see if they are in good
working order before the drug is administered.

It can be pushed, one drachm every 2 hours or so
till the action is obtained and then continued as long
as the spasmatic movements persist.

We now pass to consider those drugs whose action
is hypnotic and we find that numerous drugs have been
used in this direction.

Chloral in Chorea.

This drug is the most commonly used of all the
hypnotics in the treatment of chorea and is probably
the best at our disposal. Bastian and Gairdner have
both strongly advocated its use especially in the
severer types of chorea.

Bastian (Lancet Vol II page 55 1839) gives his
opinions about this drug in a clinical lecture on a
"Case of protracted and severe chorea treated by
prolonged sleep."

The case he reports was one in whom chorea had
persisted for 8 months and was very severe. After a
preliminary trial of arsenic he prescribed for her on
March /
March 4th on awakening 20 grains of chloral hydrate and 10 grains of Bromide of Potassium to be repeated on awakening, the patient only to be allowed half an hour awake at a time for the purpose of taking nourishment and the sleeping draught to be used as seldom as possible.

At first the draught was required every four hours, later on it was required on an average every six hours. Screens were placed all round the patient and she was kept as quiet as possible. The movements during sleep became rarer and rarer toward the end of two weeks. During her waking periods she complained greatly of headache and was very emotional. Her temperature and pulse were fairly constant all the time. On March 18th she was allowed to awake and the movements were distinctly less but she complained greatly of headache. On March 23rd she was again put under chloral till April 16th when she was allowed to awake: the choreic movements had decreased very considerably the hands being almost still unless she was excited. Her mental condition was however confused and remained so for 13 days. She was very depressed and emotional and suffered from delusions. On May 3rd she could knit easily and was discharged cured two weeks later. His conclusions may be summed up as follows:

(1) This treatment is not suitable for ordinary cases of chorea.

(2) In the most acute and severe cases in which the temperature is raised and the movements are violent and continuous with or without delirium or maniacal symptoms it is useful.

(3) He however considers it especially applicable to cases where there is no fever and no heart disease.
disease but where the movements are unusually severe and continuous and have been so for months.

(4) He attaches little importance to any supposed curative influence of chloral hydrate over the chorea, except through the intervention of the sleep which it produces.

Gairdner (Lancet 1889 vol II p. 205) reports a case which he treated with chloral hydrate in 1870 when great improvement followed the treatment.

During the course of treatment with chloral hydrate an overdose of 60 grains was accidentally given which kept the patient in a very somnolent condition for 24 hours. After recovery from this she never showed any signs of the disease even after the effects of the excessive dose had completely disappeared.

His conclusions as regards the usefulness of chloral hydrate are as follows:

(1) It sometimes succeeds absolutely in chorea where other drugs fail.
(2) It can be depended on as a rule in very severe cases to initiate a treatment which may successfully be carried out otherwise.
(3) In such cases it has an almost absolute power of suspending or controlling spasm during the persistence of its deep hypnotic action and is therefore invaluable as a palliative (care being taken to avoid poisoning acute or chronic.)
(4) This or other limitations will interfere with the curative action of the remedy in some very in-veterate cases; the failure of the chloral hydrate /
hydrate in these cases being however common to it with all other remedies.

In some cases chloral hydrate is undoubtedly very useful, but cardiac depression and gastric irritation has to be carefully watched for. It also sometimes fails as the movements return after the drug is stopped and it may mean a very protracted treatment. The mental disturbance also which may occur after it lessens its usefulness.

Hutchison (Index of Treatment page 639) considers that for the most serious cases, the most valuable drugs are chloral hydrate and bromide of potassium, and that it is seldom that any signs of cardiac failure present themselves. The chief difficulty is in leaving off the chloral for experience shows that when it is omitted there is considerable nervous disturbance. This is most likely to occur when the physician has been alarmed by signs of collapse and on this account has abruptly stopped the chloral. In most cases the difficulty can be overcome by cautiously diminishing the dose and substituting occasionally a small dose of another hypnotic such as veronal. For a child of 7 years he gives chloral hydrate grs VII
Potassium bromide grs X aqua chloroformi $\frac{3}{4}$ ss
He gradually increases to 10 grains of chloral if necessary and also gives a stimulant with it. After this treatment he gives arsenic.

Chloral may also advantageously be administered per rectum in 10 - 30 gr doses.

Essex Wynter (British Medical Journal Vol II 1908 p. 912) states that since Autumn 1905 he has used /
used chloretone. With such success that he talks of it as the specific in chorea.

He describes first the chemical and physical characters of the drug. It is a permanent chemical compound, not decomposed by heat or light and stable in the presence of dilute acids and alkalis. It is a white crystalline powder with camphoraceous odour and taste.

He considers the best vehicle in which to administer chloretone is petroleum emulsion or glycerine.

Physiologically chloretone acts in therapeutic doses as a powerful sedative to the nervous system without depression of the circulation or respiration.

He states that he used it in 5 grain doses in half an ounce of petroleum emulsion, sometimes sweetened by glycerine, every 4 - 6 or 8 hours according to the severity of the attack and the age of the patient. When the movements lessen after 2 or 3 or 4 days the dose is to be reduced to one half and it may also be given less frequently.

In this series of 50 cases treated by chloretone the duration of treatment averaged 9 days, covering the period of choreic movements. In the majority 4 - 6 or 7 days sufficed. There were several cases which lasted 12 to 20 days and were instances of cases where the disorder had resisted treatment for a long period. In some of his later cases he had cases to whom the drug was given for two days in 5 grain doses and for two other days in 2½ grain doses, a four days in all.

