The Falling Sickness

A Treatise on

Ideopathic Epilepsy

By

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"The Falling Sickness"

A Treatise on Idiopathic Epilepsy.

I.

Epilepsy in History and Literature.

Epilepsy is a disease, the knowledge of which extends into the realms of antiquity, and this is not surprising because the terrible manifestations of the 'Grand Vais' attack could not escape the most casual observer.

In those times it was thought that the attack must be the work of some agent of demoniacal power and strength.

Perhaps the most concise and vivid description ever written to describe the paroxysm is that in the ninth chapter of St. Luke's Gospel:

"And, behold, a man of the company cried out, saying, "Vulcan, I beseech thee, look upon my son, for he is mine only child."

"And, lo, a spirit taketh him, and he suddenly crieth out; and it teareth him that he foameth again, and bruising him, "hardly departeth from him"."

Examples of this idea are common in the New
Testament and elsewhere. The man who wandered among the tombs bound in chains was very probably an epileptic maniac.

The theory of demonic possession as a cause of epilepsy was not confined altogether to ancient times. Dr. Bucknill holds that even in Shakespeare's time the ceremony of exorcism was not uncommon, and even much later Dr. Webster wrote that at St. Symphonia's shrine at Sheel, exorcisms were practiced, the story being that St. Symphonia, having offended her father, an Irish king, fled for refuge to Belgium. She was overtaken at Sheel and had her head cut off, several lunatics who were present being cured by the horrible sight.

In less civilized countries the belief is still held. In Abyssinia there are 'Boudas' or sorcerers whose business it is to drive demons to whose vagaries diseases especially of an epileptic nature are ascribed.

The word 'Epilepsy' is derived from the Greek ἐπίληψις meaning to seize. It is a very good name in view of the suddenness with which people in seemingly perfect health are struck down. Different ages have had different
names for this disease, and in Tossi's 'Recherches historiques sur l'épilepsie' interesting attempts at explanation of some of these names may be found. The Romans called it 'illustre comitialis' because if any person present at a meeting (comitia) was struck down with this dread disease, it was thought unlucky to continue the meeting.

Another name was the 'morbus Herculeus', probably because Hercules was supposed to be afflicted with it. Epileptics are often very strong and Sir Edward Burnett regards Samson as an epileptic.

Other names were, morbus saecer, morbus mensalid, morbus daemonico, morbus australis.

Not only in holy writ, but in more secular writings of all ages, do we find beautiful descriptions of or references to this disease.

In his 'De Rerum Natura', Lucretius gives the following very vivid picture of the grand mal attack:

'Oft, too, some wretch, before our startled sight
Struck as with lightning by some keen disease
Drops sudden: By the dread attack improwned.
He foams, he groans, he trembles and he faints.
Now rigid, now convulsed, his labouring lungs
Have quick, and quivers each exhausted limb.'
Spread through the frame, so deep the dire disease
Disturbs his spirit, as the long main
Trouns through each wave beneath the tempest-tide,
The growns, since every member smarted with pain
And from his inmost breast with wontless toil
Impuick and hoarse, articulation springs.
The caves since soul and spirit are alike
Disturbed throughout, and secular lack from each
As urged above distracted by the bane.
And when at length the morbid cause declines,
And the fermenting humours from the heart
Flow back - with staggering foot first tread
Led gradual on to intellect and strength.

Coming to poets of more recent date we have
Kennyon who describes in 'The Princess' an attack
Of moral delirium occurring in the hero who suffered
From a sectarian nervous affection.

And, truly, waking dreams were, more or less,
And old and strange affection of the house.
Myself too, had weird secures, leave know what,
On a sudden, in the midst of men and clay.
And while I walked and talked as hitherto
I seemed to move among a world of ghosts.
And felt myself the shadow of a dream.
This passage describes what is very common in a
certain class of epileptic fits, namely what Crichton-Browne calls the dreamy mental state.

The treatment of epilepsy in bygone days was, in many instances, very amusing. A very popular remedy was certain roots mixed with the dust of the crushed skull of a criminal who had suffered capital punishment.

Warm milk mixed with minced snakes' heads was considered a sure and quick method of curing the disease.

An old Scotch practice was to burn a cock's comb and the parings of the patient's nails at the spot where the first fit occurred.

Many of the most eminent men the world has ever produced are said to have been epileptic.

The following, from Plutarch, referring to the well-known case of Caesar is of interest:

"But they were astonished at his patience under this, so far in all appearances above his bodily powers: for he was of slender make, fair, of delicate constitution and subject to violent headache and epileptic fits. He had the first attack of the falling sickness at Corinna. He did not however make this disease a pretext for his infirmities, endeavoring to strengthen
his constitution by long marches, by simple diet and by seldom coming under covert. Thus he intended with his discretion and fortified himself against these attacks.

Caesar went to Spain as Governor in the year B.C. 60. He was therefore about 32 years old when besieged with the attack at Corunna.

Of the great soldiers, Marlborough and Napoleon were epileptic. When we read of these cases we are almost led to infer that epilepsy is essential to great military genius.

Very probably the history of the world would have been greatly altered if Mohammed had undergone a course of Peruvian Potassium; for there is no doubt that the prophet suffered from the falling sickness. He is described as subject to attacks in which he fell to the ground foaming at the mouth and bellowing like a bull, and he has himself related how he saw sudden visions of light and heard voices. Many people now believe that it was during these epileptic conditions that Mohammed received the utterances which he afterwards wrote partly on skins and partly on the shoulder blades of sheep and from which was formed the Koran.
The names of Elliot and Sheridan, Balzac, Flaubert and other eminent writers who have been epileptic would almost suggest that this disease is a valuable ingredient in literary talent. But we must regard such cases as very exceptional because it cannot be denied that as a rule epilepsy is a blighting, a crippling, and a destroying disease.

But passing on to the later history of epilepsy, the novel theory after that of demonic procession was that "animal spirits" were the cause of the malady.

With regard to the later and more scientific investigations these will be referred to in their proper place under Pathology. Suffice it at present just to mention some of these.

Husain, Vener, Brown-Séquard and Schäff and others established the anatomical starting point of the fits as being situated in the pons and medulla oblongata.

Brown-Séquard in his later investigations maintained that loss of consciousness was due to contraction of the cerebral vessels caused by stimulation of the vaso-motor nerves supplying them.
In 1870 Fritsch and Hitzig found that by stimulation of part of the parietal lobe about the fissure of Rolando or by removing the cortical centre for the anterior extremity, they were able to produce convulsions similar to those of epilepsy.

J.Hughlings Jackson, the prince of neurologists whose researches have thrown so much light on diseases of the nervous system, propounded the theory that the nerve cells were like Leyden Jar and gave off nervous discharge at intervals. Thus, he said, in epilepsy there is an instability in these cortical cells and when they receive some very slight outside stimulation they discharge and cause a convolution.

Nearly every investigator has brought forward some new theory, but we know little more about this obscure disease, than was known by our Great Grandfathers.
II.

The Etiology of Epilepsy.

The causes of epilepsy have been classed under different heads by all authors on the subject from the time of Galen onwards, and there have been almost as many differences of opinion as to the most important factors bearing on the question of etiology.

At the outset I referred to the ancient belief that demoniacal influences were the cause and even up to comparatively recent times there have been those who held this view.

But even in ancient times there were those who held that epilepsy was not caused by demons. Hippocrates was one of these:

"The disease called sacred arises from causes like others - namely those things which enter and quit the body, such as cold, the sun, and the winds, which are ever changing and are never at rest: and these things are divine, so that there is no necessity for making a distinction and holding this disease to be more divine than the others, but all are
divine and all human, and each has its own particular nature and power, and none is of an ambiguous nature or irremedial.

As we have grown more enlightened the view of demon possession has been gradually given up and we now look elsewhere for the causes.

I think the best way is to divide the question of etiology into two parts, namely:

(1). The influences that produce the "epileptic change" in the central nervous system, and,
(2). The influences that produce the outbreak of the symptoms.

Influences producing the epileptic change (Predisposing)

It is pretty generally admitted that there must be some inherent instability of the cortex, or in other words, an epileptic diathesis. We may compare this to a barrel of gunpowder, which only requires the application of a spark to cause an explosion.

I do not believe that every person who has the condition of brain of the epileptic has convulsions, but I believe that epilepsy might develop in them with the application of a suitable excitant.

I. Heredity. Chief among the predisposing causes we must place heredity. This is often very difficult to arrive at, as many families try
It is hard to give a definition of this kind, and moreover it is almost impossible to ascertain the state of health of the collateral branches of a family.

Although ancestral epilepsy is the most common hereditary cause of epilepsy in the offspring, nevertheless, insanity and alcoholism also exert a very appreciable influence.

Out of 250 male cases of epilepsy that have been treated in this asylum; in only 90 of these have I been able to investigate the question of these hereditary influences. In 163 female patients I have also been able to investigate these influences.

Table I showing epileptic heredity

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Mother</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Sister</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Brother</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Aunt</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Uncle</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Grandmother</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>First cousin</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>No epileptic history</td>
<td>76</td>
<td>34</td>
</tr>
<tr>
<td>Total</td>
<td>90</td>
<td>43</td>
</tr>
</tbody>
</table>
From this table it appears that out of 90 male patients, there was a family predisposition to epilepsy in fourteen cases or 15.5 per cent, while out of 48 female cases 9 or 29.9 per cent showed a similar heredity. This gives a mean percentage of 18.15.

It also shows that heredity has more influence on the female offspring than on the male, which most investigators believe to be the case.

The following table (No. 7.) shows along with the percentage of those with an epileptic heredity, those who show a family history of Insanity and Intemperance.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Total Cases</th>
<th>Percentage</th>
<th>Total Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M  F</td>
<td>M  F</td>
<td></td>
</tr>
<tr>
<td>Epilepsy</td>
<td>14 9</td>
<td>15.5 20.9</td>
<td>18.15</td>
</tr>
<tr>
<td>Insanity</td>
<td>9 5</td>
<td>10.0 11.6</td>
<td>10.8</td>
</tr>
<tr>
<td>Alcoholism</td>
<td>9 4</td>
<td>10.0 9.3</td>
<td>9.6</td>
</tr>
<tr>
<td>No known heredity</td>
<td>58 25</td>
<td>64.5 58.2</td>
<td>61.1</td>
</tr>
<tr>
<td></td>
<td>90 43</td>
<td>100 100</td>
<td>100</td>
</tr>
</tbody>
</table>

The importance of Intemperance causing a hereditary influence towards epilepsy in the offspring, seems to be much greater in France than in this country.

F. Louise Robinowitch (1882) found in the
majority of cases in the Asile St. Anne, Paris, a history of alcoholism in the parents. Of 140 patients, 90 were descendants of alcoholic parents, a proportion of sixty-four per cent.

Of 315 patients admitted to the London Epileptic colony at Oswel, a faulty heredity was present in nearly 50 per cent, distributed as follows:

- Insane heredity 19 per cent,
- Epileptic heredity 17 per cent,
- Alcoholic heredity 10 per cent.

These results agree in the main with my own observations, except that in the Oswel colony insanity seemed to be the chief of the hereditary influences (19 per cent), whereas in my cases it was present in only 10.8 per cent.

The results of various writers vary.

Echeweria out of 306 patients had 90 with a hereditary tendency or 26.1 per cent. Sevitt found that epilepsy prevailed among the number of the families of his patients in 13.4 per cent.

Sowers, in Allbutt's System of Medicine, gives the combined hereditary influences at 44.1 per cent, while W. A. Jevons who studied the question in 676 epileptics gives it as 37.2 per cent.

Although these results vary considerably, still
the figures show that heredity must be a very
important factor in the etiology of this dreadful disease.

If epilepsy is hereditary the first appearance
of its symptoms is generally early. From my cases
I find that in those showing a family history of
epilepsy, insanity or intimitism, the average age
at which the disease first manifested itself is 12½ years.

Echeverria holds that in these cases it generally
arises before puberty.

If an individual with a hereditary predispos-
tion remains healthy up to the 20th year, he runs a
good chance of escaping epilepsy after that. Others
say that about 75 per cent of hereditary cases arise
during the first twenty years.

Of other diseases, I don't think any stand
in a definite relationship to epilepsy. Some writers
hold that phthisis is connected with the disease
by some hereditary mechanism, but in the epileptics
in this asylum the ratio of phthisis in the families
was not seen to be larger than in the families of
patients who are not epileptics.

Stupors, chorea and neurasthenia have no
direct bearing on the production of epilepsy, but
still they are of some importance as indicating
the neuropathic tendencies of a family.
II. Age.

In many cases it is difficult to ascertain when the disease actually commenced, as the patients may have suffered from petit mal attacks which were disregarded, before they had a typical convolution.

Another difficulty is that they may have had convulsions in infancy from which they recovered, and the question arises whether we are to regard these convulsions as epileptic or no. If they have arisen from periphrase mutation such as teething or worm and have given way to treatment, I don't think we can call them epileptic, though if allowed to go on untreated I have no doubt they might develop into typical epilepsy.

The following table (p. 16) shows the age at the commencement of the disease in 166 male cases who have been treated in this asylum.

From it we are able to deduce the following conclusions:

1. The greatest number of cases of idiopathic epilepsy begin during the first year of life (3.8 per cent).

2. Almost half the cases - 48.1 per cent - begin during the first decade of life, and slightly over three quarters (76.3 per cent) begin under 21 years of age.
Table showing age at commencement of the disease in 166 male cases of Epilepsy.

<table>
<thead>
<tr>
<th>Age</th>
<th>Number</th>
<th>Percentage</th>
<th>Age</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 1 year</td>
<td>23</td>
<td>13.8%</td>
<td>2-2.5 yrs</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>2-2.5 yrs</td>
<td>9</td>
<td>5.4%</td>
<td>2.5-3 yrs</td>
<td>3</td>
<td>1.8%</td>
</tr>
<tr>
<td>3 yrs</td>
<td>5</td>
<td>3.0%</td>
<td>3-4 yrs</td>
<td>11</td>
<td>6.6%</td>
</tr>
<tr>
<td>4 yrs</td>
<td>3</td>
<td>1.8%</td>
<td>4-5 yrs</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>5 yrs</td>
<td>0</td>
<td>0%</td>
<td>5-6 yrs</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>6-7 yrs</td>
<td>5</td>
<td>3.0%</td>
<td>6-7 yrs</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>7 yrs</td>
<td>9</td>
<td>5.4%</td>
<td>7-8 yrs</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>8 yrs</td>
<td>5</td>
<td>3.0%</td>
<td>8-9 yrs</td>
<td>4</td>
<td>2.4%</td>
</tr>
<tr>
<td>9 yrs</td>
<td>3</td>
<td>1.8%</td>
<td>9-10 yrs</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>10 yrs</td>
<td>3</td>
<td>1.8%</td>
<td>10-11 yrs</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>11 yrs</td>
<td>3</td>
<td>1.8%</td>
<td>11-12 yrs</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>12 yrs</td>
<td>6</td>
<td>3.6%</td>
<td>12-13 yrs</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>13 yrs</td>
<td>6</td>
<td>3.6%</td>
<td>13-14 yrs</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>14 yrs</td>
<td>12</td>
<td>7.2%</td>
<td>14-15 yrs</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>15 yrs</td>
<td>2</td>
<td>1.2%</td>
<td>15-16 yrs</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>16 yrs</td>
<td>4</td>
<td>2.4%</td>
<td>16-17 yrs</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>17 yrs</td>
<td>5</td>
<td>3.0%</td>
<td>17-18 yrs</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>18 yrs</td>
<td>2</td>
<td>1.2%</td>
<td>18-19 yrs</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>19 yrs</td>
<td>4</td>
<td>2.4%</td>
<td>19-20 yrs</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>20 yrs</td>
<td>3</td>
<td>1.8%</td>
<td>20-21 yrs</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>21 yrs</td>
<td>4</td>
<td>1.2%</td>
<td>21-22 yrs</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>Grand Total</td>
<td>131</td>
<td>100%</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table IV showing age incidence in 166 male cases of epilepsy divided into quinquennial Periods.

<table>
<thead>
<tr>
<th>Age</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-10</td>
<td>31</td>
<td>30.7%</td>
</tr>
<tr>
<td>11-15</td>
<td>29</td>
<td>17.4%</td>
</tr>
<tr>
<td>16-20</td>
<td>29</td>
<td>17.4%</td>
</tr>
<tr>
<td>21-25</td>
<td>18</td>
<td>10.8%</td>
</tr>
<tr>
<td>26-30</td>
<td>10</td>
<td>6.0%</td>
</tr>
<tr>
<td>31-35</td>
<td>6</td>
<td>3.6%</td>
</tr>
<tr>
<td>36-40</td>
<td>5</td>
<td>3.0%</td>
</tr>
<tr>
<td>41-45</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>46-50</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>51-55</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>56-60</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>61-65</td>
<td>2</td>
<td>1.2%</td>
</tr>
<tr>
<td>66+</td>
<td>1</td>
<td>0.6%</td>
</tr>
<tr>
<td>Total</td>
<td>166</td>
<td>100%</td>
</tr>
</tbody>
</table>

3. The second largest number of cases beginning in any single year, arise between the ages of 11 and 15 — 17.4% cent. — This corresponds to the period of puberty.

