EPILEPTIC INSANITY.

Being a Thesis for the Degree of M.D.
of Edinburgh University.

presented by

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April 1899.

EPILEPTIC INSANITY

In writing on the subject of Epilepsy and its Insanity, I am constantly reminded of the difficulty of the subject. In studying this form of insanity one frequently meets with something which seems incompatible with one's previous ideas or observations. This has a tendency to dter one at the outset. However, knowing as I do, that the smallest observations of one man, insignificant as they may seem to him, may yet be of great value to another whose powers of deduction may be greater, I do not hesitate to commit to paper some observations of my own concerning this fell disease. Here in this large asylum where I come into daily contact with some 200 insane epileptics, I have opportunities not afforded to many. Opportunities which I hope I have made good use of.

In the first place, I may state that I will try to make my paper as simple as possible without the aid of any unnecessary padding.

I shall start by giving a rough classification of some forms of this epileptic insanity I have met with here. I do not intend this to be an arbitrary classification, but to serve as a guide. One is too apt, now-a-days, to slump all the insane epileptics under the the term "dement". How much this may mean

or how little! What a large field of imaginative is left to the hearer! "Oh he's only an epileptic dement", we hear one say, as if that was the beginning and the end of his case. Here then is my classification.

Epileptic Insanity.

(a) With pronounced physical weakness.

(1) Acute Epileptic Delirium.
(2) Acute Epileptic Stupor.

(b) Without pronounced physical weakness.

(1) Epileptic Melancholia (1) acute (2) subacute.
(2) Epileptic Mania (1) acute (2) subacute.
(3) Epileptic Monomania.
(4) Epileptic Circular Insanity.
(5) Epileptic Dementia.
(6) Epileptic Imbecility.

Epileptic Dementia.

I shall consider this first as it is the commonest form of epileptic insanity. By epileptic dementia I mean the chronic enfeeblement of mind occurring in epileptics, which results from repeated fits and occurs often after many years of epilepsy, and tends towards absolute destruction of intellect. This dementia may be primary, arising de novo, or secondary (terminal) following other forms of epileptic insanity.

The fits in these cases have as a rule been frequent and have spread over a period of years. It is

interesting to note here an observation of many writers that petit mal may cause as great and even greater dementia than grand mal. I am quite at one with them as far as my experience goes. So it is not the exhibition of motor phenomena but the effect on the cells controlling consciousness, we have to fear. Another observation I have made is that in epileptics who have night fits only the dementia is less than in those who have day fits or in those who have fits both day and night. The latter exhibit, as one would expect, a graver and deeper dementia.

I take it that epilepsy ocurring during the night is a less departure from state of mental health than epilepsy ocurring during the day. Perhaps I have some support in this theory given me by the fact that night terrors and nightmare, which I believe to be closely connected with epilepsy, occur in people who are quite sane. Furthermore epilepsy often manifests itself first during the night and as the disease advances it shows itself during the day also. Very often also when an epileptic patient has been free from fits for several months and is again giving way to the epilepsy, the fits occur first during the night.

Again epileptics who fit at night are as a general rule longer in becoming insane.

Now it may be a very important thing to know whether any given epileptic is really demented or not. As I shall say later on Epileptic dements are liable to maniacal outbursts. Now suppose an epileptic is admitted into an asylum insane in a state of mania, how are you to tell whether this is mania occurring in a demented epileptic or a case of what I call Simple (acute) Epileptic Mania? It is impossible to tell in the majority of such cases on admission. You have to wait till the mania is over. You can then test this epileptic carefully and for some considerable time. The best test is that of memory. If a patient has a good memory he cannot be demented, but if he has a bad memory he may be so. I say "may be" because if you test epileptics sane or insane after a fit you may find their memory gone or very bad. Hence choose your time and opportunity well. Most cases of dementia are evident, some are on the borderline, and these are the ones we must watch. I am sure there are many epileptics now in asylums who could live perfectly well and much happier outside. One is far too apt to think once an epileptic is insane, he will always be insane. No one will deny that there is a great tendency to recurrence of the insanity, but after an

epileptic has been rational for a year or even six months even though the fits continue why keep him in the asylum?

It is important that the symptoms belonging to epilepsy proper are not taken as symptoms of insanity.

The various symptoms of the demented epileptic are well known, their irritability and want of inhibitory power; their religious emotion and fondness for lying; their violence and hypochondria and hysteria. These demented epileptics are liable from time to time to break out into a state of acute mania. Many of them are then uncontrollable. In a true epileptic maniacal outbreak the patient is only semiconscious of his acts, or it may be quite unconscious of them. However, there are other maniacal outbreaks where the patient is quite conscious of his acts and knows perfectly well what he is doing. I have noted this repeatedly. As regards the termination of epileptic dementia, there is no recovery. The most that can be hoped for is that the patient may as time goes on, become manageable and quiet so that he can be allowed home if desired. Some of them remain practically the same for years, others become more and more stupid, others again pass into a state of chronic stupor.

Many of these epileptics become so weak and paretic that they have to be kept continually in bed. Epileptic dements generally die from intercurrent diseases, especially diseases of the chest. (See tables) They may smother themselves in bed in a fit. They may die from status, delirium, or exhaustion from frequent fits. I shall now detail a typical case.

M. B., aged 32, Card-room hand. Admitted 9th January, 1897.

The history is that patient developed epilepsy when she was twenty one, before that she was a healthy woman. There is no heritary history of any sort, and her home surroundings are good.

Her friends report that she gradually became excited after the fits and seemed to lose her memory. She was always worse when she was six or seven weeks without fits. She is said to have been insane for nine months. About a week before admission she failed to recognise her mother, sister, and others. She also at that time became very rough and unmanageable.

The medical certificate reports that she looks dazed and refuses food; refuses to answer questions; says she has lost her eyes, though she can see; in answer to questions why she won't work says her work

is both at home and abroad; she threatened a man with a jug and he had to get assistance to get rid of her. She is reported to be epileptic, suicidal and dangerous.

On admission her bodily condition was good and there was nothing physically wrong to be made out.

On January 9th she threatened to injure those around her but had made no attempt to injure herself. She was stupid, heavy, and demented and could give no coherent account of herself.

On January 14th she was reported to the Commissioners as being demented; having no memory; making confused statements; saying her head was dizzy and it was working down into her throat; saying she was troubled with bad thoughts of things she had seen in America and since her return.

On January 24th she was reported stupid and idle, disinclined to work, confused in her ideas and does not speak spontaneously.

On February 14th she was reported to be very noisy and excited and had to be secluded. When they went to open the room next morning she violently assaulted the nurses. She threatened to murder one of them.

In June she was reported as having been much quieter for some time. She was then on bromide.

She still broke out at times.

In December she was reported as being demented, excitable, quarrelsome, especially when having fits. Her bodily health kept good.

In May she was reported unimproved bodily or mentally, but quieter. Was then on epileptic medicine (Potassium Bromide twenty grains thrice daily.)

In August she was quieter.

In December she was reported to be noisy and excitable, constantly walking about giving orders and demanding things; violent and agressive and not to be reasoned with; dirty and destructive; and in moderate bodily health.

She is now a quiet demented and very stupid epileptic who occasionally becomes maniacal, when she has delusions of persecution and is hypochondriacal.

EPILEPTIC IMBECILITY.

As a classification of Epileptic insanity would not be complete with this variety left out I include it but my observations here have not been extended over so many cases so my deductions cannot be so accurate.

There are many varieties and in most of them some organic change is demonstrable after death in the brain. Many of these cases show the results of infantile paralysis.

In the statistics I have made it will be noticed that in some cases the fits commence immediately after birth, and the imbecility and fits are coincident, in others the fits do not appear till the age of two or three and are sometimes associated with dentition.

These cases are disgusting, hopeless, and degraded. They often have violent maniacal outbursts. Vanity is a frequent mental attribute. Many of them are intensely erotic and I was struck in reading over the cases with the number who said they had "lots of sweethearts".

The imitative faculty is largely developed and many of them learn to swear and masturbate from the society into which they are thrown. This I think is a sufficient reason why imbeciles should be in a separate asylum, or at any rate, in a separate ward from other patients.

This is the sort of case one meets with here. C.W., female, aetat 16; no occupation; admitted April, 4th, 1896. The history is that she was born

an imbecile and became epileptic at 12 years of age. Her father, brother, and sister were all epileptic. She was not suicidal or dangerous.

The certificate of admission said "Talks in a wild rambling manner; gives contradictory answers; says one of her fathers has gone to Heaven with a little sister, that another father has got a diadem; thinks one of the nurses is her mother."

On admission she resisted examination, screamed, spat, and bit when touched. Articulation was indistinct, she was noisy and dirty. She could take her food but could not dress herself. The report to the Commissioners said "She is idiotic and very excitable, noisy, destructive, "wet and dirty", and fights and struggles if interfered with in any way.

April 15th. Very violent and unmanageable. Fights, scratches, and bites everyone indiscriminately whether she is molested or not. Has a harsh rough voice, shouts, screams and sings from morning to night. Sleeps in a single room and requires seclusion sometimes by day.

April 25th. Will pull down every one within her reach, and fix her teeth into them. Smashes extensively. Has had two or three showerbaths and

is becoming more subdued.

July 12th. Has a phlegmonous inflammation of the upper left eyelid to which warm boracic dressing is applied. Mentally is noisy, unreasonable, tawdry, destructive, and dirty. General health fair.

September 15th. At present in bed in hospital where she has been for some time owing to restlessness and stupor following on some epileptic seizures. Throws herself about, will not keep her clothes on, and lies about on the floor.

September 17th. No change: still confined to bed: noisy and confused.

September 25th. Very noisy and excited, will not stay in bed, and pulls the clothes off the other women.

November 28th. Is up but very noisy and excited and will not keep her clothes on.

December 23rd. Is in a single room off the hospital as she is very restless and excited and will not keep on her clothes. Her heel and the skin over the sacrum are abraded, where she has picked off the epidermis. If she escapes from the single room she goes about the hospital pulling off the other patient's clothes and otherwise interfering with them. Is con-

tinually shouting and screaming.

1897. January 7th. No improvement. Is still excited and screams in a mechanical way. Is resistive, picks herself, and has bursisis over the left elbow. Is very dirty in her habits. Is getting extra diet and porter and a draught at night.

January 26th. Still maniacal and restless. Is getting thin and emaciated. Screams, throws off her clothes, requires to be fed and is very dirty in her habits, smearing herself with excrement and also eating it. Is getting milk and stimulants.

March 21st. She is "wet and dirty," noisy, mischievous, tears and destroys, shouts and screams all day and is very troublesome. Her bodily condition is fairly good.

April 3rd. Secluded and given sulphonal for being noisy and excited.

July 31st. Continues to be troublesome, noisy, "wet and dirty." Bodily condition improving.

1898. January 16th. Sent to bed in hospital. She is getting very thin and having more fits. Her appetite, which as a rule is voracious, is now poor.

March 18th. She is quite incapable of rational conversation and unable to appreciate her

surroundings; is very noisy and dirty in her habits. Requires to be fed and dressed. Her bodily condition is fair.

May 31st. For three weeks has been suffering from purging and pyrexia, but seems somewhat better now. Has been and is in bed.

June 4th. Patient who has remained in bed since the beginning of May died rather suddenly from phthisis.

The Post Mortem Examination revealed the meninges thickened and opaque. Cerebral hemispheres generally congested and soft. Ventricles dilated with excess of fluid. Both pleurae adherent in places. The lungs showed patches of consolidation and some small cavities and a few caseating tuberculous masses in both upper lobes. Both lower lobes congested.

The heart was small and there was commencing atheroma of the arch of the aorta. The left ventricle was hypertrophied and the mitral valve thickened.

There was a solitary tuberculous ulcer in the small intestine.

The liver was fatty. The kidneys were congested.

Epileptic Monomania.

By this I mean a delusional form of insanity occurring in epileptics in which the delusion forms the principal and sometimes the only symptom of insanity. It is as a rule a fixed delusion and in many cases the person may be taken for sane if you do not know his delusion. Considered from other standpoints his intellect stands perfect or nearly so. Epileptic monomania may be associated with some mental enfeeblement, but in that case I would classify it is dementia. Now many of these fixed delusions arise from the sensations which are associated with epilepsy. They are as it were intensifications and misrepresentations of the aurae and other sensory disturbances met with in epilepsy. We would not call an epileptic insane who told us that often he felt a feeling as if cold water were running down his back, which often preceded a fit, but if this sane man persisted that he had an iceberg on his back or that he had a cold knife in his spine or that people were continually "acting" on his back we would begin to doubt his sanity.

These various and curious sensations often lead the epileptic to believe he is being persecuted by some unseen agency. It may be he thinks it connected

with electricity or "wireless telegraphy" as in one of my cases.

The delusion generally persists and in most cases the powers of the mind fail altogether and the patient passes into dementia.

Many of these cases are of a most violent type. They often fix on some patient, nurse, or attendant who they consider acts on them and wreak their vengeance on such people whenever they get the chance.

I shall now quote a case:-

Case of Epileptic Monomania.

M. R., aet 37, housewife, admitted 12th December, 1898. There is no history of a hereditary nature. The medical certificate reports. "She thinks there are bottles in her inside put there by the Doctors; that people have cut her tongue out and others have tried to put her eyes out; that she hears voices. She is subject to epileptic siezures and is very violent at times, attacking nurses and others about her." She was reported to be dangerous but not suicidal.

On admission she was in good bodily health except for her epilepsy, and had a slight eczema on her arm. She could talk fairly rationally on many subjects. Had a good memory. She, however, suffered from the

monomania of persecution and she commenced at once to explain to me that the eczema on her arm was the work of an enemy. She also stated that some enemies had cut her tongue for her, the facts being that she had bitten her tongue while in a fit some days previously. Every little ailment she had she misconstrued as the work of enemies.

She was certified to the Commissioners as being deluded and as having auditory hallucinations; as saying that people cut her tongue and illused her while she was asleep.

She has remained quiet since admission except once when she became violent, thinking some of the other patients were the enemies she thought were acting on her.

In this case the delusions of persecution make up the whole insanity and account for every insane act of the patient and that is why I classify it as epileptic monomania.

We will now pass on to the more acute forms of Epileptic Insanity.

Epileptic Mania.

By this I mean the acute exaltation and excitement occurring in an epileptic not otherwise mentally enfeebled - not demented.

Now the neurosis epilepsy is calculated to so impair the mental stability that causes, insufficient to cause insanity by themselves, acting in conjunction with it may turn a person insane, such causes being perhaps capable by themselves if intensified of causing insanity in a person not epileptic. Let me explain myself further. I want to convey to you the impression that a cause which would not cause mania or melancholia in a person not epileptic may cause mania or melancholia in a person not epileptic because that person is epileptic. But I wish you also to understand that an epileptic may become insane apart from his epilepsy altogether from causes which would act were he not epileptic all the same. Now we may have epileptic mania resulting from the epilepsy itself; or we may have epileptic mania resulting from epilepsy plus some other cause (as drink, domestic worry etc.); or we may have simple mania occurring in an epileptic.

In the first case we notice that the fits bear some relation to the mania, frequently the mania

following a "bout" of fits, sometimes preceding this.

In the second case we always find some other cause and not necessarily any relation to the fits though these may become more frequent (or in some cases cease) when the primary cause has begun to act.

In the third case there is no difference in the fits. They do not become any more or any less frequent.

Now in the first form if we can relieve the epilepsy we may do some good.

In the second form we may treat the epilepsy also with good result, but the result will depend a good deal on the other cause. If one cause ceases to act the patient may recover.

Now epileptics are apt to become insane after a bout of drinking. Now it is the drink and the epilepsy that causes insanity. When the effect of the drinking has worn off, the patient may become quite himself again.

In the third case also the patient may recover.

Nevertheless recovery in epileptic mania is not common, but I believe the second class of cases are the most likely to recover.

The mania of the epileptic is indeed terrible

to behold. The incessant talking and shouting, the motor excitement, the utter depravity are very terrible. There are many and various hallucinations and delusions and sleep is impossible.

As far as my experience goes I have found that it is the exception for epileptics to have fits when they are in a state of acute mania. This is indeed a blessing as we can more safely put them into single rooms under these conditions.

This also applies to the mania which occurs from time to time in epileptic dements.

There is one form of epileptic mania I should like to refer to, and that is a transitory maniacal condition occuring in an epileptic not formerly insane. This bears a similar relation to epilepsy that "Mania a Potu" bears to alcohol and like it is a condition which often passes off in a few days or at most weeks, leaving the patient little the worse for the attack. In many of these cases there is a good deal of hysteria also.

I shall now give four examples of cases such as I have mentioned above.

Mania from Epilepsy.

E. McG., aet 20, cotton operative, admitted 7th March, 1898. The hereditary history is that her father was a drunkard. There is no other family history. The history of the present illness shows that in February she had more fits than usual and complained of great pains in her head. Since then she gradually became worse mentally. The medical certificate says, "Incoherent: says she is the Virgin Mary; violent at times and uses her teeth freely when she gets the chance." She was stated to be suicidal and dangerous.

On admission she was talking incoherently, paying little or no attention to questions asked her. Her brain was stimulated by objects catching her eye. Hence when she saw the curtains she said "White curtains, mother." When I examined her the next day she was restless and rambling. When asked what a pencil was she said "A pen and ink." She maintained that she was in a church. Her mania had a somewhat religious tinge.

She was reported to the Commissioners as being restless, excitable, quarrelsome and violent; fighting, biting, tearing and scratching the other patients; being incoherent in conversation; having no memory;

saying she had been seven years here; and not knowing where she was.

A fortnight after admission she was reported much improved, quiet and well behaved.

Three weeks after admission she was still better, and worked a little and amused herself. She remained so for three weeks, and increased in weight and bodily strength. The fits, which were frequent, began to occur only at night.

In October she was reported as being in good bodily health except for fits and as being calm and quiet.

One year after admission she was reported as being rather weak minded and childish; becoming excited at times, talking and laughing away to herself in a silly manner; being quarrelsome and aggressive; but in fair bodily condition. She remains so at present. She will probably gradually become more and more demented.

The next case is one where Epilepsy was complicated with drink as a cause.

A. C., Farmer, aet 49; admitted 4th January 1898. There is a hereditary history of drunkenness and his mother had three "strokes." The medical certificate

stated, "He says the school children told him about a lot of money in some boots, out of which he can make £100 a year. Is coherent." His son reported that he threatened to murder his sister. He bought stock he had neither room nor use for. He was reported to be dangerous but not suicidal.

On admission he was excited and noisy, and had to be confined in a single room. He had to be kept in a single room for more than a week after admission. He said he was persecuted by devils which flew about the room. He was then very noisy and excited, and required constant watching. He was reported to the Commissioners as being maniacal restless and incoherent; as having delusions, saying his blood had nothing in it; rambling about large sums of money.

On the 24th January he was reported quieter but still hallucinated.

In February he was quiet enough to be allowed in the smoke room, but was still very incoherent.

At the end of February he was weak bodily and mentally confused.

He remained practically in the same condition till April when he developed erysipelas of the face. He recovered from this in a fortnight and was sent back to the day room.

In June he was transferred to a quiet ward.

Here he remained and gradually became demented, one year after admission, being reported dull, heavy, demented and stupid, being unable to converse, nothing but the briefest answers being got from him.

This case shows the grandiose delusions which may exist in epileptics and especially in those cases associated with the drink habit. These cases may easily be confused with General Paralysis.

The next case of mania is one where the person became insane independently of epilepsy.

A. McC., aet 23, charwoman, admitted 30th October, 1897. The history is that she developed epilepsy when sixteen years of age. She had few fits however but these did not bother her much. There was no change in her fits just before she became insane, nor did they increase or decrease in number after that period. There was no hereditary history of a neurotic nature.

The medical certificate on which she was admitted says,"Talks in a wild rambling manner, won't answer questions, laughs when spoken to, says she has been married several times, sometimes shouts when spoken to and then sings quite loudly, has a wild expression, and requires restraint in a padded room.

On admission she was silly and erotic, laughed at everything said to her. Said she naver had any fits, which was not true. Was, however, fairly quiet. Whenever she was questioned she answered by a lot of silly nonsense.

She was reported to the Commissioners as follows. "She is insane, complains of the police misrepresenting her and of the nurses at Withington trying to make her tell lies; says they told her "if you do not answer this or say that you will be turned out of here;" says they excited her and made her break windows; has behaved quietly since admission but she is evidently still unsettled in mind and when speaking to me laughs at intervals without cause." In November she was reported quiet and well behaved, but otherwise not much improved mentally.

On the 27th November at night she had an epileptic fit in bed and was unconscious for an hour and a half after it.

In December she was reported a good worker, cheerful and well behaved.

On the 10th December she escaped but was brought back at night.

In February she was free from delusions and improved bodily and mentally.

In April she was discharged recovered. In this case the epilepsy had probably little or nothing to do with the insanity which I believe was only a simple mania brought on by adverse circumstances and want.

Epileptic Melancholia.

Instead of an epileptic becoming maniacal he may become melancholic. This may occur in an epileptic previously sane or may tinge the dementia of epileptics or in some instances may precede an acute attack of mania in an epileptic. An epileptic melancholia is one of the most miserable of mortals. He will pay no heed to comfort, will refuse his food and walk about or sit in a corner brooding over his delusions. If disturbed he may lose his selfcontrol altogether and attack his would-be-friend.

In many epileptic melancholics the delusions refer to the thoracic, abdominal, and pelvic viscera and I have no doubt that there are in real truth sensations and perversions of function in these organs which are misconstrued by the epileptic.

Thus hypochondria is a common symptom. Delusions concerning the bladder and especially the uterus are

frequent. This is important as in many of my epileptics here there is often menorrhagia and metrorrhagia, often associated with uterine displacements. I have one such woman in whom there is no such foundation for delusion, however, who continually affirms she has a wound in her uterus and a tremendously enlarged bladder. She was under many specialists before admission but never could any of them make out anything abnormal in the parts mentioned.

The function of menstruation is much altered in epileptics especially when they are having fits. Some of my patients have frequently to be put to bed for floodings. (See tables).

I have no doubt that the peculiar sensations epileptics are subject to are quite sufficient to cause melancholia in them. The unsuccessful treatment, the mystery attached to their disease, the hope deferred all tend to strain the patient's strength of judgment and mind. I have noticed in several cases a train of religious emotionalism which stamps them.

In many epileptics this is the case but especially so in the melancholic ones. Their delusions partake of this nature. They have offended God, they have committed the unpardonable sin, they have lost the

Pope's blessing. I remember one case who on admission kept shouting, "Oh those angels, I see the Almighty God. Take me to church, Church, Church, O Father P----! Oh God! Oh God! Oh Christ!" Then covered her face with her hands and wept bitterly.

Neuralgia is a common symptom. Gastric troubles and symptoms are common.

Epileptic melancholia does not have such a close relation to the fits as epileptic mania. It does not so frequently preceed or follow a series of fits. Like mania it may tinge the outbursts of the demented epileptic. I believe as a general rule epileptic melancholics have fewer fits than epileptic maniacs. Hysteria is often associated with the melancholic outbursts. Epileptic melancholia is not so grave as epileptic mania as regards prognosis especially if the epilepsy is not the only cause of the insanity.

In my tables I found that about 25% of epileptic melancholics recovered as against 18% of epileptic maniacs.

I append an illustrative case.