The average stay in hospital was three weeks even /
even where there was valvular heart disease due to previous rheumatism. He only knows of a relapse in one case after three months. The value of laxatives in assisting the action of the chlorretone is emphasised. They doubtless accelerate the absorption of the chlorretone.

He also enjoins continuous rest in bed, liberal diet, isolation behind screens and encouragement to do some finger work when the involuntary movements have subsided. After the chlorretone, steel wine with cod liver oil or arsenic was given.

In speaking of the sedative effect of the drug on the nervous system he says that it is liable to extend to a condition of stupor if the drug is given in excessive doses too frequently or over too long a period. This occasionally occurred, the child sleeping profoundly till roused to take food or for other purposes and permitting the limbs or body to remain in almost any attitude in which they were placed as in catalepsy but without either actual rigidity or loss of muscular tone, the state passing off within a few hours of discontinuing the drug."

Erythema and peeling of hands and feet may also occur in rare cases.

The 50 cases he had were fair examples of the disease, some were unable to speak or feed themselves and a few required padded cots on account of the violence of the movements.

Chlorretone.

Voelcker (Folia Therapeutica April 1903) after a trial of arsenic, ergot and Sodium Salicylate in large doses, considers that in uncomplicated chorea, sedatives /
sedatives give the best results. Chloretone especially he found very useful, but it is apt to make children too drowsy, and sometimes produces an erythematous rash and the eyes get a puffy appearance as in whooping cough but without albumenuric.

Bromural has not seemed more useful than chloretone.

Antipyrin.

It was introduced by Walner in 1887 who described the action of this drug. It was given in doses gradually increasing up to two drachms in the twenty-four hours. Its chief use is during the acute stage and even children tolerate it for weeks. Apart, however, from the more usual signs of intolerance, the urine must be carefully watched for the appearance of albumen and if this appears the drug must be stopped.

Antefibrin, asaprol etc. have also been used but show no advantage over antipyrine.

Moncorvo (Journal de Clin. et de Therap. Paris 1896 IV page 967) is prominent among those who have advocated the use of this drug, Antipyrine.

Hubrect (These de Paris 1895) also recommends this drug. It is chiefly useful in cases of moderate severity with no complications. It should at once be discontinued if signs of intolerance and especially of albumenuric appear, but these events appear to be rare and were not observed in Hubrect’s cases; marked anaemia malnutrition and active cardiac and rheumatic complications are contra-indications to the use of this drug.

Jules Conby (Archives de Med. des Enfants April, 1899)
1899) recommends this drug also.

He gives it in large doses 14 grains for every year of the patient's life. He considers that doses of 4 - 8 - 15 grains according to age should be given at the commencement and repeated 2, 3 or 5 times a day. He states that it may be continued for 3 weeks without ill effects (La France Med. et Paris Med Sept. 6th 1895).

Leroux (Revue Mem des Mal. de L'Enfance) states that antipyrine had a beneficial effect in 49 out of 59 cases but in three fourths of these the affection recurred. Where the drug failed, it was due to intolerance or cutaneous eruptions, but in a few cases it seemed to have no effect. It was necessary to give large doses, doses from 1 to 14 drachms which were well tolerated for some weeks.

S. D. Hopkins (Philadelphia Med. Jour. Aug. 19th 1899) says that satisfactory results are to be got when the drug is given in increasing doses beginning with 1 grain for every year of the patient's life and increasing 1 grain per day. In the mildest cases antipyrine is only given at night and the child may be allowed to sit up during a part of the day but when the case is severe, absolute rest in bed is essential, the antipyrine being given three times a day. The drug is stopped as soon as the choreic movements cease or greatly diminish. Fowler's solution and Iron are given for two or three weeks after the cure appears to be complete.

In giving such doses (20 grs t.i.d.to a child of 8 years) and the child must be kept in bed and very carefully watched.
He considers that is should not be given where there is heart disease or any fever. He reports rapid cures in 19 cases who were treated after this plan.

Langovin (New York Medical Journal Feb 8th 1906) recommends gradually increasing doses of antipyrine but this must be stopped if albumena, weakness of the pulse or other toxic manifestations appear.

**Exalgin.**

Collins (Har's System of Practical Therapeutics vol II p. 763) speaks very highly of exalgin. He has pushed the dose up to 30 grains in the twenty four hours. He admits however that the administration of large doses is sometimes attended by very serious symptoms, while its haemolytic action is well known.

**Brometone.**

This has been strongly recommended and is given in doses of 2 grains thrice daily to children of 6 years.

**Apomorphine.**

Tull (Pediatrics August 1905 p. 523) reports a case where in spite of veronal, opium, valerian and arsenic, the violent movements continued until apomorphine gr. one fortieth was administered hypodermically. The movements rapidly subsided and the apomorphine was continued one twentieth gr. by the mouth every 3 hours with arsenic.

**Tartar Emetic.**

Grenet /
Grenet (Arch. Gen. Med. May 1915) recommends emetics in chorea, but would use Tartar Emetic as less dangerous than apomorphine for young children. He gives it once daily for 3 consecutive days in doses respectively of 0.02 c. gms, 0.03 c. gms and 0.04 c. gms. then after an interval of 3 to 5 days another series of doses are given for 3 days 0.03 c. gms, 0.04 c. gms 0.05 c. gms. It may be necessary to give even a third course.

Sulphonol is not a very satisfactory drug, its action is uncertain and depressing.

Trional.

Vogelker (Folia Therapeutica April 1908) considers trional to have a very beneficial effect in some cases of chorea. The only unfavourable result was rather vivid dreams.