14. About the age of seven there is also a slight rise — 5.4% cent. — and this is a very common age at which children begin school life, and what is more important, perhaps, it is
the age when the second examination commenced.

5. Less than one quarter of the cases—23-7 per cent. begin after twenty years of age.

These results correspond in the main with those of most observers, but I notice that W. H. Burnes says that twice as many cases arise between the ages of ten and twenty-three, as arise between birth and nine years of age. My own observations are different, more of my cases arising between birth and nine years of age than during the second period. These results will be seen from a glance at the following table.

**Table V.**

<table>
<thead>
<tr>
<th>Own results</th>
<th>Durand's results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth to 9 years</td>
<td>46-3 per cent</td>
</tr>
<tr>
<td>10 to 23 years</td>
<td>33-7 &quot;</td>
</tr>
<tr>
<td>24 to 70 years</td>
<td>19 &quot;</td>
</tr>
</tbody>
</table>

This is quite to be expected as my cases are drawn from asylum patients, whereas his were for the most part ordinary hospital cases: and I think it is not going too far to say that all patients in whom the disease starts in early life become affected mentally and a great many of them drift into asylums. This would account for the large proportion of my patients in whom the disease began in infancy or childhood.
I think the table proves that idiopathic epilepsy may make its appearance at any age, but as three quarters of the cases arise under twenty one years of age it must be regarded as a disease of early life.

It is during the period of growth and development of the central nervous system, and the period during which the reproductive organs become active that the disease commonly starts. So it is certain that these systems must play a part in its production.

3. Sex.

Taken altogether, I think it is the general opinion that as many males as females are the subjects of epilepsy. Power says that for every twenty males, twenty one females suffer from epilepsy, and that female epileptics exceed male epileptics in early life, but that in adult life more males are attacked.

In the Registrar General's report for 1904, 1557 males and 1357 females died from epilepsy in England and Wales, and other years show a similar proportion.

Seockett says that out of 100 cases, 55 are males and 45 females. "W. R. James states the same view, his statistics showing that out of 1000 cases, 559, 55.9 per cent—were males and 441, 44.1 per cent—females. The same view also denies that female epileptics exceed male
epileptics in early life, and shows that at practically all ages the males exceed the females.

4. Natural Conditions

Epilepsy is a disease of all countries and climes and it seems to be as common among the untaught savages as among the civilized nations.

The approximate ratio of epileptics varies in different countries from 1 to 3 per thousand of the general population.

It seems to be less common among the natives of India than among Europeans, nearly four times as many British soldiers being attacked as natives.

In Norway and certain districts in the Alps, epilepsy appears to be very common.

But it is an extremely difficult matter to arrive at a definite conclusion as to its frequency in different nations.

From the foregoing facts it will be seen that epilepsy spares no condition, age or sex, and yet it is a disease which we are often powerless to alleviate, much less to cure.
Determining causes of Epileptic Attacks.

The must consider the foregoing to be the chief causes producing the epileptic diathesis, if it might be so called. They must have produced some change in the central nervous system and it only requires some exciting cause to change the mere predisposition into the actual disease.

And even in some cases it would appear that an exciting cause is not necessary to start the disease, though in the majority of cases, I believe that the first convolution has been caused by some definite excitant which may have been very trivial but quite sufficient to act as the spark which ignites the gunpowder.

1. Infantile Causes.

A considerable proportion of cases of epilepsy date from infantile convulsions, and although these convulsions are not to be considered as epileptic in all cases, still if the infant has a neuropathic tendency these convulsions are very apt to go on to puberty and then become definitely epileptiform, or they may cease during childhood for several years and then recommence about the time of puberty.

A healthy infant may develop convulsions but they cease when the cause is removed and are not
to be regarded as epileptic.

Reflex causes are important as determining factors in the production of epilepsy in infants with the neuropathic anæsthesia, and of theseething is very important. Powers does not believe that such causes have much influence, the chief factor being the general retardation of development. In rachitic children in whom convulsions are common there is this retardation and the highest centres which normally are slowest to develop suffer most. The lower centres including the motor cortex and reflex centres of the cord therefore take on excessive function, and from this, ordinary neuropathic epilepsy may develop.

Infantile hemiplegia due to injury to the cortex at birth is a fairly common cause. Turner gives this as the cause in 5.7 per cent. of his cases.

The onset of the hemiplegia is usually noticed in by severe convulsions, but they may cease for years, to return again later. In these cases, as a rule, the plegia affects the paralyzed limb more than the others, and there is often an area beginning on the affected limb. In many cases the amount of mischief is not sufficient to cause total paralysis, but in cases which develop epilepsy in early infancy we must suspect this as a cause.
especially if the birth has been tedious or if instruments have been used.

The damage to the brain may not have occurred at birth but later, from thrombosis of the cerebral vessels leading to softening and cystic formation in the cortex. This may not be observed at the time, but many epileptic brains after death show a condition of herniephry and the cause has probably been thrombosis.

Rare causes are embolism and acute encephalitis.

2. Reflex Causes.

Some reflex causes have already been mentioned among the infantile causes, but even in adult life we can often trace the first convulsion to them.

(a) Eye Strain: Among the reflex causes, the subject of eye strain as an exciting cause of epilepsy has received a good deal of attention of late.

A very large number of epileptics suffer from error of refraction. Gould and Bennett published an interesting report on their researches in this direction. They examined the eyes of 71 epileptics and in only three were they normal. Of the remaining 68, 67 had refractions, and of these 33 or 50 per cent had asymmetrical refractions, a defect with it is said to produce injurious results on cerebral functions.
Dr. Wallace, in his same paper describes a very severe case of epilepsy being cured with unsuitable lenses. It is quite possible therefore that seeing, reading with uncorrected refractions and unisometropia may share in the causation of epilepsy as it does in strabismus and although it may be a relatively infrequent cause, still its treatment should never be neglected, as being a possible agent.

(b) Teeth: The teeth of epileptics are, as a rule, very carious and they seem to suffer a great deal from toothache. In examining the epileptics under my care, I could not find one with a good set of teeth and the great majority were extremely bad. I do not think that this can be of much importance as an etiological factor, but still it is worth while seeing that the teeth are properly attended to.

(c) Nose and Ears: Adenoids, polypi or foreign bodies in the nose, or middle ear disease may occasionally give rise to epilepsy seizures.

(d) Digestive disturbances including parasites, worms and constipation. Ticinia, round and thread worms are frequently the cause of epileptiform convulsions which may persist even after the cause has been removed. Kraffmann says that he does not think that the worms cause the fits directly, but by producing a chronic catarrh
of the gut. This question, however, will be more fully treated into when discussion the auto-intoxication theory. Several cases are on record in which the fits always occurred after a meal, more especially if meat had been eaten.

(e) Genital Organs: The relation between epilepsy and the sexual organs deserves special attention, as we have already seen that the physiological evolution of these organs is a predisposing cause, so that it is very probable that marked states of the genital organs may create attacks in persons with an epileptic tendency.

Epilepsy has been known to follow the act of cohabitation, but it is so rare that it scarcely requires to be taken into account, and moreover in some cases it seems to have the opposite effect, as there are cases reported of bad epileptics who seem to have been cured after marriage.

Writers differ regarding the influence of masturbation on epilepsy. It is such a wide-spread view that a causative relation is hard to establish, but still I am certain that its effects on the nervous system are very marked.

Nothenkold says that examples of epilepsy following on onanism are very rare and Turner is doubtful.
if it is ever an exciting cause of epilepsy. I have inquired rather carefully into this subject in my cases here and I find that the majority have practiced the vice too. Success and their sexual feelings are undoubtedly very strong, so I have come to the conclusion that persons predisposed to epilepsy are particularly prone to vicious indulgence, and that in a small proportion at least, the exciting cause was certainly masturbation. This is the opinion of several of the older writers. Leuret found that on 12 out of 106 cases, onanism was to be regarded as the cause of the disease.

Phimosis, with retention of smegma often causes convulsions in boys and circumcision is frequently followed by great improvement.

Disorders of menstruation especially when they arise about the period of puberty have a close relation to the onset and relapses of epileptic seizures. Another point of interest is that in females, the seizure commonly occurs in series just before or after the period.

The above are the chief reflex causes of epilepsy and it is easily understood that disturbance in sensory nerves may give rise to convulsions as these are the initial nervous operators, and all
motor actions are in response to afferent impulses.

A ligature applied tightly round a limb may
inhibit an epileptic paroxysm, and if a peripheral
stimulus can produce such an effect, it may be
inferred that stimulants of another kind and degree
are capable of generating a dizziness.

3. **Damage to Peripheral nerves — cranial and spinal.**

This must be considered as a very rare
exciting cause of epilepsy, but as the reflex causes are
somewhat closely allied to these cases of nerve injury, it
is well to consider them here.

The commonest nerves by which it is
produced are the sciatic and the trigeminal, and
the lesions are usually traumatic, acting on trunk,
branches and cutaneous twigs. Less often the
nerves are injured by the pressure of tumours or
by neurinoma. In these cases the first seizure
generally occurs some considerable time after the
injury and there is usually an area which may
be either sensory or motor, proceeding from the
affected part.

4. **Infective Diseases.**

Scarlet fever, measles, whooping cough,
rheumatic fevers and influenza have all been given
as the cause of fits in patients under my charge.
According to Powers the only one of any importance is scarlet fever, and when the first fit occurs during or after this disease, there are no renal sequelae.

Since the time of Hippocrates some have believed that the acute infectious diseases in many cases cured epilepsy and some even go so far as to say that epileptic patients should be inoculated with malaria, and when the disease has stopped the fits, the malaria could be cured by quinine. Various successful results attributed to this mode of treatment have been published, but cases where the treatment had the opposite effect are carefully withheld.

Hippocrates stated (de morbo sacer) that intermittent fever replaced or mitigated epilepsy, at least temporarily, whence the adage:—

"Quartana epilepsiae vanitatis appellatur"

I have certainly seen cases where the disease appeared to be arrested for the time being, during an acute illness, but they have never remained permanently cured and I am rather inclined to believe that the cause of the arrest is not so much the fever as the changed conditions in which they are living. They are in bed in a sick
wards and for the most part living on a light diet which certainly has a beneficial influence on epilepsy.

A short time ago I had a patient (A.J.P.) in bed as a result of falling in a fit and fracturing his clavicle. During the time of his treatment in the sick ward which was about three weeks he had no fits although previous to that his fits had been averaging more than one per day. Shortly after his discharge from the sick ward his fits began again and have continued since.

At the present time I have another case (J.W.) in bed suffering from pleurisy with effusion. Since his illness began which is about a month ago he has not had a fit, although previous to that his fits were averaging five per week.

Other toxic influences may sometimes be concerned in etiology, such as tobacco intoxication, alcoholic intoxication, lead poisoning and syphilis. These, however, are comparatively rare as exciting causes, though they are all potent in causing relapses.

Sleep.

This is a point to which patients are not likely to advert spontaneously as a cause of the disease,
because it is not sufficiently tangible: but to medical men it certainly is an adequate and efficient cause in many cases.

During sleep the respiratory process is carried on with less vigour and the pulse is reduced. Thus we have the blood more charged with carbonic acid and the heart acting with diminished contractile power. On this account the blood circulates with less rapidity through the brain and as it contains less of carbonic acid it may act as a poison to the nerve cells and cause a discharge of nerve energy. This however can only be applicable in a certain number of cases.

From the following table (p.31) compiled from 54 epileptic patients at present in my wards, it will be seen that in severe cases or 11.1 per cent, the fits only occurred during sleep, while in four cases or 7.4 per cent they occurred only during the day. The remaining 81.5 per cent had fits occurring both by day and night.

Taking the total number of fits, it will be seen that slightly more occur during the day than during the night, 1605 being diurnal and 1544 nocturnal.

Another curious fact showing that the state of
### Influence of Sleep on Epilepsy

#### Table No. VII. showing the proportion of nocturnal and diurnal fits

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**Grand Total, Diurnal 1605. Nocturnal 1544. Total 3149.**

The above table shows the number of fits taken by 574 epileptics during a period of 24 weeks, beginning March 10th 1907 and ending August 18, 1907.
Sleep has an influence on the incidence of the attack is, that if a patient who only takes fits at night as a rule, falls asleep during the day, he is liable to an attack. Probably my table would also show a larger proportion of nocturnal epileptics if this could have been taken into account in its compilation.

The nocturnal type is far more frequent at the commencement of the disease, and this is one reason why epilepsy is often not diagnosed at its commencement, as a patient may not be aware that he is having the nocturnal attacks.

6. Emotional Disturbance:

Close and continued mental excitement, long continued repressing emotions and fright occasionally give rise to epilepsy in a cerebrum of weak and unstable balance. There is a well known case recorded of a schoolmaster who suffered from epilepsy, and several of the scholars who saw him in a fit developed the disease.

(16) Powers tells of a man on duty beside a churchyard, developing epilepsy by getting a fright from seeing a white goat, which he thought to be a ghost running through the graveyard.

Powers regards this as one of the most potent
of all the immediate causes of epilepsy. Out of the 166 cases in which I studied these factors, I was able to get a history of emotional disturbance in only 5 or three per cent. But these were all male patients and according to some writers these influences seem to have a greater effect on females, in producing epilepsy.

I have not of course referred to the so-called Jacksonian epilepsy which arises from gross damage to the brain, but there appears to be some cases where trauma has been the alleged cause of the disease and in which no damage to the brain is apparent. Probably trauma acts as an etiological factor in the same way as fright or mental shock. If this is the case we naturally expect the disease to show itself within a few weeks of the injury and where there has been an interval of several months, I do not think that we can assign the epilepsy to the trauma.

In concluding this part of the subject I should just like to mention a case who was treated in this asylum for a short time and in whom the disease could not be traced to any of the above factors.
A young man, W.T. aged 26, was admitted on February 23rd, 1906 in a state of epileptic fever. He had been well until a few months before admission. One day he got very much overheated playing football and took a prolonged drink of cold water when he immediately fell down in a fit. The fits continued for the next few months rather frequent and severe. He became irritable and depressed, on one occasion drinking turpentine with suicidal intent and at times he was very violent to his relatives.

Such was his condition on admission, but he soon settled down and the fits entirely ceased. He was discharged on May 12th, 1906 quite recovered. In this case none of the ordinary predisposing or exciting causes could be traced and he showed no degenerative or neurophatic stigmata.
The Clinical Study of the Epileptic Fit.

The clinical picture of epilepsy is chiefly drawn from the nature of the individual seizures. But there are also symptoms belonging to the interval, though they are not characteristic of epilepsy in any special sense. Still the fit must not be regarded as the disease but merely as a symptom.

"Epilepsy is the flower of arosenic weed. We may be able to recognize the flower by its colour or smell when we have once seen it, but we shall fail to prevent the development of other flowers and seeds in the same plant and on other plants, unless we recognize the plant itself by all its characters and are thus enabled to pluck out the whole weed by the roots wherever it may be discovered."

Definitions:

Sauvage's defined epilepsy as follows: "Epilepsia, quae in omnibus humanis genere sexurum chronicus et periodicos, cum acutum seseutone in paroxysmus et antecitum oblivione."

That the Lapse of centuries has not materially
affected the views that prevail on the subject may be seen from studying the definitions of some of the more modern writers.

(3) Dr. Copland defines the disease as "a sudden loss of sensation and consciousness, with spasmodic contraction of the voluntary muscles, quickly passing into violent convulsive distortions, attended and followed by effort, recurring in paroxysms more or less regular."

I do not think a more complete definition could be devised.

(4) Seator says, "Epilepsy is a recurring sudden brief discharge of nerve energy in some part of the cerebral cortex, not due to the normal cause of such discharge."

(5) Broadbent's definition is that "epilepsy is a disease characterized by recurrent attacks of general convulsions attended with or preceded by loss of consciousness, usually sudden."

(6) Clouston defines it as "the periodic recurrence of general convulsions begun and accompanied by unconsciousness."

Classification:

I divide symptomatic epilepsy into three classes depending on the nature of the paroxysms. But there is no hard and fast rule between them as many persons suffer from both 'grand mal' and 'petit mal', and although the disease often begins with attacks of petit mal, in the majority of cases sooner or
later a typical grand mal attack ensues.

1. Grand Mal or Epilepsia Grandes

2. Petit Mal or Epilepsia Epileptica

3. Irregular forms of seizures.

1. Grand Mal:

Description of Seizure: Innumerable accounts of the epileptic seizure have been written. Some of them almost thrill us on the reading from their dramatic vividness, and the disease with its violent symptoms, cry, the sudden downfall of the affected person and convulsive movements almost provoke such a description.