Case of an Epileptic Melancholia.

E.B. aet 29, charwoman; admitted 5th April, 1897. There is no history forthcoming. The medical

certificate stated, "Talks in a rambling, disconnected way, contradicts herself, says she has been here a long time, thinks one of her children has been here to-day, says her food has been tampered with."

She is reported to have refused her food and have been very depressed; to be suicidal but not dangerous.

On admission she was in moderate bodily health, but was very depressed and melancholy and could give no reason for being so. When questioned she burst into tears. There were no delusions ascertained. She now explains that she refused food because she found it impossible to take it.

In April she was reported to be calm and quiet and to know that she was in an asylum.

At the end of April she was reported cheerful. She remained rather better in mind till October 17th when she spat blood and signs of Phthisis were elicited in the lungs at both apices.

On the 28th April, 1898, she was reported emotional and hysterical and said she would be far happier dead and in Heaven. She was sent to the Hospital for consumption, when I lost sight of her till February, 1899.

She was then, I thought, rather enfeebled mentally and became excited and violent at times.

Her consumption gradually progressed, but at the same time her mental condition became somewhat better. At the present time, two years after admission, she is irritable and querulous; melancholic, weeps for trivial reasons; is excitable, fretful, and very quarrelsome; collects rubbish and secretes it about her clothing; she has chronic phthisis.

Epileptic Circular Insanity.

Perodicity is a marked symptom in epileptic insanity of whatever form. Very often in women the maniacal and melancholic outbursts arise regularly at the time of the menstrual flow or take its place. Then there are some insane epileptics who always have their outbursts at night and are quite quiet the next day, and the same thing continues for several nights if not checked by drugs. But there is more or less definite form of epileptic insanity to which I choose to give the above name in which the symptoms of insanity constantly recur at a more or less regular interval with a period of sanity, to all intents and

purposes, intervened. Now these are the very cases that have to be watched most carefully as one is liable to think that they have recovered. As a rule the most marked cases are in young epileptics, especially in women. Often in the quiet or sane period the patient is free from fits though this is not invariably the case. I have one such girl where there was a period of ten months in which there were no fits and the mental balance of the patient seemed restored. We promised her discharge in a month should she keep well but unfortunately before that time had elapsed she had passed into a state of status epilepticus from which she tardily recovered. However, she did recover and is less insane now than she was when she was admitted. An interesting point in this case is that whereas formerly she used to have fits day and night but very much oftener during the day, she now has fits only at night and they are described by the nurse as being very slight fits.

In epileptic circular insanity I have not noticed the regular advent of melancholia, mania and comparative sanity one sees in the typical Folie Circulaire. But there is always a period of

comparative sanity preceded by mania or melancholia with in nearly every case a stuporose condition.

Thus we get circles made up of various mental states. We may have mania for a few weeks, followed by stupor for a long or shorter period, then a period of comparative sanity which always lasts for a good time, never less than one or two weeks. Then the same regular course is followed next time the patient goes "off it".

Melancholia may take the place of mania and we get melancholia, stupor, comparative sanity. I have noticed other varieties thus in one case I find, Melancholia with delusions of persecution, weeping etc., lasting for a week in a more or less acute form, gradually tapering off to comparative sanity. The latter lasts for months when the patient works well and is a very useful person in a ward. Next time she bursts out she will have a maniacal attack and will be very violent and excited, knocking the other patients about and will be a very dangerous individual. Then follows the calm which lasts a long time. After some months she becomes melancholic again, and so the process goes on.

I have other cases in the wards of regularly recurring stupor which is not followed or preceded by mania or melancholia. These seem to me to be cases where the mental powers are completely exhausted by the fits. When the stupor is actually on, at least in the worst cases, the fits are absent or very infrequent. This came as a surprise to me when I first noticed it. However, I am more reconciled to it now having noticed many cases. The brain motor centres are too exhausted to pass into the state necessary to a fit. I have noticed that the advent of the comparatively sane interval in these cases is heralded by a fit or a series of fits after which the patient becomes more or less rational. I may mention here that in these cases I have found the bromides worse than useless. They seem to deepen the stupor and cause the patient to become feeble on her legs and much more liable to the contagion of phthisis, erysipelas, typhoid, influenza, or such like diseases which may be about at the time.

In pure cases of circular epileptic insanity the prognosis is fairly good compared to the other forms of epileptic insanity. Out of six cases noted in my tables, two recovered.

Here is such a case.

M. R. ,aet 24, domestic; admitted 30th October, 1898. There is practically no history of a hereditary nature except that her uncles was insane. She commenced to have fits at the age of eighteen, and since that time her mind has gradually become impaired. She was not suicidal or dangerous. The certificate of admission states that "She is low spirited and depressed; keeps her face averted and her eyes downcast; keeps repeating "There's nothing the matter with me;" declines to answer when spoken to; refuses food."

On admission she was in a state of nervous tension and said people were persecuting her. She was agitated and seemingly frightened for something indefinite. She had a fit on the night after admission. The report to the Commissioners says, "She is melancholic: says something has gone wrong at home; she is quite sure of this but no one will tell her what it is; she sees proof of this in three patients who are lying in bed near her; says that they have come here and to that pass through this affair; is unable to converse rationally."

Soon after this on November the 8th she was reported cheerful and better, but in a month she passed into a state of mania, was deluded and hallucinated,

struck the other patients thinking they were going to shoot her. This lasted three months, during which time she had frequently to be secluded.

In June 1898 she was reported to have remained well for three months but was beginning to get upset and agitated after fits.

She got gradually worse till September, when she had another melancholic outburst. She then settled again but in February last she became violent, maniacal, and excited, losing all selfcontrol. This lasted for one month, and she is now quiet again.

We now pass on to those cases whose characteristics include great physical weakness.

Epileptic Delirium.

From a clinical standpoint this may partake either of the nature of mania or melancholia.

One feature of these cases is their comparatively sudden onset. We are told by the friends that the fits never affected the patients mind till quite recently. The fits are now absent altogether, as a rule. A fatal termination is very common. Pneumonia and bronchopneumonia frequently cause death.

The patient when seen is generally chattering incoherently and continues to do so often for days, even though draughts and sedatives are administered.

The utter want of object consciousness and the oblivion of the attention are very marked. Hallucinations flit across the patient's senses in a cinematograph-like manner. The rapidity of expression is bewildering to the hearer. One cannot follow the ravings. The extreme exhaustion and typhoid like state which follow are very marked. The pulse is accelerated, the tongue black and parched, the mouth and teeth offensive and covered with foul sordes. There is complete constipation. There may be suppression of urine. I have seen cases where on passing the catheter no urine could be drawn off. There may be retention of urine which must be instantly treated. The abdomen is often collapsed and the abdominal wall. There is a tendency to vomiting, retching, and hiccough. These latter I have always found to be the forerunners of a fatal termination, if they do not subside but become continuous as it were. The patient cannot swallow, partly from the dryness of the

throat, partly I believe from nervous influence.

The skin is dry, hard, and burning to the touch. The temperature is elevated as a rule, though generally not over 102oF if the case is uncomplicated. Frequently it varies from 99oF in the morning to 101oF at night. The patient has generally to be put in a single room, often refusing to stay in bed. Darkness and quiet are indicated. Stimulants are needed. Laxatives, and enemata if possible, must be given.

The patient has often to be fed by the stomach pump. Milk and eggs make the best diet. Warm baths if possible should be tried. Hypnotics as a rule are hopeless, but the best is paraldehyde, which may be given in IV doses at night.

It is well to avoid letting the nurse spoon-feed these cases if they show much resistance as they often "swallow the wrong way" and bronchopneumonia results, which in such a condition is fatal.

Let us look at such a case.

M. D., housewife, aet 62; admitted 24th January, 1899. This is the first attack of insanity. The date at which she became epileptic is unknown. She has been

insane fourteen days. There is no further history except that she has been very anxious about the illness of her husband, and this is given with epilepsy as the cause of her insanity.

On admission she showed fairly extensive bruising. Her pupils were equal; tongue foul; bowels constipated; appetite fair; sleep bad; expression ecstatic.

January 27th. Patient cannot converse as she is entirely occupied in singing an incoherent chant all day. At times she can be got to answer monosyllabically. She is noisy at night and has had to have draughts at bedtime. She is epileptic, suicidal, and dangerous. She has frequently to be removed to a single room as she is uncontrollable in bed.

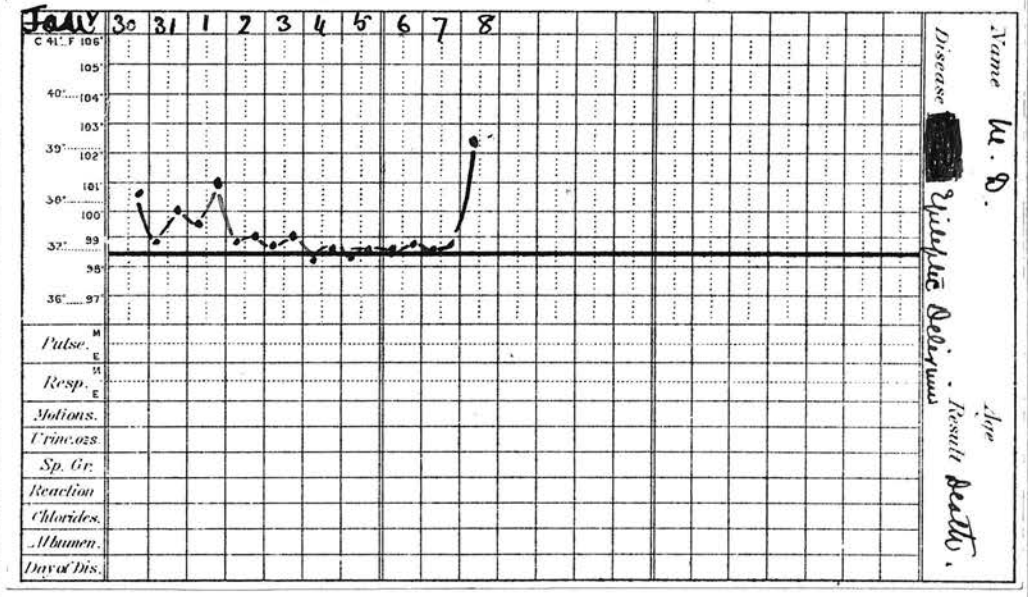
January 31st. She is restless, incoherent, noisy and quite unable to answer questions or converse; refuses food and is wet and dirty. She is rather feeble and haggard looking. Her temperature was 100-6F last night, normal this morning.

February 7th. Is much quieter now though restless. Her temperature is now normal. Has been in bed since last note.

February 8th. I was called to see the patient yesterday. She was reported to be swallowing badly.

When I saw her she was in a more or less torpid condition. There was some fluid food at the back of her throat which I removed. She was given two ounces of brandy per rectum. At night (5-30) I found her very weak. Pulse 125 per minute. Respirations 40, regular. Pupils normal in size and react to artificial light. Crepitations over left apex in front. Atrophine Sulphate $\frac{1}{100}$ grain was injected and one ounce whisky given per rectum. Seen again at nine and found somewhat better. Temperature however was 102.4F. Seen again at 11-30 when $\frac{1}{50}$ grain atrophine was injected. Slept till late this morning. Is now dyspnoeic, pulse rapid and irregular; breathing deep but regular. Brandy one ounce every half hour given by rectum. She died in the afternoon.

I append the temperature chart.



At the post mortem examination the following changes were found.

1. Brain and its coverings.

Skull-cap thickened; diploe deficient; heavy. Dura Mater very much thickened, not adherent. Pia-arachnoid strips readily from subjacent convolutions. Brain firm and hard. Brain substance contains a lot of serous fluid. Ventricles not enlarged. Nothing obviously pathological in the hemispheres, ganglia, cerebellus, pous, or medulla.

11. Chest.

Heart of average size; nitral stenosis; left ventricle not dilated; some thinning of the wall of the right ventricle. Left lung shows its lower lobe much congested, and passing into a state of consolidation. Right lung also shows congestion of lower lobe.

111. Abdomen.

Liver slightly enlarged; shows nutmeg appearance. Spleen very soft and friable. Kidneys apparently normal to the naked eye.

Acute Epileptic Stupor.

Stupor following nervous discharge occurs in epilepsy to a greater or less extent after every fit. The length of the stuporose state depends on the rapidity with which the cells in the brain take to energise again. This condition is a merely transient one, however, in most epileptics sane and insane. But the acute epileptic stupor I refer to is more a state of coma. It may occur in an already demented epileptic constituting a form of nervous disorder allied to if not a variety of status epilepticus. I shall refer to it again under that head.

Acute epileptic stupor, however, occurs sometimes in epileptics, previously quite sane, as the first warning of insanity. The patients lie completely exhausted and prostrated in a more or less typhoid like condition.

There is a condition of insensibility present from which the patient cannot be completely aroused. The limbs are not moved or only on reflex stimulation. The pulse is weak and often hardly perceptible. The respiration is shallow. The skin has a dusky hue. The patient passes his water and urine involuntarily. Now and then I have noticed these patients groan

deeply and disturb others who may be asleep in the same ward. Now every now and then at comparatively long intervals, generally of some hours, these patients arise from their mental torpor and shew a certain amount of mental activity. They become restless, sleepless, and hallucinated. The hallucinations being indicated by the patient following unseen objects with their eyes and turning their head also by clutching at imaginary objects. If the patient gets a little better he will try to get out of bed but not being able to co-ordinate his movements, he will fall and then roll about on the floor in his efforts to regain his feet.

If we examine the reflexes we generally find the knee jerk present but very sluggish and not elicited by one tap alone. Ankle clonus I have never been able to obtain. Plantar reflex is generally weak.

The temperature in these cases is often subnormal, but sometimes it is as high as 101oF.

The patient can sometimes swallow but often he cannot, and should then be fed by the pump, otherwise there is always the danger of his getting bronchopneumonia from inspiring some of the food.

The bowels are very constipated and enemata

should be used for this. This form of epileptic insanity is fortunately very rare. I have only seen one genuine case. As regards prognosis, that of mental recovery is absolutely bad the patient passing into a state of chronic dementia, and generally is confined to bed for the remainder of his life. As regards life these patients may hang on for a year or two in a more or less stupid condition.

I have been struck with the resemblance of these cases to sunstroke, except that the temperature is not so high.

The fits as a rule are not frequent and I have noticed that the worse the patient is the fewer are the fits, and if the patient shows a certain amount of mental activity the fits occur.

Here are notes from my case:-

J.P., aet 35; female; no occupation; admitted 3rd November, 1898.

She is stated to be epileptic but not suicidal nor dangerous. She had a fit after getting a fright at a fire when three years old. She has had fits ever since. There is no family history of nervous disease.

On her admission her physical condition was poor. No further bodily disease was elicited. The following are the notes of her condition since then.

November 5th. Patient is very stupid and has no attention, hence cannot answer nor understand questions. She has had several severe fits since admission, and has been very stupid after each one. She has remained quietly in bed not showing any mental or motor excitement. The first cardiac sound is impure at the apex.

November 7th. She is utterly incoherent and unable to answer questions or speak intelligently. She is wet and dirty in her habits and person.

November 15th. Remains very stuporose and dull. Pulse and heart very weak. On citrate of caffein V grains thrice daily. Four ounces of whisky given daily.

November 17th. Has had several fits since last note. Heart sounds are stronger but are reduplicated at apex.

December 8th. Has been much better lately. Takes milk much better. Fits have ceased.

December 16th. Has been very restless lately. Is troublesome and keeps getting out of bed.

December 27th. Has been very restless and is more noisy and troublesome. Bodily condition somewhat better. Is having more fits.

January 17th. Still in bed and is at times noisy, always troublesome. Bodily health better. Remains wet and dirty and stupid.

I append some further notes on the treatment adopted.

November 12th, 1893. Put on ovarian tabloids (gr V) one daily.

November 13th. Increased to three daily. Had one fit at night.

November 14th. Had one fit.

November 15th. Taken off ovarian extract. Very weak. Pulse regular but feeble. Face cyanotic. Attempts to vomit. Injected with Atrophine Sulphate gr $\frac{1}{100}$. Had five fits during the day and three during the night.

November 16th. Put on Citrate of Caffein five grains thrice daily. Very stuporose and feeble. Toxaemia seems to be established. Given soap and water enema. Had 3 fits to-day. At night very much better. Had 2 fits during the night.

November 17th. Had 3 fits this morning, slept well last night. Citrate of Caffein doing well. Heart sounds at apex purer. Reduplicated second sound. Had 3 fits during the night.

November 18th. Had 3 fits during the day. Rather dull. One fit at night.

November 19th. Had 3 fits during the day and 2 at night.

November 20th. Had no fits during the day but 5 during night.

November 21st. Had 2 fits during the day and 2 at night.

November 22nd. Citrate of Caffein stopped. No fits in day; 3 at night.

November 23rd. Remains unimproved. 5 fits during the day, 6 at night.

November 24th. Is worse and duller. 2 fits during day, 5 at night.

November 25th. Worse still. Legs reported "stiffened" during the morning. Pupils contracted, react, equal. Crepitations in lungs. Indrawing of lower intercostal spaces. Pulse weak, 100 per minute. Reduplicated second sound. Twenty minims Acetic Ether injected. Better at 5 o'clock p.m. 2 fits during the day, five at night.

November 26th. Remains much better. 10 fits to-day.

November 27th. Temperature has gone up to 100oF. Otherwise as before. No fits.

November 28th. Tendency to syncope marked. Injected again with Ether 20 minims. Temp. 98oF. No fits.

November 29th. Better this morning. Eating better. Injected with 90 minims of Quinine solution (equal 9 grains Quinine Sulphate). Much better in the evening. Seems brighter. Drinks better. Pupils react better to-light. Temperature normal. No fits.

November 30th. Keeps better. Responds to stimuli more. 1½ gr. Quinine Sulphate injected. Pulse good 100. No fits. In the evening brighter. Face less livid. Not injected. Has had no fits since first injection. Pulse good. Regular.

December 1st. Shows wonderful improvement. Responds to name. Feels the injection more now. Pupils active. Pulse 98. No fits since Quinine started. 2 syringes full injected - 4 gr. Quinine. Is very constipated.
In the evening. Much better. Eats and drinks tremendously. Given this afternoon Castor Oil one ounce Croton Oil two minims. Not moved at 5 p.m. Injected with equal to 1½ gr. Quinine. Pulse rather feeble. No fits.

December 2nd. Remains much brighter. Nurse reports that she takes notice of what is going on around her. Swallows much better. Bowels have been well moved. In the evening - Keeps improved; mouth cleaner; skin clearer; eye brighter; pulse slow. No fits.

December 3rd. Not injected. Pulse weak. Is generally brighter. Takes food well. No fits.

December 4th. Injected (1½ gr. Quinine) Keeps much the same. Not so wet and dirty now. No fits.

December 5th. No fits.

December 6th. Not injected. Much the same. No fits.

December 7th. Still no fits or syncopal attacks. Rather more dusky about the face. Perhaps not so well to-day. Still eating well. No more injections. Syringe.

December 11th. Since last note has not been injected and is much worse. Much more restless and tries to throw herself out of bed. When she gets out of bed she falls. Does not eat so well, and does not look so well. Fits have not returned.

December 13th. Injected (4½ grs. Quinine Sulph.) Seemed better after. No fits.

December 14th. Much better last night. Restless this morning. Injected (1½ grs. Quinine sulph) At night - Has been less troublesome to-day. No Fits.

December 15th. Not injected and very restless all night. Had a fit at night.

December 16th. Injected (4½ gr. Quinine Sulph.) Much brighter in the evening.

December 17th. Not injected. Brighter.

December 18th. Not injected. Keeps brighter. Had a fit to-day.

December 19th. Injections stopped. She seems to have got as far as she will towards mental health. Had a fit at night.

It will be noticed that she always seemed better after being injected with Quinine. During the interval when she was not injected she was worse. It was found, however, that the Quinine if pushed seemed to be too depressant, so it had to be stopped from time to time. Permanent good did not seem to be established. Probably in a less advanced case quinine would act better. It is, however, remarkable how the fits disappeared while quinine was being used.

The Relation of the Fits to the Mental Processes.

Taking 137 typical cases I obtained results as follows:- Firstly, that 23 or about 14% were unaffected by fits at all. Secondly, that 16 or about 11% exhibited phenomena before fits alone, 12 being excited and 4 being stupid. After the fits they became their everyday selves. Thirdly, that 32 patients or about 23% show phenomena both before and after fits, 20 being excited before and after fits, 7 being stupid before and after fits, some alternated, 3 being excited before and stupid after fits, and 2 were stupid before and excited after.

Fourthly, some 66 patients, or about 48%, show mental change after fits only. If these 38 were stupid after fits and 28 were excited after fits.

Summing up the whole situation we find that mental change before fits occurred 48 times, 35 being excited and 13 stupid. Mental change following fits occurred 96 times, 48 being excited and 48 stupid. Thus taking

all the cases showing mental change either before or after fits or both we see, that the mental change generally takes the form of excitement (this occurring 83 times to 61 of stupor); that the pre-epileptic change is generally one of excitement when it exists; that the post epileptic condition may equally be one of stupor or excitement; stupor as a rule occurring when there is no mental change before the fits as well and excitement when there is such change.

The Diagnosis of Epileptic Insanity.

In several other forms of insanity the occurrence of fits takes place. Thus we meet with fits in patients suffering from general paralysis, alcoholic insanity, in some cases of melancholia, and in some cases of brain tumour and other gross brain disease.

It is only in those cases of epileptic insanity in which the fits are comparatively infrequent that we have any difficulty in diagnosis. I have no doubt that if my hypothesis is correct that the examination of the blood will be the surest diagnostic sign. I have not found similar changes in the blood in other forms of insanity (see the section on the blood).

From general paralysis there may be some difficulty in diagnosing epileptic insanity if the fits occur early in the former. However, in epileptic insanity we do not meet with the motor tremblings so frequently noted in general paralysis. The size and reaction of the pupil is nothing to go by, as many epileptics show changes in size and reaction of the pupil exactly similar to general paralysis. The pathogneumonic speech of the general paralytic may be of great help, but it is in those cases of general paralysis where most of the prominent symptoms of general paralysis are absent that there is the most difficulty. The delusions afford little help because the epileptic may have delusions of grandeur and the general paralytic may be melancholic. The general history of the case from the beginning is the best guide. The occurrence of fits for years before the insanity, and an hereditary history point to epilepsy, the occurrence of the insanity before the fits to general paralysis.

The effect of the fits on the patient is an important point, the general paralytic being as a rule far more affected by the fits and becomes rather worse mentally after the fits. The occurrence of a state of excitement after the fits seems to me to be one

of the chief points in favour of a case being epileptic.

In alcoholic insanity we have the history of drinking, but of course this may exist in epileptics too, and there is a distinct form of epilepsy associated with drinking as a cause. The fits in alcoholic insanity are very infrequent and as a rule the case is a history of insanity existing for years before fits. If the case be one of mania or melancholia the great improvement that takes place in alcoholics under asylum treatment is of great diagnostic value in cases of doubt.

It is not, however, in cases of the insanity of major epilepsy that the most mistakes in diagnosis are made but in cases of insanity of minor epilepsy. In these cases we are apt to mistake the syncopal attacks of heart disease and angina pectoris for petit mal. I have seen this happen often. The cases become more involved when we remember the frequency of heart disease and cardiac pain in epileptics, and also when we remember that Trousseau considered epilepsy to be one of the causes of angina pectoris.