It may be prescribed thus.

Px. Trional grs XV.
   Pulv. Sacch. All. II
   Gum Tragacanthae III
   Gum. Arab. III
   Aq. Flor. Aurantia II gs.
   Aq. Lauroserasi 3 s.s.

M. ft. emuls. One third part to be taken in milk or water as a single dose.

Five grains of trional may be given three times a day to a child over four years, but the dose should soon be increased to 5 grs every six hours, or even every four hours.

It is more advantageous to give the smaller doses at
at shorter intervals than larger doses at longer intervals.

Paraldehyde has not shown itself to have any special advantages and the difficulty in masking the taste renders it unsuitable for administration to children especially. A small piece of asaccharine pellet is perhaps as good as anything to help to get over this difficulty as far as it possibly can be surmounted.

Chloralose has also been used in much the same way as chloral.

Hyosine.

Hyoscynamine, Hydrochlorate or Hydrobromide of Hyosine administered by hypodermic injections have also been found useful in severe cases.

Taylor (Nervous Diseases in Childhood p. 260) states that he used it with excellent results in less acute cases which had proved intractable under other treatment. He used Hyosain hydrobromide and injected one hundredth grain three times a day. "As a rule the injection was immediately followed by wide dilatation of the pupils and slight flushing and sleep lasting from one to two hours. It is contraindicated in acute cases and in cases where there is endocarditis or where the patient is badly nourished. Indeed in all such cases food and alcohol are the only drugs admissible."

Bromides.

They are of little use in the treatment of this disease
disease except in combination with chloral when they may avert the headache which chloral is apt to produce. Good results have however been obtained from them in Chorea Gravidarum.

Chloraldehyde.

This drug has been found efficacious as a hypnotic in 10 to 30 grain doses. Garrod (Clinical Journal Feb. 8th 1905) has used it, and states that it seems to quiet the patient and induce sleep, a very important matter in severe chorea. He considers chloral the most useful drug in bad cases of chorea. He also states, however, that in bad cases of chorea where drugs are being pushed, a sudden cessation of all treatment is sometimes followed by immediate and rapid improvement.

Hedonal.

This has been used by Varras (Annales de Med. et Chir. July 15th 1834). He gave the drug in doses of 7 to 15 grains in the form of a powder suspended in a little sweetened water. He gave it to older patients in the form of cachets. In some cases he states that cure resulted within a week.

Camphor Bromide.

Bourneville and Katz (Progress Med. June 16th 1833) reports a severe typical case of Sydenham’s Chorea rapidly cured with camphor bromide increasing from 1 to 2½ drachms a day during twelve days, again decreasing to 1 drachm during the next fifteen days. They also recommended that the dose should be 3gs. given in /
in capsules.

**Monobromate of Camphor.**

Bernardo (Il. Polieo. Oct. 4th, 1903, p. 1267) relates an instructive case where this drug proved very useful. In a child of 9 years arsenic had been tried for a week and failed to arrest the disease, and then monobromate of camphor was given 15 grains daily and pushed to a maximum of 30 grains daily. After four or five days the movements began to abate. The monobromate of camphor was then stopped and arsenic substituted: the patient at once relapsed. After a few days the camphor salt was resumed and again pushed rapidly to 30 grains daily. Improvement at once took place, and in the course of 8 weeks the patient was cured, no relapse having occurred two years later when the case was published.

**Physostigmine.**

J. W. Russell (Birmingham Medical Review Sept. 1903) reports upon the use of this drug in two extremely violent cases of chorea. His results, he states, were better than those usually obtained by arsenic. The extract of physostigma was given in doses of one-sixteenth grain three times a day.

**Morphine.**

This drug should if possible be avoided in all cases.

Churton, (British Medical Journal March 24th 1894) has recorded a case in a girl of seventeen years, in which after the failure of chloral in doses of 20 grains
grains, frequently given, to produce sleep and restrain the movements, he succeeded in controlling the movements completely by giving morphine hypodermically followed by the inhalation of chloroform for a few minutes. He began with one-sixth grain of morphine but had to increase the dose to a grain before he secured the desired result.

**Chloroform Inhalation.**

This may be necessary in the very gravest cases when the movements are violent and uncontrollable. It is necessary to get the patient well under otherwise he would soon awaken and the amount of chloroform used would be greater.

*Cimicifuga and conium* are of use in the treatment of chorea, the former having been chiefly used in America.

Whitley states that the conium is very useful from its sedative properties and he considers it kept a child alive until the arsenic had time to act in one case in which he used it. Its action however is very evanescent.

**Alcohol.**

Sleeplessness is the rule in severe cases and Hughlings Jackson considers this to be due to exhaustion caused by incessant movement and lack of nourishment.

Taylor (Nervous Diseases in Childhood and Early Life) supports this view. He says "Enquiry will generally /
generally show that if a child has not slept well for three nights, it has had no adequate nourishment for four days. In this sleeplessness of starvation, sedatives such as morphia, chloral and bromides are useless and may be harmful, but the administration of a proper meal by the nurse combined with a full dose of alcohol is in most cases followed by sleep.

The use of drugs for sleeplessness in chorea is to be avoided and is for the most part not necessary. Indeed these may do positive harm and should be rigidly avoided in severe conditions. Chloral however is sometimes of value in less severe cases where sleep is broken.

Anti streptococci serum.

Stresser (Pediatrics Feb. 1916) used this serum with benefit. Fifteen injections of the serum were sufficient to cure a case where streptococci were present in the blood. Arsenic, chloral, and bromide had been previously tried without effect. He recommends the use of polyvalent anti streptococcal serum.