I divide the epileptic paroxysm into four stages:

(a) The aural stage (b) Stage of loss of consciousness with tonic spasm of the muscular tissue (c) Stage of clonic convulsions (d) Stage of coma.

In addition various prodromal signs may occur but these are not part of the paroxysm itself.

All the four stages of the fit may not be present, the main element being loss of consciousness which always occurs in a grand mal attack.

Prodromal Symptoms:

The patient may know that a fit is impending for several hours or days beforehand by symptoms which are for the most part psychical but which are occasionally sensory or somatic. Prodromal
symptoms are rather less frequent than the immediate
warnings or aura.

Psychical Prodromata: Among the insane especially un-
attendant can often tell that a fit is impending by
the patient who at other times perhaps is bright and
cheerful giving way to melancholic gloom and despondency.

According to Seale Brown another common prodromal symptom is
that the patient takes hypochrondriacal perversions.

P.B. for a day or even two days before he has a
fit sits in a corner of the ward and if any one
approaches him he will strike or kick. At this time
also he has a delusion that his food is poisoned.
At other times he is a cheerful man and a good worker.

Some epileptics often show signs of great
irritability for a few days before a fit and are
apt to get excited over trifles and take extreme steps.
Headache, dizziness and insomnia are also common
prodromata. In rarer cases the patient may
show a condition of great lethargy and somnolence.

Serious prodromata are rarer and I
have never come across a case manifesting them.
There may be a feeling of numbness, pain or pricks
and needles. Eusthen describes areas of
anaesthesia corresponding to the segmental areas
from the 8th cervical to the 14th dorsal spinall segments.
Somatic symptoms commonly precede an epileptic fit and by treating them the fit may sometimes be averted. Common symptoms are epigastric flatulence and constipation and Onion holds these very important as indicating the autotonic origin of the fit. Certainly I have seen cases where the fit seems to have been averted by a dose of castor oil. Reynolds describes a peculiar dark colouration of the skin, especially of the face and neck about twelve hours before a seizure.

In my experience premonitory occur only in a small proportion of cases and the aural stage is of more interest and importance.

The Aural Stage.

Since the time of Galen, the word Aura has been in use to denote the symptoms that are sometimes present immediately before a fit. The meaning of the word has received several interpretations. It is probable, as Cusen says, that it received this name from the idea that was prevalent at that time that the arteries contained air and that the warning was due to the ascent of some unnatural vapour in the vessels.

Others, including Esquinald, say the term aura meant that the patient actually felt a sensation like
a breeze passing over their body.

"Le propagant comme une vapeur le long des membres, du trone, du cou vers la tête, et lorsque cette vapeur est arrivée au cerveau l'accès éclate."

Among my patients I have been unable to find one who described the sensation of a breath of wind.

The aura when present, is of great importance as it标志着 the site of origin of the discharge of nerve energy. It is generally believed that the aura originates in the central nervous system and is referred to the periphery, being the subjective aspect of the nervous discharges in the cortical center.

The aura is much less frequently present among insane epileptics than among the mentally sound. Out of 200 cases I could only get a history of an aura in 31 cases or about 15 per cent. In epileptic patients outside asylums it is present in from thirty to fifty per cent.

In general the sensation is uniform in the same case, though occasionally it may undergo a change. The aura as a rule only lasts from fifteen to thirty seconds, but it may last for five minutes. It is of great benefit to the patient when it occurs as it enables him to place himself in a position of safety.
Dr. Pierre Clark believes that the longer and more elaborate the aura, the less disposed is the motor cortex to act convulsively. It shows that there is a resistance of the other cerebral centres to participate in the fit.

The various forms of aura may be grouped under five heads: (a) Psychical, (b) Special sense, (c) Visceral, (d) Sensory, and (e) Motor.

Psychical Warning. This is rather a rare form of immediate warning. It may show itself as a sudden disturbance of mind, for instance a sudden acceleration of the imagination preceded sometimes by slight confusion of ideas. It may be in the form of an emotion which is always one of fear or a feeling of dread of something about to happen. It may be fear of an imaginary person and the patient may actually start to run giving rise to a form of seizure which has received the name of epilepsia curiosa. One of my patients runs across the ward and then turns with a diabolical grin, finally falling down unconscious.

Dreamy mental states in which the patient feels a sense of unreality in his surroundings may occur as an aura in a grand mal fit but are commoner in minor attacks and will be considered later.
Auras referred to the organs of special sense may be visual, auditory, olfactory or gustatory.

Visual auras are the most common of these. They may be simple such as colours, flashes of light, stars or sparks or they may be more complex such as faces or scenes and these complex visual warnings are often accompanied by auditory symptoms. When the warning consists of a colour, it is usually blue or red.

Case: Mr. H. previously to a fit coming on sees two faces which he describes as black and ugly. He has never seen them at any other time.

Mr. D. sees old faces which he has not seen for years, then objects begin to move round him and he remembers no more.

Mr. S. feels a very strong inclination to burst out laughing and then sees a wheel of light turning round at great speed in his right eye. His head then turns towards the right and he falls down unconscious.

An interesting point about this case is that for the last month, he has been having attacks in which he does not lose consciousness, but bursts out laughing without cause and loses all power of controlling his laughter. I saw him have such an attack the other day while eating his dinner. It lasted less than a minute.
Pierreau describes an interesting case of a young man in which the aura took the form of his seeing a carriage driving up at a gallop and with great noise, containing a little man in a red bonnet. Fearing to be thrown by the carriage he fell down without consciousness.

Sometimes the warning is a sudden loss of vision when everything becomes dark, and this is sometimes followed by seeing stars. This is an interesting form as showing inhibition followed by discharge.

Even in sleep dreams may indicate the approaching paroxysm. Trousset relates the case of a man who dreamt that he was pursued by a bull, and soon after waking was seized with a fit.

**Auditory Warnings.**

Auditory auras which are subjective sensations of sound come more in frequency, and as I said before they may be associated with visual auras. They may be simple noises such as hissing, or a crack or the sound of waves dashing over rocks.

J.E. heard a noise like a steam engine before he had a fit, gyrates round and round and then falls unconscious.

J.D. has definite hallucinations of hearing and says he hears voices talking to him which he never hears at any other time.
As is the case with the visual centre, so we may have sudden inhibition of the auditory centre, a strange stillness preceding the loss of consciousness.

Olfactory and gustatory warnings are rare but are interesting in connection with a class of fits which John Hughlings Jackson believes to depend on discharge lesions of the uncinate gyrus.

In these cases at the onset of the paroxysm there is a crude sensation of smell or taste often accompanied by movements of chewing, smacking the lips and sometimes reflex ptyalism.

Perris showed that the representation of smell and taste is in the uncinate gyrus; and excitation of this area produces similar movements as they are to be regarded as motor or "reflex" consequences of an epileptic discharge in the neighbourhood. They are often accompanied by an epileptic sensation and a dreamy mental state.

Vaso-motor aurae which are extremely rare might be mentioned here.

Dr. has a momentary feeling of warmth on his neck, chest and face, and marked dilatation of the cutaneous vessels can be observed. Nothenagel describes a vaso-motor aura with a feeling of numbness or weight especially in the fingers or toes of one extremity and
the affected part becomes pale. I have never seen an example of this form.

(1) **Torsorial Warning**

This is the commonest of the remaining forms of aurae and often forms it to be present in fifteen percent of his cases. It usually consists of a deep seated burning sensation in the epigastrium. It may amount in some cases to a feeling of nausea, though very rarely is there vomiting. One of my patients describes it as a feeling of "circulation", which mounts from the epigastrium to the throat and then to the head after which he remembers no more.

Sometimes the aura may be referred to the heart, lungs or intestines, or perhaps anywhere where impression reaches the centre through the vagus. If the head it may take the form of a sudden angina pectoris or palpitation and in the lungs it is often a feeling of suffocation.

S. Willis describes a case where a patient was treated for angina pectoris owing to a sudden pain occurring in the chest. When carefully watched he was found to be epileptic.

J.L. has a feeling of constriction about the lower part of his chest and a stabbing pain in the heart.
Sensory Warnings.

Sensory auras are often referred to the limbs and may be unilateral or bilateral. They may consist of a feeling of pins and needles or a prickling sensation or a feeling of numbness. It may be a sense of movement, e.g. twisting, as was the case in J.C. who describes his aura as "a twisting feeling in the legs, which goes down to the feet."

J.C. has a momentary warm sensation in his head; or O.W. a general diffuse indescribable feeling in his head.

J.C. has an aura of an acting feeling on the right side of his head over the costal angular process followed by anaesthesia, and a peculiar sensation in his nose, "as if it were going to draw me up."

Motor Warnings:

Motor auras are much more common in Jacksonian epilepsy, but they are occasionally to be found in nonepileptic epilepsy.

Paresthesia or dizzying which is very common should be regarded as a motor phenomenon, being due, according to Powers, to the influence on consciences of the greater energy of the centres of one hemisphere. This causes the patient to seem to turn towards the side most convulsed and costal objects seem to turn in the same
direction.

It is immediately before a fit starts up and down the ward and assumes a defiant air, keeping a vigilant eye on the nurse by.

Just as prodromal symptoms may not go on to a convulsion, so the attack may end with the aura.

Pituitary attacks often simply consist of an aura and momentary loss of consciousness.

Though of great interest scientifically, the only real benefit which the patient derives from the presence of an aura is that he can seek a place of safety before he falls down.

Table No. 111 showing percentage and forms of aura in

205 cases of epilepsy.

<table>
<thead>
<tr>
<th>Form of Aura</th>
<th>Number of Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Psychical</td>
<td>2</td>
<td>0.98</td>
</tr>
<tr>
<td>2. Special Sense</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) Visual</td>
<td>4</td>
<td>1.95</td>
</tr>
<tr>
<td>(b) Auditory</td>
<td>2</td>
<td>0.98</td>
</tr>
<tr>
<td>(c) Olfactory</td>
<td>1</td>
<td>0.48</td>
</tr>
<tr>
<td>(d) Gustatory</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>(e) Peco-Motor</td>
<td>1</td>
<td>0.48</td>
</tr>
<tr>
<td>3. Visceral</td>
<td>8</td>
<td>3.9</td>
</tr>
<tr>
<td>4. Sensory</td>
<td>6</td>
<td>2.42</td>
</tr>
<tr>
<td>5. Motor</td>
<td>3</td>
<td>1.46</td>
</tr>
<tr>
<td>Degraded</td>
<td>4</td>
<td>1.95</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>31</strong></td>
<td><strong>15.1</strong></td>
</tr>
</tbody>
</table>
The Fit.

We now come to the objective part of the fit, the phenomena of which are so well known as to require little description. The fully developed fit consists of: (a) the stage of tonic spasm, (b) stage of clonic convulsions and (c) stage of coma.

Consciousness is immediately lost. If there has been no warning to enable him to reach a place of safety, he falls down as if struck by lightning, the muscles being for the moment in a state of relaxation. This is one of the main diagnostic features of idiopathic from organic epilepsy. In the latter the spasm generally begins before consciousness is lost and in some cases consciousness is never lost. The discharge commences at the adynamia shot and spreads thence and it is only after it has spread widely that consciousness is involved. In idiopathic epilepsy unconsciousness precedes the spasm, the discharge spreading widely in the brain and being extensive from the first.

(9)

Powers says that the fall is due to involuntary spasm of the muscles, inconsistent with the maintenance of the upright position, but in the great majority of cases, I am certain that the
muscles are in a state of relaxation and it is only after the patient has fallen to the ground that spasm begins.

The unconsciousness is very intense, the pupil being insensitive to light and the conjunctival reflex absent. The pupil is, as a rule dilated, though I have seen cases in which it alternately contracts and dilates under the influence of the involuntary muscular fibres. The knee jerks are absent at this stage.

The face is said by some authors on this subject to become very pale, but I have been unable to detect any change in the colour of the majority of my patients. In some the face is dusky from the beginning especially if spasm of the throat muscles prevail with compression of the jugular veins.

Before the spasm commences there is often a sudden cry, which is probably due, as Bowes suggests, to the forcible expulsion of air through the spasmodically closed glottis, by the sudden contraction of the thoracic and abdominal muscles, and not, as Kerpel thought to sudden pain, fright or surprise.

It is often a low tremulous groan but at other times it is, as von Berg described it,
"a cry, shrill and terrifying to man and beast."
This is by no means a universal symptom; the vast majority of patients uttering no sound. It probably only occurs when the spasm of the thoracic and laryngeal muscles is simultaneous.

Then begins the stage of tonic spasm affecting to some extent all the muscles in the body, but as a rule one side of the body is more affected than the other.

The spasm of the respiratory muscles causes arrest of respiration and the face becomes very cyanosed. The tongue is congested and swollen and often forcibly protruded between the teeth. The veins of the neck also are very much swollen.

There is conjugate deviation of the eyes to the side most affected and the whole body tends to roll to that side.

Echeverria and others said that at this stage the fundus of the eye showed a condition of anaemia, but Powers denies this to be present. The pulse is small and rather slow and in some it is quite imperceptible.

The arms are flexed at the elbows and the fingers are tightly clenched, the thumb being flexed and adducted across the palm.
The legs are extended, the feet plantarad, and the thighs abducted.

The involuntary muscles share to some extent in the general spasm and urine is often voided with great force from spasm of the bladder wall.

The tonic stage on an average only lasts about thirty seconds and gradually gives place to clonic convulsions which usually begin in the extremities, then the trunk, and the head being last affected. It begins as a tremulous movement in the tonically contracted muscles which gradually gets coarser and broken up into rapid rhythmical movements. The clonic convulsions are often very violent and may cause serious injury to the patient. I have a patient here who has had his shoulder dislocated thirty-nine times during fits. Usually one side of the body is more convulsed than the other. The clonic convulsions of the muscles of mastication may cause the tongue to be badly bitten and occasionally the tip has been bitten off.

The patient foams at the mouth, and the saliva may be bloodstained if the tongue has
been bitten. The peristalsis of the intestines is increased and the patient often passes flatus and feces. Respiration is quickened and stertorous and the pulse becomes fuller and more frequent. As a rule this stage lasts from half to two minutes, ending with a jerk which is often more violent than any of those which have preceded it.

The patient now lies with all his muscles relaxed, breathing stertously and in a state of deep coma which may last for a few minutes or several hours. The skin is often covered with perspiration. The face regains its normal colour and usually the patient falls into a deep sleep which is very beneficial as it restores the exhausted energies and usually prevents the intense headache which is present if the patient becomes fully conscious soon after the fit. The pupils contract and the breathing becomes quiet and regular.

The condition of the deep reflexes depends on the strength of the fit. If it has been a strong one the knee jerk is usually abolished for a short time, being followed by exaggeration which is the usual state in the inter-paroxysmal period.
After the status it is usually absent for several days. If the fit has been slight, excited knee jerk and ankle clonus may immediately follow.

The body temperature after an attack has been measured by various observers without a complete agreement having been arrived at. As a rule I have found an increase of half a degree centigrade which lasts about three quarters of an hour after an ordinary fit. If there has been a succession of fits the temperature often rises to 104°F, but after one fit of ordinary severity the elevation is very slight. The pulse remains accelerated for about the same time.

As a rule there is polyuria after a fit but only very rarely is albumen present. Klein found a faint trace of albumen in 15 out of 23 patients and chloride where in excess in 14 cases.

Others have found large quantities of acetio-lactic acid and consider this due to poverty of oxygen during the attack rather than to disturbance of liver function.

Post paroxysmal motor paralyses may follow a severe fit and it usually affects the side which has been most convulsed.oughings

Jackson believes it to be due to exhaustion of the
cortical centre, but this is doubtful as it has been known to occur after a sensory aura which was not followed by convulsions. It may last for several minutes or several hours.

(23) Rose says that even in the interparoxysmal periods most epileptics show a condition of muscular hypotonia and he believes it to be due to exhaustion of the cortical cells. He writes, "After an epileptic attack there is complete muscular relaxation, but as the patient is at the present time unconscious, the degree of paralysis present cannot be estimated. Even after consciousness is regained general muscular weakness remains for a time, which though not called paralysis is really paralytic in nature."

Temporary sensory paralysis is found in a few cases and may last hours or days after the attack. The loss of sensibility is chiefly referable to touch and painful impressions.

J.H.S. has fits followed by prolonged stupor and unconsciousness, when he is quite insensitive to all external stimulation. On one occasion the stupor was followed by complete hemanesthesia of the left side and the sense of taste was absent on that same side of the tongue. There was no motor change and no loss of temperature sense.
Anomaly in the form of constriction of the visual fields has been described by Thomsen and Oppenheim.

3. Petit Mal or Minor Epilepsy

This is a rarer type than the preceding and only occurs in a pure form in about four per cent. of cases. There are many cases in which the two types are combined and often the disease begins with attacks of petit mal.

Often the only characteristic of these seizures is sudden loss of consciousness without any visible spasmodic element. They may or may not be preceded by an aura and when it is present it is usually similar to that which the same patient has before a grand mal fit.