The occurrence of a distinct aura of a physical nature points to epilepsy. I do not include noises in the ear or flashes of light before the eyes, or

sensations of numbness, giddiness, or feelings of impending death and such like phenomena, but aurae which partake more of the nature of a delusion or hallucination, such as the sight of a distinct form or the hearing of distinct words.

If the examination of the candiovascular system yields no results, such as the presence of cardiac enlargement or murmur of arterial sclerosis, the case is probably one of epilepsy but the reverse does not hold good.

Careful watching and examination of the case from time to time will generally clear up the diagnosis. The examination of the blood I would especially advocate as some of my best specimens are taken from cases of epilepsy minor.

Treatment.

In treating epileptic insanity we have to remember firstly to treat the epilepsy itself and secondly to treat the phenomena associated with the insanity. Let us then consider the treatment of the epilepsy.

Now in treating a case of epilepsy it is well to remember that in this disease the ailimentary tract is apt to be deranged and even if this is not the

case it is well known that some of the most potent remedies upset the digestive system and the absorbing powers of that system. Hence we should always seek to put the alimentary system in as good a state as possible before proceeding to combat the epilepsy itself. The condition of constipation should be overcome at the outset. Constipation not only seems to aggravate the condition of epilepsy by increasing the number of fits, but it will in some cases even induce the condition of status epilepticus. The relief afforded by enemata and purgatives when a patient is having an excessive number of fits is sufficient proof of this. I have no doubt that there is an antointoxication resulting from the absorption of some substance from the alimentary tract in epilepsy. I find the well known mixture of nitric acid and quassia an excellent one. This with the use of saline laxatives is as a rule quite sufficient to regulate the patient's digestive functions. Having done this we may either go on at once to treat the epilepsy itself or if the patient be rather more amaemic than usual we may give a course of iron or arsenic first. In the anaemia accompanying epilepsy I have found potassium permanganate given as a two

grain pill followed by a large potation of water the first thing in the morning to be of use. Whether this acts as a haematinic directly or merely as an intestinal antiseptic I am unable to say. In some cases it certainly causes a diminution in the number of fits.

The main treatment, however, consists in steadying the patient's nervous system. Here undoubtedly the bromides are the best drugs to use. And I am old-fashioned enough to prefer potassium bromide before all others. Now in chronic epilepsy associated with insanity, where there is little hope of getting rid of the fits altogether, moderate doses of potassium bromide are all that is required. I find that I never need to exceed the dose of twenty grains thrice daily in cases which will be benefited by potassium bromide. Cases requiring more than this are best treated by means of a combination of potassium bromide and chloral, fifteen grains of the one and fifteen minims of the other thrice daily. Potassium bromide is with advantage combined with many other drugs. Thus in cases where we have a weak circulation we can combine potassium bromide with digitalis. This is a very useful combination.

to employ and it often seems to do better than bromide alone in cases where the heart is unaffected. Ten to fifteen grains of bromide with from five to ten minims of tincture of digitalis seem to be quite sufficient.

The use of bromide together with belladonna is to be advised in some chronic and run-down epileptics who need something more of a stimulating than of a depressing nature. Belladonna itself is known to be useful in epilepsy but I like to combine it with bromide in doses of fifteen minims of the tincture to fifteen grains of bromide.

Now many cases of epilepsy suffer much from anaemia and here we can administer a haematinic with the bromide. Now the best one is arsenic. I generally start with ten grain doses of bromide and two minim doses of Fowler's solution thrice daily, and gradually work up to full doses of arsenic. The bromide I generally keep at fifteen grain doses. Epileptics seem to bear arsenic well.

I am not an advocate of the continuous use of bromides. I find that the cases which need this are very few. Some cases, which one learns by experience, undoubtedly need bromides continually for if the

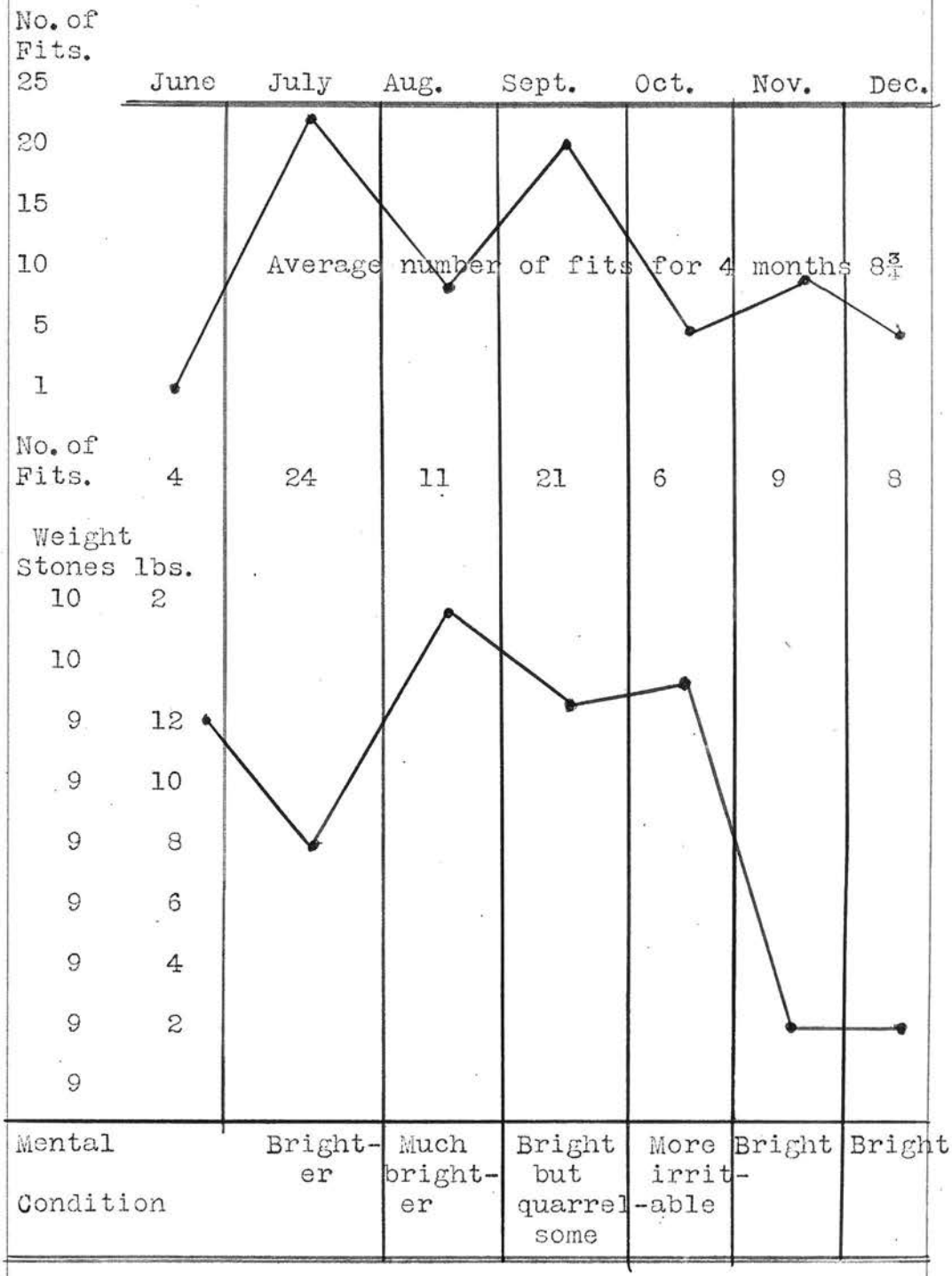
bromide is stopped the patient becomes worse bodily and mentally and may even pass into a condition of status epilepticus.

In most cases, however, the patient may be made much less dull and more cheerful by stopping the bromides for several months. Perhaps it is best to stop them gradually but I find that no harm results in stopping the bromide suddenly in such cases. I had to do this in cases which had become as it were supersaturated with bromide.

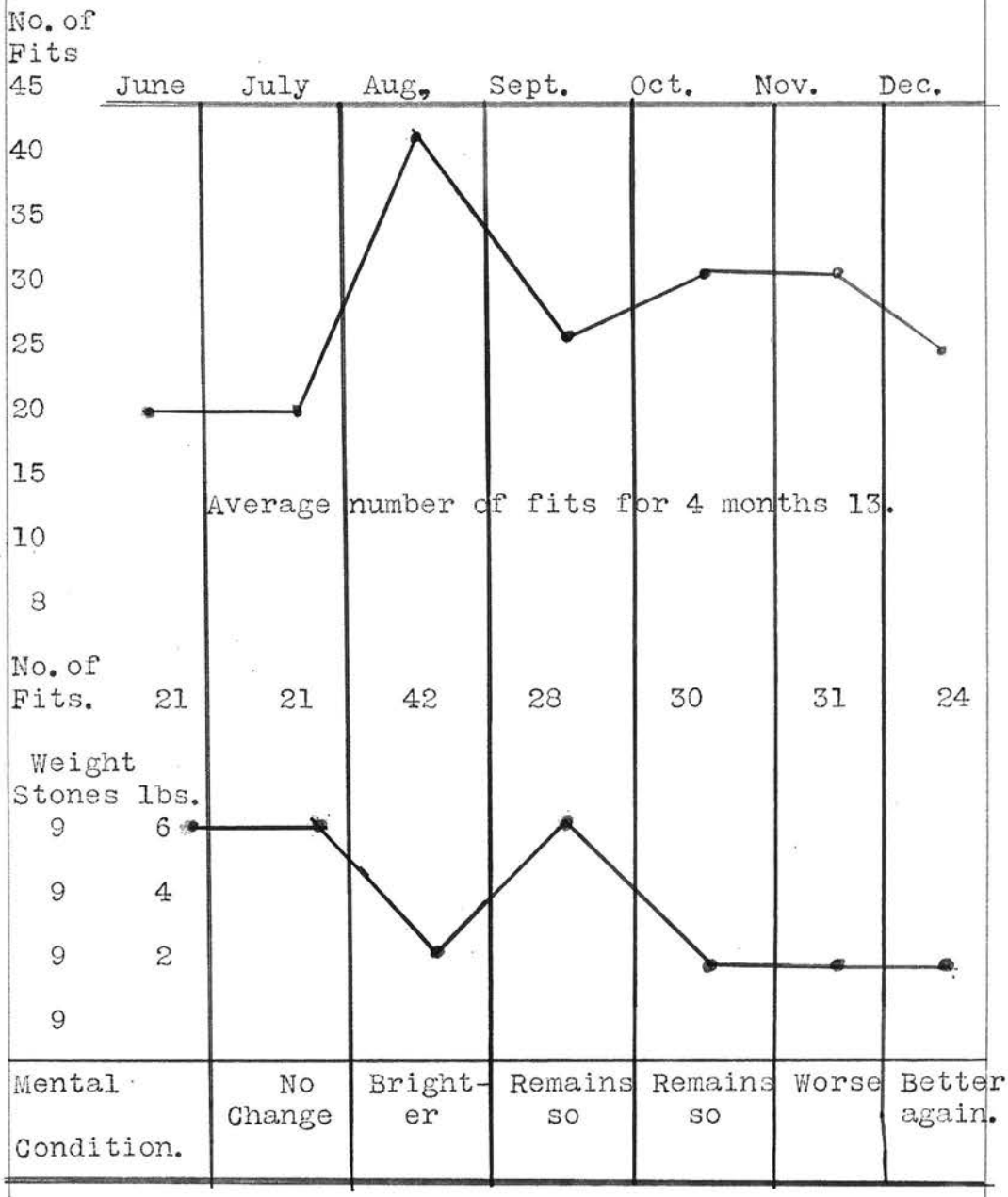
When we stop the bromides there are certain changes take place in the patients mental and physical health and in the number of fits. Thus there is always a rise in the number of fits.

With this as a rule there is a fall in body weight (this may rise again). After several months there is a certain level reached both in the number of fits and in body weight, and this remains more or less constant. The mental condition brightens. It is this brightening of the mental condition we should aim at. I have drawn out tables of five cases for reference.

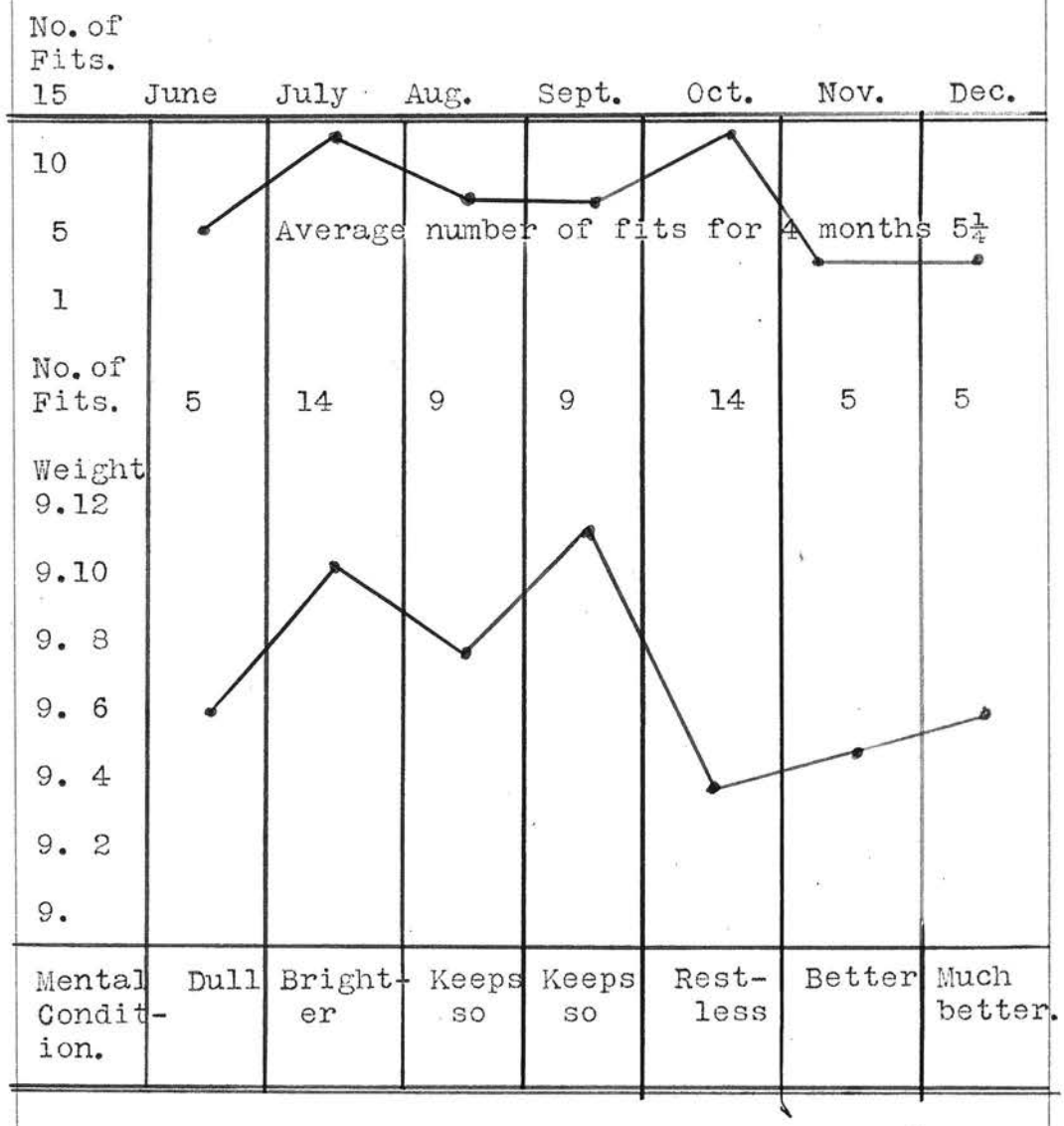
In each case the treatment by bromide was stopped in June. The dose of bromide was twenty grains thrice daily and had been used continuously for many months.



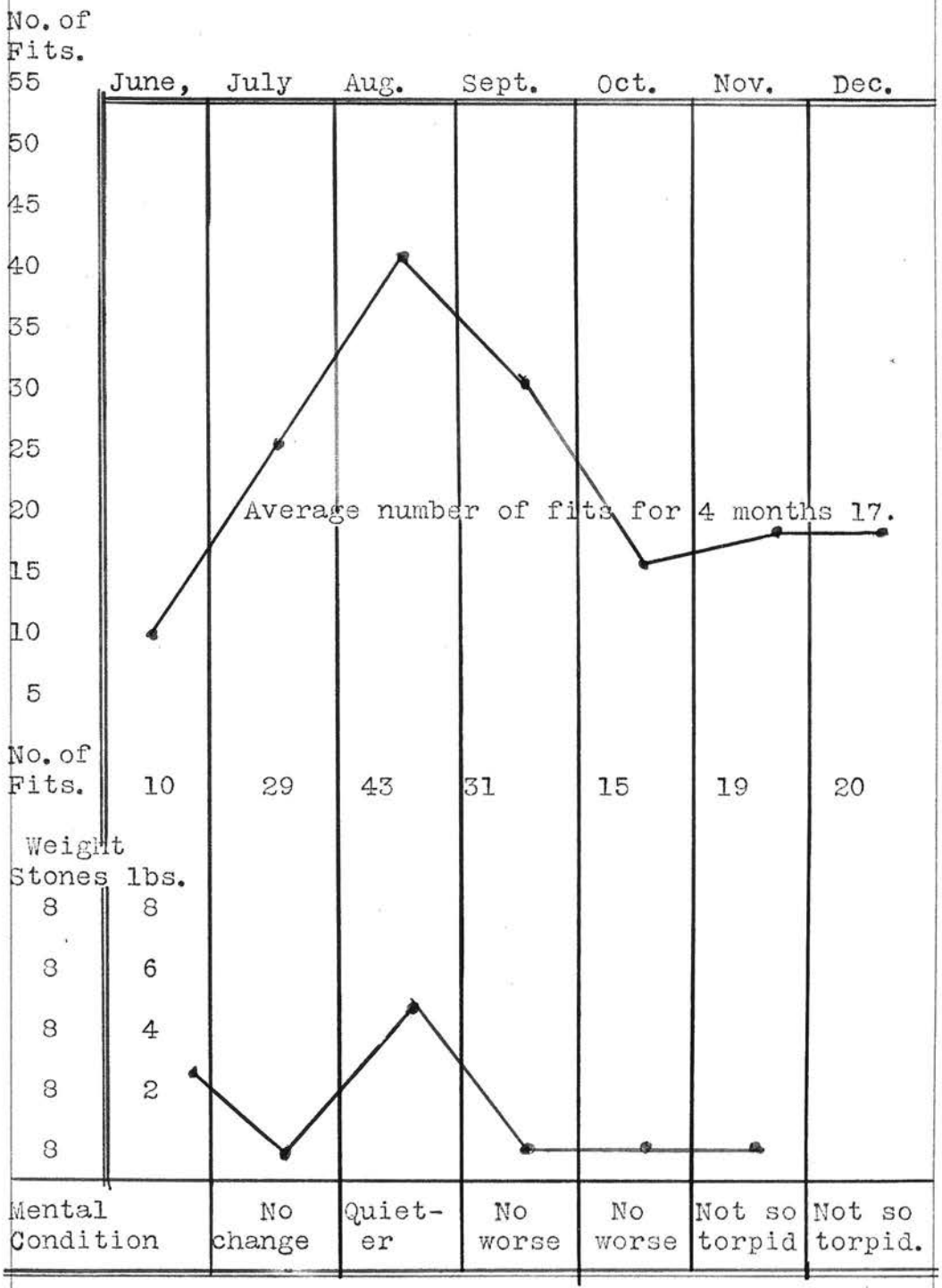
Case of L.R. an epileptic dement aged twenty-nine years. On stopping the bromide the fits increased in number. Six months afterwards the average monthly number of fits was about 12 as against 8¾ formerly.



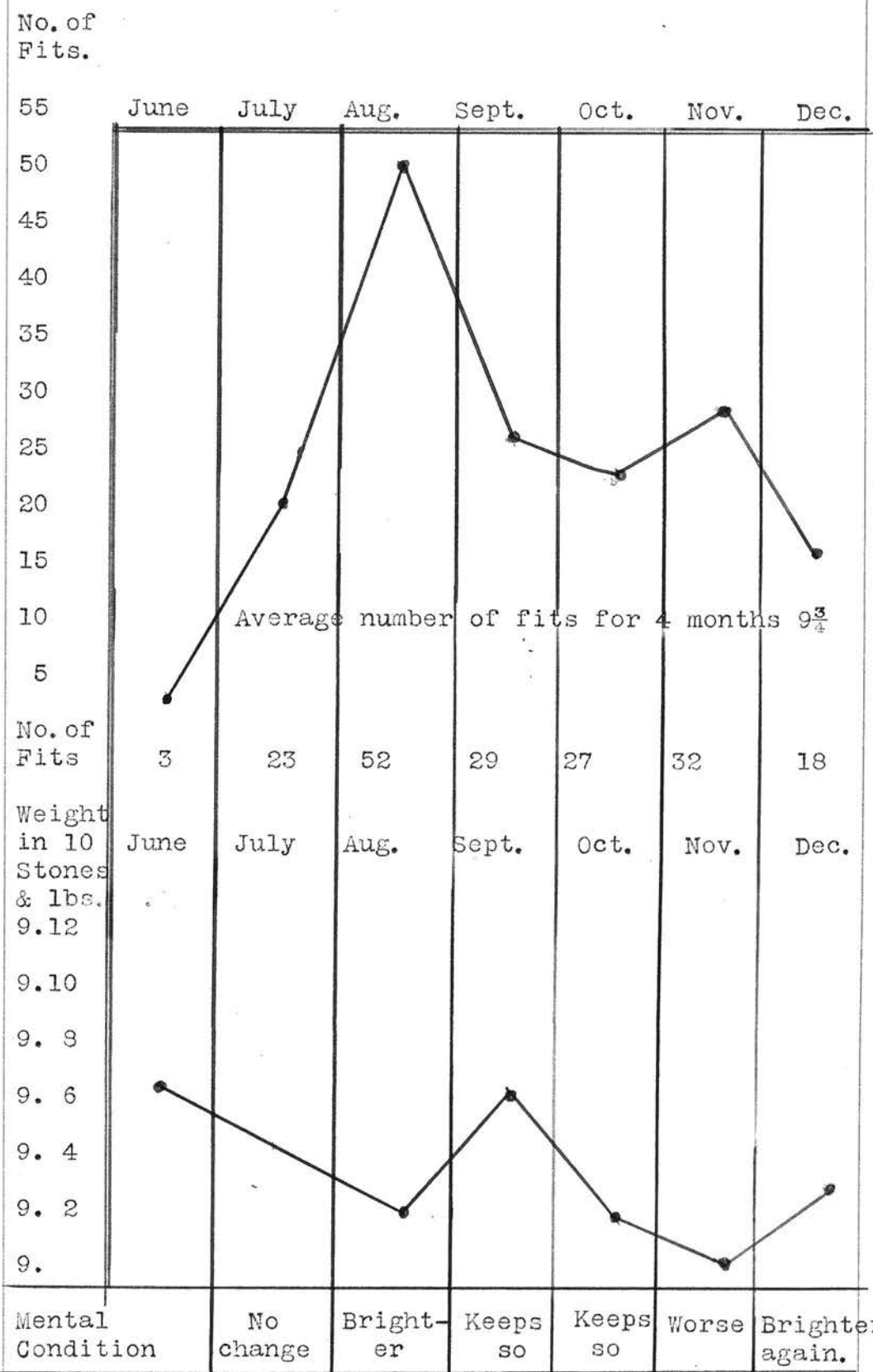
Case of M. B, an epileptic dement, aged twenty-two years. Six months after stopping the bromide, the average monthly number of fits had arisen from 13 to 29 1/3.