Lymphatic gland extract.

Vidal (British Journal of Diseases of Children, May 1906, p. 244) reports that he used with success hypodermic injections of lymphatic gland extract in a girl 11 years of age who was suffering from severe chorea. No relief had been obtained from antipyrine, wet packs, and tonics.

Counter-Irritation to the Spine.

Freezing the skin by the ether spray are not beneficial /
beneficial and not to be recommended. No good seems to have resulted from Hypnotism.

Intra-Spinal Injection of Magnesium Sulphate.

Marinescu of Bucharest (in British Medical Journal vol II 1908 page 1707) recommends the use of magnesium sulphate by intra-spinal injection.

Weltzer and Auer had recommended this treatment for Tetanus (Journal of Exp. Medicine, December 1906 p. 629).

Marinesco employed their 25% solution of the salt but lays stress on its being crystallised and pure and on the solution being prepared shortly before it is required for use.

The dose is that recommended by Weltzer and Auer namely 1 c.c.m. of the solution for each 25 lbs of the patient’s weight. He tried it in 4 cases.

In case (1) He gave two injections of 3.5 c.c.m. but the disease lasted ten weeks.

In case (2) Choreic movements ceased three days after injection of 5 c.c.m. of the solution.

In case (3) The duration of the disease was just about the average length.

In case (4) He gave two injections to a girl who had been ill for some time, and a cure resulted in 7 days.

In two of these cases there is evidence that the remedy had some direct and almost immediate effect.

The injection caused headache, which was persistent and required hypodermic injections of morphia for its relief.

There //
There were other complaints of tingling, numbness and inability to move the legs and nausea; the temperature occasionally rose one or two degrees and in one case there was somnolence.

He suggests the advisability of using a weaker solution and proposes one of 7.3% strength which is isotonic with the cerebro-spinal fluid — 3.2 c.c.m. of the solution equalling 1 c.c.m. of the 25% solution.
Marinescu (Sem. Med. November 1903) records other four cases of chorea which he treated by introspininal injection of Magnesium sulphate. He used a 25% solution of freshly prepared crystalline magnesium sulphate and generally speaking he withdrew some cerebro-spinal fluid before he injected his solution.

Case (1) A girl, aged 14 years, with general chorea which had lasted for over a month, had injected into her spinal canal on July 5th 3 c.c.m. of the solution and within three quarters of an hour the choreiform movements had greatly diminished. The movements however returned and persisted and on July 30th another 3 c.c.m. was injected. Half an hour later the patient complained of headache and formication in the limbs and back. By August 2nd the movements had completely disappeared.

Case (2) A girl, aged 22 years, with general chorea had 5 c.c.m. of the solution injected into spinal canal. Her pulse soon fell from 104 to 63 per minute and in one and a half hours her lower extremities became numb and she could not move them. A few hours later the patient slept and her pulse rose again to 106 and her temperature was also raised. Three days later the numbness disappeared and the choreic movements did not return.

Case (3) A girl, aged 15, who had had chorea for two months, had injected intro-spinally 3 c.c.m. of the solution on August 14th. The choreic movements diminished but had not gone by August 26th when other 3 c.c.m. were injected preceding /
preceded by a hypodermic injection of morphia. The choreic movements disappeared the following day and did not return.

Case (4) A girl, aged 11 years, with moderate chorea which had lasted for two or three weeks, had injected into her spinal canal on September 11th 2½ c.c.m. of the solution, this being preceded by a subcutaneous injection of morphia. The movements lessened considerably. Another injection was given on September 13th, and by September 18th the movements had completely disappeared.

He considers that this is an excellent therapeutic measure in Sydenham’s Chorea and may be employed in both mild and severe cases and recommends that a subcutaneous injection of Morphia should be given before the intra-spinal injection. He has never seen urinary troubles in any case in young subjects.

**Electrical Treatment.**


In what he calls the habit cases of chorea, induced by some trivial local disturbance or reflex influence, not of central origin, static electricity if applied early is universally successful.

He considers it to act in two ways—(1) It lessens the irritability and (2) it acts as a powerful suggestive influence when systematically employed. He further states that most cases of central origin are not due to any traceable organic defect but are induced by functional derangement. Such he states are capable /
If not of too long standing.

"For treatment, a metal electrode covering the affected muscles, is applied and held in position by the hand and the wave current is employed with as long a spark gap as can be used without causing painful muscular contractions. Sparks to the region will also render the treatment more effective in some cases. If the condition is suspected to be of central origin, a large electrode should be applied to the back or the abdomen (as in epilepsy) for an additional 15 minutes for its general effect. Under this régime there are few cases of not more than two years standing that will not yield."

W. F. Somerville (Med. Electrol and Radiol. May 1904) considers that high frequency currents have an undoubted effect in subduing choreic movements.

Electrical treatment has not, on the whole been followed by good results and it is apt to frighten the child and so aggravate the disease. It may however in the later stages of parietic chorea be useful in the form of mild galvanic current applied to the muscles but is not so useful in these cases as massage which in fact may be distinctly useful as an aid to general nutrition in the convalescent stage.

Exercises.

Exercises are not to be used during the acute stages. They are however of service in chronic cases of chorea, where although the patient has regained his general health, the movements still persist. They may be designated Residual Chorées.