The duration is often very brief and may pass unnoticed by other persons in the same room. The patient may or may not fall down and in some consciousness may not be completely abolished though it is always obscured. These seizures have received different designations among the laity, such as "sensations", if an aura is present, "faints" if the patient is completely unconscious, "turns" or "spells".

A very common symptom is micturition and
Gowers says it is commence in women than in men. It may occur with loss of consciousness too brief to be noticed.

A typical attack consists of a momentary loss of consciousness. If the patient is at dinner he drops his knife and fork, if engaged in conversation he stops for a second and then may go on from where he left off, as if nothing had happened. Automatic actions such as walking or playing the piano may not be interrupted at all. The eyeball may appear fixed, but there is no pallor of the face, though this may succeed an attack.

In some slight fits the patient turns blue or purple and this cannot be due to pressure of the chest. Gowers says that in all animals there is a spot situated to the outer side of the olfactory tract, just in front of the junction of this tract with the uncinate, irritation of which produces arrest of respiration. So probably petit mal attacks with this symptom correspond to the grand mal attacks which Engelmann Jackson believes to be due to discharge lesions of the uncinate gyrus.

Engelmann Jackson records a very interesting case of a child who experienced and epigastric sensation which rises to the neck. He says, out, "I am ill, I am ill" and then starts chewing movements.
and his mouth fills with water in spite of noisy efforts at deglutition. Irregular movements in the arms— which are not tremors or jerks— tend to carry the hand to the head. His face becomes livid or purple. He does not fall and if offered a handkerchief he takes it and attempts to wipe his lips, showing that consciousness is not completely lost. After the attack has lasted from one to three minutes he is well again.

Still a patient in this asylum has a similar kind of fit. He goes very red in the face, turns his head to the left and says "Well! what!" He stroke his forehead and hits his chops. Finally he turns very pale and the fit is over, but is followed by a severe headache.

Another patient has fits which are so slight as to be imperceptible. If they occur when walking he does not fall, but usually gives an inarticulate cry and stops speaking for a moment. It is all over in a second or two and he resumes his conversation where he left off.

In spite of their mildness these attacks are said to cause more rapid impairment of intellect than grand mal attacks and this according to Jackson is because in petit mal, the disease affects
chiefly the highest nervous arrangements, that
is to say those which have the greatest integ-
ration, — the substrata of consciousness.
Sensations preceding or attending minor attacks.

The curse are similar to those of Grand
mal fits. One of the commonest is vertigo, which
may be subjective or objective or both. Objects may
appear to be moving as the patient may actually turn
round and round.

Of great interest in this connection are
the dreamy mental states of Sir T. Brichon Browne.
It may consist of a feeling of strangeness or of
an impression that the passing moments of life
have once been lived before and that surrounding
objects have been seen before although the patient
may never previously have been in that place.
Everyone has experienced this sensation in a minor
degree. Thomas Hardy in "A pair of Blue Eyes"
says: "Everybody is familiar with those strange
sensations we sometimes have that our life for
the moment resides in duplicate, that we have
lived through that moment before or shall again."

These states rarely occur in children or in persons
with less than average mental intelligence.
Brichon Browne says "the most general description
of them is that they are indescribable and transcend all common experience, and the crudest analysis discloses this much—that they consist in an exaltation of subject consciousness and a degradation in the power of attention for the time being, and are almost invariably concerned, however vaguely, with those ultimate scientific ideas—space, time, matter, motion, force and the like—which are beyond the domain of certain knowledge and according to Spencer, unthinkable. In Tennyson’s Princess, quoted at the outset, another example of dreamy mental state is given.

These dreamy state auras are common in the hippocampal seizures. Dr. Jackson & D. Stewart record a case of epilepsy in a doctor, the aura consisting of strong sensations of smell like camphor or ether. The smell was succeeded by an intellectual aura, a condition in which he felt as if he were saying, doing and looking at things which he had experienced before and people and things seemed to be far away. There were no twitchings and apparently he did not lose consciousness.

Sensations referred to the special auras are common in minor epilepsy.
It may be a momentary darkness or a flash of light or stars. Auditory sensations also occur in those cases in which they form the aura of a severe attack. Odours usually unpleasant are not uncommon. General sensations such as a sudden thrill passing up the body are common, as is also the epigastric sensation which has previously been described.

After States

It has already been stated that a severe attack is usually followed by a deep sleep which is nature's method of restoring the exhausted centre and if the patient is roused he frequently has a very severe headache.

A very common sequel in this institution is an attack of acute mania of transient duration and this bears no relation to the severity of the attack. The mania is characterised by ungovernable violence and frenzied fury. These patients constitute a very dangerous class in an asylum.

Other patients become very delirious after their fits and even if they have had delusions in the interparoxysmal period, after a fit they are more
serious, as then the moral self-control is withdrawn, and the delirious influence the patient's conduct.

Mr. A. after a fit is seized with a desire to strangle someone. He says that he feels compelled to do it and he attacks anyone near. He remembers it afterwards like a dream.

S.D. was brought here by the police. He had assaulted a woman with a knife and inflicted serious injury. The police surgeon at the trial, said the prisoner was certainly of weak intellect and was not responsible as he was the subject of epilepsy. In the course of conversation the prisoner said "They know what I am and showed leave me alone." The father said that on the morning of the assault, he was had had several epileptic fits.

The fits may be followed by stupor lasting several days as in the case with E.Y. who when free from fits is a pleasant and agreeable man, but after fits which he usually takes in series he becomes stupid and pronounces most obscene and violent language. When he recovers which is usually in about a week, he has no recollection of the intervening period.

There is a tendency for some patients after a fit to lie in bed to turn round on their face. The limbs are so deep that even the asphyxial does
Post epileptic automatism is an important subject medico-legally, and many serious crimes have been committed in this state. Several amusing instances of post epileptic automatism are also on record. (29) Rousseau reports the case of a judge who suddenly in the midst of a session rose from his bench and armchair in a corner, afterwards resuming his seat as if nothing had happened.

This automatism usually follows the slight attack and is due to the withdrawal of the control normally exerted by the highest centres. Taking off the clothes is very common, as was the case with a music master who had to give up his profession on account of this proceeding before young ladies. "Kleptomania, Pyromania and Depomania are also common.

The patient after a slight epileptic attack may pass into violent hysteroid convulsions, but this never occurs after a grand mal attack. It is commonest in young women under thirty-five years of age, but also occurs in boys and occasionally in young men.
The Status Epilepticus (État de Mal).

By this is meant a succession of fits, on the intervals between which consciousness is not restored. It is a common cause of death in epilepsy. It may appear at any time during the life of the epileptic and nothing is known of its immediate causes although occasionally it seems to arise from accidental circumstances such as the sudden stoppage of bromides, strong excitement, the puerperium, or a fall or blow on the head. Its occurrence is usually preceded by an increase in the number of seizures, although sometimes it is of sudden onset.

The patient rarely dies in the convulsions but usually in the after state of coma and exhaustion, developing symptoms of the typhoid state.

During the Status the temperature may range from 104° to 107°, often being highest on the side most convulsed, and falling most rapidly on that side till a balance is established. The pulse is usually very accelerated, beating about 150 to the minute.

The pupils are dilated and insensitive to light and the body is often covered with a cold sweat.

If a had a succession of 522 fits in four days, dying on the fifth day. The fits were distributed as
follows: On February 13th he had 18 fits, on the 24th, 89 fits, on the 25th, 122, and on the 26th, 363.

Table VIII, showing weekly number of epileptic fits taken by twelve patients during the 24 weeks from March 10 to Aug. 18, 18—.

<table>
<thead>
<tr>
<th>Year</th>
<th>April</th>
<th>May</th>
<th>June</th>
<th>July</th>
<th>August</th>
</tr>
</thead>
<tbody>
<tr>
<td>1SB</td>
<td>010211311312030100310</td>
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</tr>
<tr>
<td>2MP</td>
<td>511181412827253421120</td>
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</tr>
<tr>
<td>3GR</td>
<td>792312113779451171571081410613897</td>
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</tr>
<tr>
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</tr>
<tr>
<td>5JB</td>
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</tr>
<tr>
<td>6JG</td>
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</tr>
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<td>7JK</td>
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<td></td>
</tr>
<tr>
<td>10JR</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11JS</td>
<td>0010001000040000240000004</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>12AG</td>
<td>111320001117200222471027107</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. The above twelve patients were picked out at random from the fit charts and give a good idea of the weekly number of fits, that asylum patients have. Number 4 shows a fairly common form in which a sort of wave is produced, the fits slowly increasing in number and then receding again. Nos. 5, 10, 11 are examples of...
3. Irregular forms of Seizure.

Psychical epilepsy or larvated epilepsy in which there is no convolution or sensory disturbance, but in memory is for the time being in abeyance and consciousness is disturbed. There are some writers who do not believe in this form and say that the symptoms which constitute it are preceded by some form of incomplete epileptic attack which may pass unnoticed.

Larvated epilepsy usually occurs in persons who have occasionally grand and petit mal attacks and very often there is a certain amount of periodicity in them.

It may be preceded by prodromal symptoms of heaviness, irritability or fear. The phenomena manifested in masked epilepsy are practically identical with those which may follow an attack of grand mal and which we have already described.

The patient is not entirely unconscious and may be partly aware of his surroundings, but he has no subsequent recollection of what has occurred during the attack. The attacks may be of sudden onset and only last a few seconds or minutes, but may be much more prolonged.
Dr. Henry of Philadelphia reports an interesting case of a man who was subject to these attacks. He suffered from Bright's disease and had been a heavy drinker. Describing an attack, he said that in the office he began to feel stupid and heavy and very irritable. He felt sick but did not vomit. After leaving the office he wandered in an automatic way about the streets till after midnight and at 2 A.M. found himself on the outskirts of the city and boarded a car. He had no recollection of intervening events.

He again had an attack of mental confusion and partial unconsciousness and was carried far past his destination and found himself on another outskirts. He finally got home very sleepy and exhausted.

Ten days after, he had a similar attack, when he went to call on a friend and tried to open the door with his own latchkey and attempted to eat soup with a fork. In an attack a few days later, he became irritable, violent and incoherent.

From the above case it will be seen that semi-purposive actions may be performed.

I have already referred to manacel attacks following a fit, but occasionally a patient without any obvious fit will become extremely manacel and even homicidal. It may be an uncontrollable impulse devoid of any motive that drives him to
cases of violence or it may be the outcome of temporary delusions.

Mr. X. takes such attacks of sudden impulsiveness when he rushes about the ward over chairs and tables and will knock down anyone who tries to stop him. In his ordinary state he is quiet and well behaved and says that he is conscious in a dazed sort of way of these attacks, but has no power to control himself.

The manic acut attack are usually followed by a period of lassitude and sleep and when the patient wakens, he has as a rule no recollection of what has happened, though he may remember in a confused way as was the case in the patient above described.

Paroxysmal headache, dreamy states, nausea and vomiting, attacks of pain etc. are sometimes mentioned as occurring in the interparoxysmal periods and resembling epileptic "equivalents", but not probably they are aware which have not been followed by a complete attack.

In some cases sleep may constitute a form of masked epilepsy. Walker records the case of a young lady, act. 19 who gradually became insensible, and when roused fell asleep again, and the sleep lasted several hours. No convulsions were observed before the coma occurred.
On being questioned she said that before these attacks came on, she sometimes experienced a strange feeling in her right arm, leg and face. Attacks of paroxysmal laughter or sneezing may occur as interparoxysmal phenomena, when I think we are to regard them as epileptic equivalents. Perhaps T. S. described on page 426 is an example of this.

Sudden paroxysmal rise of temperature may constitute irregular attacks, H. takes frequent grand mal attacks. On several occasions his temperature has run up without cause to 104°F, with pulse of 120, the temperature returning to normal in two days. No signs of any pulmonary disease could be detected and after these attacks he is almost immediately well again.

The origin, course and termination of epilepsy

We have mentioned casually before that epilepsy may commence in one of four ways.

1. It may start with fits of the major type with frequently recurring seizures.

2. As petit mal attacks which may run a course of years before a grand mal attack occurs.

3. As nocturnal attacks which often escape diagnosis for long periods and may be unknown to patient himself.
4. As a single seizure which may not be repeated for several years. This often constitutes a difficulty in treatment as the patient thinks that he is cured and is averse to continue medicine.

The disease having commenced in one of these ways, runs a more or less definite course. The fits may recur daily, weekly, monthly, quarterly or yearly, but the majority of cases have average one or more fits per week. This is what would be expected, as it is those with frequent fits who are most prone to mental deterioration.

Sometimes a patient may go for a month or more without having a fit and then have a series of several. This constitutes a definite type called serial epilepsy.

Long remissions may occur during the course of epilepsy. Remissions of several years may occur between the first and second fit, during the years of childhood, and even after the disease has been thoroughly established a long remission may occur, arising either spontaneously or as a result of treatment.

Examples of the types of epilepsy may be seen in the table on page 64 compiled from patients at present in the institution.
Only about ten per cent of epileptics are cured altogether of their fits and the great majority steadily become more confirmed and progress towards dementia. If the fits continue the mind is almost sure to become affected sooner or later and in this way another load is added to the already heavy burden which the epileptic has to bear.

The average age at death in 34 cases that have died here is 35.2 years. Spathing gives it as 29.4 years. In the London County Asylums it was found to be 40 years and in the Manchester Epileptic Institution 39 years.

<table>
<thead>
<tr>
<th>Table IX showing causes of death in 84 cases of epilepsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Status Epilepticus</td>
</tr>
<tr>
<td>Sudden death as result of a fit</td>
</tr>
<tr>
<td>Suffocation during a fit</td>
</tr>
<tr>
<td>Acute Lobar Pneumonia</td>
</tr>
<tr>
<td>Congestion of the lungs</td>
</tr>
<tr>
<td>Pneumonia Pneumonic</td>
</tr>
<tr>
<td>Acute Bronchitis</td>
</tr>
<tr>
<td>Aneurism of Aorta</td>
</tr>
<tr>
<td>Heart disease</td>
</tr>
<tr>
<td>Softening of the brain</td>
</tr>
<tr>
<td>Acute Colitis</td>
</tr>
<tr>
<td>Pyelitis</td>
</tr>
<tr>
<td>Tuberculo Pneumonia</td>
</tr>
<tr>
<td>Abscess of Kidney</td>
</tr>
<tr>
<td>Typhoid Fever</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

It will be seen from the above table that the commonest cause of death is the Status Epilepticus, twenty one
cases or 2.5 per cent succumbing to this complication. Suffocation was the cause of death in five cases and was due either to the patient turning over on his face in bed or to food entering the air passages.

Thus only 29 cases out of the 84 died of epilepsy; the remainder dying from an intercurrent affection.

It will also be noticed that a large number, 30 per cent, died from affections of the lungs, especially phthisis and acute lobar pneumonia.

These figures correspond pretty closely to those of Sprattling who studied the causes of death in 150 cases. He found the 23 per cent died from the status, 5 per cent from suffocation, 24 per cent from pulmonary diseases and 10 per cent from organic heart disease.
IV.

The Interparoxysmal Period:—The Epileptic Temperament.

That a healthy mind may coexist with epilepsy is a well known fact, and the many cases in which gifted men have been epileptic confirm this. But alas! the majority after a time become enfeebled in mind, and the best care and treatment often fails to prevent mental decay.

Some patients may have fits for years without the mind suffering to any appreciable degree, while others after a few months begin to show some loss of memory and signs of dementia.

We do not yet know whether the mental degeneration is due to brain injury caused by the fits, or to the advance of the morbid process that caused the epilepsy to the parts of the brain associated with the intellect. Thus the epileptic fits and the mental accompaniments are believed by some to be equally effects of the same cause.

Dr. Radcliffe pertinently observed that epilepsy as often begins as ends in madness. Russell Reynolds says "the duration of epilepsy is, per se, without influence upon the mental condition of the epileptic."
There are however several factors associated with the disease which seem to increase the tendency to mental alienation.

In epileptics who are the offspring of insane or epileptic patients, the probability of the superintention of dementia is increased.

Turner has shown that if the disease has lasted more than ten years there are only a very small percentage who remain intellectually normal, whereas if the disease has lasted a shorter time there are many who are quite unaffected mentally. This seems to disprove Reynolds's belief.

The same holds good with regard to the frequency of seizures. The more frequent the attacks, the more common and profound is the associated dementia.

It is generally held that minor epilepsy is more crippling to the mind than major epilepsy and the reason usually given is that in minor epilepsy, the very highest centres, i.e. Wülfling-Jackson's highest level of the central nervous system are those from which the discharge takes place. Whether this is so or not I have been unable to determine as in asylum patients where the disease is very advanced, petit mal unassociated
with grand mal is rare.

Mental enfeeblement sometimes begins when fits which have been going on for years cease spontaneously. The cause of this is probably the repression of the discharges to which the brain has grown accustomed.