Case of E.B, an epileptic dement aged thirty-two years. Six months after stopping the bromide the average monthly number of fits had arisen from 5¼ to 9 1/3.



Case of C.D, an epileptic dement aged thirty-nine years. Six months after stopping the bromide the average monthly number of fits had risen from 17 to $26\frac{1}{6}$.



Case of M.C. an epileptic dement. Six months after stopping the bromide the average monthly number of fits had risen from 9¾ to 30 1/6.

In treating the phenomena associated with the mental condition, there are three groups into which we may divide epileptics.

1. The maniacal.
2. The melancholic or hypochondriacal.
3. The stuporose.

First let me consider the maniacal epileptic. Chronic epileptic maniacs are best treated by means of potassium bromide and chloral at the time of their outbursts. Sulphonal, trional, and hyoscine I do not find nearly so potent. When an epileptic passes suddenly into a condition of mania you can best treat it by a good dose (30 or 40 grains) of chloral hydrate by the mouth. You can repeat this in four or five hours and in bad cases sooner. I firmly believe in secluding these cases in single rooms and keeping a nurse at the door to watch them. The darkness and quiet seem to quieten them. Sometimes, and now more and more frequently, I give paraldehyde at the outset of the mania, starting with a dose of two drachms or two and a half drachms to which another drachm may be added in an hour if the patient be no better. Paraldehyde in two drachm doses may be given to a patient who has already had a dose of chloral hydrate one hour afterwards.

I find that by thus using paraldehyde or chloral

at the outset I can get time to start the bromides which always require some time to act.

Hyoscine by the mouth and subcutaneously I do not find of much value, even in fairly large doses, in checking an epileptic maniacal outburst. Given by the mouth in small doses for some weeks ($\frac{1}{100}$ or $\frac{1}{150}$ grain thrice daily) it is more valuable and in some cases seem to act well.

Treatment of the Melancholia and Allied Conditions.

Though some cases of epileptic melancholia yield more or less to the use of bromide and in some agitated cases to bromide and chloral, there are many cases where this treatment seems to be too depressant. I find that the use of belladonna is attended by much good in some of these cases. Atrophine itself may be tried in small doses. 2 to 4 minims of the Liquor Atrophinae Sulphatis freely diluted with water thrice a day is a good form of prescription. In cases where neither bromide nor atrophine seem to do good I have found benefit accrue from the use of Opium, atrophine and chloral.

I prescribe it thus.

Chloral Hydratis vi
Tinct. Opii. vi
Liq. Atropin. Sulphi. m xlviii
Aquamad vi

Of this I give from i to ii twice or thrice daily according to the amount of mental disturbance.

Now many cases of Epileptic Melancholia suffer from hypochondriasis and these cases show a good deal of hysteria as a rule. In such cases the use of tincture of Valerian in drachm doses with about twenty grains of potassium bromide twice or in bad cases thrice daily, is very useful.

We now come to the consideration of the cases of stuporose epilepsy. Now in these cases the stupor, if it is due principally to the fits, may give way to the use of the bromides. In other cases it does not, however, and in these the use of quinine sulphate in 5 grain doses thrice daily sometimes seems to clear the patient's mind. I have also in these cases used Ovarian extract with benefit. I shall now relate such a case.

L. M., a female epileptic dement who shows a great deal of dulness and stupor.

She was admitted in May 1898 and was nine stone in weight. She had no fits in May.

In June she weighed 10 stone 8 lbs. and still had no fits.

In July she weighed 10 stone 5 lbs. and had one fit.

In August she had ten fits and weighed ten stone.

In September she had one fit and weighed 10stone 3 lbs.

In October she had six fits and weighed 9 stone 10 lbs.

On November 2nd. she was given Ovarian Tabloids (5 grains of the extract once daily). Up till then she had been very dull and stupid and had no treatment. After that the following notes were made on her condition.

November 5th. Excited and sleepless.

November 10th. Much better. Headache which she complained of ever since admission has gone.

November 11th. Tabloids increased two daily.

November 14th. Keeps well: much better.

November 16th. Is so well that she wants to go home. Her pulse is, however, very weak and of low tension though regular.

November 24th. Has been taken off tabloids as they are depressing her.

At the end of November she weighed 9 stone 1 lb. She had no fits in November at all, but had then lost 9 lbs.

In December there was a reaction and at the end of December she weighed 9 stone 13 lbs. and was much better.

The Status Epilepticus.

By this I mean a condition epileptic patients are liable to characterised by frequently recurring motor convulsions with loss of consciousness, or by loss of consciousness alone, generally accompanied by a rise in temperature. In the interval between the fits the patient is either unconscious or only semiconscious. If complete consciousness exist between the fits the name of status epilepticus is not so applicable, and many people call death resulting therefrom death from epileptic exhaustion. In the latter condition there is as a rule no rise of temperature and there is also generally complete consciousness between the fits. Both of these conditions partake of the same nature, however. I have said above "or of loss of consciousness alone" and I hope to show that we can have a status of petit mal as well as of grand mal.

In my tables of statistics you will find a group of cases of status epilepticus ending in death and some cases of epileptic exhaustion.

Excepting two cases of melancholia the mental condition was one either of dementia or imbecility. The less acute the mental phenomena the greater the tendency to status is, I believe, the rule.

Another point worth noticing is that these cases

have as a rule, a stronger heredity of disease than the average epileptic.

Now in these cases of status epilepticus you will notice that it is not the epileptics who have frequent fits who suffer most but those who, for some time at any rate, have had few fits.

I shall now give you six examples, taken at random, to show this. You will notice that there is generally a period of a month or so immediately preceding the status, during which time there are no fits at all. Another point to notice is that the condition of status may come on abruptly and suddenly or be preceded by a few days on which fits are more or less frequent.

The first case shows a latent period of 46 days preceding status.

L. T., died 21st October, 1895.

During October there were no fits till the 21st. During September one fit on the 4th. During August and July no fits.

The next case shows a latent period of at least 27 days.

E. T., died 28th May, 1896.

On the 28th of May she had 42 fits. During the preceding part of May she had no fits. She was not here before that date.

The next case shows no distinct latent period for any length of time but during the month preceding status only 13 fits.

H. D., died 7th June, 1896.

On June 1st. she had 8 fits.

On June 2nd she had 12 fits.

On June 3rd she had 13 fits.

On June 4th she had 8 fits.

There were no more fits till status set in on the 7th. During May she had 13 fits thus, on the 12th two fits, on the 22nd one fit, on the 30th three fits, on the 31st seven fits.

You will notice that the period of latency is wanting here but also that the fits, which generally followed each other at intervals of days, became more frequent so that from the 31st of May up to the 4th of June, the fits occurred daily.

The next case shows a latent period of 22 days.

E. W., died 31st January, 1897.

In January she had no fits till the 23rd when she had one fit. On the 26th she had two fits; on the 27th two fits; on the 30th four fits. On the 31st she had one fit before she passed into status and died.

The next case shows a latent period of practically two months.

E. C., died 2nd June, 1897.

During May she had no fits till the 29th when she had two; on the 30th she had four; on the 31st she had 29 fits. In April she had no fits. In March she had one fit on the 30th. In February she had two fits. In January she had no fits.

The next case shows a period of five months without fits.

A. G., died May 2nd 1898.

On that day she had 136 fits. For five months previously she had no fits. The month before that (November) she had one fit on the second day of the month.

From all this we may argue that a period during which the fits are quiescent must always be regarded suspiciously and precautions must be taken against status. Constipation especially should be guarded against and the patient should be put on suitable diet and perchance medicine.

As we must look from all points of view I will now give an exceptional case in which there is no latent period.

M. C., died June 17th, 1898.

Starting with January of the same year she had 13 fits. During February she had eleven fits; during March 9 fits; during April 5 fits; during May 16 fits; then in June she had no fits on the first; on the second one fit; on the third no fits; on the fourth one fit; on the fifth two fits; on the sixth one fit; on the seventh and eighth no fits; on the ninth three fits; on the tenth three fits; on the eleventh 2 fits; on the twelfth two fits; on the fifteenth four fits; on the sixteenth twenty four fits; and on the seventeenth 28 fits.

Here though there is no distinct latent period the fits became more frequent during the half of the month immediately preceding status and in that time there were more fits than the patient usually had in a whole month, and even more fits than the average number for the preceding five months. The mental symptoms preceding status show a certain amount of conformity in most of the cases. In the statistical tables I append I have noted the mental condition of the various patients briefly and it will be found that generally before status the patient has been quiet and

harmless for a longer or shorter time. He is often dull in mind and needs more attention than usual. In those tables notice that the hereditary tendency to neurotic affections is much greater than in the ordinary epileptic. Every case in which the friends gave any information there was some hereditary tendency. In six out of the seventeen cases, however, no friends gave any information.

None of the cases were recurrent cases showing that the tendency to get well mentally is quite absent. From the age at which their epilepsy started no conclusions can be drawn as these are most varied.

I will now give a sketch of the condition after which I will consider the condition more in detail and discuss its treatment and rough pathology.

What then is this status epilepticus? Let us take a typical case. You are called to a patient who has had several fits following one another, say about a dozen, or it may be you are called earlier. At this early stage you may be able to tell whether the patient is really going to have status or not. If you examine the patient you may observe that there is nothing very noticeable about her except that she may be less bright after the fits than usual. This

in itself will not help you much as the fits may be "very strong" according to the nurse's account. You examine the patient's pulse and you will probably find that it is quicker than usual after fits say 120 to 140 beats per minute. This is a sign you should not lay much stress on as I have often seen patients who have had several fits and in whom I have suspected status with a pulse like this, but these cases did not invariably end in status. Yet this quickening of the pulse is the first sign to arrest your attention. The next sign I would draw your attention to is a slight, or it may be a considerable rise in temperature. The greater the rise the more sure status. At this stage you will often only have a temperature of 99oF, but the same night it will probably rise to 102o or 104oF.

The next symptom is the occurrence of sweating in large amount. This is also an important sign. If the patient be sweating and have a high temperature and a quick pulse, the probability is she will have status. The next sign, but not of much frequency, is vomiting and retching. If this is frequent it is also important. With such symptoms present you must treat the case at this early stage as you may abort the attack.

Let us follow out the case. Perhaps for one day the patient does not alter much, but at the end of that time you will mark that the stage of consciousness is getting less and less, and the number of fits increasing in a given space of time - that is to say the interval between the fits is decreased. The patient now becomes unconscious in the interval or only halfconscious. When the latter is the case the patient can take, and will, milk or other fluids the nurse gives her. But in most cases the power of swallowing, even reflexly, is disturbed and I take this as a very important sign and one we must attend to very carefully. I believe this arises from a central change in the deglutition centre in the medulla. This loss of the power of swallowing is often seen when the patient is quite conscious between the fits and then the nurse tries to get her to swallow and finds out that she cannot. You will notice at this stage that the sweating is getting more profuse and this is not a bad sign in itself. I rather fear those cases in which this does not take place. You will find that the pulse is now very rapid and of very low tension, what might be called a "running pulse". The respiration also is increased. The one from 160 to 200 per minute, the other 30 to

50 in bad cases. The patient now is completely unconscious and those reflexes which were obtained before are not obtained now. These are the contraction of the pupil to the light, the conjunctional reflex and deglutition. Also the organic reflexes may become impaired and the patient may pass water and foeces though this is not constant. The face has often a dusky hue and the heart sounds are feeble. The respirations become stertorous and may assume the cheyne-stokes type. The temperature will probably keep up and hyperpyrixia is the rule.

This state may last for one or two days increasing or lessening in intensity. I have noticed also that now the fits become weaker, they may not be general as they were before. They may be only indicated by a twinging of the eyelids, a drawing of the head to one side, or a monospasm of the arm or leg muscles. Still they occur frequently and may alternate with somewhat stronger and more general convulsions. The patient is now in a state of utter collapse and is not going to recover. The condition of coma deepens and the pulse becomes quicker and feebler till death takes place. The temperature goes up to 107oF, say, and the patient dies in a condition of hyperpyrexia.

Or the patient may have fewer fits and look as if she were going to recover and the temperature may go down to 100oF or even 99oF and then suddenly the heart gives up the struggle and the patient dies from exhaustion.

There is also another termination which is that of bronchopneumonia. This a serious affair and often arises from feeding the patient by the mouth when she is not able to swallow, being in fact a deglutition pneumonia. I have noticed this over and over again.

Now there are several varieties of Status Epilepticus.

1. The ordinary form with many fits, high temperature, and coma.
11. Where there is no hyperpyrexia.
111. Where there are few fits but deep coma.
1V. Where there are no fits observable at all, but coma and hyperpyrexia.
V. Where pneumonia or bronchopneumonia or other complications arise early.

1.

Here is an example of the ordinary form.

A. G., aged 72, suddenly passed into status on May 2nd, 1898 after having had no fits for months. Her temperature went up to over 105oF. She had 136 fits altogether that day and night. She was treated by a

drachm of chloral per rectum in an ounce of whisky, after a soap and water enema.

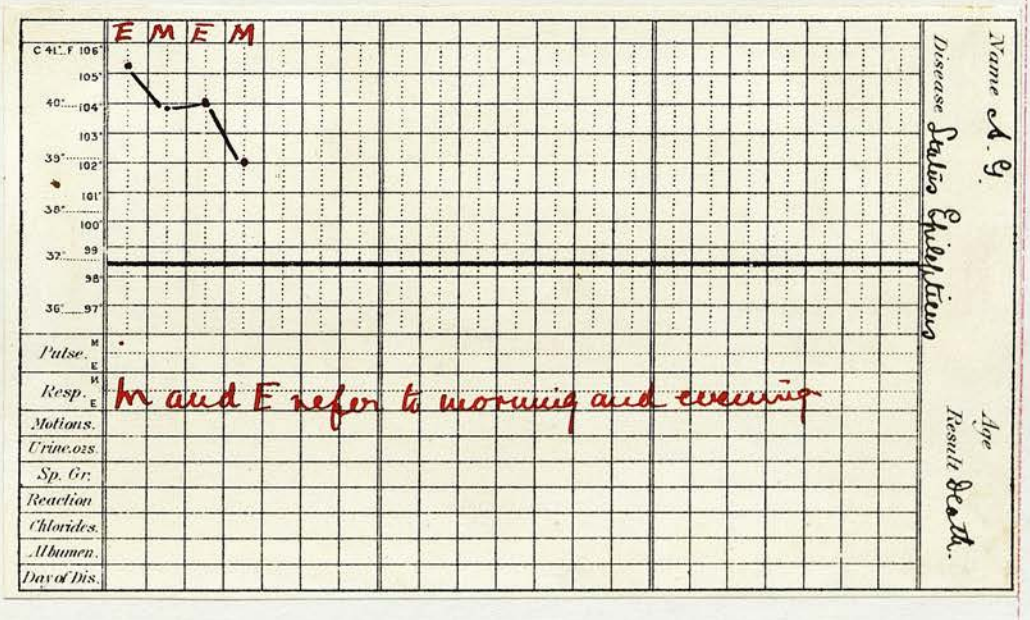
In an hour she had 5 minims of Liquor Atropinae Sulphatis injected. The fits stopped.

On the 3rd her temperature was about 104oF; she was unconscious and very weak. She had brandy and beef tea per rectum. There were no more fits.

On the 4th she was worse; tenperature 103oF. No more fits. She died that day.

Here the fits completely exhausted the patient who was an old woman of 72.

I give a temperature chart.



Temperature chart of a case of ordinary Status Epilepticus.

11. Where there is no hyperpyrexia. These cases partake more of the nature of very frequently recurring fits than of true status. There is not often deep coma. Cases of death from Epileptic Exhaustion belong to this group I believe.

I append a case.

Case of M. E. S., aet 17. Imbecile.

Has fits frequently as a rule (3 to 8 daily).

November 8th. Had 36 fits.

November 9th. Had 55 fits notwithstanding 80 grs. Potassium Bromide. Also 15 grs. Chloral per rectum after soap and water enema.

November 10th. 43 fits. Put on Ovarian Extract gr. V one daily.

November 11th. Had 53 fits. No bromide. Ovarian Extract given. Sleeping peacefully at visit. Hydrarg. subchlor. gr. 2½.

November 12th. Had 33 fits. Pulv. Glych. Co. two ovarian tabloids: 5 grs. Caffein Citras.

November 13th. Had 21 fits. At 5.30 trional gr. XV.

November 14th. 35 fits. Better last night, not so well this morning. Taken off ovarian tabloids.

November 15th. 48 fits. Not so well. Headache, listless. Given i dose spiritus Thymolis (R. Thymol. gr. viii. Spirit Vini Rectif. m 80. Aguamad ii). Blood taken.

November 16th. Had 47 fits. Given i Thymol mixture and whisky ii

November 17th. 150 fits. i spt. Thymol. No better. Very bad to-day, also given 40 gr. Pt. Bromide. Temp. for first time 100o to-night.

November 18th. Fitting continuously. Given chloral Hyd. gr. 22½ Tinct. Opii m 22½ Liq. Atrop. Sulph. m 3. At night much better since having medicine. Fewer fits. Slept and found sleeping at round. Medicine repeated.

November 19th. Fitting continuously. Given chloral gr. xxx. Liq. Atrophine Sulphate. m 4 Tinct. Opii. m xxx.

November 20th. Semicomatose all day. Fitting continuously. Temp. 99.80oF. Pulse 128. Respiration regular, 25 perminute. Swallowing badly.

November 21st. Somewhat better, though fitting all night. Pulse 120 regular, fair tension. Respiration regular; given nothing. Had 88 fits.

November 21st. Night much better, pulse 84.

November 22nd. 66 fits, given Tincture of Aconite m. 8.

November 23rd. Better. Bowels well moved by Liquorice Powder. Morphia ¼ gr. Atiopia Sulph. gr. 1/150 injected. 42 fits during the night.

November 24th. Slept well, otherwise no better. Had 31 fits at night.

November 25th. Had 24 fits during night. Given no medicine.

November 26th. & 27th. No fits. Brighter.

November 28th. 82 fits. No better.

November 29th. Last night vomited much. Temp. normal this morning. Pulse weak 90. Fits getting weaker. Had 76 fits.

November 30th. Much the same. Pulse 120. Had 59 fits.

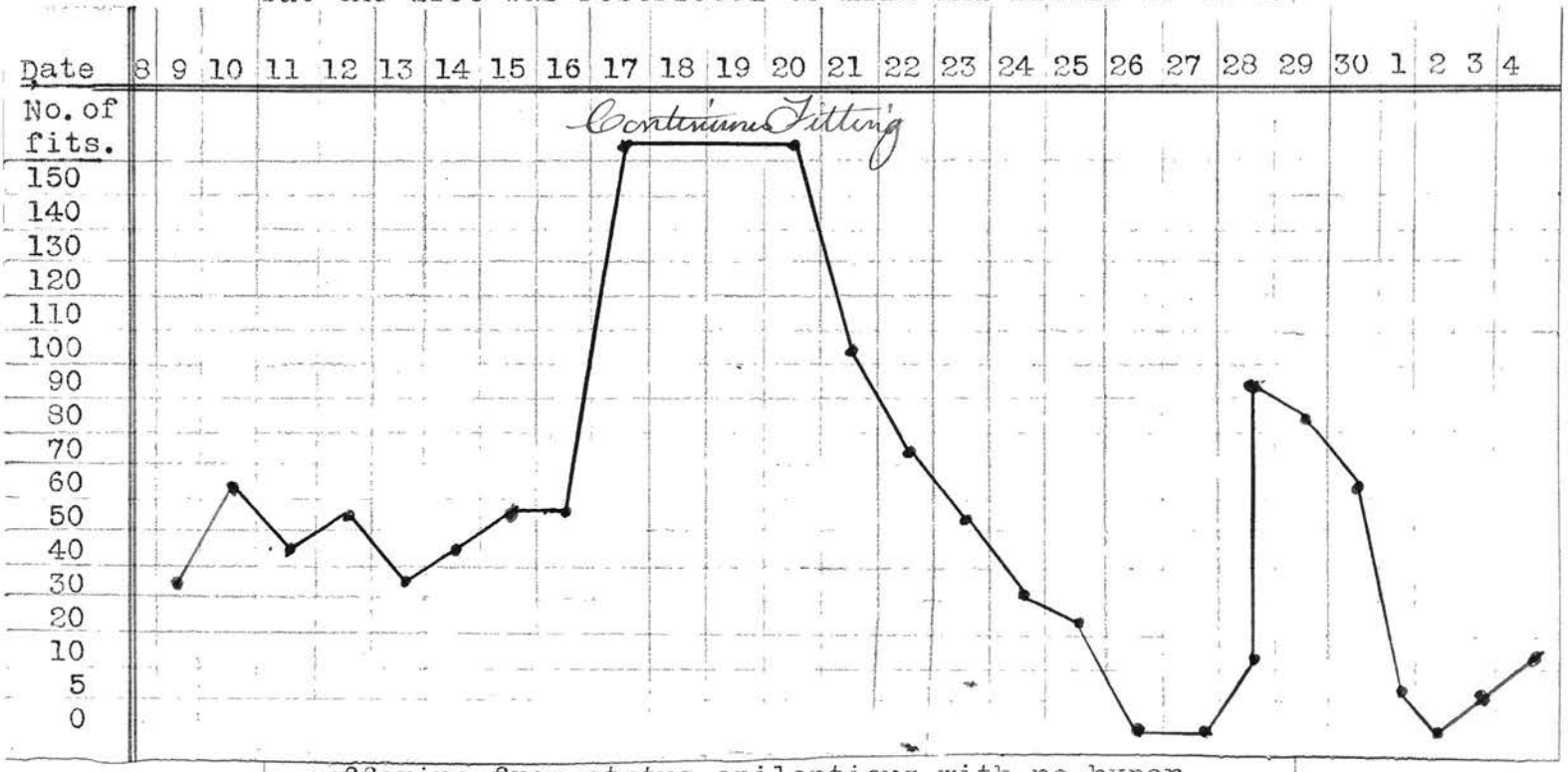
December 1st. Much better, pulse 80. 5 fits at night. Fits are increasing in strength.

December 2nd. 4 Fits.