In Hutchison's Index of Treatment -- Chronic chorea -- it is recommended that the graduated exercises should /
should be very simple at first. "Taking and crowning the pieces arranged on a draught-board or building houses with small wooden blocks should be practised. Freehand drawing and writing letters with coloured chalk on a blackboard: touching, at word of command, small coloured balls hung at varying heights by strings to a frame: aiming with a pointer at the different circles of a target, are all useful and easy exercises as a commencement. More difficult are building card houses and setting backgammon pawns in holes drilled in patterns into a board. Threading needles, sewing and writing in small text should be left to the last. No exercise should be employed if it tires or bores the patient.

For slouching, clumsy gait, ungainly attitudes lumped shoulders, dropped chins — (all of which are sometimes the sequelae of chorea) — ordinary drills are essential."

General Considerations as regards the use of drugs.

Believing that chorea is a manifestation of rheumatism in the great majority of cases, it naturally follows that the treatment, at least at the outset, should be vigorously anti-rheumatic, in the hope that it will not only subdue the choreic movements but also lessen the tendency to permanent endocardial changes. After a preliminary purge, the patient should, as early as possible be put on salicylate treatment, either sodium salicylate with double the quantity of Sodium Bicarbonate or preferably aspirin. The dose of aspirin, to begin with, should be small but rapidly increased, if it agrees, and it should be continued until /
until the patient has been placed completely under
the action of the drug, careful watch being maintained
for any toxic symptoms. As Williamson points out the
dose should at least be 10 - 15 grains, given at
first perhaps twice a day and then more frequently,
according to the severity of the case and the age of
the patient. During this treatment, the patient
should be confined to bed and given a milk diet. The
bowels should be carefully regulated and a daily
motion secured. Hypnotics, if it is at all possible,
should be avoided and careful feeding at short inter-
vals, (small quantities at a time being given) . .
attended to. If sleeplessness is a very con-
spicuous symptom and required special attention then
a luke warm bath at bed time may first be tried, and
if drug treatment become essential then chloral in
10 grain doses with some bromide of potassium may be
given. It is interesting when on the subject of
sleeplessness to note that Taylor considers it often
to be due to starvation and if so, he recommends a
good meal to be carefully administered along with a
full dose of alcohol at bed time.

If after this treatment the movements still con-
tinue then chloretone might be used as suggested by
Essex Wynter in the belief that, after the rheumatic
poison has been subdued by the aspirin, the persisting
choreic movements are due to the instability of a
nervous system irritated by the rheumatic poison.

In cases which are very severe from the outset
then the use of sedative drugs may at once be necessary
and of these chloral seems to be as good as any. It
must be used freely, watching the heart, and not given
up /
up too abruptly, and it is well to give some stimulants when this treatment is resorted to. The "wet pack" in these cases is often very soothing.

Gentle massage may be used all during the attack unless it is very severe but exercises should never be commenced until the patient's general health is completely re-established.

Prognosis of Chorea.

The prognosis in uncomplicated cases of chorea in children is almost invariably good and this applies even to paralytic cases. Of the 439 cases in the British Medical Association Investigation Report only 9 deaths occurred, a death rate of only 2%. While the immediate prognosis is very good as regards recovery from the manifest symptoms of chorea, in the shape of irregular spasmodic movements and psychical disturbance, the ultimate prognosis is not nearly so satisfactory. Osler has shown how frequently in after life those people who have suffered from chorea, develop definite organic disease due to the endocarditis so often occurring in the choreic attack. Batten has also shown how often other definite signs of rheumatism occur subsequent to an attack of chorea in those patients who had no previous history of rheumatism.

According to Anstey the disease is more grave after puberty, but as Taylor (Nervous Diseases in childhood and Early Life, p. 255) points out, the statistics of the in-patients treated at the National Hospital between the years 1883 and 1903 do not support this view.

Sturges /
Sturges considers the most fatal period to be about puberty and especially in those cases which have shown a definite rheumatic affection.

The prognosis is also grave in chorea in pregnancy. The majority of deaths from chorea in pregnancy result from abortion, whether spontaneously or artificially produce, or because of the extreme debility which commonly ensues in such cases. A labour at full term may be fatal.

Sturges considers that the more pronounced the psychical disturbances, the more serious is the prognosis. He also considers that chorea as a fatal disease belongs almost exclusively to puberty and especially female puberty. Even however, in cases where great psychical disturbance is present, many such cases recover. The danger is that some permanent mental deterioration may follow, but mental defects have been known to persist for months or even years, and ultimately to pass away. It must also be remembered that epileptoid attacks during chorea may develop into true epilepsy.

Apart from complications the chief dangers are exhaustion from want of sleep due to the never ceasing violent movements, and interference with the patient's nutrition and even with the very act of taking food. The prognosis in those severe cases where there is difficulty in feeding, because of constant excessive movement and where sleeplessness is also present, and where the movements are so liable to cause wounds, depends greatly on the skill of the nurse. By good nursing even those cases may make a good recovery.

The duration of the disease is very variable. The usual duration is supposed to be 6 - 10 weeks.
weeks or even more. First attacks usually last longer than subsequent ones. Sir William Gowers also shows that the more severe an attack, the longer will be its probable duration; that a recurrence will probably terminate sooner than a first attack and that the etiological conditions of age and sex and preceding rheumatism or heart disease and present endocarditis afford us no indication of the probable duration of the disease.

Paretic choreas are often very obstinate cases to cure although they present little or no danger to life. Post-chorea paralytic is sometimes very well marked but in the absence of evidence of occlusion of a cerebral vessel complete restoration may confidently be expected.

Hysterical cases often persist for a long time but never end fatally.

The prognosis in a recurrence of chorea is rendered more grave if there has resulted from the previous attack or attacks any cardiac organic disease. The gravity of the prognosis depends upon the extent of heart lesion and the compensatory powers which are present.