The forms of mental defect chiefly met with in epileptics are (1) Mania 8, Melancholia 12, Dementia 14, Congenital mental deficiency and idiocy.

On referring to the case books I find that out of 205 epileptics admitted to this institution, the form of mental disorder on admission was as follows:

Mania 67: Melancholia 12: Dementia 69: and congenital mental deficiency and idiocy 57.

Thus it will be seen that dementia is the commonest form of mental disorder and it may exist in varying degrees, from slight loss of memory to drugged idiocy.

I am certainly of opinion that where cause or inebriety is complicated with epilepsy, they do not bear any relation of cause and effect but march side by side and have a common origin. Such patients also usually have a hereditary history of epilepsy, insanity or idiocy.

Those admitted as suffering from mania may be fairly rational between the fits, and merely
Have maniacal outbursts before or after the fits, but the majority of them also show some impairment of memory and as time goes on they become demented.

Melancholia is a rare form of mental disease accompanying epilepsy and was only present in twelve out of the 205 cases or in rather less than six percent.

Let us now look at the mental and bodily characteristics commonly met with in epilepsy. Two better words could not be found to express the epileptic psychosis than those used by Clouston, namely, Irritability and Impulsiveness. They may exist in varying degrees from slight irritability of temper to dangerous homicidal impulses. The character and disposition are profoundly altered. Irritability of temper is often the first symptom noticed by the friends, but it is soon followed by weakening of memory especially for recent events and things that have happened long ago remain quite fresh. Sometimes the memory is perverted so that very trivial and unimportant matters are distinctly remembered.

(3) Strangor says "the memory decreases, the imagination grows dull, the fancy loses its richness of colouring, its intensity and warmth, and the
spirit is withered.

In course of time the reasoning powers yield to the debilitating influences which destroy the healthy activity of the cerebral lobes.

A common feature is a strong religious fervour which contrasts in a marked degree with their general behaviour and actions. One of my patients who is constantly reading the Bible, without provocation will come out with appalling oaths and many of his actions are most immoral. They are often egotistical and self-laudatory.

They are moderate liars and bring all sorts of charges, often based on their own hallucinations against their attendants and fellow patients. They have a high idea of their own importance and will never acknowledge themselves in the wrong. Very furtive, they will attack any offending person and when charged with it they will always try to make out that they were the aggrieved party.

Mycrochamasiis is common. They imagine that they suffer from all kinds of disorders and have great faith in quack medicines.

Delusional states are not uncommon and as mentioned previously, probably account for many
charges of assault and murder.

Want of initiative is characteristic and although many are good workers they require constant supervision.

It is these characteristics, described by Bevan Lewis as the "epileptic neurosis" which add tenfold to the terror of the disease, and after several years the patient often sinks to the level of one of the lower animals and the mental life merely consists of the lower animal instincts and passions.

We are accustomed to observe, in the inter-episodic period, the facial expression—or rather the want of it. The 'facies epileptica' is so characteristic that on entering a ward one can be almost certain which are the epileptic patients. According to Spratling it results from a combination of dementia, facial scars and bromide acne. There is an expression of great dullness and heaviness and a want of the finer shades of mobility which express the emotions.

The lips are thick and the skin coarse and oily and often covered with acne papules. If the patient is taking bromide. A pasty, leaden or livid hue of the skin is characteristic.

The pupils are commonly dilated and the
eyes have a metallic glint. Sometimes the pupils are unequal and present the condition of anisocoria. The nose is broad and flattened. They usually have abundant hair which gets very moist when the fit occurs. This has often struck me as a curious fact and among the many epileptics in this institution, I do not know one who could be described as bald and most of them have a very luxuriant crop of hair. They often receive terrible cuts and bruises during their fits and it is an interesting fact that their injuries heal most readily.

The speech is slow and drawling. Amnesia, aphasia, and scholalia are also common. By repeating the questions he is asked the patient tries to stimulate the slowness of his memory. If asked to recite a poem he does it all right as if it were or less mechanical. They nearly all deny that they have any difficulty in speaking. Temporary aphasia appears sometimes as an aura and sometimes as the immediate consequence of a fit.

The characteristic slow speech is probably due to the tongue being large and atonic and to the impairment of memory. Slight paralysis of the laryngeal, tongue and face muscles may assist.
In extreme cases there is dribbling from the mouth from the half-closed state of the lips.

Their physical development may not differ from normal, but though robust and muscular they are often undersized and thickest. As a rule they are very strong and often require several attendants to hold them during a fit, and I have seen one after a fit break a thick door panel with his fist.

Their gait is slow and shuffling, and they seem unable to hold up their head and they sit in a characteristic slouching attitude.

Sometimes I have noticed constant fine muscular tremblings affecting special groups of muscles, and Reynolds says this is extremely common.

The pulse is usually quick, feeble and soft, but in the more serpentine type it may present no variation from normal. The cutaneous circulation is feeble and the patient often suffers from cold extremities.

The gastro intestinal system is often disordered, the tongue being furred and the bowels inactive.
They have large appetites and are coarse feeders and require apenic medicine once or twice a week.

Stigmata of degeneration have been carefully studied by Turner, who finds in them indications of a neuro- or psychopathic hereditary tendency. Such abnormalities are microcephaly and other cranial deformities, prognathism and cranio-facial asymmetry, defective development of the hard palate, irregularities and displacements of the teeth.

I have found deformities of the ears to be common and hematomas auris especially so.

According to Turner, such stigmata of degeneration are "structural or psychical deviations from the normal, occurring during the course of growth and development of the central nervous system, in those who are the subjects of a hereditary degenerative predisposition."
V.

Pathology and Morbid Anatomy.

That there is some morbid change in the cell elements of the cortex, I think there can be no doubt, but our knowledge on this subject is very elementary, and nearly every investigator has discovered some new pathological change in the nervous elements.

It may be as Sir Wm. Powers says that the changes which occur are of such a fine kind, that they are not discoverable by means of microscope or any scientific means and are only revealed by altered function. It is quite possible that the nutrition of the cell protoplasm might be very greatly modified, without showing itself as an evident morbid change.

These changes, whatever they may be in the nervous elements are very probably not the direct or locating cause of the fits, but I think they are certainly preponderating causes and that is the view I intend to adopt here.

I will therefore first attempt to describe some of these morbid changes which have been found in the brains of epileptics and then go on
to discuss the existing factors.

Some of the demonstrable changes in the brain are very probably not the "pons et origo nati" but the "front et propter hoc".

A great advance has certainly been made in narrowing down the seat of the disturbance to the cortical matter of the cerebral hemispheres. Nothnagel believed that minute lesions in the medulla or pons evoked generalized convulsions, but if this is the case it certainly does not bear on ideopathic epilepsy. Since the discovery of Fritsch and Hitzig and the observations of Ferrier there have been many experiments made by electrical stimulation of the cortex.

Van de Kolk and others believed that the starting place of the fit was in the medulla and that it was there that the changes should be looked for. The reason for this belief was that respiration is affected early in a fit and this would point to the medulla as the part chiefly affected as it contains the respiratory centre. But now we know that the change in respiration is not produced by direct stimulation of the respiratory centre, but by the tonic condition of the muscles of the chest wall.
at the commencement of the convolution. Further
than this Horley showed that the floor of the
ventricle is not the only centre for respiration
but it also has a direct representation in the
cortex and also in the cord. The same
observer also found that in persons who bit their
chance during a fit the blood vessels along the
course of the hypoglossal fibres in the brain were
much more dilated than those along the course of
the vagus. Conversely in non tongue bites he found
that the vessels along the course of the vagus were
more dilated than those along the course of the
hypoglossal.

Of course some might say that the whole
nerve was a nerve might be the starting point of the
convulsions. Horley proved that the spinal
cord could not initiate the discharge of energy, by
sailing the bulb and carrying on artificial respiration.
We injected amphetamine and got convulsions from the
nerve centres above but not below the point of section.

We know that the cerebrum is the seat of
consciousness and this function is so early
abolished that if the starting point is not here
it must be very early involved.

If the cord is electrically stimulated we
get clonus produced in the muscles supplied and not tonus, or if we get tonus it is not followed by clonus when the irritation is withdrawn.

Simple electrical stimulation about the Rolandic area has been found to evoke tonus followed by clonus, while if the cortex is cut off (Francé & Petrid) and we excite the subjacent fibres of the corona radiata we only get tonus not followed by clonus.

Further, if during a fit, the cortex is cut off from the underlying part, the fit ceases.

The rest of the nervous system merely conduces the impulses which start in the cerebral cortex and it is here that we expect to find the morbid change.

The cerebral cortex can be divided into five layers and according to Brain-Lewis definite changes of a highly characteristic nature can be observed by careful examination in the second and third layers but chiefly in the former. This normally consists of a thin layer of a large number of closely packed nerve cells, small in size, and pyramidal in shape. The nucleus of these cells is the first part affected, the rest of the cell body appearing normal for a considerable time, and being only secondarily involved.

A bright, refractile, spherical body usually
of fatty nature appears in the centre of the nucleus, which increases in size till the nucleus is no longer apparent in the cell. This droplet is usually spherical, but may be crescentic, oblong or irregular.

When half the nucleus is occupied by this body it refuses to stain by ordinary aniline dyes and is only faintly differentiated from the surrounding cell. Later on this fatty change proceeds to vacuolation, due to the globule bursting out of the cell body and leaving a cavity with a sharp cut marginal contour. Pale floccus now appear in the cell protoplasm which show that it also is beginning to undergo degeneration.

This vacuolation may not only affect the second layer, but even those succeeding as far as the spindle-celled layer, but always to a far greater extent in these small pyramidal cells. The cells ultimately break down, and finally very few can be found, their place being occupied by fragments of cell processes or of destroyed nerve elements.

A. W. Campbell showed that these cell changes could be found in various other diseases such as tuberculous and pneumonia, and Lewis himself admits that a similar change is met with in alcoholic brain disease, but never so marked or
so limited to cortical areas as in epilepsy.

The majority of investigators have made
the brains of congenitally defective persons the
basis of their investigations as to the morbid
anatomy of epilepsy, as the disease is so
common in those individuals and among
others Dr. John Turner of the Essex County Asylum has
recently, working in this direction added much to
our knowledge.

He agrees with Raven Lewis that the cells of
the second layer of the cortex are diminished in
number and also show signs of degeneration,
but he does not attach much importance to vacuolation
of the nucleus.

He has found change in the Betz cells of
the ascending frontal convolution in seventy-seven
per cent of cases, in some cases the majority of
these cells being affected, in others just a few.
The changes in these cells are as follows. The nuclei
bodies in the more central part of the cell are in the
form of fine granules while those at the
periphery are normal. The cell is swollen and
the nucleus instead of being at the centre
is at the side or up in the apex.
Lewis also describes these cells as being swollen
and globose and says that they may retain their lateral or basal processes but have no apical processes, or merely a stunted one attached. These cells are found normally in the brains of some of the lower animals and when they occur in the human brain it suggests a regression to a more primitive type. It is quite possible that in persons whose ancestors have been insane, epileptic or drunkards, the brain may undergo a retrogression.

In the white matter just below the cortex, Penfield pointed out that there are numerous nerve cells. They are found normally in the lower vertebrates throughout life and in newborn infants but they normally disappear towards adult life. They are found to persist in imbeciles whether they are epileptic or not.

Ford Robertson believes that most of the changes described as occurring in the brains of epileptics are the result of the fits and are at any rate non essential to the production of epilepsy.

There are other more obvious mental appearances generally found post mortem, but most observers are agreed that they are secondary to the disease.

In my experience I have found that as
a rule the dura mater is much thickened and often adherent at parts to the pia arachnoid. Moreover, the membranes are usually congested to some extent in all cases and to a very marked degree in the cases that have died from status epilepticus. The vessels in the membranes often show some indurations of their walls.

In every case that I have examined I have found a frothy exudate lying on the surface of the cortex. It is probably an albuminous exudate from the vessels. T. G. Armes believes that this material by undergoing fatty changes may act as a direct poison to the nerve cells by setting free decomposition products.

In a large proportion of the cases I have examined here there was atrophy of the convolutions, but whether this is the case in epileptics who are not insane of any kind I am not prepared to say.

Some observers lay stress on a difference of weight between the two hemispheres. On an average I find the difference to be twenty-two grammes and I do not think this is more than is ordinarily found in any case of insanity. In all cases there is an overgrowth of neuroglial tissue which is palpably evident on
cutting into the brain. A very dense and broad layer is often found in the cortex immediately beneath the grey matter. It is said also to be very marked in the cornu ammonis and some pathologists look on this with great significance. Hooper only found it to be present in one out of thirteen cases, whereas Jollesma found it in twenty-four percent of his cases, but both observers affirm that it was only a somewhat marked degree of a general sclerosis throughout the white matter.

Anglade by special staining methods affirms that the nerve cells and especially the pyramidal cells are intact, even in patients who have died from the status, but that the neurophagial elements are never normal either as individual cells or in their relative proportion as a brain constituent. The neurophagial cells showed increase and proliferation in the form of patches of hard consistence on various parts of the brain, conspicuously in the hippocampal region, pons and bulbo and even the spinal cord.

The general consensus of opinion is that the scleroses of the cornu ammonis is not constantly present in epilalsy and therefore it is not essential to its pathology.

After death from the status, the brain is
always intensely congested throughout and the convolutions flattened.

Small haemorrhages, or evidence of such haemorrhages in the shape of haematoaoidin corpuscles are commonly to be found.

Such then are the changes which are found either microscopically or macroscopically in the brains of epileptics and we now come to consider the exciting cause of the attack. Of theories on this subject, their name is legion and we are perhaps no nearer the solution of the problem than Hippocrates was.

"We must all agree with Broadbent that "convulsion is the result of an excessive and disorderly liberation of nerve energy, as an ordinary paroxysmal action is the result of a duly limited and orderly discharge". This energy, whatever it is, is probably generated in the nervous grey matter and not in the nerve cells themselves, whose function according to Schäfer and others is purely nutritive."

We must now consider the various views held regarding this sudden discharge of nerve energy.

Cerebral anaemia: Brown, Kirwan believed that
cerebral anæmia caused by contraction of the cerebral vessels in response to stimulation of the vasomotor nerves was a cause. Rusemane and Senner produced convulsions by ligation of all the vessels going to the brain, but this cannot be looked upon as a proof, because by so cutting off the blood supply from the brain, the nutrition of the respiratory centre was cut off, and moreover mere convulsions do not constitute epileptic epilepsy.

Sengie's Jackson in 1864 suggested that fits were caused by blockage of small cerebral vessels. Dr. John Turner believes that the cause of a fit is sudden stasis of the blood stream resulting from the blockage of cerebral vessels by intravascular clots. This stasis correlated with defective development and probably unstable nerve cells has, according to Turner, a very definite relation to fits. He believes that injection of only substances such as absinth and curare into the vascular system of animals, sets up convulsions by causing stasis in the brain. He showed that in ninety per cent of epileptic brains there was thus intravascular clotting and in only thirty-five per cent of the
control brains, was it found and in the latter the maximum rarely surpassed the minimum found in the epileptic brains.

Blood platelets are much more numerous in the blood of epileptics than in normal blood, and Turner thinks that most of the clots found are caused by cohesion of the platelets. He has found the clots in the brains of epileptics who have had no fits for long periods before death, so that it is improbable that the clots are caused by the fits. He suggests that just before a fit, the leukocytes and platelets shed their nucleoprotein contents so that the blood platelets cohere.

Clots of different kinds are found, but the commonest are, (a) small spherical bodies, homogenous in structure and varying in size. (b) Hyaline masses clinging to the vessel wall, and (c) clots of a finely granular nature. It is believed the the spherical bodies and the hyaline masses arise from the amalgamation of platelets, for they both contain phosphorus and their behaviour to reagents is similar. The granular clots probably result from the entanglement of red blood corpuscles, and ellipsoid masses shown that red blood corpuscles are diminished...
in epileptic blood, and abundant destruction of them occurs immediately before an attack.

I have not found these clots to be present as a rule in the cerebral vessels of my patients that I have examined on the post mortem table, and though the theory is ingenious and may account for fits in some cases, I do not think it explains all the facts in the clinical history of epilepsy.

That cerebral anaemia was cause of convulsions is certainly quite proved by Kussmaul and Benner's experiments, but to say that it is therefore the cause of epileptic epilepsy is taking a great leap. Powerampioned the belief of epileptics during fits and found that the phenomena showed no anaemia, so that there cannot be a general anaemia of the brain.

I think that the theory of cerebral anaemia as a cause of epileptic convulsions has not sufficient proof to justify its acceptance. The Toxic Theory.

A theory that agrees better with the clinical facts is that the convulsions of epilepsy are caused by a toxin acting on a brain of unstable
equilibrium. A great deal of work has lately been
done in this direction by Kramský, Ceni, Voein
and Petit and other investigators.

Various toxins have been investigated but
the precise one which causes epilepsy is not yet known
though in the majority of cases it is probably
produced in the alimentary canal of the individual.