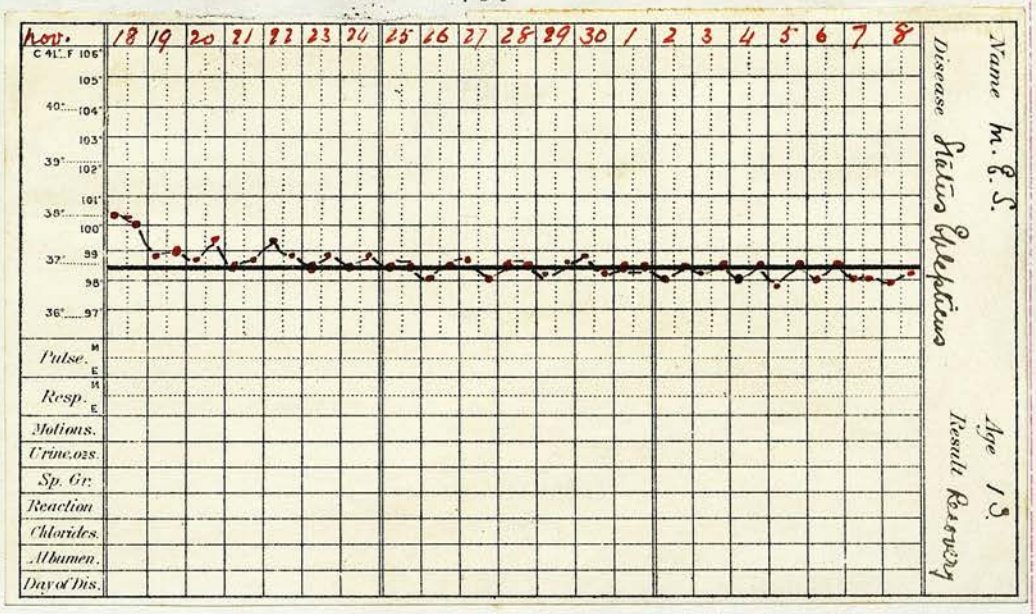
December 3rd. 5 Fits.

December 4th. Is practically in her normal condition.
8 fits.

Remarks. I append a temperature chart and a scheme of the fits. Notice that the temperature rose to 100.2°F. on the 10th November, when the patient passed into a condition of continuous fitting but gradually fell to normal in 4 or 5 days. Notice the relapse on the 28th Nov. and again a gradual fall in the number of fits. It is interesting to notice the vomiting at that time followed by gradual recovery. A point I would draw attention to is the absolute failure of the bromides. The nurse said,"Dr. I think the medicine makes her worse." The patient was receiving brandy freely all the time, but the diet was restricted to milk and little of that.



suffering from status epilepticus with no hyper-pyrexia.



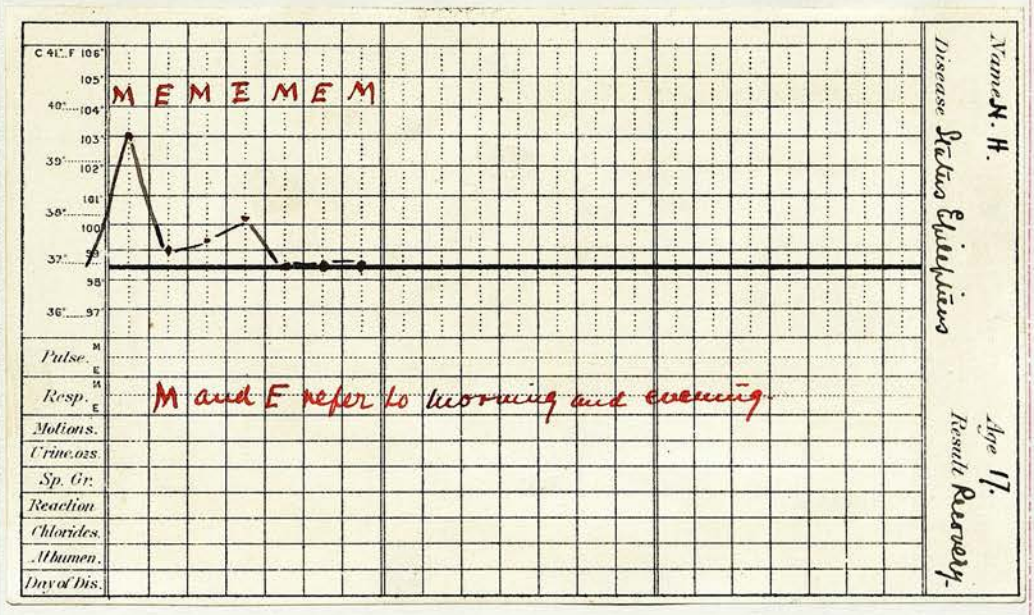
Temperature chart of M. E. S. suffering from status epilepticus with no hyperpyrexia.

III. Where there are few fits but deep coma and high temperature.

H. H., aet 17, imbecile.

On the evening of April 20th she had ten fits with a temperature of 103oF. She became unconscious. She was given chloral hydrate and potassium bromide 30 grains of each per rectum. She had no more fits. Next morning the temperature was 99.4oF; she was semi-conscious, and had two fits that day. Her evening temperature was 100.2oF. Chloral enema gr. xv given. Next morning the temperature was normal and she had no more fits. She got up in two days after that.

I give her temperature chart.



Case of status epilepticus in an imbecile where there were few fits but deep coma and high temperature.

IV. Where there are no fits observable at all but coma and hyperpyrexia. These are uncommom cases. In sone you may notice a twinging of the facial muscles every now and then like what is seen in some cases of petit mal. The patient gets more or less conscious at times and moves when you shout into his ear. After a little he relapses into a state of unconsciousness again. The temperature all the time remains up. I believe that these are cases of petit mal status. The post mortem results show changes in the brain and lungs similar to what is seen in ordinary status epilepticus. I have seen two cases and both ended in death.

I now quote a case.

E. E. S., female; aged 37; cotton winder. Admitted 18th December, 1897.

There was no history obtainable. The certificate of admission stated that she looked depressed, talked irrelevant nonsense, and appeared to speak with difficulty. She was reported suicidal but not dangerous. On admission she was noted as demented. She soon passed into a state of mania and became restless, excited, and noisy. Would not remain in bed at night without a draught. She also had now and then to be put in a single room, as draughts failed to quieten her. Her mind was a blank to all external objects. She had no attention.

The following notes were made afterwards.

December 22nd. Was called to see patient this morning. She was conscious, breathing quietly, heart weak but pulse regular. Ordered brandy ℥ii per rectum. After an hour I saw her again. She was still unconscious. There was some fluid food in her mouth. She had a cough but this was too weak to expel the phlegm. There were rales and rhonchi over the right lung at the base. Patient's breathing was noisy and there were extra accompaniments in the nose which hindered the examination of the chest. Pain on pressure over

the left inguinal region. Pulsation in abdomen. Temperature 101oF. Patient has been very constipated. Twenty minims ether were injected. Coffee enema administered but was not retained. Brandy one ounce per hour per rectum ordered.

December 23rd. She was conscious but other wise much the same.

December 25th. She died to-day at 1.40 p.m. Towards the end her breathing got very shallow and she was much cyanosed. Her temperature rose to 106oF. She had slight cough to the end. There were rales in the chest

I append her temperature chart.

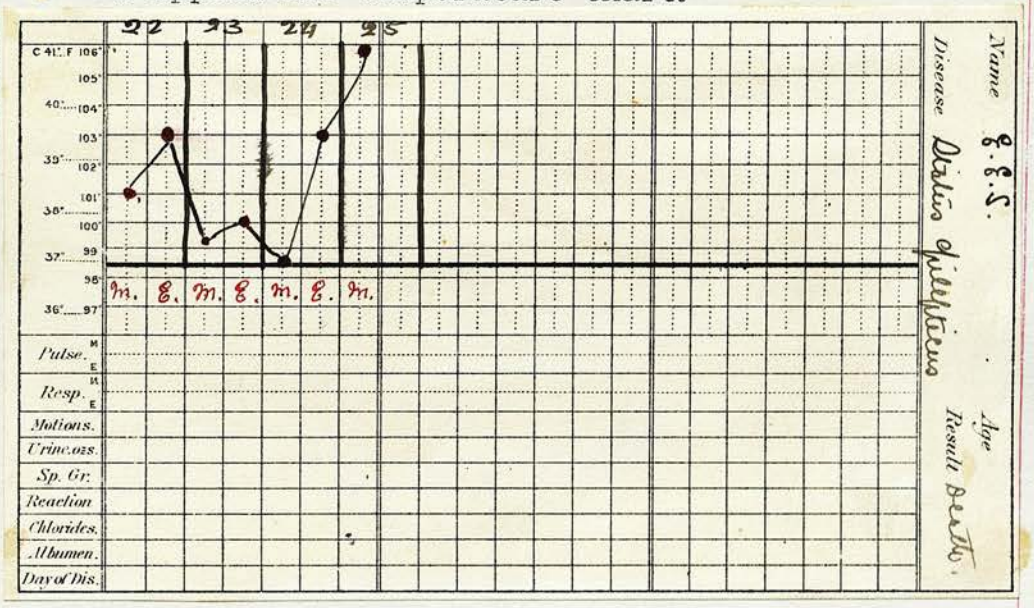


Chart of E. E. S. an epileptic who was suffering from status epilepticus in which no fits were observable.

The post mortem showed much congestion of the membranes and cortex of the brain. The condition was exactly similar to what is seen in death from statusepilepticus. There were some catarrhal patches in the lungs.

Now in this case the patient had no fits at all. On the other hand the catarrhal pneumonia was not sufficient to cause death. I believe the catarrhal pneumonia resulted from some of tne food getting into the lungs owing to the loss of the power of swallowing. Hence I believe this to have been a case of death due to epilepsy, and of the nature of status epilepticus of petit mal.

V. Where pneumonia of bronchopneumonia arises early. Here the temperature rises higher by a degree or two, and the respirations become very stentorous. The fits become less in number after the onset of the pulmonary mischief as a rule. There seems to be absoluely no hope for these cases.

I now will give you an example.

D.W., aged 46, housewife, admitted November 21st,1897. The family history shows that one brother died from consumption. The personal history shows that she developed fits at the age of forty. The cause is said to be overwork.

She is reported suicidal and dangerous, and the certificate of admission states that she is incoherent, abuses herself and her brother and her husband, and she is taciturn and morose.

On admission she was maniacal, very excited and violent. It took six nurses to hold her. One two-hundredth of a grain hyoscine hydrobromate was injected with little or no benefit. She was restless all night and banged the door of her cubicle. Next day refused food and was twice fed with the pump. Afterwards she took her tea. She then became quiet.

November 24th. Is maniacal, excited, deluded, has required padded room and feeding with stomach pump since admission. At the present moment is dull and confused. Her bodily condition is good.

November 29th. Is now much quieter. Is deluded and talks in a strange manner.

December 6th. Is quiet and well-behaved and asks continually to go home.

1898. January 6th. Had to be secluded to-day. Very violent and excited.

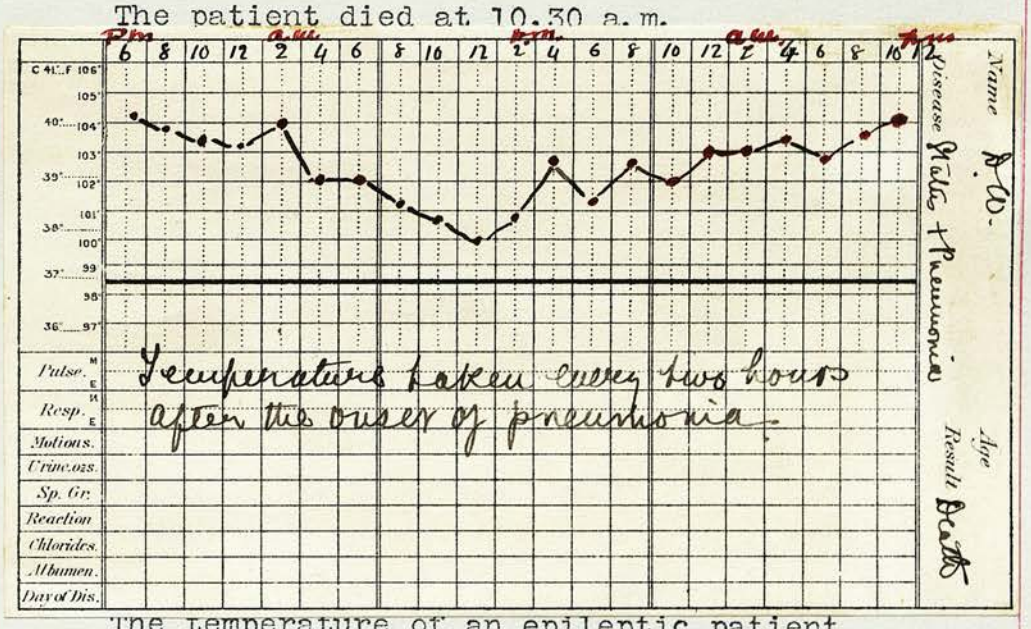
January 12th. Fed with pump, restless and excitable.

January 14th. In bed in hospital exhausted from epileptic fits.

January 15th. Last night seemed much worse. Temp. 104.6oF. Harsh breathing over lungs. Left base shows bronchial breathing over a patch of impaired resonance. There are some crepitations also. Patient is very much exhausted. There is marked dyspnoea. Skin very dry.

January 16th. Was called to see the patient at 6.15 a.m. and found her more or less comatose. Right pupil contracted, left normal. Breathing over front of chest is harsh but there are no accompaniments. Back not examined. There was marked dyspnoea. Heart was beating 108 to minute . No murmur heard. Later both pupils were contracted, hands and arms were twitching during the night, and also this morning.

The patient died at 10.30 a.m.



The temperature of an epileptic patient who developed pneumonia early when in a condition of status.

In order that you may understand my interpretation of status epilepticus I must digress a little here. In the nervous system when diseased we often notice first an excess of function followed by a diminution or paralysis of function, leading to irritative phenomena and paralytic phenomena. Notice the tinglings and pain and spasm of muscles which precede the loss of sensation and paralysis in neuritis. The same thing may be noticed when aneurysm of the aorta causes pressure symptoms resulting in dilatation and then contraction of the pupil from pressure on the sympathetic. The same thing is exemplified when previous to a hemiplegia following a "stroke" there arise convulsions.

I believe that there is a good reason to suppose this same phenomena happens in epilepsy. Notice the preliminary irritation of the highest centres resulting in the various aurae followed by paralysis of function-loss of consciousness. Next notice the irratation of the motor areas resulting in first spasm (tonic convulsions) then clonic convulsions, then loss of power (motor paralysis) during the coma. The same thing occurs in the medulla. Here the irritation causes the crowing inspiration followed by the tetanus of the diaphragm and standstill inspiration. Then follows a more or less considerable pause. The heart is

likewise first slowed from irritation of the vagus centre then it beats rapidly from weakening of this centre. Taking a typical fit as an example we thus get three stages.

1. Excitement. 2. Paresis or loss of function.

3. Regenesis or resumption of function.

Each function of the brain is thus affected but the stages are not necessarily simultaneous in each. We may make a table out as follows.

1. Stages of excitement and Paresis.

1. Psychical Excitement - Aurae.
2. Loss of consciousness.
3. Vocal irritation - cry.
4. Motor irritation - tonic muscular spasm.
5. Motor excitement - clonic muscular spasm.
6. Complete coma, total loss of sensation, motion, consciousness, and reflexes.

2. Stage of Regenesis.

The order of return I believe to be something like this.

1. Organic and other reflexes.
2. Consciousness and sensation, partly.
3. Motion partly.
4. Consciousness and sensation completely.
5. Motion completely.
6. Full consciousness, speech and other combined mental and motor functions.

Now after a fit, or more commonly after many fits as in status epilepticus, any of these functions may be disturbed for a longer or shorter time.

Fullest consciousness may be the last and slowest to return, hence we get trance - like conditions in which the patient acts automatically, and also mania from incomplete return of consciousness. Then we notice that speech is a long time in returning perfectly in many cases and we get a drunk like speech. Motor power returns slower than seusory power. You can notice this when nurses or attendants get a patient up very shorly after a fit. You notice that the patient may not be able to walk but has to be dragged or pushed along. Even for a good time after a fit co-ordination amy be impaired and the patient may not be able to co-ordinate his limbs, cannot stand or walk steadily. Now sensation and motion may be affected for a long time after a fit or number of fits and we get post-epileptic paralysis, tinglings, numbness, and other nervous phenomena. In the condition of status epilepticus the consciousness may return between the fits, but the motor power may not, and the patient lies like a log. Both may return, however, partially or completely; and the gravity of the case is to be judged by this. On the other hand neither may return (and never do so towards the end of a fatal case) in the interval. When neither return there is a condition of complete coma.

It is very difficult to come to a correct conclusion as to how all these phenomena are brought about but perhaps I may be allowed to exercise my imagination in theorising. All cells when working expend a certain amount of energy. This expenditure of energy in the cell is always balanced by an accumulation of energy by the inherent property of the cell protoplasm. All cells have a certain amount of residual energy, which, under normal conditions, remains constant, being kept so be the balance of the incoming and outgoing energy.

Let us call the power by which this residual energy of the cell is kept up the energising power of the cell. In order to energise the cell must have a certain quality and quantity of food. Sudden withdrawal of food, want of food or bad food causes weakened energising power on the part of the cell. This means eventually loss of function.

Now during the presence of a bad food supply, cells which have to work draw upon their store of residual energy and this keeps them going for some time but only for some time, and if the food supply does not improve the cell loses its function until it can accumulate sufficient energy to act again.

When a tissue or cell is in action it energises more rapidly. Now I want you to believe as I do that an attempt to energise more rapidly - that is to accumlate residue energy more rapidly - throws a tissue or cell into action.

Now a cell, or number of cells, when, owing to food supply being sufficient in quality or quantity, it has to draw upon its residual energy, attempts to energise more rapidly. This attempt to energise more rapidly throws the cell into action. It overdoes it. This is like the dying flicker of a candle. Unlike the candle, however, the cell can still accumulate energy, and if this takes place gradually and slowly the cell may regain its function. During the time the cell is regaining its function the cell is passively inactive. Let us apply all this to epilepsy. Taking the highest cells, those which subserve consciousness first we see that the effect of improper food causes weakened power and an attempt to energise more rapidly, resulting in the cell being thrown violently and suddenly into action. This causes those curious phenomena we call aurae, flashes of light and other special sense phenomena. Then follows the loss of function - unconsciousness. Not only

unconsciousness, however, but also a loss of control over the motor cell results.

Now the motor cell in epileptics is from heredity or otherwise a very unstable cell. The same process has been going on in it and it attempts to energise its more rapidly as it is losing power. This again results in the cell being thrown violently and suddenly into action - the convulsion. Then follows the loss of function, paralysis.

Thus we can explain the fits. Now if the food improve ot the cell gradually accumulates energy again consciousness and motor power return and the patient is all right till the next fit. If the cell cannot energise properly or if the food continues bad the fits recur and we get the condition of status established.

The period during which the cells are energising after a fit when they are otherwise inactive, may if prolonged give rise to the following according to the function of the cells specially affected, prolonged coma, anaesthesia, paralysis, and many other phenomena. Partial return of function may inthe same way give rise to tinglings and peculiar sensations, contractions, and inco-ordination. Now as I shall explain later

I believe that some derangement of the blood, which is the source of food for all cells, is the cause of many (I do not say all) cases of idiopathic epilepsy.

It is interesting to notice how, during status, the centres in the medulla are affected. Take the symptoms of vomiting. Now if the attack be connected with gastric irritation the vomiting may be due to this. In that case the vomiting is preliminary. But the vomiting and attempts at vomiting noticed so constantly during an attack I believe to be due either to the irritation of the sensory meningeal branch of the vagus by the congestion of the meninges, or to direct irritation of the vomiting centre in the medulla, perhaps to both factors. The progressive paralysis of the vagus centre gives rise to the difficulty in swallowing from paresis of the pharyngeal plexus. In this condition when the oesophagus becomes filled with food there is difficulty in breathing. There is also sonorous gurgling in the relaxed canal when the patient breathes.

The great quickening of the pulse is also connected with the vagal paresis. Afterwards from the same cause, we get the laboured deep and slow respir-

ation owing to which the lungs become congested because of the low pressure in the lungs during the time the thoraxis distended. This may go on to pulmonary oedema. These two phenomena are very frequently if not constantly seen post mortem. The advancing asphyxia may also account for the congestion of the lungs as the blood pressure falls in that condition.

The Gross Pathological Appearances.

On opening the skull of a person who has died from status epilepticus the first thing that strikes you is the tremendous excess of cerebro-spinal fluid over the normal. It runs out as soon as the dura mater is torn. This excess of fluid is also seen in the lateral ventricles which are generally distended with it. There is a condition of acute oedema present in the brain substance and in its membranes. Accompanying this there is a great congestion of all the vessels of the brain. Consequently on cutting into such a brain it is found to be very soft and easily disentegrated. On section many puncta vasculassa are seen both in the white and grey matter of the brain. These are most marked in the basal ganglia, pous, medulla and cerebellus. This congestion

cedema, and softening of the brain constitute the main changes met with post mortem.

The lungs also present a condition of congestion and oedema. This is very marked as a rule.

The other internal organs especially the spleen and kidneys are often congested and soft.

Treatment of Status Epilepticus.

The first indication is to try and treat the cause. As I believe that one of the main causes of this condition is a poisoning of the blood or at a least a weakening of its nutritive power arising from a deranged state of the system, we must try first if possible to relieve that deranged state of the system and secondly to improve the quality of the blood.

Now in all the cases I have seen there was a condition of constipation or at least an unnatural accumulation of foeces in the bowels. I believe that this condition is one of the predisposing causes. Whether the auto-intoxication from constipation is able of itself to cause the condition of status in an epileptic or whether as is possible there is as well a furtherance at that time of the real cause of epilepsy - be this in the blood, as I will try to show, or not - is doubtful; but there can be no doubt that

the relief following on the use of a large soap and water enema is most marked. I have seen cases nipped in the bud by means of this simple procedure followed by the administration of chloral by the rectum.

It is a curious thing what an important part the alimentary canal plays in the production of fits. Dyspepsia sometimes seems to act as a predisposing cause. I have seen status induced by the judicious overloading of the stomach with sweets and other food brought by the patient's friends. It is important to note that in many, if not all of the cases, the stomach is very much deranged and loses its power of absorption, and seemingly of motion for the food accumulates in it. This latter fact can easily be proved at the post mortem.

In these gastric cases a hypodermic injection of apomorphine ($\frac{1}{6}$ of a grain) often checks the attack and the patient falls into a refreshing sleep and is well next day. I am inclined to think that apomorphine has some peculiar action of its own in checking the occurrence of the fits, apart from its action causing vomiting, as I have seen cases improve in a remarkable way after its use.

A remedy I have sometimes found useful is quinine. I employ this as a hypodermic injection. It does not seem to lower the temperature invariably. Its real value is doubtful. Can it be that it acts as a sort of antiseptic? I use a solution of a drachm of the sulphate of quinine to a drachm of dilute sulphuric acid in ten drachms of distilled water. One hundred minims of this solution equals ten grains of quinine. This is a good dose to commence with. It is repeated once or twice daily afterwards.

We can improve the quality of the blood by means of saline solutions. I have not yet directly tranfused a patient in status but I believe it would be followed by good results. I have, however, resorted to large saline enemata or smaller ones frequently repeated, giving at the same time as a rule one or two autitoxine syringe-fulls of the same solution hypodermically.

I shall now quote a case to prove this. In this case the patient eventually died but the marked effect of the saline solution on the number of fits is worth noticing.