Prophylaxis.

The typical subject for chorea is a bright intelligent active-minded restless hypersensitive girl who as a rule is acutely anxious to do well at school and strains every nerve to do so. She is unduly affected by punishment.

Sturges maintains also that the necessity for movement is a natural one to childhood and observation or /
or attempts at suppression do not remove but aggravate it.

"What" he says is "really wanted, is not that they should be kept still which is a task beyond their years but they should learn to move with motive, that their limbs should be regarded as instruments awaiting employment and having many uses simple and complex. They will learn to use as they will learn to still first one department of the body and then another in an order which nature herself dictates."

Children of this type should not be allowed to compete at school for prizes and dropping back a year in their classes will, by diminishing the intellectual effort required, often prove of decided benefit not only for the time being but for all after life. They should be encouraged to spend as much time as possible outside engaged in healthful exercises which prove attractive. A musical accompaniment will often prove useful in interesting and encouraging girls to perform exercises.

An endeavour should be made to get them fattened up and for this purpose a plentiful, easily digested nourishing diet should be given them, as Clouston recommends.

They should also be thoroughly overhauled in order to ascertain if there are any sources of peripheral irritation which reflexly might act on their unstable nervous system.

Such neuropathic children should be carefully watched till they are about twenty years of age, dances, theatres etc. should be avoided as far as possible and golf etc. indulged in instead.

While /
While it is important to carry out these measures in all children of a neurotic temperament they should especially be observed in those where there is either a personal or family history of rheumatism. It has been pointed out by Dyce Duckworth, Still and others that rheumatic children are especially emotional and of unstable temperament, very hypersensitive and very subject to terrible frights and violent emotional outbreaks.

These children ought always to be well clad with warm woollen undergarments and well shod. Especially should they be carefully watched during the winter and spring months, and endeavours made to protect them from unnecessary exposure to cold and damp. There should be no ill-advised attempts to "harden" them by cold bathing in the winter time and the absence of thick warm stockings. Weir Mitchell (Clinical Lectures on Nervous Diseases) recommends that in the Spring months where there is the least fear of "the habit of vernal recurrence of chorea" arsenic should be administered.

Any departure from normal health should be treated at once. If they become anaemic, iron should be administered and in all the use of cod-liver-oil during the winter and Spring months is an excellent measure. They should have a liberal supply of cream, eggs, etc. and their bowels should be carefully regulated as these children often suffer from constipation.

If cod-liver-oil does not agree, Scott's emulsion or Savoury and Moore's pancreatic emulsion may be used. Any change in the child's temperament in the shape /
shape of irritability, or the occurrence of erratic behaviour, or an undue depression and lack of buoyancy with other physical signs such as increase in anaemia, of headache etc. as well as any of the usual manifestations of rheumatism, should be promptly treated with the salicylates in effective doses. D. B. Lees has shown how well the majority of these children can tolerate large doses of the salicylate when combined with twice the dose of Sodium Bicarbonate and how instead of showing depression from the use of the drug, they may become much more lively and cheery. At the same time they should be sent to bed and have an efficient laxative given and guarded from any excitement. If they suffer from any illness which lowers their vitality they should not be allowed to romp about too soon with other children.

One of the cases described at the end of this thesis shows how a girl treated for Scarlet Fever for 7 weeks, was during that time completely isolated, but immediately developed the disease when she was brought down stairs and allowed to romp about with her brothers and sisters.
RECORD OF CASES.

Name
M-- P--
age 13 years.
Address: 50 West High Street, Buckhaven,
Case taken January 15, 1909.

Previous History.
Typhoid Fever when a child.
History also of a severe fright about 2 weeks previous to the onset of the chorea. She got lost in the dark.
No personal history of rheumatism. Growing very rapidly.

Family History.
Rheumatism in the father.

Present Illness.
Spasmodic movements noticed first in her right arm, face and then leg. Difficulty in raising her knife and fork and inability to arrange her hair. She also broke several dishes before the real nature of the disease was discovered. Spasmodic movements soon became general although always worst on the left side. The paresis is not marked and came on about the same time as the muscular movements.

Temperature. Normal.
Heart. Pulse rate, 90 and not quite regular.
Definite rough systolic mitral which is to some extent propagated to the axilla and is probably due to endocarditis.

Reflexes. Normal

Psychical Condition.
Emotional /
Emotional and very easily excited. Very wilful and has frequent crying spells.

Sleep.

Sleep is apt to be disturbed by "night terrors". No movements during sleep.

Apetite is excellent and there is no anaemia.

Treatment.

Liquor Arsenicalis in 5 m. doses three times a day after food, and increased to 7 m. when it began to disagree and had to be stopped for a time and then was resumed at intervals. Later general tonics were given. Patient could not be kept in bed. No efficient nursing.

Duration. 4 months.

Remarks.

The element of hysteria seemed to be present to some slight extent in this case. Arsenic did not prove itself to be of any benefit in this case at all. She was a very bright intelligent girl. The want of rest in bed seemed to have a considerable influence in retarding the recovery from what was only a moderately severe case.
Name M--- E---
Age 7 years.
Address 3 Randolph Street, Buckhaven, case taken January 20th 1909.

Family History.
Definite articular rheumatism in her mother who died from organic heart disease.

General History for last 2 years.
Two years ago patient had an attack of chorea which lasted from March till May when she recovered completely. She went back to school in September and was very anxious about her lessons. She was given a piece of poetry to learn in order that she might recite it before the class. She became intensely excited and in a few days after developed chorea. This proved to be a very serious attack. Her speech left her. Both sides were paralysed and she had to lie in bed for weeks. She was sent to the Sick Children's Hospital and remained there for 13 weeks, being discharged well on the 29th February 1903, and has remained free from choreic movements since but still is unable to go to school and is still quite an invalid. In September of 1903 she developed Scarlet Fever from which she recovered all right, and no choreic symptoms developed after it.