Kramský believes that it is Ammonium Carbamate

(17)

Kao, believing that urine acid is the cause of all
ills that flesh is heir to, states that when it is
present in excess in the blood, it may excite the
epileptic discharge.

(18)

Donath believed it to be cholæ, while Zini believed it
to be certain cytotoxins

(19)

Hamilton of New York believes that cytotoxemia
occurs in three ways:

1. Gastric intestinal derangement which sets free from
bodies and putrefactive products (mucic acid and Skatol)
in the alimentary canal, to be taken up by the blood.

2. Presence in the blood of certain toxins such as
staphylococcus aureus and album.

3. Poisoning of the cerebral nerve cells by
degradation products of the nervous tissue, among
such toxic products being cholæ, shown to be
toxic to cats by Elliott and Walton.
The general appearance of the epileptic suggests a toxæmia, the muddy complexion, the greenish tinge and the dull expression are pointing to this. The tongue is very often furry or coated and the patient complains of a bad taste in the morning. The breath as a rule has a disagreeable odor and the bowels are constipated.

(20) Kauffmann believed that even certain reflex epilepsies may be toxæmic. For instance intestinal worms may cause a chronic catarrh of the gut which must interfere with secretion, and absorption of food stuffs. It must also to some extent lead to interference with the regular course of digestion and the formation of regular products, which will act on the tissues in a toxic manner.

A great deal of work has been done on the urine in the hope of finding some toxæme, but the results are very contradictory and confusing. Kehold and Dray injected into dogs and white mice the urine and blood of epileptics. The dogs got from 2 to 10 cc. of urine obtained immediately after a fit and (2) one hour after a fit. In no case were convulsions produced in the dogs and they had no little success with the white mice.
Vorcin and Pirron injected the urine of an epileptic obtained immediately before, during and after a fit into rabbits. They found that before the fit the urine was less toxic than normal urine, that during the fit it became toxic and that after the fit it became hypotoxic. They assumed that if this diminution of toxicity did not occur, status epilepticus and sometimes death ensued.

Klein examined the urine of twenty-three patients immediately after a seizure. In fifteen he found a faint trace of albumen and in three the urine showed albumose. Vorcin and Pirron believed that the presence of albumen depended on the degree of renal vaso-sclerosis as indicated by the facial cyanosis.

In Klein's cases chlorides were in excess in fourteen out of twenty-one cases and in six out of ten examined in the intervals chlorides were normal and in the remaining four they were diminished. Steng found diminution in urine acids before and increase after a fit.

Mason frequently found the following changes at the onset of an attack: The phosphates of alkali earths were increased, nitrogenous elimination was modified, there being diminution of urea, increase of urine acid, creatin and creatinin. The conjugate
Sulphates were increased and one or more substances were eliminated which gave the diazo reaction. The specific gravity is usually high and the reaction acid.

**The Blood.** Pagh has made researches into the chemical condition of the blood of epileptics. The neurons work under conditions of alkalinity and therefore he says it is probable that any appreciable diminution in the alkalinity of their surroundings must bring about abnormal manifestations of activity. He found that in the interparoxysmal period the alkalinity was lower than normal and that there was a sudden and pronounced fall immediately before the fit and a further fall after the fit was over, due to chemical and products of muscular metabolism such as sarcolactic and carbonate acid. It gradually rose again till it reached its maximum in five or six hours, but after a nocturnal fit it took longer. The preparatory paroxysmal fall he believed to be due to the accumulation of acid toxicines derived from absorption of lactic, acetic and butyric acids from the intestines.

The blood as a rule shows the character of the chlorotic type of anaemia with great diminution
in the amount of haemoglobin. There is a slight
leucocytosis which, according to Page, increases after
a fit. It has already been mentioned that the
blood platelets are increased and that the
blood shows increased coagulability.

As to toxicity the observations are again
very much. Some say it is more toxic and others
that it is less toxic than blood of a normal
person. Hramsky conducted many experiments
in this direction and believed that the blood of
epileptics contained carbamate of ammonia.
He found that if blood taken from epileptics
immediately before an attack were injected into
an animal, paralysis, convulsions and death
occurred, whereas blood taken in the interapontimal
period produced no symptoms. He states that
when carbamate of ammonia was injected into
animals it produced similar symptoms.

I have already stated that Illacomi said
that there was abundant destruction of red corpuscles
at the onset of the attacks. The fit is followed by
an equally active period of reformation.

As with other diseases, here also a parasite
has been thought to cause the convulsions. Bra
took the blood from the veins of the forearm
of twenty epileptics. At certain periods, viz., immediately before, during and after a fit, he discovered the
unvarying presence of a micro-organism. He
describes them as small feebly refracting points,
1 μ and under in length, seen in plasma and
occurring singly, as diplococci or in chains.
When they occurred in chains they resembled a little
worm about the diameter of a red blood corpuscle
and moving rapidly. He gave them the name
of neurococi. No other observer has confirmed
these observations and it seems hardly likely
that epilepsy can be caused by a special microbe.

The cerebrospinal fluid and the sweat
have also been found to contain these bodies,
by several observers, but their results are very
contradictory.

Many of these results are rather
antagonistic to the theory that toxins are the cause
of the convulsions, but skilful observers have found
them present so often in certain cases of epilepsy
that I think they form an important item in
its pathology.

I have again and again observed symptoms
of gastric and intestinal derangement precede a series of
fits and I am sure that in many cases I have
saved the patient from having such an attack by the timely administration of an aperient. The increased elimination of ethereal sulphates before a fit may be taken as an index of the amount of putrefactive change occurring in the alimentary canal.

We also know that convulsions in uraemia and eclampsia are caused by toxins, and these convulsions resemble epilepsy very much. My view therefore is that epilepsy is caused by some toxin acting on an unstable brain. Probably the toxin acts chiefly on the inhibitory cells in the cerebral cortex and causes degeneration of these cells and when their influence is withdrawn, the motor pyramidal cells are permitted to discharge freely and violently, causing convulsions. A normal and healthy cerebral cortex is probably unaffected by this toxin. I do not believe that fits can occur from instability alone and of all theories, the toxic one appears to me to be the most reasonable. I think the problem of the future will be to discover what the toxin is and whence its source.

Bouver Reeves believes that the cells of the...
second layer of the cortex which he found so commonly degenerated in epilepsy, pertain to the sensory type of nerve element and that a functional connection subsists between them and the large motor elements distributed at a lower level. He thinks that these sensory units have an inhibitory control over the adjacent elements, and when this control is withdrawn "their discharge will be subjected to the periodicity of the motoric rhythm."

Pierce-Clark's views are somewhat similar. He believes that the nerve storm begins in the cells of the second layer, which he also holds are sensory, and that this constitutes the aura, but as a secondary result the motor cells are temporarily withdrawn from the inhibitory influence of these cells, and discharge. He says there would be an aura in every form of epileptic fit, if the rate of diffusion of the discharge were slower and less complete than in severe cases. Supposing that our toxin acts on these cells of the second layer, this might form a theory as to the cause of the convulsion.

The question might be asked: if epilepsy is caused by a toxin, why are the manifestations
so different in different individuals? In man as in animals there is a hereditary predisposition to act differently for different poisons. Experiments were carried out by Jeffrey and Verant on various animals with various poisons and the following table gives a résumé of their results.

**Table 7. Showing the various results of different toxins on animals.**

<table>
<thead>
<tr>
<th>Animal</th>
<th>Poison</th>
<th>Epilepsy</th>
<th>Non-epileptic general convulsions</th>
<th>Local convulsions</th>
<th>Non convulsive troubles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rabbit</td>
<td>Ethyl alcohol</td>
<td>1</td>
<td>4</td>
<td>83</td>
<td>25</td>
</tr>
<tr>
<td>Dog</td>
<td>&quot;</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>29</td>
</tr>
<tr>
<td>Rabbit</td>
<td>Surpitol</td>
<td>19</td>
<td>18</td>
<td>25</td>
<td>23</td>
</tr>
<tr>
<td>Rabbit</td>
<td>Aklblyd</td>
<td>8</td>
<td>9</td>
<td>22</td>
<td>19</td>
</tr>
<tr>
<td>Rabbit</td>
<td>Antabole</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Rabbit</td>
<td>Morphine</td>
<td>11</td>
<td>9</td>
<td>10</td>
<td>8</td>
</tr>
</tbody>
</table>

In all cases the lethal dose was sensibly constant even when individual reactions varied.
The Diagnosis of Ideopathic Epilepsy.

The diagnosis of epilepsy is often attended with some difficulty, especially if one has not seen the patient in a fit and has to rely on the description of friends. But even the convulsion itself is not diagnostic of epilepsy and there are other diseases which may produce similar symptoms, and before one can absolutely arrive at a diagnosis, it is necessary to study both the paroxysmal and interparoxysmal symptoms which the patient manifests and to enter into the causes which are supposed to have led to the attack.

The etiology and symptomatology have already been discussed, and it only remains to refer to those features which are more or less characteristic of epileptic fits, before going on to contrast epilepsy with other diseases associated with convulsions.

1. The most important feature of the epileptic fit is the loss or obscuration of consciousness, and if this is not present at some stage of the disease we cannot diagnose epilepsy. Some attacks of petit

mal are not accompanied by much interference with
consciousness, but when the patient has a complete attack, consciousness is lost. This symptom is readily discovered by examining the corneal reflex and the pupillary light reflex, which are absent and return after the fit.

2. Tonic followed by clonic spasm is also an important diagnostic point, and on the account one should endeavour to find out whether the patient has micturated, or bitten his tongue during the fit. It must also be remembered that in idiopathic epilepsy, the spasm always follows the loss of consciousness and never precedes it.

3. The manner in which consciousness is restored. After a fit the patient usually falls into a deep sleep, so if he becomes conscious soon he usually shows some cloudiness of mind, irritability and erroneous ideas. There may be automatisms, excitement or outbursts of temper.

4. Nocturnal fits. If fits occur during sleep, they are almost certain to be epileptic. The patient may be quite unconscious of having had a fit, but he usually wakes up with a feeling as if he had been beaten all over, headache and lassitude. Blood may be found on the pillow if the tongue has been bitten.
5. If the patient has received any injury during the fit, such as a cut, a bruise, or a dislocation, it is highly suggestive of epilepsy.

6. Suddenness of onset, brevity of duration and spontaneity of onset should also be considered, as most other forms of convulsion are slower in their onset, continue longer, and frequently have an evident exciting cause.

But the classical features may be absent and it is almost impossible to state definitely whether the attacks are epileptic or not. Other maladies which are liable to be mistaken for idiopathic epilepsy are (1) Hyperventilation, (2) Apoplexy, (3) Masarce and eclampsic convulsions (4) Jacksonian epilepsy. (5) General Anaesthetic (6) Syncope and (7) Vertigo from eclampsia disease.

Feigned Epilepsy.

Epilepsy is sometimes feigned, but as a rule the maliqurer overdoes the symptoms, especially in the violence of the muscular spasms. He may bite his tongue, foam at the mouth, clench his hands, but he rarely shows indifference to pain, and one symptom which he can never feign is insensibility of the pupil.

However skilful he is, his pupils will dilate
when exposed to light and contract when the light is withdrawn, and the corneal reflex also is present. As a rule recovery is rapid when the police are summoned or when a strong shock from a battery is applied.

Hysteria.

Hysterical convulsions are often extremely like those of epilepsy, and it must also be remembered that attacks of petit mal especially in women and children are not uncommonly followed by hysterical convulsions.

Still there are numerous distinguishing points which will be best brought out by contrasting some of the main symptoms of the two maladies.

1. Usually there is no definite exciting cause preceding an epileptic fit, whereas an hysterical fit is often induced by emotion.

2. The aura of the former is usually easily distinguishable from the globalis hysterica of the latter.

3. In epilepsy there is sudden and complete loss of consciousness, with absence of conjunctival and pupillary reflexes. In hysteria there is no loss of consciousness.

4. Time and place may be an important factor in helping to clear up the diagnosis
The epileptic fit may occur either during the day or night, whether the patient is alone or among friends. An hysterical fit never occurs during sleep or when the patient is alone.

5. The duration of the epileptic fit rarely exceeds five minutes, whereas the hysterical fit may last half an hour or more.

6. Involuntary defecation and micturition which are common in epilepsy, never occur during the hysterical fit, though after it is over the patient usually passes a large quantity of pale limpid urine. Likewise in the former the tongue is often bitten, a symptom which is absent in the latter.

7. In the epileptic fit there is usually the definite sequence of tonic spasm of short duration followed by clonic spasm, which often affects one side more than the other and there is conjugate deviation of the eyes to that side. The patient utters no sound after the introductory cry. The hysterical fit starts with tonic spasm manifesting itself by opisthotonus, emphrophoshtosis and pleurocrathotonos, and the movements are more purposive with struggling and crucifixion attitudes. The patient often sighs, laughs or sobs, and the eyeballs roll and squint.

8. The epileptic fit is followed by sleep,
Headache, and mental dullness and immediately after the fit, the deep reflexes are often absent. The hysterical fit terminates suddenly and the patient is often wakeful and depressed. The reflexes are normal or exaggerated.

Aphoplexy

In aphoplexy we meet with loss of consciousness sometimes accompanied by convulsions. In distinguishing the aphoplectic from the epileptic fit, the age of the patient should be taken into account, as cerebral haemorrhage rarely occurs before forty.

The duration of the aphoplectic fit is much longer and it is accompanied by stertorous breathing and straining. The coma precedes and does not follow the convulsions, the pulse is slow and full, and the patient does not foam at the mouth.

Epilepsy is not commonly followed by paralysis, whereas aphoplexy usually is followed by hemiplegia.

Eclampsia and uraemic convulsions.

These are almost impossible to diagnose from epilepsy, and the difficulty is often increased by the first seizure happening unexpectedly, and unless we are aware of the patient's history, and have previously examined the urine, the true explanation of the symptoms is very liable to be missed.
Even the presence of albumen in the urine after a fit is not diagnostic of kidney disease, as small quantities have been found after epileptic fits. The presence of albumin may give no clue to the nature of the case. Neurotic fits are commonly of short duration and are preceded by symptoms.

Jacksonian Epilepsy.

Jacksonian Epilepsy due to organic disease in the neighbourhood of the Rolandic area is usually easily diagnosed. The tonic stage is usually absent, and the clonic convulsions precede the loss of consciousness, and sometimes consciousness is never lost at all.

The convulsions begin in some definite part of the body, such as the hand, and are often preceded by an aura referred to this part. The mental phenomena of ideopathic epilepsy are absent in Jacksonian epilepsy.

When due to an intracranial tumour, the patient complains of headache, coming without relation to food, and on examination of the retina of the neuritis will probably be found.

General Paralysis.

The convulsive attacks of general paralysis may resemble the epileptic fit, but as a rule the other
Symptoms present will render the diagnosis quite clear, and the age of the patient adds confirmation.

**Syncope**

A faint bears a very close superficial relationship to an attack of petit mal. Powers in his book entitled 'The Borderland of Epilepsy' says that the cause of unconsciousness in minor epilepsy and in syncope may be the same.

It shows that in syncope, loss of consciousness cannot be due to the direct effect of cardiac failure—because consciousness is not the result of blood circulation, but is in great part due to mechanical causes.

The failure of the heart's action causes diminution in the pressure of the cerebral blood vessels and this probably produces an alteration in the nerve elements. What this alteration is, we do not know, but one hypothesis is, that there is a discontinuity of conduction at the junction of the neurons, which impairs each conducting path. The dendrites have a certain power of movement, and slight though this may be, it may be sufficient to cut off the centres which subserv consciousness from all lower centres.

In simple syncope loss of consciousness is rarely so sudden as in epilepsy, unless in fuite
cases due to heart disease, and the patient seldom falls.
It is not followed by the mental confusion and
erroneous ideas of epilepsy, but the patient
immediately recovers his mental faculties,
and there is a sense of great prostration.
A transient aura referred to the heart may simulate
palpitation, thus causing another difficulty in diagnosis.
An exsudate the face gets very pale at once, whereas
this is not the case in minor epilepsy.
Vertigo due to Ménière's disease.

We have already seen that vertigo is a
very common form of aura, before an attack of
petit mal, and may in fact constitute the whole
attack, and the question sometimes arises as to
whether a patient is suffering from epilepsy or
disease of the semicircular canals of the labyrinth.
In both diseases there may be subjective,
objective or actual vertigo. Both may come on with
startling suddenness and the duration may be very
brief. Loss of consciousness even, occasionally
happens in vertigo of aural origin.

When an attack of aural vertigo has been
very brief, it is commonly followed by slight
dizziness for a time or nausea, and the
patient usually has had attacks before which we
not like epilepsy.

Then such evidence of labyrinthine irritation as tinnitus affords is always important and the patient should be carefully tested for deafness.

The patient may actually fall down in vertigo and there is often a feeling of impulsion forcing him down which is not found in epilepsy.