On Saturday March 4th 1899, I was called at 2.30 a.m. to see M.E.S., an epileptic imbecile aged 16,

who had had 50 fits. I found her unconscious and weak with a quick feeble pulse but a strongly acting heart and a tendency to vomit. She was sweating slightly. Her temperature was 103oF. Injected an autitoxine syringe-full of normal saline solution into the right buttock and ordered a soap and water enema to be followed in an hour by one pint of normal saline solution per rectum.

She had only four more fits that night.

After eight a.m., however, she began to fit again and when I saw her again at 10 a.m. She had had 54 more fits and her temperature was 101oF. I ordered her to have no food but another saline enema of a pint. She had a few fits more before this was given but at one o'clock when I visited her she had had no more fits since the injection.

At 3 p.m. she had another pint of saline solution per rectum. At 5.30 she was reported to have rejected the last injection and by that time had had 104 fits. Her temperature was 104oF. Pulse weak, 120 beats to minute. She seemed fairly strong and was conscious. Up till then had had nothing by the mouth but was then allowed a little milk and soda. At nine o'clock she was reported to have had no more

fits since 8 o'clock but was very weak and unconscious. Pulse 178. Respirations 20. Two syringefuls of normal saline solution were injected.

On the 6th at 10 a.m, she seemed weaker than ever; pulse 160; respirations 35; quite unable to swallow. She had two egg and salt enemas and three ounces of whisky per rectum that day.

At 5.30 p.m. her temperature was 104.4oF. She was ordered a cold sponge but this failed to lower the temperature.

At 11p.m. she was still having fits but only one or two an hour. Her pulse was 176, respirations 40. She seemed somewhat stronger.

On the 7th at 10 a.m. the fits were reported to be getting few in number, she had only 8 the previous night since 8 o'clock. She had had two since seven this morning. Temperature 102oF. Pulse 176, respirations 44. She was reported to have slept nearly all night. She was fed by the rectum with salt and eggs and brandy.

In the evening the temperature went up to 103.6oF and she died.

I generally give a rectal injection of a pint of tepid water containing from i to ii of common salt

half an hour after the enema of soap and water. This is repeated from time to time. The patient generally retains this and as a rule after the first administration has no more fits for an hour or two, or sometimes as long as 12 hours. The following injections seem to lose their effects. The pulse improves and the patient's face seems to clear wonderfully, the lips becoming less blue and the cheeks much better in colour. The eliminative processes are stimulated and there is free diuresis and sweating.

In trying to stop the number of fits, which is not the same thing as stopping the cause from acting, there is no drug of so much use as chloral. I always give it by the rectum, firstly because the patients often cannot swallow well, secondly, because it upsets the gastric functions, and thirdly, because it seems to act better that way. An injection of i is quite safe in a child of 15.

As pointed out by Dr. Bevan Lewis the administration of atrophine at the same time hypodermically lessens the risk of chloral. But I would go further and say that the atrophine aids the chloral in some way. It had been known for a long time that belladonna is a valuable remedy in epilepsy and I believe this is due

to its alkaloid atrophine. Chloral is best avoided after the first 24 hours of status as it tends to be dangerous when the pulmonary circulation is getting impeded. I have never seen it do any good after the first day, so that there is no need in giving it on the second. If the patient gets over the third day, however, it may be used at intervals of a day in doses of from fs to i.

The action of the chloral is not at all impeded by the use of brandy or whisky with it and I often give ii of one or the other along with it. In some cases this seems to be better than chloral alone.

The temperature does not seem to be influenced by cold sponging. In all the cases I have tried it remained up.

Cold sponging, however, aids the action of chloral as this drug acts much more strongly when cold coexists.

In the later stages of the process we must reject the use of depressant remedies altogether and here I believe in the subcutaneous use of atrophine sulphate which keeps the heart going and also, as I before stated, acts beneficially on the morbid processes. (Dose $\frac{1}{100}$ - $\frac{1}{60}$ gr.) I have also tried digitalin and strychnine in the ordinary hypodermic doses but have

not had much success.

At this stage we can use Paraldehyde or Amylene hydrate by the rectum. These seem to be of considerable value and do not depress the heart. Doses from ii to iv according to the frequency and strength of the fits. Trional and Sulphonal I find useless. Bromide of Potassium is worse than useless because in some cases it makes the patient much worse. I used it for some time and the nurse always told me it made the patient worse in her opinion. She volunteered this statement without being asked, and thus directed my attention to this fact.

As regards keeping up the patient's strength by feeding I believe in doing this solely by the rectum. The danger of feeding by the mouth is that owing to the difficulty in swallowing the patient may get food into the lungs and die from pneumonia. On account of the extreme irritability of the stomach and great tendency to vomit I do not use the stomach tube.

If you have a good nurse who can notice when the patient begins to swallow saliva as she is getting better, you may allow her to feed the patient carefully with a little milk by the mouth.

Pancreatised milk may be given by the rectum. Brandy and beef tea (or bovril) pancreatised is also very good. I believe in giving a good deal of brandy by the rectum varying with the strength of the patient. I find Hunter's method of giving the white of one egg with 15 grains of common salt a very good one for feeding the patient per rectum. I have fed a patient in this manner alone with brandy per rectum and a very little milk by the mouth for a fortnight, after which time she made a good recovery. I generally give three eggs daily. In the milder cases and especially in those due to gastric and intestinal derangement I do not believe in feeding the patient at all the first day. I always find these mild cases do best on this treatment.

During convalescence the patient is often troubled by attacks of vomiting. I find the best remedy for this in permanganate of potash in the form of a two grain pill followed by a copious drink of water every night and morning.

The Causes of Death.

The following table of Causes of Death extends over a period of about 5 years and includes 105 deaths.

The largest individual cause is Phthisis with 22 deaths. Status follows closely with 19 deaths. Exhaustion from Epilepsy has 9 deaths. Suffocation in fits 6 deaths. Thus out of 105 deaths only 34 were due more or less directly to the epilepsy, a little over 32%.

The post mortem notes from which I drew these statistics are incomplete but it is a significant fact that in the last 37 deaths (of which the notes are complete) there was heart disease or atheroma of the aorta (generally both) in 16 out of the 37 cases a little less than half of them that is.

D signifies Dement; I, Imbecile,
M, Male; F, Female.

Sex.	Age.	Mental Condition.	Cause of death.
M.	-	D.	Suffocation in fit in bed.
F.	17	I.	Status.
M.	48.	D.	Exhaustion from fits.
M.	17.	I.	Exhaustion from fits.
F.	41	I.	Cerebral Haemorrhage.
M.	19	D.	Status.
M.	38	I.	Status.
F.	51	D.	Status.
F.	48	D.	Status.
F.	33	D.	Phthisis.
F.	25	D.	Enteric Fever.
F.	17	I.	Asphyxia in fit.
M.	42	D.	Cerebral Haemorrhage.
F.	-	D.	Acute Purulent Meningitis.
M.	24	I.	Status.
F.	16	I.	Status.
M.	72	D.	Exhaustion from Epilepsy.
F.	37	D.	Status.
F.	42	D.	Colitis.
F.	54	D.	Cerebral tumours(Cysticercus)
M.	41	D.	Exhaustion from Epilepsy.
F.	55	D.	Chronic Bright.
M.	31	I.	Catarrhal Pneumonia.
M.	32	D.	Suffocation by bread in fit.
F.	33	D.	Ulcerative Colitis.
F.	36	D.	do. do.
M.	39	D.	Phthisis.
F.	73	D.	Cancer of Stomach.
F.	28	I.	Pneumonia.
M.	25	D.	Status.
M.	15	D.	Pneumonia.
F.	36	D.	Bronchiectasis.
F.	24	D.	Middle Ear Disease.
F.	38	D.	Phthisis.
F.	50	D.	Phthisis.
M.	44	D.	Pneumonia.
F.	29	D.	Status.
F.	79	D.	Exhaustion from Epilepsy.
F.	48	D.	Status.
M.	25	D.	Empyiema.
F.	23	D.	Phthisis.
M.	21	I.	Phthisis.
F.	43	D.	Phthisis.
F.	23	I.	Colitis.
F.	32	D.	Catarrhal Pneumonia.
F.	37	D.	Exhaustion from Epilepsy.

Sex.	Age.	Mental Condition.	Cause of Death.
F.	22	D.	Asphyxia in bed.
F.	47	D.	Heart Disease.
F.	22	D.	Phthisis.
F.	47	D.	Bronchopneumonia.
F.	62	D.	Pneumonia.
M.	34	D.	Bronchopneumonia.
F.	37	D.	Exhaustion from Epilepsy.
F.	22	D.	Phthisis.
F.	45	I.	Heart Disease.
F.	37	D.	Status.
M.	34	I.	do.
F.	17	I.	Phthisis.
F.	59	D.	Heart disease.
F.	18	I.	Phthisis.
M.	35	D.	Gangrene of Lung.
F.	35	D.	Status.
F.	24	D.	Phthisis.
F.	49	D.	Heart disease.
F.	52	D.	do. do.
F.	30	I.	Phthisis.
F.	22	I.	Enteric Fever.
M.	17	I.	Status.
F.	30	D.	Phthisis.
F.	39	I.	Status.
F.	21	I.	Phthisis.
M.	12	I.	Exhaustion from scalds.
F.	69	D.	Colitis.
F.	21	I.	Phthisis.
M.	56	D.	Chronic Bronchitis.
M.	45	D.	Exhaustion from Epilepsy.
M,	37	I.	Suppurative Arthritis.
M.	39	D.	Cellulitis.
M.	54	D.	Death in a fit.
M.	35	D.	Cerebral tumours.
M.	42	D.	Status.
M.	33	D.	Phthisis.
M.	37	D.	do.
F.	41	D.	Cellulitis.
M.	45	D.	Chronic Bronchitis.
F.	18	D.	Status.
F.	27	D.	Dysentery.
M.	27	D.	do.
M.	39	D.	do.
M.	34	Mania	Acute Mania.
F.	36	D.	Dysentery.
F.	67	D.	Phthisis.

Sex.	Age.	Mental Condition	Cause of Death.
F.	50	D.	Status.
F.	73	D.	Cirrhosis of Liver.
F.	55.	D.	Heart Disease.
F.	47	D.	Bronchopneumonia.
M.	24	I.	Status.
F.	47	D.	Pneumonia.
F.	37	D.	Phthisis.
M.	-	D.	Epilepsy?
F.	32	D.	Bronchitis.
M.	24	I.	Phthisis.
F.	36	D.	Pulmonary Gangrene.
M.	68	D.	Exhaustion from Epilepsy.

I now come to the statistical tables and the various results obtained from them.

Taking the ages of admission first.

These represent the ages at which the patients became unmanageable and had to be sent to the asylum. They do not represent the ages at which the patient became insane for this reason and al so because they were not all first admissions.

So we cannot learn very much from these ages. This is how the two hundred were distributed.

Ages.	Number admitted.
5 - 10	3
10 - 15	19
15 - 20	15
20 - 25	37
25 - 30	27
30 - 35	31
35 - 40	19
40 - 45	10
45 - 50	18
50 - 55	5
55 - 60	3
60 - 65	8
65 - 70	2
70 - 75	2
75 - 80	1
	200.

The first attack or not.

Of the two hundred cases 183 were attacked for the first time; 8 for the second time: 7 for the third time, and 2 for more than the third time.

From this we see that cases may not be entirely hopeless even though they have been insane once or twice before.

The Suicidal and Dangerous Propensities.

Fifty-three cases were suicidal; a little over 25%. One hundred and thirteen cases were dangerous; somewhat more than fifty per cent.

Thus in epileptic insanity both these tendencies are more marked than in other forms of insanity and the dangerous tendency is especially characteristic of epilepsy. The impulsiveness and lack of self-control of epileptics probably account for this latter fact, and also the great frequency of delusions of persecution.

The Mental Conditions were as follows.

Epileptic Dementia	91
Epileptic Imbecility or Idiocy	37
Epileptic Melancholia	31
Epileptic Mania	27
Epileptic Circular Insanity	6
Epileptic Delirium	4
Epileptic Stupor	2
Epileptic Monomania	2
	200.

Dementia is thus the most frequent form of epileptic insanity. It is interesting to note the closeness in numbers of Melancholia and Mania. The Circular form of epileptic insanity, the Delirious form, the acute Epileptic Stupor and the Monomania are shown to be comparatively rare forms. They are none the less interesting, however.

The bodily condition.

The bodily condition was fair in all except 18 cases, showing that as a rule the effect of epilepsy on the bodily condition and nutrition is not markedly bad.

The age at which Epilepsy started is found to be as follows:

Age.	Cases.
1 - 5	49
5 - 10	19
10 - 15	36
15 - 20	21
20 - 25	11
25 - 30	9
30 - 35	4
35 - 40	3
40 - 45	5
45 - 50	7
50 - 55	2
55 - 60	2
60 - 65	2
65 - 70	1
70 - 75	1
75 - 80	28
Unknown	200.

Thus the great majority of cases, more than half, occur before the age of twenty. Thus we find that epilepsy is specially a disease of early life or of early adult life. After the age of five most cases occur between the ages of ten and fifteen. Possibly the developing sexual functions have something to do with this. From the age of thirty to the menapause there is a fall. At that time there is a slight rise in the number of cases till the ages of fifty from which period cases become very infrequent.

The age at which Insanity started was ascertained in 199 cases out of the 200.

This is how the cases were proportioned.

Between the ages of	1 and 5	there were 30 cases.
	5 - 10	2
	10 - 15	12
	15 - 20	14
	20 - 25	28
	25 - 30	25
	30 - 35	24
	35 - 40	17
	40 - 45	10
	45 - 50	17
	50 - 55	4
	55 - 60	4
	60 - 65	7
	65 - 70	2
	70 - 75	3
	75 - 80	0
		199.

The first five years of life are thus found to present the most cases. There is a big drop during

The next five years. From the age of ten there is a gradual rise in the number of cases till the ages between 20 and 25 when the second largest number of cases, namely 28, is reached. From then till 45 there is a gradual fall, the number then being ten. From then to the age of 45 there is a slight rise to 17 cases. From the age of 45 there is a sudden fall to 2 cases. There is only one more slight rise and that is between the ages of 60 and 65.

Thus after the first five years, when the cases of insane epileptics are most numerous, we get a period of twenty years during which there is a gradually increasing number of epileptics becoming insane. Followed by another period of twenty years during which the tendency of the epileptic to become insane is gradually decreasing. After forty-five there is a period of five years during which there is a greater tendency for epileptics to become insane.

It is interesting to notice that this tendency to become insane at certain ages seem to increase with the gradually developing sexual activity (to 25) abd decreases from then to the menapause (45) after which there is a slight rise in numbers.

This is an interesting point to consider along with the fact that many female epileptics are much worse at the monthly period. It would seem to indicate that in many cases the sexual habit has an influence on the tendency to epilepsy.

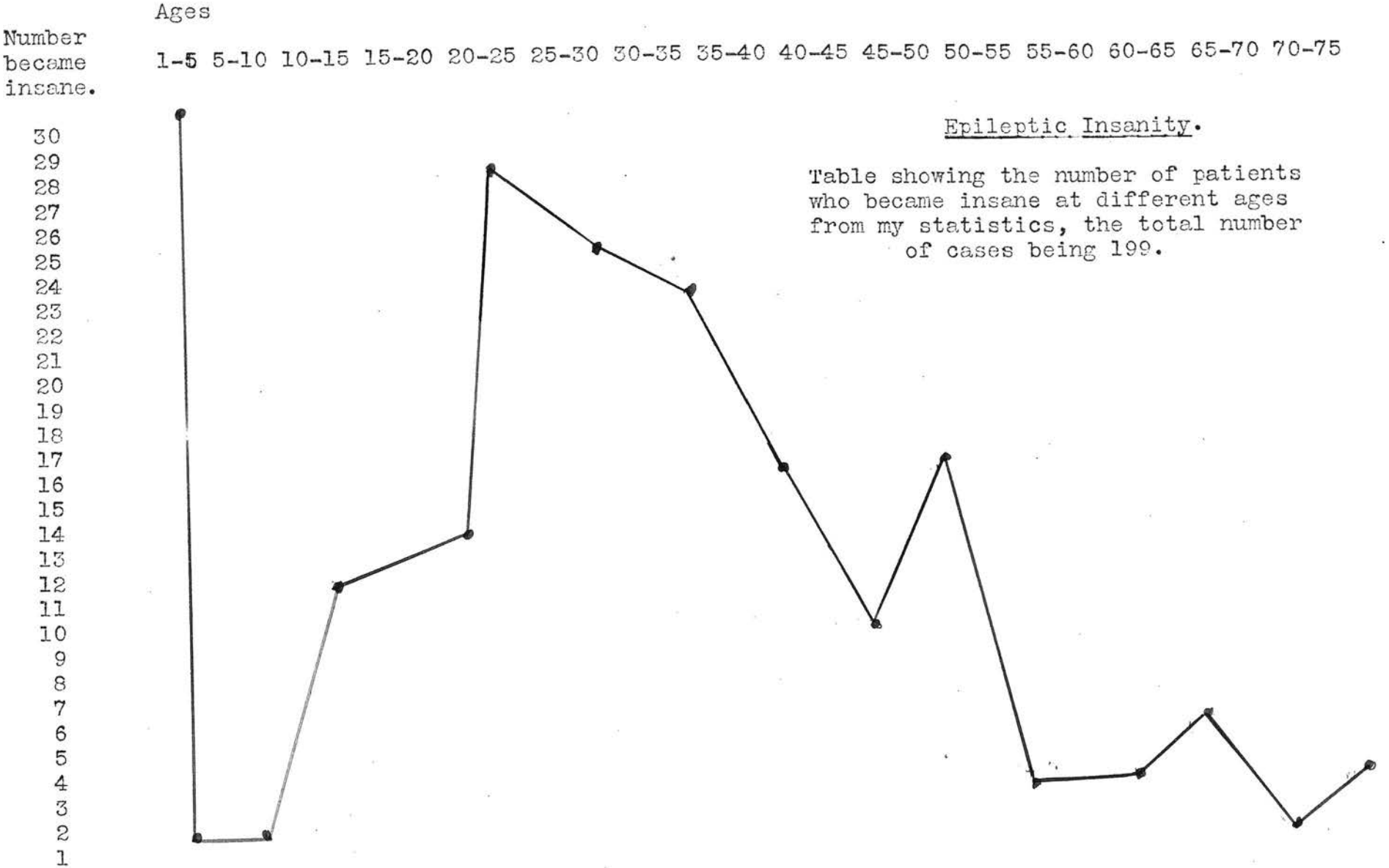
The difference in years between the onset of the insanity and the onset of epilepsy gives the period of sanity existant with epilepsy in each case.

I find that there are three main groups thus:-

The insanity as a rule comes on after the epilepsy. This is the first group. There is a second group in which the epilepsy followed the insanity, principally in imbeciles. There is a third group where the insanity and epilepsy occurred simultaneously or almost so. Now out of my 200 cases I was unable to ascertain the onset of the epilepsy in 29 and the onset of insanity in 1, making 30 cases in which I could not find out the sane period, if it existed. That left me 170 cases to divide into the three groups I have mentioned above.

The results were as I represent below.

In the group of cases where the insanity and the epilepsy started simultaneously 7 cases occurred at birth and 14 cases occurred ar other ages. Thus two occurred within the first five years of life; two at the age of twelve; two between 20 and 30; two between 30 and 40; three between 40 and 50; two between 50 and 60; and one at the age of 72.



Epileptic Insanity.

Table showing the number of patients who became insane at different ages from my statistics, the total number of cases being 199.

The fits came on after insanity in 22 cases; of these 19 were imbeciles and 3 were not.

In the cases of the 19 imbeciles the period without fits ranged from 9 months to 22 years. They were thus divided:- One had a latent period of 9 months; 4 had a latent period of 2 years; 5 had a latent period of 3 years; one had a latent period of 7 years; one had a latent period of 10 years; two had a latent period of 11 years; one had a latent period of 12 years; one had a latent period of 14 years; one had a latent period of 15 years; one had a latent period of 21 years; and one had a latent period of 22 years. Of the three who were not imbeciles, the epilepsy followed the insanity at periods of 3 months, 5 years, and 1 year respectively.

In the group of cases where the insanity followed the epilepsy there were 127 cases.

The average period between the onset of epilepsy and the onset of insanity was found to be just under 14 years. Thus it takes a considerable time, far more than we would expect, for the average epileptic to become insane.

The cases were found to occur as follows.

Difference in years between the onset of Epilepsy and Insanity.	Number of Cases.
1	7
2	7
3	5
4	8
5	9
6	6
7	3
8	4
9	5
10	8
11	3
12	5
13	1
14	8
15	3
16	3
17	1
18	3
19	3
20	2
21	2
22	2
23	4
24	2
25	5
26	1
27	2
28	1
29	3
30	1
31	1
34	1
35	2
36	1
37	1
38	1
41	1
44	1
46	1
60	1

Fancy a period of sixty years between the onset of epilepsy and that of insanity! Does this not tend to convince one of the fact that insanity may occur in Epileptics *per se* and not on account of the epilepsy?

Hereditary Factors in Epileptic Insanity.

The chief of these are shown to be Drink, Consumption, Nervous Disease, Insanity, and Epilepsy.

Now I have found a hereditary history of one or more of these in 93 cases out of the 200, but there were 22 cases of the 200 in which the hereditary history was not denied but was unknown. Thus we find 93 cases out of 178 give a hereditary history, that is about 52%.

In considering the hereditary history I have found it convenient to divide it into what I call Direct Heredity and Indirect heredity. By direct heredity I mean that of the parents or grandparents; by indirect heredity I mean any other heredity history. I have subdivided this into Lateral Heredity - that of uncles and aunts (and a few other maternal and paternal relatives except their parents) - and Contemporary Heredity - that of the insane person's contemporaries, sisters, brothers and cousins. Lateral heredity I sometimes call Indirect Heredity A and Contemporary Heredity Indirect Heredity B.

1. Drink.

There was a history of drink in 41 cases. 40 of these were direct and one indirect. Thus I found

the father was a drunkard in 24 cases, the mother in 9, both father and mother in 3, and the grandparent in 4 cases, the other case being inderect.

II. Consumption.

There was a consumptive history in 33 cases. Of these 18 were direct and 15 inderect.

The father was affected 9 times the mother 5 times, both parents twice, and the grandparent twice.

Of the 15 inderect cases, group A had 9 and group B had 6 cases.

Nervous Disease.

This as a rule took the form of"strokes" There was a total of 32 cases. There was a history of direct heredity in 25 cases.- Mother 9 times; father three times; and a grandparent 13 times. There was an inderect heredity in 7 cases, group A 3 cases and group B 4 cases.

Insanity.

There was a total hereditary history of 20.

Direct heredity had 6 cases, father twice , mother twice and grandparent twice. There was indirect heredity in 14 cases,group A having 11 and group B having 3 cases.

Epilepsy.

There was a history of hereditary epilepsy in twenty cases. 4 of these were direct; the mother once, the father twice, and the grandparent once. Indirect heredity occurred in 16 cases group A having 3 and group B having 13 cases.

Summing up the whole aspect of heredity in epileptic insanity we find.

There was a direct heredity of Drink 40 times; of nervous disease 25 times; of consumption 18 times; of insanity 6 times; and of epilepsy 4 times.

Hence epilepsy itself can hardly be called a hereditary disease per se.