Heart Condition.
Presystolic and systolic mitral murmurs; left heart dilated. Definite Endocarditis.

Remarks.
The second attack was a very severe parotic attack and what is of interest, the occurrence of Scarlet Fever in a child very much predisposed to chorea did not produce any fresh attack.
Name: W.--- T----
Age: 8 years.
Address: Randolph Street Buckhaven.
Case taken: February 5th 1909.

Previous History:
Very mild attack of Scarlet Fever 8 weeks before onset of present attack of chorea. History of fright a week before disease occurred. Rheumatic history also. Nervous type of child.

Family History:
Mother very neurotic.

Present Illness:
Right side affected chiefly. Could not lift a cup, winked and pushed out his tongue. Movements always very slight and paresis also inconsiderable.

Temperature: Normal

Heart:
A little increased in frequency and systolic mitral murmur appeared at 4th week which was not propagated to the axilla.

Psychical Condition:
Very capricious about food. Has crying spells.

Sleep:
It is very good and no movements occur during it.

Pain:
Complains of pain in his axilla which is aggravated on moving his shoulder joint.

Treatment: Rest in bed.
Duration: 6 weeks.

Remarks:
A very mild case. Scarlet Fever followed by arthritic pains probably rheumatic. Stammering from which /
which the patient never suffered previous to the onset of the disease, occurred and persisted for 5 weeks after the spontaneous movements had disappeared.

Name N----- A-----
Age 8 years.
Address, Randolph Street Buckhaven. Case taken February 5th 1909.

Previous History.

Rheumatism when a child and in November 1907 chorea. Very nervous temperament. Had just gone to school. She was confined to bed for six weeks and recovered under arsenical treatment. She was then sent for a holiday and came back quite well.

In October 1908 she took Scarlet Fever and during that time (for six weeks) she was isolated and showed no signs of chorea but exhibited chorea immediately after she was allowed down stairs and her brothers and sisters were allowed to play about the bed in which she was lying.

A case of average severity. The choreic movements commenced on the right side, right arm, then gradually became general. As she improved the left side showed considerable paresis and she drags her left foot. The paresis is largely hemiplegic as she can write with her right hand.

Heart.

Heart's action rapid pulse 100 and irregular. Soft systolic mitral, not propagated to axilla. No heart enlargement.

Reflexes /
Reflexes diminished somewhat on the left side.

Temperature. For first week there was a slight daily rise of temperature of only one or at most two degrees.

Speech. Jerky.

Psychical Condition.

Very moody and difficult to please as regards food.

Sleep.

Gets a fair amount of sleep. The movements cease during sleep.

Treatment

Rest in bed and isolation and general tonics. The triple syrums. Cod-liver oil and malt.

Duration 10 weeks.

Remarks.

There was no appearance of post scarlatinal articular rheumatism. While the child was kept quiet and secluded no choreic movements appeared. The excitement which was occasioned by seeing her brothers and sisters after six weeks seclusion seemed to be an exciting cause.

Scarlet Fever would seem also from this case to be a predisposing cause.

Name J---- D----

Age 11 years.

Address 19 Bridge Street, Leven.

Case taken February 15th 1909.

Family History.

Very neurotic parents.

Previous Illness.

Tonsillitis /
Tonsillitis occurred 5 days before onset of the movements.

Present Illness.

He became dull and listless, lost his appetite. Movements first noticed in right arm and then right leg. Very fidgety. The left side never affected to any extent. Movements chiefly confined to the right arm.

Sleep.

Very broken because of night terrors.

Psychical Condition.

Very wilful and capricious about his food and very irritable.

Reflexes. Normal

Treatment.

Liquor Arsenicalis given in 5 drop doses three times a day for 3 weeks and movements practically have gone.

Remarks.

A very mild case. The occurrence of tonsillitis immediately before the attack is especially interesting as indicating the Rheumatic diathesis.

Name M---- G----
Age 9 years.
Address 47 Rosie, Buckhaven. Case taken February 20th 1939.

Previous Illnesses.

Chorea when 6 years old.

Family /
Family History.

History of Rheumatism in the parents.

Cause.

Fright supposed to be the exciting cause but no definite history of it was obtainable. First attack occurred 3 years ago.

It was noticed that she had difficulty in lifting a cup up to her mouth. She was ill for a considerable time and about two months after the onset she is said to have lost her speech completely. She gradually got weaker and took little food. She slept well at night, but about 6 months after the onset she lost all power in her legs and arms in the following order: Right hand and arm, then right leg, and then the other side in the same order as they were attacked by the choreic movements. The choreic movements practically completely disappeared 6 weeks after the onset.

Her memory was very bad. She was treated at home for 2 years and was then sent to Stirlingshire where she greatly improved. She then went to Royal Infirmary Edinburgh in June of last year and remained there for twelve weeks and came home quite recovered.

She has remained well until a few weeks ago when choreic movements had returned to the right side and also to some extent the left side. They are at present not very severe.

Heart.

Pulse 80 and quite regular.

At the mitral area a soft systolic bruit not propagated to the axilla. There is no enlargement of the heart.

Knee /
Knee Jerks.

They are diminished somewhat on both sides.

Psychical Condition.

She is dull and listless and takes little interest in anything.

Treatment.