In idiopathic disease, attacks of vertigo may occur during sleep which renders the diagnosis still more difficult. Power explains this nocturnal vertigo by supposing that the equilibration centre can stand receiving stimuli for a certain time, but that it is a cumulative action. The stimuli at last overturn the balance and the patient has an attack of vertigo. If the sensory elements of the semicircular canals are specially disturbed by the horizontal position, the equilibration centre may be brought beyond its stable point, and this is probably how attacks occur in sleep.
VII.

Prognosis of Epilepsy.

Epilepsy is undoubtedly one of the severest diseases which flesh is heir to, not only on account of the terrible seizures which from their suddenness interfere altogether with the patient's social and business life, but also from the way it resists every kind of treatment, and the mental disturbances which are so liable to ensue.

Some writers have even gone the length of saying that it is incurable, but I think we must admit that a certain percentage of cases are absolutely cured and very many are at all events greatly relieved by treatment.

Treatment if it does not effect a cure, will in all probability as diminish the severity and number of attacks as to transform the life of the patient from one of perpetual torment to a comparatively happy and useful one.

In giving a prognosis in epilepsy there are various factors to be taken into account.

1. Age at onset: Epilepsy commencing under ten years of age is as a rule very intractable,
and if they do grow up, the great majority become incurables or dementes.

The most favourable issue is to be expected when the disease begins between puberty and twenty years of age, while from this age onwards to thirty five, there is a steady diminution in the number of cures. Senile epilepsy is perhaps the most favourable of all, nearly all of which can be greatly improved under treatment and a great many cured.

2. Duration of the Disease. If the malady has lasted for several years, there is much less prospect of a cure, than if treatment is begun at the commencement. Under this heading might also be taken the number of relapses, because naturally the prognosis is worse in a man who has had a hundred relapses in a year, than in one who has only had ten in that space of time.

This leads me on to the question of the influence of habit. As Suckling says, "for good or for bad, the repeated occurrence of the same acts facilitates their recurrence." In ordinary everyday matters habit begins as a voluntary act but by frequent repetition it becomes automatic,
though never removed altogether from the control of the manual if he chooses to exercise it. The organs of the body are prone to put on disease action and though this cannot be said to be under the control of the will, nevertheless it can be checked by physiological influences which he can bring to bear on it.

Scofield says "in a disease like epilepsy, habit plays an undoubted and very important part. Every successive attack strengthens the habit, and renders the individual more atrocious to future seizures: every arrest or postponement of a seizure is as much gain in favour of the patient, not only by avoiding the pain and the risk of the related paroxysms, but still more by diminishing his future liability to the disease."

3. Character of the attacks. I do not think that there is much difference between the major and the minor attacks with regard to curability, although W. Turner says that the major attacks occurring alone are the most curable, the combined major and minor fits, while the minor occurring alone are the most incurable.

4. Heredity: Broadbent believes that heredity must enter largely into our consideration in giving
a prognosis. A family history of not only epilepsy, but also insanity and alcoholism is certainly a great factor in the etiology of the disease, but so far as I can make out from various authorities on the subject, it does not influence the prognosis to any great extent. (3) Turner's tables show that as large a percentage of those with a family history are cured as those without, although as regards improvement more is to be expected from those who have no hereditary predisposition.

5. Condition of patient at onset. If along with the epilepsy there is imbecility or moral depravity, the prognosis is gloomy. As also if there are stigmata of degeneration present. Some authors also lay stress on the condition of the pulse. If of low tension the prognosis is bad, whereas if it is of high tension the prognosis is more favourable.

6. Marriage, Pregnancy and Dyspepsia. Marriage does not have any appreciable influence on the course of the disease. In some instances it has an unfavourable influence as sexual intercourse has been known to bring on an attack. "There is an old Latin adage, 'Citus brevis epilepsiae est.' Although marriage may not influence the
patient himself, it is necessary before advising a patient to take this step, to consider the effect on the offspring.

During pregnancy there may be a temporary arrest, but it is seldom permanent. The juniperum is favourable for recurrence and the period of lactation often has an offsetting influence on its production.

4. Caution: If we are able to discover what was the cause of the first fit, we may in many instances be able to give a more favourable prognosis. If it has been some peripheral cause, such as intestinal worms, we hope that when they are removed the fits will cease. But we must be very careful here, because if the fits have lasted some time a habit has been formed and their cure will be more doubtful. If there is no definite exciting cause the prognosis is gloomy.

I therefore regard the following as the most hopeful cases.

1. Those arising about infancy or adolescence.
2. Those in which treatment was begun early after the disease manifested itself.
3. Those cases in which the patient has
a book of bodily and mental vigour, with healthy organs and a strong full pulse.

4. Those in which there has been a definite exciting cause for the fits. Reynolds lays it down as a general proposition that those cases are least amenable to treatment in which the source of the disease is involved in the greatest obscurity.

In no case must we give up hope of a cure and if this is not possible we must remember that nearly every case can be greatly improved and rendered happy by appropriate treatment.
VIII

Treatment of Epilepsy.

The tardy advance made in the therapeutics of epilepsy during the past two or three generations must be attributed to our inadequate knowledge of its pathology and etiology, which necessarily renders it well nigh impossible to remove or antagonize the cause.

Changes of a degenerative kind have been found in the brains of epileptics, but as yet opinion is divided as to whether these morbid changes are the cause of the clinical manifestations or merely evidences of the destructive effects of the storm.

In the same way, with regard to etiology, the balance of opinion is in favour of an autoimmune origin in many cases of idiopathic epilepsy, but this is not yet proved and we are still in ignorance of the essential cause of epilepsy. On this account its treatment must be empirical to a great extent and as yet no rational treatment has been discovered.

We will consider the treatment under the headings:

1. The course to be pursued in the interparoxysmal periods.
2. The proceedings to be adopted during the fit, and
the means by which, in some cases, it is possible to arrest an attack.

Reference will also be made to Psychical treatment and treatment in colonies.

1. Treatment in the Interauricular period.

a. Medicinal Treatment:

Scheuchz concludes the chapter in his book on treatment by observing that "there is not a substance in the materia medica, there is scarcely a substance in the world, capable of passing through the gullet of man, that has not at one time or other, enjoyed a reputation of being antiepileptic." Since the time when these words were written, many drugs, then unknown, have been tried, and from time to time it has been thought that a curative process had been discovered, but the reaction soon comes again and the search begins anew.

The causal treatment is unfortunately possible only in very few cases, as the etiology is so a rule so obscure, and besides even when there does appear to be a definite cause, its removal does not always lead to cure.

Reflex epilepsy which arises from some source of peripheral irritation may be cured by removing the cause. Removal of a tight pinafore may lead
to arrest. Errors of sight should be corrected and
diseases of the ear and nose: pharynx should
have suitable treatment. If the patient is
suffering from worms, these should be got rid of by
suitable medicine. When the exciting cause of
the fit is to be a lesion of a peripheral nerve,
the source of irritation should be excised.
Even in these cases, however, treatment should be
carried on after removal of the local disease.

In 1867 Sedgwick read a paper before
the London Medical and Chirurgical Society, advocating
bromide of potassium in epilepsy, and although the
bromide must be regarded as the most potent agents in
the treatment of this disease, there is a tendency for
the practitioners, when he diagnoses epilepsy to prescribe
tromides, and rest in the assurance that he has
done his duty ammonium atem. But this
should not be so, as, although the bromides may act
like a charm on one patient, another may show little
or no improvement under their influence, and when
large doses are given, harm may be done.

The potassium, podmum and ammonium
bromides are those usually prescribed and I do
not think that there is much to choose between them,
and the combination of the three salts does not appear
to possess any special advantage.

At the National Hospital for Paralysed and Epileptic, nearly a ton and a half of bromides are used annually.

Experiments carried out on animals by Albertoni, show that while under the influence of bromides, the brain does not respond to quickly to electrical stimuli. They lessen the irritability of the central nervous system and diminish reflex activity. Hence when a patient is under the influence of bromides trivial causes such as gastrointestinal disturbances, which might otherwise excite a fit, are much less capable of doing so. Thus the value of the bromides depends on their influence in suppressing the convulsions and not on curing the disease by removing the cause.

Drawbacks: In some cases which are however rare I have found the bromides as harm rather than good. In one patient grand mal attacks were replaced by more frequent petit mal attacks. Here it might be said that bromides have much less influence on petit mal than on grand mal, and as a rule other remedies have to be resorted to.

The administration of large doses of bromides tend to make insane patients more irritable, dirty and
difficult to manage.

The ammonium and sodium salts are said to cause less depression than the potassium one, but I have not been able to discover any appreciable difference in this respect. Even in moderate doses it causes slowing of the heart's action, and dulls the intellect.

If more than 100 grains are given in the twenty-four hours, the symptoms of bromism are very apt to be induced. There is catarrh of the alimentary and respiratory passages, the patient gets into a dull apathetic state and all his movements become feeble and slow. There is nausea and diarrhoea and the respiration is shallow and imperfect.

These disorders appear to be commoner in women than in men. Alone it is very liable to develop and bromine has been found in the nostrils. It may even lead to a local dermatitis and the development of ulcers. Patients who are prone to a certain skin disease may have their own special eruption brought out.

I have found that three minims of liquors arsenicalis with each dose prevents these eruptions to a great extent.
If the large doses are persisted in, the speech becomes slurred, the palatal and pharyngeal reflexes are abolished, there is intense headache and great mental dulness.

Soppe says there are four classes of epilepsy who tolerate the usual bromide treatment badly:

1. Those persons whose secretory organs function badly;
2. Those whose cardiac and arterial organs are incapable of performing their functions properly;
3. Those whose epilepsy is complicated with organic brain lesions;
4. Those whose gastric functions are insufficiently carried out and who suffer from hyperacidity and motor insufficiency.

Administration: When bromides be succeeded in stopping the fits, the drug must not be stopped when this result is attained, but continued for at least two years after the last seizure and then gradually stopped. I have seen the status epilepticus brought on by too sudden cessation of the drug.

The dose should never exceed ninety grains in the twenty-four hours, and if a smaller dose does not keep the fits in check, some other remedy should be tried. As a rule doses of ninety grains a day can be tolerated for years without any ill effects.
and if there is a tendency to skin eruptions
the addition of Fowler's solution is invaluable.

If the fits are solely nocturnal, a single
dose should be given an hour before bed time
and this dose may be gradually increased up to 0.5 grains.
If the fits recur about the same hour in the day, a
good method is to give a similar dose two hours
before the attack is due. In other cases in which
the fits occur at any hour of the day or night, I
give the drug in twenty grain doses, three times
a day.

Maniacal excitement is best treated by
Thirty grains of the former with fifty minims of
the latter given three times a day generally
subdues the excitement.

Where the pulse is of low tension and
where there is valvular disease of the heart — which
is not uncommon, especially mitral stenosis — the
addition of Tinct. Digitalis in small doses is
advantageous.

By attention to detail in this manner,
great improvement takes place in the majority
of cases.

The following table shows the results of treatment
By bromide of Potassium in six fifteen cases of neurotic epilepsy. For three months the patients took no medicine and the following six months they were treated with Potassium bromide in twenty grains doses threes times daily. The cases selected were those who did not take a very

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<th>Large number of fits.</th>
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Bromide of Strontium of recent years has had many advocates since it was introduced by the late Dr. Roche.
Roche recommended twenty grains of the strontium salt with from five to ten grains of Bromide of ammonium or sodium nitrate and morning, diluted with water. He increased the dose of the strontium bromide rapidly until he was giving one dram twice daily where smaller doses did not control the attack.

Dr. N. Sullivan recommends large doses of strontium bromide in combination with syrup of orange peel and infusion of calumba, and if no irritability of the stomach exists he adds ten grains of borax to each dose. He gives for an adult, dram doses of the bromide, four times a day, and he says this may be continued for an indefinite period without affecting the mental condition or producing depression.

My own experience is that very large doses of the strontium salt are required to keep the fits in check, and it exerts its influence much more slowly than the potassium salt and is not so lasting in its effect. On the whole, I am inclined to think that the bromide of potassium is the more useful salt.

Borax is a very serviceable drug, either alone or combined with bromide, but is unfortunately liable to cause gastric trouble with flatulence and anorexia. This is to some extent got rid of by giving the drug after food and increasing the
dose very gradually. An itching skin eruption is sometimes induced and the drug is said to cause alopecia. In cases where there is hyperacidity of the stomach, Apple finds both very useful and beneficial.

Ten to thirty grains of bromide may be given three times a day, or four to ten grains of the hydrate of soda as recommended by Powers. The addition of arsenic alleviates distressing skin eruptions.

A combination of Bromide of Potassium, hydrate of soda, and arsenic, as recommended by Dr. Byron Bramwell, I have found very beneficial in cases where the bromide alone has no effect. The prescription is:

- Br. Arsen. Amy. 30 grs.
- Pot. Brom. 30 grs.
- Soda Bicarb. 30 grs.
- Hyd. Aurant. 30 grs.
- Inf. Int. Bo ad 30 grs.

Mise. H. Mkt.

A dessertspoonful at 9 AM and 3 PM, and a tablespoonful at bedtime.

Heck's treatment has little to recommend it, and I think the dangers connected with the administration of opium, outweigh its advantages.
Gowers reports a case in which death occurred from the administration of a quarter of a grain of morphine a short time before a fit. The influence of the narcotic together with the deep coma that so often follows a fit, may result in death. Thus it seems especially dangerous to administer opium in any form for the treatment of the status.

If morphia has to be given to an epileptic, the dose should not exceed ½ grain.

Schein's treatment consists in preparing the nervous system for subsequent bromide treatment by the administration of opium. Any preparation may be given, but the extract is the one recommended, and its administration is continued for six weeks, in doses gradually increasing to fifteen grains per day. The opium is then stopped and large doses of the bromide from go to 120 grains are substituted, the dose being gradually diminished, till only about thirty grains per day are being given.

During the opium treatment, the body weight almost always shows a diminution and often the fits are increased in frequency. I am of opinion that when improvement does occur under this method of treatment, it is the bromide that
Belladonna is one of the most successful drugs in the treatment of epilepsy when bromide fails, and its combination with bromide is often very beneficial, especially in cases where the patient suffers from both grand and petit mal. The tincture in doses of ten minims is the best preparation. The alkaloid atropine appears to have no beneficial influence on epilepsy.

Zinc has for long been a favourite remedy in France. The salts may be given as oxide, valerianate or lactate, the latter of which was introduced by Staph and given in doses of from five to seven grains three times a day. The oxide is the one best borne by the stomach and it is usually given in full form in doses of three grains which may be cautiously increased to five grains three times a day. The zinc salts are especially useful in the treatment of petit mal. Staph, who was their great advocate relates that he cured twenty out of forty two persons by oxide of zinc. The fulvus antiepilepticus consists of oxide of zinc, radix valerianae, and extractum belladonnae or lycograve.
Copper Salts: Of the copper salts, the ammomin-
sulphate is the one recommended by Brown-Séquard. It is best given in full form, and the dose to begin
with should not exceed a quarter of a grain, and its administration should not be carried on beyond
three or four months.

Other drugs which have been used, need only be mentioned. Camphora monobromata in
doses of five grains is said to be useful in
hypochondriac epilepsy. Probably all the benefit is due
to the camphor, as the amount of bromine is small
in such doses as can be tolerated.

Silver salts were the favourite remedy in the
pre-bromide days, and algypria was not uncommon.
Reynolds relates how patients have come to him,
whose skins were coloured a blackish blue by
its use, whose epilepsy however remains unchanged.

Sulphate of Prussic, Phosphorus, Berum, Bricel,
Lithium, Rommim, have all had their advocates.
Cidvalli and Gianella (9) recommend sulphate of
Duboisine in epilepsy associated with psychical
disorders. They give it in doses of 1/20 grain increased
to 1/60 grain.

Towers recommends rubidum eriine especially
in children with minor attacks, the dose being
a half to one and a half minims of the one per cent. solution.

'Ovarium' (ovarian extract) was reported by Redon to be useful in cases of epilepsy, which appear to be connected with the menstrual functions.

Infusional extract in glycerine has been recommended by Still, and thyroid extract also seems to diminish the number of attacks in some cases.

Bronipin, Bromalin, and Bromacol are patent medicines, which all depend for their action on the bromine they contain.

The above is only a small portion of the list of drugs which have been used in epilepsy, and glowing accounts of which help to swell medical literature from year to year.

TREATMENT OF THE STATUS EPILEPTICUS

If a patient is threatening to pass or has already passed into the status epilepticus, my routine treatment is to give a large soap and water enema, followed by a thirty grain dose of chloral hydrate by the bowel, or if the patient can swallow, by the mouth. A smaller dose may be repeated in a few hours. I have lately seen three cases of the status on
which the fits were quite arrested for the time being by this treatment. Bromides are quite useless here.

In one case which did not appear to improve under the enema and chloral treatment I found very great benefit resulted from the inhalation of Chloroform, but I do not care to use this as a rule.