There was an indirect heredity of epilepsy 16 times; of consumption 15 times; of insanity 14 times; of nervous disease 7 times; and of drink once.

Thus, with the exception of consumption, the greater the direct heredity the less the indirect heredity. In the cases of drink, consumption and nervous disease, the direct heredity always exceeds the indirect. In the cases of insanity and epilepsy the indirect heredity is greater than the direct.

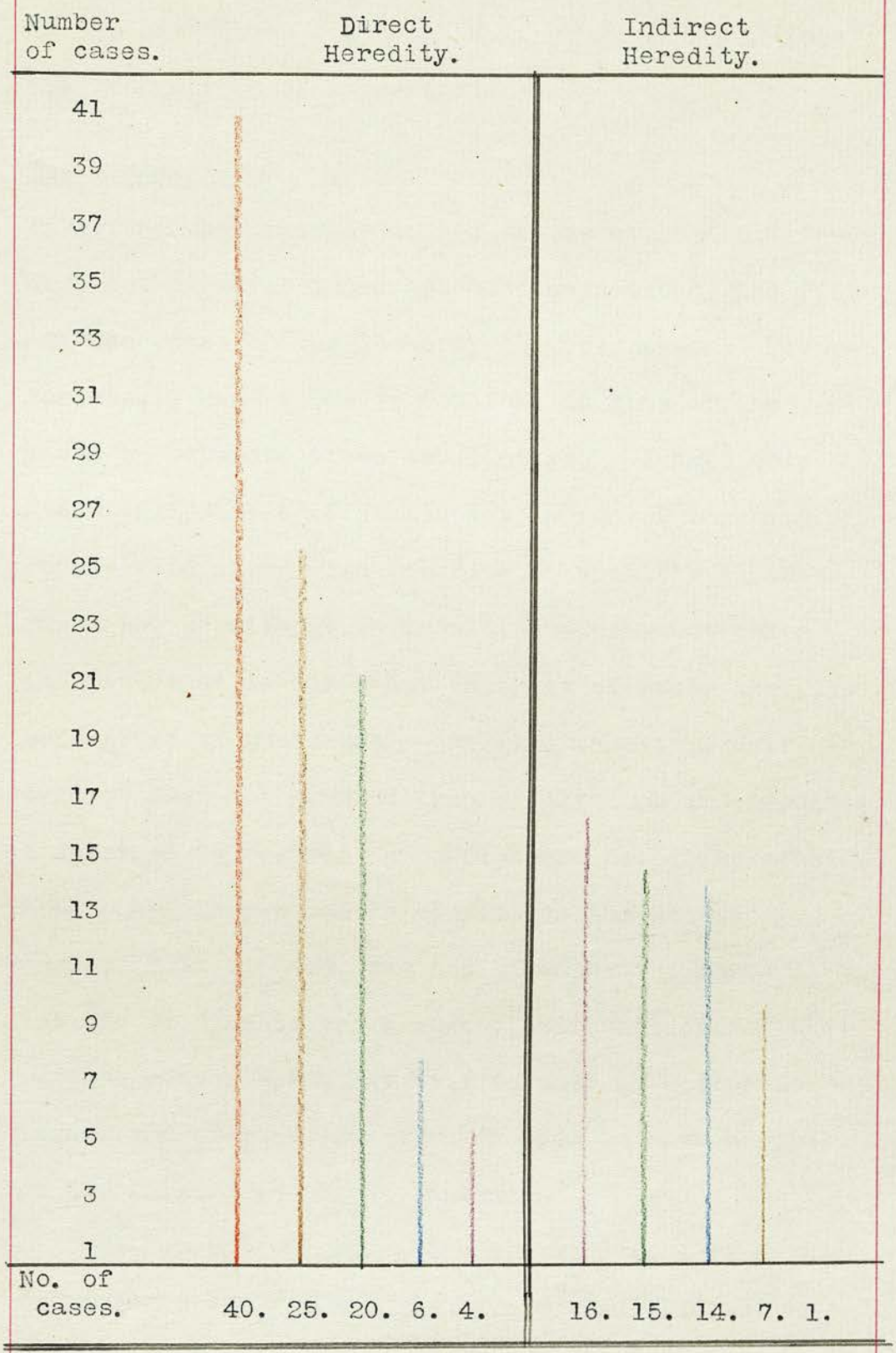


Table showing the hereditary factors in Epileptic Insanity, the direct and indirect nature of each, and their differences.

I have drawn out a table in order to facilitate the recognition of these facts.

The Causation.

In giving what they estimated as the cause the friends have not differentiated between the cause of the fits and the cause of the insanity in every case. Nevertheless, I have found it possible in some of the cases to separate these two factors. I have only noted in my table of statistics causes of insanity which acted along with the fits or possibly in some cases per se without their aid, and have not taken the fact that in the great majority of cases the fits were given as the cause. We will take this self-evident fact for granted then. Although the results I obtained as regards causation are not to be considered complete because in many cases the accessory cause, if it existed, was not given or was unknown, yet one is able to get a pretty fair estimate of the various causes which may help to make an epileptic insane and of some causes which seem to have brought on the fits in the first instance.

As regards the causation of the fits I find the following given. Head injury occurred oftenest as a factor of causation 9 cases being so noted. How

far this may be taken as a direct factor is doubtful for many children fall and suffer severe head injury and in some cases the fits arise afterwards from other causes. Many of these cases I find, however, arose in adults who fell from scaffolds or other structures, and the fits seem in these cases to be undoubtedly due to the fall, coming on shortly after the accident. Now these cases were not always cases of Jacksonian epilepsy but were cases closely resembling idiopathic epilepsy in the nature of the fits, which were general. Some cases were Jacksonian in nature. However, in all cases the resulting insanity bore all the symptons commonly seen in epileptic insanity. So the cusation of the fits has little effect on the form of insanity except in the case of alcohol.

Alcohol was the second largest cause of the fits noted. There were 8 cases showing this causation. Now these cases show that some persons who become hard drinkers develope epilepsy. The epilepsy comes on a year or two after the drink habit is formed .
The fits as a rule are few in number, one a week or so. They may even occur at longer intervals. Months may elapse between the fits. Very often these cases

develope delusions of grandeur, though they may develope delusions of persecution. Now these are the very cases which are most apt to be mistaken for general paralysis. The motor tremblings, the delusions of grandeur, and other symptoms closely simulate that condition.

Fright was the third largest cause and had 7 cases to its credit.

The fourth cause was fever. There were two cases where the fits arose during Scarlet Fever, in one case the fits arose during measles, amd in one case during an attack of smallpox.

Pregnancy and confinement accounted for 4 cases.

Teething was only given as a cause in two cases. A suprisingly low number it would seem as this is a cause most readily accepted.

Sunstroke accounted for two cases; vaccination for one; brain tumour for one; premature birth for one.

Now we come to consider the causes which may aid and abett the epilepsy in causing insanity or which may cause insanity in an epileptic per se.
The moral causes were found as follows.

Domestic worry and grief	12 cases
Disappointment in love	4 cases
Business worry and anxiety	4 cases
Poverty	2 cases
Religious anxiety	1 case

The bodily causes were found as follows.

Irregularity of courses	2 cases
Self abuse	2 cases
Confinement	1 case
Bodily weakness	1 case
Heart disease	1 case
Drink	5 cases

Now all these are accepted causes of insanity and I shall only now refer to the last one, drink.

Now in these cases drink was not the cause of the fits but the cause or accessory cause of the insanity. It is well known how many epileptics become maniacal and uncontrollable from small quantities of alcohol. Now many epileptics take to drink. They seek to drown their care and misery thus. The drink instead of helping them to bear their affliction causes them to become insane.

Now we saw that drink may cause fits by itself. Hence the drink habit exaggerates the epilepsy. In both classes of insanity thus formed the ultimate state of the patient is identical, and you cannot tell whether the fits or the drink occured first in the history of the case unless you are told.

Whether these epileptics would have become insane if the accessory causes were absent or not, of course we cannot say, but it seems more than probable

that in most cases they would either not have become insane at all or would have become insane only at a much later period.

The occurence of Status Epileptics.

Status epileptics occured 39 times (some of the patients having more than one attack) of these 19 ended in recovery, and 20 ended in death.

Of chronic bodily disorders epileptics are not very susceptible. Phthisis, heart disease, and chronic anaemia are the most frequent.

The Menstruation.

In epilepsy the menstruation is often disturbed, but this often occurs in insanity. However, I have ascertained the menstrual histories as far as possible before the patient became insane but was epileptic, with the following results.

40 cases were regular.

31 cases were regular.

14 cases had either no menstruation or it was altered in quantity.

The rest of the cases had no history.

The Delusions.

Although in many of the imbeciles and dements there were no definite delusions ascertained, and

these are not necessary to constitute insanity in the insane epileptic, in 79 cases there were definite and more or less fixed delusions ascertained.

The largest group of all was the group consisting of delusions of persecution; there were 39 cases.

Closely allied to this form of delusion are the delusion of poisoning and delusions of suspicion, the former occured twice and the latter 5 times.

Nine patients presented delusions of a religious nature.
Six patients had delusions of grandeur.
Six patients had delusions of identity.
Three had delusions of a hypochondriacal nature; 4 of an erotic and hysterical nature; and 7 of various other natures.

The Prognosis.

Out of the 200 cases of epileptic insanity recorded 15 or about 7½% recovered.

Five cases of mania or about 18% of all cases of mania recorded recovered. Of the melancholics eight cases or about 25% of all the melancholics recovered. Of those suffering from circular epileptic insanity two cases of 33% recovered. Of the cases of true monomania one case out of the two recorded recovered. Epileptic dementia, delirium and stupor showed no recoveries.

Females.

Number.	1.	2.	3.	4.
Age	32	28	15	20
Attack	1	1	1	1
Suicidal	No	No	Yes	No
Dangerous	Yes	Yes	Yes	Yes
Mental Condition	Circular form	Dementia	Dementia	Mania
Bodily Condition	Fair	Poor	Poor	Fair
Epilepsy started	6	17	7	13
Age, 1st Attack of Insanity.	25	18	12	20
Hereditary History of: Drink	No	No	No	No
Consumption	No	No	No	Uncle
Nervous Disease	Mother (strokes)	No	No	Mother
Insanity	No	Grandfather	No	No
Fits	No	No	No	Cousin
Cause given	----	Disappointment in Love.	Fright	Onset of courses
Status Epilepticus	No	No	No	No
Chronic Bodily Disorder	Nil	Nil	Nil	Nil
Menstrual History	Irregular	Irregular	Regular	Regular Anaemia improved under Bromide.
Delusions	Of suspicion			Of persecution
Notes	Gradual development from Epilepsy minor to Epilepsy major and on to insanity.			

Females.

Number.	5.	6.	7.	8.
Age	21	35	23	23
Attack	----	----	1	1
Suicidal	No	No	No	No
Dangerous	No	No	Yes	Yes
Mental Condition	Imbecility	Dementia	Imbecility	Imbecility
Bodily Condition	Fair	Feeble	Fair	Fair
Epilepsy started	7	16	12	1½
Age, 1st Attack of Insanity	11	35	12	9
Hereditary History of Drink	No	No	No	No
Hereditary History of Consumption	No	No	No	No
Hereditary History of Nervous Disease	No	No	No	No
Hereditary History of Insanity	No	No	No	No
Hereditary History of Fits	No	No	No	No
Cause given	----	Irregularity of courses.	Fall	Teething
Status Epilepticus	No	No	No	No.
Chronic Bodily Disorder	Nil	Nil	Nil	Nil
Menstrual History	Regular	Irregular	Not regular	Not regular
Delusions		Of identity		Of persecution
Notes				

Females.

Number.	9.	10.	11.	12.
Age	38	24	47	48
Attack	3rd	2nd	1st	1st
Suicidal	No	No	No	No
Dangerous	No	No	Yes	No
Mental Condition	Dementia	Dementia	Dementia	Mania
Bodily Condition	Poor	Fair	Fair	Poor
Epilepsy started	Unknown	18	Not known	Now known
Age, 1st Attack of Insanity	33	23	47	48
Hereditary History of: Drink	No	No	Not known	Father
Hereditary History of: Consumption	No	No	Not known	No
Hereditary History of: Nervous Disease	No	No	Not known	Sister
Hereditary History of: Insanity	No	Uncle Insane	Not known	No
Hereditary History of: Fits	No	No	Not known	No
Cause given	----	Irregular Menstruation	----	Family Trouble
Status epilepticus	No	No	No	No
Chronic Bodily Disorder.	Nil	Nil	Nil	Psoriasis Anaemia
Menstrual History	Regular	Irregular	Regular	Regular
Delusions	Of persecution	Of persecution	Of persecution	Of grandeur
Notes	1st attack, pregnant at the time and was discharged recovered.			

Females.

Number	13.	14.	15.	16.
Age	23	46	13	37
Attack	3rd	1st	3rd	1st
Suicidal	No	Yes	Yes	Yes
Dangerous	Yes	Yes	Yes	No
Mental Condition	Mania	Delirium	Imbecility	Delirium
Bodily Condition	Fair	Poor	Fair	
Epilepsy started	16	40	5	
Age, 1st attack of Insanity	21	46	7	37
Hereditary History of: Drink	No	No	No	
Hereditary History of: Consumption	No	Brother	No	
Hereditary History of: Nervous Disease	No	No	No	
Hereditary History of: Insanity	Not known	No	No	
Hereditary History of: Fits	No	No	No	
Cause given	----	Business worries	Scarlet Fever	
Status Epilepticus	No	No	Once	No
Chronic Bodily Disorder	Cardiac Disease	Nil	Nil	
Menstrual History	Regular	Floodings	Not regular	
Delusions	Of persecution	Nil		
Notes	Cardiac Epileptic Discharged recovered.	Ep. Delirium Pumpfeeding Died of Pneumonia 2 mos. after admission.		Died from exhaustion and pneumonia, 4 days after admission.

Females.

Number.	17.	18.	19.	20.
Age	39	48	62	20
Attack	1st	1st	1st	1st
Suicidal	No	No	Yes	No
Dangerous	No	No	Yes	Yes
Mental Condition	Melancholia	Dementia	Delirium	Mania
Bodily Condition	Fair	Poor	Poor	Fair
Epilepsy started	Unknown	Unknown	Unknown	12
Age, 1st attack of Insanity	39	48	62	20
Hereditary History of — Drink	No	Father		Father
Hereditary History of — Consumption	No	No		No
Hereditary History of — Nervous Disease	Uncle (stroke)	Sister (stroke)		No
Hereditary History of — Insanity	No	No		No
Hereditary History of — Fits	No	No		No
Cause given	----	----		----
Status Epilepticus	No	No		No
Chronic Bodily Disorder	Nil	Psoriasis	Heart Disease	Nil
Menstrual History	Regular	Floodings	Absent	Regular
Delusions				Of a religious nature
Notes		2 children died of consumption.	Died from Heart Disease congestion of Lungs and exhaustion 14 days after admission.	

Females.

Number.	21.	22.	23.	24.
Age	23	11	25	46.
Attack	1st	1st	1st	1st
Suicidal	No	No	No	Yes
Dangerous	Yes	Yes	No	Yes
Mental Condition	Imbecility	Imbecility	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	3	7	18	Unknown
Age, 1st attack of Insanity	3	Birth	21	45¼
Hereditary History of — Drink	No	No	No	
Hereditary History of — Consumption	No	Grandfather Sister Brother	No	
Hereditary History of — Nervous Disease	No	No	No	
Hereditary History of — Insanity	No	No	No	
Hereditary History of — Fits	No	No	No	
Cause given	Fall	----	Grief	
Status Epilepticus	No	No	No	
Chronic Bodily Disorder	Nil	Nil	Nil	Nil
Menstrual History	Regular	Absent	Regular	
Delusions			Of a hypochondrical nature.	Of persecution
Notes.		Had fits at 7 years old none since.	Fits (epilepsy minor) came on after confinement 2nd child.	

Females.

Number.	25.	26.	27.	28.
Age	22	79	50	18
Attack	1st	3rd	3rd	1st
Suicidal	No	No	Yes	Yes
Dangerous	No	No	No	Yes
Mental Condition	Dementia	Dementia	Melancholia	Mania
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	12	57	36	4 weeks ago
Age, 1st attack of Insanity	22	57	36	16 " "
Hereditary History of Drink	No	No	No	No
Hereditary History of Consumption	No	Father	No	No
Hereditary History of Nervous Disease	No	-	No	No
Hereditary History of Insanity	No	-	No	No
Hereditary History of Fits	No	-	No	No
Cause given	----	----	Family Troubles	Puerperal
Status Epilepticus	No	2 attacks 2nd fatal.	No	No
Chronic Bodily Disorder	Nil	Atheroma	Five Miscarriages.	Nil
Menstrual History	Regular	Absent	Regular	Absent
Delusions	Of persecution	Of identity	Of a Political nature.	Of persecution
Notes	Died from suffocation in a fit.	Status 2 mos. after admission and again in 2 months. One child had fits. Died in Status of 14 days.	Discharged recovered 4 months.	Discharged recovered 5 months after.

Females.

Number.	29.	30.	31.	32.
Age	37	27	27	36
Attack	1st	1st	1st	1st
Suicidal	No	Yes	No	No
Dangerous	No	Yes	Yes	No
Mental Condition	Dementia	Melancholia	Dementia	Melancholia
Bodily Condition	Feeble	Fair	Good	Fair
Epilepsy started	7	Unknown	Birth	Unknown
Age, 1st attack of Insanity.	37	27	$26\frac{3}{4}$	36
Hereditary History of: Drink	No		No	
Hereditary History of: Consumption	No		"	
Hereditary History of: Nervous Disease	No		"	
Hereditary History of: Insanity	No		"	
Hereditary History of: Fits	No		"	
Cause given	Measles at 7	----	Poverty	----
Status epilepticus	Died in Status.	No	No	No
Chronic Bodily Disorder	Nil	Nil	Nil	Acne
Menstrual History	Regular	Regular	Irregular	Regular
Delusions		Of persecution	Of persecution	
Notes	Died in Status 1 day.			Procursive Epilepsy.

Females.

Number.	33.	34.	35.	36.
Age	29	9	37	22
Attack	1st	1st	2nd	1st
Suicidal	Yes	Yes	No	No
Dangerous	No	Yes	Yes	No
Mental Condition	Melancholia	Imbecility	Dementia	Mania
Bodily Condition	Fair	Fair	Feeble	Fair
Epilepsy started	Unknown	2 yrs. old	2 but stopped till 16.	16
Age, 1st Attack of Insanity	29	Birth	37	21¾
Hereditary History of: Drink		No	No	No
Consumption		No	Father	No
Nervous Disease.		No	Mother	No
Insanity		No	Maternal Uncle, Brother.	Maternal Uncle.
Fits		No	Brother	No
Cause given	----	----	----	Family Troubles
Status Epilepticus	No	No	1 attack	No
Chronic Bodily Disorder	Heart Disease and Phthisis.	Infant Paralysis	Nil	Hysteria
Menstrual History	Regular	None	Regular	Irregular
Delusions				Of an erotic and hysterical nature.
Notes.			Fits stopped between ages of 2 & 16.	Fits started after Scarlet Fever at 16.

Females.

Number.	37.	38.	39.	40.
Age	13	26	14	39
Attack	1st	1st	1st	1st
Suicidal	No	No	No	No
Dangerous	Yes	No	Yes	No
Mental Condition	Imbecility	Imbecility	Imbecility	Melancholia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	2 years	2 years	9 months	?
Age, 1st attack of Insanity.	Birth	Birth	Birth	33
Hereditary History of — Drink	Grandparents on both sides.	No	No	
Hereditary History of — Consumption	Grandmother (maternal)	No	No	
Hereditary History of — Nervous Disease	Grandmother (maternal)	No	No	
Hereditary History of — Insanity		No	No	
Hereditary History of — Fits		No	No	
Cause given	----	Vaccination?	Teething	
Status epilepticus	Yes recovery.	No	No	No
Chronic Bodily Disorder	Nil	Nil	Infant Paralysis	Heart Disease
Menstrual History	Never changed	Regular	No menses	Irregular
Delusions				Of a hypochondriacal nature.
Notes.				Great variation in mental condition sometimes happy.

Females.

Number.	41.	42.	43.	44.
Age	26	62	21	31
Attack	2nd	1st	1st	1st
Suicidal	No	Yes	No	Yes
Dangerous	No	Yes	Yes	Yes
Mental Condition	Circular form	Melancholia	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair	Poor
Epilepsy started	19	18	?	9
Age, 1st attack of Insanity	23	62	13	30¾
Hereditary History of — Drink	Mother	Father		Father
Hereditary History of — Consumption	No			Sister and Brother
Hereditary History of — Nervous Disease	No			
Hereditary History of — Insanity	No	Sister and Brother		
Hereditary History of — Fits	No			
Cause given	----	Change of life.	----	Family Troubles
Status epilepticus	No	No	No	No
Chronic Bodily Disorder.	Phthisis Ague.	Heart Disease.	Nil	Nil
Menstrual History	Irregular	Menapause	Regular	Irregular
Delusions	Of a religious nature.		Of persecution	Delusions about womb. Hysteria of a Hypochondriacal nature.
Notes.	Quite rational at times, discharged recovered 3 yrs. ago.	At times violent & excited.		Child died from fits. Took drink, cut throat on admission.

Females.

Number.	45.	46.	47.	48.
Age	34	32	45	17
Attack	1st	2nd	1st	1st
Suicidal	No	No	No	Yes
Dangerous	Yes	No	No	Yes
Mental Condition	Dementia	Dementia	Dementia	Mania
Bodily Condition	Poor	Poor	Fair	Fair
Epilepsy started	3	5	7	15
Age, 1st attack of insanity.	32	22	45	17
Hereditary History of: Drink	Grandfather		No	No
Consumption	2 aunts (paternal)	Maternal Aunt & Uncle	No	No
Nervous Disease		Maternal Grandfather	No	No
Insanity	Paternal uncle		No	No
Fits			No	No
Cause given	----	Sunstroke at 5 years.	----	Love affair
Status Epilepticus	No	No	No	Slight
Chronic Bodily Disorder	Anaemia Menorrhagia.	Anaemia	Dysentery	Nil
Menstrual History	Irregular	Regular		Regular
Delusions	No definite delusions	No definite delusions	No definite delusions	Of a sexual nature.
Notes.				

Females.

Number.	49.	50.	51.	52.
Age	14	28	21	19
Attack	1st	1st	1st	1st
Suicidal	No	No	No	Yes
Dangerous	Yes	No	Yes	Yes
Mental Condition	Imbecility	Dementia	Imbecility	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started		8	Infancy	17
Age, 1st attack of insanity	1	28	Infancy	19
Hereditary History of Drink		No	No	No
Hereditary History of Consumption		No	No	No
Hereditary History of Nervous Disease		No	Yes	No
Hereditary History of Insanity		No	No	No
Hereditary History of Fits		No	No	No
Cause given	----	----	----	----
Status Epilepticus	No	No	No	Yes
Chronic Bodily Disorder	Cardiac Disease and Infantile Paralysis.	Heart.	Rickets	Nil
Menstrual History	Regular	Irregular	Regular	Irregular
Delusions	"Lots of sweethearts"	Of a melancholic nature.	Of persecution, and erotic, lots of sweethearts.	Very erotic and religious.
Notes.	Erotism marked.			

Females.