Iron used because of a very considerable degree of anaemia and later aspirin in 10 grain doses twice daily, when the movements ceased in 2 weeks after the commencement of the drug.

Remarks.

A case of paretic chorea or chorea mollis where the paresis appeared gradually and after the spasmodic movements. The child has never really recovered since the onset of the disease. She walks now but is capable of no sustained exertion and after some time drags her legs when walking. Aspirin proved very beneficial and was taken without any inconvenience.

Name  K---- W----
Age  6 years.
Address  38 Denbeath, Mathil. Case taken February 23rd 1909.

Previous History.

Nothing special to note. Child of very nervous temperament. No history of fright. History of "Worms".

Family History.

Drunken parents. No rheumatic history discoverable.

Present /
Present Illness.

First left arm affected. She let things fall when attempting to lift them.

Her left leg then became affected. Spasmodic movements considerable and paresis marked. She dragged her leg. Her face next became affected and then her right side showed a few spasmodic movements.

Very little paresis on right side.

Temperature Normal.

Heart

No murmurs could be detected but heart's action always rapid, pulse 110.

Reflexes. All normal.

Psychical Condition.

Very ill-natured. Crying spells frequent.

Memory defective.

Sleep.

Sleeps very badly. No movements during sleep.

Remarks.

This case was interesting on account of the rapid improvement which followed the administration (with the necessary preliminaries) of antonin and the expulsion of several nematodes. The choreic movements at once abated to a marvellous extent so much so that no other treatment was deemed necessary. The improvement was so rapid and occurred so soon after the removal of the nematode worms as to leave no doubt that they indirectly at least were a cause of the chorea.
Name: W---- A---- (boy)
Aged: 12 years.
Address: Waggon Road, Leven.
Case taken March 16th 1909.

Family History.
Mother neurotic and father rheumatic.

Previous Illnesses.
Nothing special to note. No rheumatic history obtainable. History of "worms".

Present Illness.
The boy was very restless and could not long sit still: the movements were not definitely choreic but certainly were approaching that. Very cross and irritable.
Temperature was 99°.

Treatment.
Patient at once confined to bed had santonin given after 12 hours light diet which had been preceded by a purge and some round worms were expelled. He then got aspirin grains 10 three times a day and this was kept up for 3 days, the patient meanwhile being confined to bed. He made a complete recovery in this time. There was no appearance of restlessness and the boy’s nature had completely returned to his usual normal condition.

Remarks.
This certainly seems to have been a case where an attack of chorea was aborted by the instant removal of the reflex irritation and the prompt employment of aspirin in effective doses.
Name     W---- C----
Age      15 years.
Address Methil Hill, Methil, Fifeshire.
         Case taken April 5th 1939.

Family History.

No family history of rheumatism discoverable. One sister committed suicide. Another sister has suffered from recurring attacks of great severity of Dermatitis Herpetiformis which is a nervous skin affection.

Previous History.

Patient had his first attack of chorea when five years old and previous to this there had been no rheumatism. The attack was of sudden onset and came on the day after he had an abscess opened by his doctor. The attack was severe and lasted for six months and during all that time patient was confined to bed. Both sides were affected and speech was also lost for some time. The element of paresis was very marked, he had great difficulty in walking for some time after he got out of bed. He improved slowly but recovery was complete and he presented no signs of endocarditis or rheumatism.

He had a second attack three years ago. For several weeks previous to this attack he was very nervous and easily frightened and would not sleep in a room alone even although he was 12 years of age. His left side was chiefly affected and his speech was also affected but paresis was not a marked feature during this attack which lasted three months.

He had a third attack about a year ago (when 14 years old). Choreic movements again chiefly on left side with some loss of speech. He was treated with /
with salicylates and arsenic and recovered completely in eight weeks. Within several weeks of recovery from the third attack of chorea he developed manifestations of acute rheumatism articular and endocardial. He complained of pain in pericardial region and was very ill for six weeks being confined to bed all that time.

Present Condition.

Well developed, works at a pit, plays football. Rather dark in complexion and a little anaemic. Complains occasionally of shortness of breath and palpitation.

Circulatory System.

Heart is considerably enlarged. Apex beat is in 6th interspace 5 inches from the mid-sternal line. There is an Aortic diastolic murmur and a Systolic and Presystolic mitral murmur.

Remarks.

This case shows how recurring attacks, at least regarding the spasmodic movements and paresis, are less severe than previous ones. The rapid onset of rheumatism after the third attack is also very suggestive. The family history shows that he came of a distinctly neurotic family.

Name        T---- M----
Age         26 years.
Address     Jackson Street, Penicuik.

Family History

Mother very neurotic. One sister takes epileptic fits.
fits.

**General History of Patient.**

When a boy he often complained of "growing pains" and sometimes his joints were swollen and painful. When 15 years of age he took chorea. It was at first a right-sided chorea and later became general. It was a very severe attack. His speech was quite unintelligible and the choreic movements persisted during sleep. The paresis was also considerable. The attack lasted for six months. He was treated with salicylates and arsenic. His mother noted especially a peculiar change in his temperament which occurred during this attack and has persisted. He became very irritable, easily excited and sometimes is very passionate. When 29 years of age, while playing football, as he was in the act of throwing in the ball from the touch-line he took an epileptic fit, falling with the ball clasped in his hands. He however returned and took his place again in the game after an interval of half an hour or so. He now suffers from recurring fits which occur at longer intervals as he is under treatment with bromides.

**Remarks.**

This case is interesting because of the neurotic family history and also especially because of the occurrence of the epilepsy after the severe choreic attack. Sir William Gowers has called attention to this combination.
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