Hyosine Hydrobromate is sometimes effectual given hypodermically in doses of 1/100 or 1/1000 grain.

Ephedria is strongly recommended by some, but as already mentioned, I do not care to use this drug in epilepsy.

Injection of the bromides has been recommended by Morton; sterile solutions of thirty grains to the ounce are used, and 10 to 15 c.c. of the cerebral spinal fluid being withdrawn, 10 c.c. of the bromide solution is injected.

In the aftertage of deep coma, in which many patients die alcohol should be given freely, and strychnine and digitalis are also useful.

The strength must be maintained, and if the patient is unable to swallow, feeding with the stomach tube should be tried.
Usually give one pint of milk, two whipped eggs and half a pint of beef tea, and it is advisable only to give half this quantity at one time, as it is apt to be vomited if too much is put into the stomach.

If it is impossible to pass a stomach tube, I try nutrient inenata, but however carefully administered they are very apt to be returned.

**General Treatment and Hygiene.**

Toxæmia probably enters largely into the causation of epilepsy. Hamilton of New York believes that the occurrence of the fit, due to the accumulation in the body of some particular toxic agent in quantities sufficient to excite the cerebral nerve cells to convulsive discharge. Eclampsia and the functional epilepsy produced by Atropine drinking illustrate the action of poisons on the brain.

Since coming here, I have admitted over twenty epileptics, some of whom have been maniacal, some demented. Each case has shown some evidence of gastric, intestinal disturbance, such as coated tongue, foul breath, anorhexia, or constipation. My first care is always to get the alimentary canal into a healthy
condition, and in several of my cases this has led to great improvement, both as regards the number of fits and the mental condition. I cannot lay too great stress on the importance of getting all the excretory organs into healthy activity.

Epileptics are very prone to suffer from constipation and they should be treated with aperients, so as to have a good evacuation of the bowels daily. Proper action of the skin also is important and Turkish baths are useful for this purpose. If it is not possible to give Turkish baths, frequent hot baths act almost as well.

The patient should lead a regular life and alcohol in any form should be prohibited. He should have plenty exercise, and games in which there is no danger of injuring himself should be encouraged. His amusements should be those of any healthy person, but dancing ought to be avoided, especially in girls as the excitement and fatigue are very liable to bring on an attack. Crowded rooms are to be avoided as much as possible. Exercise such as swimming, riding, and cycling are dangerous.
He should be given some occupation and here is seen the benefit of the colony system. Needless to say engineering and all dangerous occupations must be avoided.

Marriage ought to be discouraged, not because it has any marked effect on the sufferer, but when we consider the effect it may produce on the offspring, it is the duty of a medical practitioner to discourage an epileptic patient from taking this step.

The education of epileptic children is an important and difficult problem. If the disease has commenced through overstrain, the patient should have complete rest from study. As a rule they should be educated in special schools, as they usually show some mental impairment and are incapable of competing with the normal child of the same age.

They should be taught various forms of manual work such as carpentering, basket-making, and gardening, and the should have both outdoor and indoor recreations.
Diet:

A very important matter in relation to treatment is the dieting of epileptic patients. The majority of epileptics have a great craving for food and overloading of the stomach often precipitates a seizure. Besides the quantity, the quality of the food must be considered, and the fluids allowed in these cases also call for some attention. Spratting says "I know of no single factor in the treatment of this obstinate affection that proves so quickly and permanently beneficial, as proper food properly prepared."

Fleury in the Journal de Médecin, strongly believes in the antitoxic origin of epilepsy, and considers that diet is one of the most important elements in its treatment, and should be combined with methods tending to free the gastro-intestinal tract from toxic materials.

I believe that small meals at frequent intervals are best and they may be given as follows: Breakfast at 8 A.M., Lunch at 11, Dinner at 1 P.M., Tea at 4, and supper at 7. Naturally this is rather difficult to carry on in an institution and as this, but in the cases in which I have tried it, I found there was a great diminution in the
number of fits, and the patients were less troublesome and violent.

As to the kind of diet, opinions differ. I believe that there is no harm in giving meat occasionally, but the amount of nitrogenuous food should be limited. Toulon, who considers that epilepsy is caused by excess of urea acid in the blood, says that all nitrogenuous food should be cut off, so as to limit the amount of urea acid in the system.

An ingenious modification of the bromide treatment was introduced by Dr. Ed. Rehet and Dr. Ed. Toulon in 1889. Their idea was that by diminishing the amount of chloride in the diet of epileptic patients who were being treated by the bromides, the cells would have increased attraction for the similar salt, i.e., the bromide, and its action would thereby be increased. About 210 grams of sodium chloride are consumed daily by ordinary persons, and this can easily be reduced to 30 grams. Toulon's diet was as follows:

Milk, 35 oz.
Beef, 10 "
Potatoes, 10 "
Flour, 7 "
Sugar, 1 3/4 oz.
Coffee, 154 grains
Butter, 1/2 oz.

This diet was given in the following manner,
7 A.M. 9 oz. milk.
11 A.M. 2 Pancakes made with eggs, flour and milk, Sugar and Coffee.
2 P.M. Porridge made with flour and sugar, diluted with milk.
5 P.M. Broth, boiled beef and potatoes poached with butter; all without salt. The remainder of the milk drunk with water.

Bromide of sodanum was given in doses of fifty grams daily and they found that under this regime there was a diminution of 80 per cent of the number of fits.

G. Balint recommends the following diet:
Milk, 1 1/4 to 2 1/2 pints.
Butter, 1 1/4 to 1 1/2 oz.
Eggs, 3.
Bread, 9 1/2 to 12 1/2 oz.

These articles of diet were given without the addition of cooking salt, and contained less than 30 grains of sodanum chloride. So the bread less added 45 grains of sodanum bromide.
instead of the usual chloride. He found that under
this treatment the fits ceased altogether in 80
per cent. of cases, while in the remaining 20 per
cent. the intensity and number of fits materially
receded.

R. Meyer has given bread, which he calls
Bromopan, containing sodium bromide instead
of sodium chloride. Bromopan contained
one gramme of sodium bromide per 150 grammes
of bread, and each patient received 450 grammes,
that is 3 grammes of sodium bromide per day.

Of the patients whom I have treated by
these dietetic measures, the results have not
been so satisfactory, but I think this may be
accounted for by the fact that they were all
confirmed epileptics. During the first
month of treatment the fits certainly were
diminished in number, but beyond this
period improvement was not maintained.

Purin Free Diet: The purin bodies are
substances constructed on a base C5N4 and are
contained in many of the most common articles of
diet. When a purin free diet is combined
with a chlorine free diet, some observers have
noted a diminution in the number of severity of
the fits, and the type of convulsions most favorably influenced is the major severe, but there is little benefit on the minor seizures. Fluids to be allowed.

Alcohol in any form is to be strictly forbidden, and it is certain that it is often an important factor in the causation of the disease. I do not think that it is the direct stimulating action of the alcohol that brings on the fits. It is more probable that it weakens the resisting power of some of the cortical cells, and when the train which has been formed, probably in the intestinal canals, reaches these cells, they are not able to resist it. As mentioned before, it is necessary to give alcohol sometimes in the coma following the status epilepticus, and I believe that Champagne is the best form, half-since does being given as the case requires.

Milk and soda or beef tea sipped hot often relieves the craving for drink and satisfies hunger. Large quantities of fluid should not be taken at one time.

As regards tea and coffee, these beverages should only be taken in moderation
and China tea is the one to be preferred, being the least rich in purin elements.

I think the chief points to be attended to in the dieting of epileptic patients are:

1. Limitation of the quantity of red meat, which should certainly never be taken more than once a day and preferably not so often.

2. Avoidance of overeating and the meals should be taken slowly, and the patient should rest after them.

3. The last meal of the day should not be taken immediately before going to bed, but there should be time for completion of the digestive processes before the patient retires.

B. Treatment of an attack and means by which an impending attack may sometimes be averted.

In cases where the patient has a well-marked aura, it is sometimes possible to avert an impending seizure. It is impossible where the paroxysm breaks out suddenly.

An old method of arresting an attack was compression of the carotids, but favourable results have only been reported very rarely.

Where there is an aura referred to one of the
limbs it is sometimes possible to prevent the fit going any further by applying a ligature round the limb above the point where the aura is felt. A useful measure is that the patient should wear a strap round the limb, which can be tightened whenever the sensation is felt. It is, however, a question whether this may not only result in the postponement of an attack, which when it does occur may be exceptionally strong.

A powerful act of mental concentration on some instances aborts the impending fit. A patient under my care, who has an aura of dizziness, is sometimes able to avert an attack by concentrating his mind on some definite subject or by a determination to overcome the attack. Lately, however, he has begun to show signs of increasing dementia and his fits have greatly increased in frequency.

In like manner, a sudden muscular effort or forced extension in a direction opposite to the warning sensation is sometimes effectual. By this I do not mean the practice of extending the thumb after a seizure has already commenced, which is quite useless.

Powers recommends the inhalation of nitrite of amyl, especially in fits with a cephalic aura.
and if these it seems to be most useful where the aura is referred to the sense of smell.

Inhalation of ammonia also has been used successfully. Strong pressure over the pit of the stomach or drinking cold water are methods which are sometimes useful in cases with an epigastric aura.

Very often the minor fit is merely the aura of a major attack, so it is difficult to say whether these abortive measures really do prevent an attack from coming on, or whether they are only minor seizures which would not have gone on to convulsions.

"The seizure when once begun, wears itself out; we may banish the idea of interrupting it, for the welfare of the epileptic patient so greater the more complete the seizure, particularly after a long interval has elapsed." This dictum of Romberg still retains its significance.

Nothing brings home to a medical man his helplessness, more than to be called upon to treat an epileptic fit.

The only thing to be done during the seizure is to prevent the patient injuring himself. He should be laid down flat on the floor and the clothes about the neck and chest should be loosened. It is only seldom possible to
prevent the biting of the tongue, as when once the fit has commenced, it is impossible to get anything between the teeth, but where there is any aura, a Cork or tongue repressor should be inserted between the teeth, care being taken that such objects do not get into the throat and cause suffocation.

McConaghy describes a method of arresting a fit, by turning the patient on his left side during the tonic stage. In the majority of his cases as treated, the muscles became flaccid and the colour of the face improved. They still breathed heavily and remained unconscious, but no tonic spasm supervened. My results have not been so good, but it certainly often reduces the intensity of the fit.

An epileptic fit in itself very rarely causes death, but there is always the risk of the patient passing into the condition of the status, which must be looked on as a very serious condition. In patients who have nocturnal seizures there is always the danger of suffocation, caused by rolling round on their faces, and this can only be guarded against by attention till the fit is
over. If the patient falls asleep after the
paroxysm, he should be left undisturbed, as it
renders the headache which is liable to follow a fit
much less severe. The frontal headache is often
greatly relieved by a ten grain dose of phenacetin.

Treatment by Psychical Methods

If Campbell Thompson has lately recommended
the treatment of epilepsy by psychical methods,
we have already seen that some fits may be
arrested by an effort of the patient, such as by
tying a cord tightly round a limb as soon as the
aura is felt. In some cases they can be arrested
by an effort of will and it is certainly true
that fits are far less frequent when attention
is being held by any special occupation or other
mental effort.

Borod Sidis has tried to put the hypnothe treatment
of epilepsy on a rational basis. He considers that
in the functional psychoses, the fundamental
reason is a dissociation of groups of neurons,
wherby they become separated from the conscious
mind, although they still exist in the subconscious
mind. His object is to reassociate the
dissociated neurons and to do this the patient
is hypnotised and while in this state an
endeavour is made to make him reproduce all the details of the fit and what went on during that time, the memories of which are, of course, completely lost when he is in his ordinary state.

Thompson believes that fits are arrested by tightening the cord, not by inhibitory impulses being carried up a sensory nerve to the brain, but by inhibition starting in the brain itself and being brought about by the concentration of mind upon the action of tightening the cord. If this is so, the cord method should be useful in cases other than those in which the aura starts in the arm and Thompson says this is so. He has modified Borsic's method, and instead of using hypnotism to reassociate the neurons, he makes the patient use voluntary efforts of memory during the period of a fit which precedes unconsciousness.

Reassociation in this manner would seem to be more of a physiological process than that produced by hypnotism, because in the voluntary method reassociation would take place from the higher to the lower centres, while in the hypnotic method it is sought to unite the subconscious to
the conscious, that is the lower to the higher.
Thompson recommends that the patient be instructed to remember every detail at the commencement of each fit, and when the fit has passed, he should carefully write out all the phenomena he has observed. The power of memory gradually increases and extends further into the fit with practice.

Surgical treatment.

Various surgical procedures have been advocated in the treatment of ideopathic epilepsy, but as a rule all are useless, with the exception of those which should certainly be carried out where some reflex cause appears to be exciting the convulsions. Thus circumcision, removal of a polypus or foreign body from the nasal or anal cavities may cure the disease. Excision of scars where a nerve is involved likewise may result in cure.

Ligation of the vertebral arteries, division of the sympathetic nerves in the neck and ocular tenotomies have had their advocates, but are now looked on as quite unjustifiable.

Sulphur in the skull has sometimes been followed by a cessation of the attacks, but only
rarely is the arrest permanent. The fits may cease for a time after an operation just as they sometimes do after an acute illness, during pregnancy or after an injury.

S. J. W. White considered that in this connection there were four ways by which benefit might have been attained.

1. Influence of the anaesthetic
2. Psychical influence or so-called mental impression
3. Relief of tension
4. Reflex action or the reaction of traumatisms.

He believes that it is by mental impression that the arrest is brought about.

Kocher recommended trephining with incision of the dura under the belief that in epilepsy there was increased intracranial pressure, but there is no evidence of this being the case.

In certain traumatic cases in which there is not a strong hereditary predisposition to epilepsy, there is occasionally improvement from operative measures, but even in these cases, if the fits have lasted for any length of time, permanent arrest is rare.
Every case of idiopathic epilepsy should be treated by medicinal and general means in the first place, and operative measures on the skull should only be performed in cases where nothing so grave and no chance of good too small, to counterbalance the influence of the disease on the life of its subject.

There are rare cases of apparently idiopathic epilepsy, in which during the course of the disease, symptoms arise, pointing to the existence of a new growth, and if this is situated in an operable locality, an operation for its removal is certainly indicated.

Treatment in Colonies.

It is only of recent years that institutions have been provided in this country for the exclusive benefit of epileptics, whose claims were much overlooked both by the state and philanthropic agencies. Many epileptics are unfit to be at large. They are a burden to themselves and to their friends. Parents get discouraged by long medical attendance and longer bills, and treatment is in many instances given up just at the time when it is most required.
The asylum or workhouse was the only place to which the poor epileptic could retreat, when unable to hold his own in the struggle for a livelihood with his fellow men.

The colony system was first started in Germany at Bielefeld, and was afterwards largely taken up in America.

In this country now we have such institutions as: (1) The Magdall Institute, near Liverpool, (2) Meath Home, Surrey, (3) Chalfont St. Peter, Buckinghamshire, (4) David Lewis Epileptic Colony, Manchester, (5) Epsom, Surrey, in connection with the London County Council Asylums, (6) A small one at Bridge of Weir, Scotland.

The object of the colony is to enable epileptics to maintain themselves, while living under suitable conditions for the treatment of their malady. They can earn from 7½ to 2½ per week.

The young epileptics get an education suited to them. The epileptic boy at an ordinary school is made a butt for the sallies of his schoolfellows and even the master looks on him as a trouble and
annoyance.

Litchworth says, "The children are taught according to their capacity for the reception of knowledge, either by means of kindergarten methods, embracing the ordinary object lesson system of teaching, or by the common school methods of instruction in reading, writing etc.

They are also taught useful occupations.

It is essential for Britain's welfare that many more homes be erected for the housing of epileptics. It is a reflection both on the humanity and common sense of the twentieth century, that an epileptic has to qualify as a lunatic before the state will take charge of him.

With regard to medicine in these institutions, the bromides are reduced to a minimum and usually stopped where possible. The patient to keep regular hours, their diet receives special attention, the mind is occupied by games and the amount of work done is carefully regulated.

The principles of treatment in colonies are:

(a) Removal of the epileptic from town and from his family circle.
(b) Regulate and congenial employment under experienced persons.

c) A well ordered and regular mode of life with abstinence from alcohol and abundance of simple nourishment.

(d) Reduction of medicinal remedies to a minimum.

The foregoing resume of the various methods of the treatment of epilepsy will, I hope, suffice to show that much can be done for the majority of sufferers from this dreadful malady. In every case nothing should be left untried until there is improvement, and it must be remembered that the longer the disease lasts, the greater is the liability to dementia.

There are enormous numbers of epileptics in this country today who are, to use Sir T. Lushington-Browne's words, as far as medical treatment is concerned, left to jolt down the hill of futility, heading and heedlessly, or with only the temporary application of the bromide brake now and then.
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