Number.	53.	54.	55.	56.
Age	30	22	32	62
Attack	4th	2nd	1st	4th
Suicidal	Yes	No	No	Yes
Dangerous	Yes	Yes	Yes	No
Mental Condition	Circular form	Dementia	Melancholia	Melancholia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	14	16	29	Unknown
Age, 1st attack of Insanity.	26	21	32	62
Hereditary History of Drink	No	No	No	No
Hereditary History of Consumption	No	No	No	Son
Hereditary History of Nervous Disease	No	No	No	Brother & Sister (Apoplexy)
Hereditary History of Insanity	No	No	No	2 sisters commited suicide.
Hereditary History of Fits	No	No	No	No
Cause given	----	Fright	Pregnancy	Family troubles.
Status Epilepticus	No	No	No	No
Chronic Bodily Disorder	Heart Dis. Phthisis & Hysteria	Phthisis	Anaemia	Heart Dis. Phthisis.
Menstrual History	Irregular	Regular	Regular	Irregular
Delusions	Of persecution of food being poisoned.	Of persecution		Of a melancholic nature. Of persecution.
Notes.		Died of Phthisis 2 years after admission.	Discharged recovered after six months.	

Females.

Number.	57.	58.	59.	60.
Age	15	19	29	27
Attack	1st	3rd	1st	1st
Suicidal	No	Yes	No	No
Dangerous	No	No	No	Yes
Mental Condition	Imbecile	Mania	Melancholia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	10	Unknown	3	7 months
Hereditary History of: Age, 1st attack of Insanity	Birth	15	29	Unknown
Drink	No	Mother	No	No
Consumption	No	No	No	No
Nervous Disease	No	No	Paternal Grandfather	No
Insanity	No	No	No	Maternal Aunt
Fits	No	No	No	No
Cause given	----	----	----	Fright
Status Epilepticus	Yes	No	No	No
Chronic Bodily Disorder	Nil	Nil	Nil	Nil
Menstrual History	Absent	Regular	Regular	Irregular
Delusions		Poisoning	Hypochondriacal	
Notes		1st attack insanity 9 days, 2nd 2 months.	Discharged relieved in 3 months.	

Females.

Number.	61.	62.	63.	64.
Age	28	33	38	27
Attack	1st	3rd	1st	1st
Suicidal	No	No	No	No
Dangerous	No	No	No	Yes
Mental Condition	Dementia	Circular form	Mania	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	6 months	3	37	4
Age, 1st attack of Insanity	27	25	38	27
Hereditary History of Drink	No	No	No	No
Hereditary History of Consumption	No	No	No	No
Hereditary History of Nervous Disease	No	No	No	No
Hereditary History of Insanity	No	No	No	No
Hereditary History of Fits	No	No	No	No
Cause given	----	----	Family Troubles.	----
Status Epilepticus	No	No	No	No
Chronic Bodily Disorder	Nil	Dyspepsia Hysteria.	Nil	Nil
Menstrual History	Irregular	Irregular	Regular	Irregular
Delusions	Of identity	Of a religious nature, also of hypochondriacal nature.	Of persecution.	Of persecution.
Notes		Discharged recovered twice from other asylums.	Discharged recovered 7 months after.	

Females.

Number.	65.	66.	67.	68.
Age	12	16	31	57
Attack	1st	1st	1st	1st
Suicidal	No	No	Yes	No
Dangerous	Yes	No	Yes	Yes
Mental Condition	Idiocy	Imbecility	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	10	At birth	6	24
Age, 1st attack of Insanity.	12	"	31	57
Hereditary History of: Drink	No	No	No	No
Hereditary History of: Consumption	Paternal Grandfather	No	No	15 cousins
Hereditary History of: Nervous Disease	No	Maternal Grandmother (Apoplexy)	No	Mother (stroke)
Hereditary History of: Insanity	No	No	No	No
Hereditary History of: Fits	No	No	No	No
Cause given	----	----	----	----
Status Epilepticus	Frequently	Once	No	No
Chronic Bodily disorder	Anaemia	Phthisis	Hysteria	Nil
Menstrual History	Not changed	Regular	Irregular	Stopped at 24 and then fits came on.
Delusions				
Notes	Largest Monthly number of fits in Asylum. Has since died in Status.	Died from Phthisis.		

Females.

Number.	69.	70.	71.	72.
Age	50	18	28	21½
Attack	1st	1st.	2nd	1st
Suicidal	No	No	No	No
Dangerous	Yes	No	No	Yes
Mental Condition	Melancholia	Idiocy	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	9	11	1½ yrs.	15
Age, 1st attack of Insanity	50	Birth	20	21
Hereditary History of: Drink	Father	No	No	No
Hereditary History of: Consumption	No	No	Father	Father
Hereditary History of: Nervous Disease	Father (stroke)	No	No	No
Hereditary History of: Insanity	No	No	No	No
Hereditary History of: Fits	No	Brother	No	Mother's cousin
Cause given	Drink		----	----
Status Epilepticus	No	No	Once	No
Chronic Bodily Disorder	Nil	Phthisis	Nil	Nil
Menstrual History	Regular	Not changed	Regular	Irregular
Delusions	That relations came to see her at night.		Of persecution	Of persecution
Notes.			Fits started when teething.	

Females.

Number.	73.	74.	75.	76.
Age	28	34	28	26
Attack	1st	1st	1st	1st
Suicidal	No	Yes	Yes	No
Dangerous	No	No	No	No
Mental Condition	Dementia	Melancholia	Melancholia	Dementia
Bodily Condition	Fair	Fair	Poor	Fair
Epilepsy started	12	34		15
Age, 1st attack of Insanity	12	34		26
Hereditary History of Drink	No	No		No
Hereditary History of Consumption	No	No		Cousin.
Hereditary History of Nervous Disease	No	No		Both Grandfathers (stroke)
Hereditary History of Insanity	No	Child		No
Hereditary History of Fits	No	No		No
Cause given	Scarlet F.	Family Bereavement	----	Fright
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Nil	Nil	Phthisis	Hysteria Heart disease.
Menstrual History	Irregular	Regular	Irregular	Regular
Delusions	That she has lost her relatives and of persecution.	Of persecution and identity	Of Poisoning	Of identity
Notes	Fits started in Scarlet Fever.		Died from Phthisis.	Choreiform movements at times.

Females.

Number.	77.	78.	79.	80.
Age	36	62	33	55
Attack	1st	1st	1st	1st
Suicidal	No	No	Yes	No
Dangerous	No	No	No	No
Mental Condition	Melancholia	Dementia	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	20	Unknown	28	15
Age, 1st attack of Insanity	36	62	33	55
Hereditary History of — Drink	No		No	No
Hereditary History of — Consumption	No		No	No
Hereditary History of — Nervous Disease	No		No	No
Hereditary History of — Insanity	No		No	No
Hereditary History of — Fits	Brother		No	No
Cause given	Disappointment in Love.	----	Fright	----
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Hysteria Anaemia	Heart Disease.	Nil	Heart Inf. Paralysis.
Menstrual History	Regular		Regular	
Delusions	Says everybody here is married.			
Notes.		Died from Ascites.		Fits began with menstruation, and have become less in number since the change in life set in.

Females.

Number.	81.	82.	83.	84.
Age	36	23	31	24
Attack	1st	1st	1st	1st
Suicidal	No	No	Yes	No
Dengerous	No	No	No	No
Mental Condition	Circular form	Dementia	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	Birth	18	20	10
Age, 1st attack of insanity	36	23	31	24
Hereditary History of — Drink	No	No	Mother	Brother
Hereditary History of — Consumption	No	No	No	Mother
Hereditary History of — Nervous Disease	No	No	No	Father
Hereditary History of — Insanity	No	Mother Grandmother	No	No
Hereditary History of — Fits	No	No	No	No
Cause given	----	Confinement of illegit. child.	----	Fright
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Heart Disease	Nil	Nil	Nil
Menstrual History	Regular	Irregular	Regular	Regular
Delusions	Concerning her heart.	Of persecution	Concerning her husband.	
Notes.				

Females.

Number.	85.	86.	87.	88.
Age	36	32	36	32
Attack	1st	2nd	1st	1st
Suicidal	No	Yes	No	No
Dangerous	No	No	Yes	Yes
Mental Condition	Dementia	Stupor	Dementia	Dementia
Bodily Condition	Fair	Poor	Fair	Fair
Epilepsy started			2	21
Age, 1st attack of Insanity	36	31	36	32
Hereditary History of: Drink			No	No
Hereditary History of: Consumption			No	No
Hereditary History of: Nervous Disease			No	Maternal Grandfather (stroke)
Hereditary History of: Insanity			No	No
Hereditary History of: Fits			No	No
Cause given	----	----	Dis. in Love Illeg. Child.	----
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Nil	Nil	Nil	Anaemia
Menstrual History	Irregular	Regular	Regular	Irregular
Delusions	Of persecution		Concerning her paramour	Of persecution
Notes		Died from purulent meningitis and pneumonia		

Cases of Status ending in Death.

Number.	89. (female)	90. (Male)	91. (Male)	92.
Age	16	19	14	51
Attack	1st	1st	1st	1st
Suicidal	No	No	No	No
Dangerous	Yes	Yes	Yes	Yes
Mental Condition	Idiocy	Dementia	Imbecility	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	12	Childhood	3	
Age, 1st attack of insanity	Birth	15	Birth	51
Hereditary History of — Drink	No	No		Father
Hereditary History of — Consumption	Mother	No		No
Hereditary History of — Nervous Disease	No	No		No
Hereditary History of — Insanity	No	Father		Maternal Grandmother.
Hereditary History of — Fits	No	No		No
Cause given	Weakness	Fall		Religion
Status epilepticus	Twice or thrice.	Yes		Yes
Chronic Bodily Disorder	Anaemia	Heart Dis. Headache.		Heart Disease.
Menstrual History	Never unwell.			
Delusions				
Notes and History of case	Very stupid, complained constantly of pain in head. Duration of status 3 days. Preceded by jerkings and signs of cerebral irritation for a fortnight. Died 15 mos. after admission.	Very stupid & dull. 1st symptom loss of power in legs. Duration 2 days. Died 6 months after admission.	1. Excited & violent. 2. Dull & stupid 3. Free from fits. 4. November 18 cereb. Haemor. May 27, Status, duration 1 day. 5 yrs. after admission.	Nearly choked 26th May (paresis) 29th May, died in status. 42 fits, Temp. 103°F. 4½hrs. 16 days after admission.

Cases of Status ending in Death.

Number.	93. (Female)	94. (Male)	95. (Female)	96. (Female)
Age	44	24	16	37
Attack	1st	1st	1st	1st
Suicidal	No	No	Yes	Yes
Dangerous	No	Yes	Yes	Yes
Mental Condition	Dementia	Idiocy	Idiocy	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started		Birth	12	23
Age, 1st attack of insanity	44	Birth	$15\frac{1}{2}$	37
Hereditary History of — Drink		No	Father	No
Hereditary History of — Consumption		Mother	No	No
Hereditary History of — Nervous Disease		No	No	No
Hereditary History of — Insanity		No	No	Brother
Hereditary History of — Fits		No	No	No
Cause given	Fits came on after confinement.	----	Fright	Confinement
Status epilepticus	Yes	Yes	Yes	Yes, twice.
Chronic Bodily Disorder	Heart Disease	Nil	Nil	Uterine Myomata.
Menstrual History			Not commenced	Irregular
Delusions				
Notes & History of case.	1. Excited and violent. 2. Dull & Stupid 3. Cheerful 4. Dull & stupid 5. Fits less frequent. 6. Status, 3 days Temp. 104°F. 4 yrs. after admission.	Died from status, 3 days duration. 1 yr. after admission.	Died in status of 12 hrs. duration, 1 yr. after admission.	1. Violent. 2. Quiet and few fits. 3. Status, recovery. 4. Quiet, Demented & Dirty. 5. Status, 70 fits, 3 days. 10 years after admission.

Cases of Status ending in Death.

Females.

Number.	97.	98.	99.
Age	48	72	25
Attack	1st	1st	1st
Suicidal	No	No	Yes
Dangerous	No	No	Yes
Mental Condition	Melancholia	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair
Epilepsy started	48	72	3
Age, 1st attack of insanity	48	72	25
Hereditary History of — Drink	No		No
Consumption	Father and Mother		No
Nervous Disease	No	Not given	Brother
Insanity	No		No
Fits	Sister		Father and Sister
Cause given	Grief		----
Status epilepticus	Yes	Twice 2nd Fatal	Yes
Chronic Bodily Disorder	Nil	Bronchitis Heart Disease.	Nil
Menstrual History	Regular		Irregular
Delusions.			
Notes & History of case	Patient found in yard in a state of convulsions, head turned to right. Convulsions general Temp. 105.2°F. Fell to 103°F. after sponging. Died in 8 hours.	May 3rd. Has passed into status epilep. Last night after 70 fits had ½ i chloral per rectum & ½ i Brandy. In an hour 5 mins. atrop. sulph. injected. Fits stopped then having had 90. Very weak, Died 4th May, 2 days.	1. Depressed & violent. 2. Fits frequent and dull. 3. Quiet and Demented. Died in status, June 17/98. 11 yrs. after admission. Duration 1 day.

Cases of Status ending in Death.

Number.	100. (Male)	101. (Female)	102. (Male)
Age	12	38	21
Attack	1st	1st	1st
Suicidal	Yes	No	No
Dangerous	No	No	Yes
Mental Condition	Imbecility	Idiocy	Mania
Bodily Condition	Fair	Fair	Fair
Epilepsy started	3	14	Birth
Age, 1st attack of insanity	Birth	Birth	21
Hereditary History of — Drink	Father	Father	Not given
Hereditary History of — Consumption			Not given
Hereditary History of — Nervous Disease	Father's sister		Not given
Hereditary History of — Insanity		Paternal Aunt	Not given
Hereditary History of — Fits			Not given
Cause given	----	----	----
Status epilepticus	Yes	Yes	Yes
Chronic Bodily Disorder	Nil	Hydrocephalus	Nil
Menstrual History			
Delusions			
Notes & History of case.	1. Mania 2. Fits less frequent. 3. Quiet and Imbecile. 4. Status, Temp. 101°F., 6 yrs. after admission Duration 3 days.	1. Destructive and violent. 2. Somewhat quieter. 3. Status 1 year. after admission. Duration 3 days. (128 fits).	1. Mania with violence. 2. Quiet and frequent fits. 3. Stupid & Demented 4. Status of 3 days duration, 22 years after admission.

Cases of Status ending in Death.

Number.	103. (Female)	104. (Female)	105. (Male)
Age	17	50	14
Attack	1st	1st	1st
Suicidal	No	No	No
Dangerous	No	No	No
Mental Condition	Dementia	Melancholia	Imbecility
Bodily Condition	Fair	Fair	Poor
Epilepsy started		36	Birth
Age, 1st attack of insanity	17	50	Birth
Hereditary History of: Drink	Not stated	Mother	Not given
Hereditary History of: Consumption		Father	
Hereditary History of: Nervous Disease		No	
Hereditary History of: Insanity		No	
Hereditary History of: Fits		No	
Cause given	----	Drink	----
Status epilepticus	Yes	Yes	Yes
Chronic Bodily Disorder	Nil	Nil	Nil
Menstrual History	----	-----	----
Delusions	----	----	----
Notes & History Of case.	1. Demented and dull 2. Quiet, useful, fits frequent. 3. Fits infrequent Died in status, 1 day, 1 yr. after admission.	1. Melancholic 2. Somewhat better 3. Status, Fits 50 Temp. 106°F. 9 months after admission, duration 3 days.	1. Weak & having many fits. 2. Few fits, more cheerful & normal. 3. Working, bright and cheerful. 4. Went out on trial, 6 yrs. after admisn. 5. Brought back in 5 months. 6. Getting worse. 7. Status, Duration 4 days, 11 yrs. after admission.

Deaths from Epileptic Exhaustion .

Males.

Number.	106.	107.	108.	109.
Age	48	70	13	25
Attack	1st	1st	1st	1st
Suicidal	No	No	No	No
Dangerous	No	Yes	Yes	Yes
Mental Condition	Dementia	Dementia	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	40	From fall 64	11	10
Age, 1st attack of insanity	41	70	13	19
Hereditary History of — Drink	Father & Uncle	Not stated.	Not known.	Not known.
Hereditary History of — Consumption	No			
Hereditary History of — Nervous Disease	Mother (stroke)			
Hereditary History of — Insanity	Mother			
Hereditary History of — Fits	No			
Cause given	Alcoholic Epilepsy.	----	----	----
Notes & History of case.	Jan. 23, 1896. Last night patient was reported to be convulsed; when visited was lying on his back in bed. Left sided convulns. Pupils dilated. On attempt to examine him next morning convulsions came on again. Jan. 26, Has been having fits more or less ever since. Jan. 29 died. D. 4 days. 2 yrs. after admission.	Died quietly, having fits 2 yrs. after admission.	Died more or less stupid, having fits 3 days. 4 yrs. after admission. Fits followed blow on head when 11 yrs. old.	48 days. 17 yrs. after admission. Fits followed blow on head at ten years old.

Males.

Number.	110.	111.	112.	113.
Age	15	27	28	34
Attack	1st	1st	1st	1st
Suicidal	No	Yes	No	No
Dangerous	No	Yes	No	No
Mental Condition	Idiocy	Melancholia	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	?	2	28	30
Age, 1st attack of insanity	Birth	27	28	34
Hereditary History of — Drink	Not given.	No	No	Father & Brother.
Hereditary History of — Consumption		No	Paternal Aunt.	No
Hereditary History of — Nervous Disease		Grandmother (stroke)	Mother's Mother (stroke)	No
Hereditary History of — Insanity		No	No	Father and Grandfather.
Hereditary History of — Fits		No	No	Father
Cause given	----	----	Alcoholic Epileptic	Alcoholic Epileptic.
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Rickets	Nil	Nil	Nil
Delusions		God speaks to him.		
Notes.				

Males.

Number.	114.	115.	116.	117.
Age	15	23	41	30
Attack	1st	1st	1st	1st
Suicidal	No	Yes	No	Yes
Dangerous	No	Yes	Yes	Yes
Mental Condition	Idiocy	Dementia	Melancholia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	2	8		23
Age, 1st attack of insanity	Birth	23	35	29
Hereditary History of: Drink	No	No	No	No
Consumption	No	No	No	No
Nervous Disease	Maternal Grandmother	No	Maternal Aunt (Stroke)	No
Insanity	No	Grandfather	No	No
Fits	No	No	Brother	No
Cause given	----	----	Worry	Blow on head.
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Profound Anaemia	Nil	Nil	Nil
Delusions				Concerning the Queen
Notes			Died from Asphyxia by choking	Traumatic epilepsy from blow on head.

M a l e s.

Number.	118.	119.	120.	121.
Age	25	37	31	42
Attack	1st	1st	1st	1st
Suicidal	No	No	No	Yes
Dangerous	Yes	Yes	No	Yes
Mental Condition	Imbecility	Dementia	Dementia	Mania
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	21	12	17	42
Age, 1st attack of insanity	Birth	37	31	42
Hereditary History of — Drink	No	No	No	No
Hereditary History of — Consumption	No	No	No	No
Hereditary History of — Nervous Disease	Grandmother (stroke)	No	No	No
Hereditary History of — Insanity	No	No	No	No
Hereditary History of — Fits	No	No	No	No
Cause given	----	----	----	----
Status epilepticus	No	Once	No	Once
Chronic Bodily Disorder	Nil	Nil	Nil	Nil
Delusions				
Notes.				Died from Cerebral Haemorrhage.

Males.

Number.	122.	123.	124.	125.
Age	24	14	6	20
Attack	1st	1st	1st	1st
Suicidal	No	No	No	No
Dangerous	Yes	Yes	No	No
Mental Condition	Dementia	Imbecility	Idiocy	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	15	11	1	14
Age, 1st attack of insanity	24	Birth	Birth	20
Hereditary History of: Drink	No	No	No	Father
Consumption	No	Mother	No	4 cousins
Nervous Disease	No	No	No	No
Insanity	No	No	No	Maternal uncle
Fits	No	No	No	"
Cause given	----	----	----	Self abuse.
Status epilepticus	No	No	Once, recovery	No
Chronic Bodily Disorder	Phthisis	Nil	Rickets	Nil
Delusions				
Notes.	A case of Traumatic Epilepsy from blow with cricket ball. Died from Empyaema.			

Males.

Number.	126.	127.	128.	129.
Age	61	39	62	49
Attack	4th	1st	1st	1st
Suicidal	No	No	Yes	No
Dangerous	Yes	Yes	Yes	Yes
Mental Condition	Mania	Dementia	Mania	Mania
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	60	25	51	47
Age, 1st attack of insanity	56	39	62	49
Hereditary History of: Drink	No	No	Father	Father
Consumption	No	No	Mother	Father
Nervous Disease	No	Grandmother (strokes)	No	Mother (stroke)
Insanity	No	No	No	No
Fits	No	No	No	Sister
Cause given	----	Loss of Relatives	Heart Disease	Drink Alcoholic Epilepsy
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Chronic Rheumatism		Heart Disease.	
Delusions		Of persecution	That he is dead.	Of grandeur.
Notes.			Died from Heart Disease.	

Males.

Number.	130.	131.	132.	133.
Age	48	26	15	34
Attack	1st	1st	1st	1st
Suicidal	No	No	No	No
Dangerous	No	Yes	Yes	Yes
Mental Condition	Dementia	Mania	Imbecility	Mania
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	46	After a fall age not stated	3	11
Age, 1st attack of insanity	46	26	Infancy	34
Hereditary History of: Drink	No	No	No	No
Consumption	No	No	No	Father
Nervous Disease	No	No	No	No
Insanity	No	No	No	No
Fits	No	No	No	No
Cause given	Drink Alcoholic Epil.	Fall	----	----
Status epilepticus		No	Once recovery	No
Chronic Bodily Disorder	Nil	Nil	Nil	Nil
Delusions		Of persecution		
Notes.	Died from Cerebral Haemorrhage.			

Males.

Number.	134.	135.	136.	137.
Age	45	30	65	44
Attack	1st	1st	1st	1st
Suicidal	No	Yes	No	No
Dangerous	No	Yes	No	No
Mental Condition	Monomania	Dementia	Mania	Mania
Bodily Condition	Poor	Fair	Fair	Fair
Epilepsy started	?	21	Childhood	32
Age, 1st attack of insanity	45	30	65	44
Hereditary History of: Drink	Mother	Father	No	No
Consumption	No	No	No	No
Nervous Disease	No	Mother & Sister Died of(strokes)	No	No
Insanity	No	No	No	No
Fits	No	Brother	No	No
Cause given	Alcoholic Epilepsy	Poverty	----	Loss of Employment
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Heart Disease	Nil	Nil	Nil
Delusions		Of persecution	Of persecution	Of persecution
Notes	Discharged recovered after 8 months.			

Males.

Number.	138.	139.	140.	141.
Age	24	24	32	31
Attack	1st	1st	1st	1st
Suicidal	Yes	No	No	No
Dangerous	Yes	No	Yes	No
Mental Condition	Melancholia	Dementia	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	14	20	3	3
Age, 1st attack of insanity	24	24	32	31
Hereditary History of: Drink	Father	No	No	No
Consumption	No	No	No	No
Nervous Disease	No	Grandfather (stroke)	No	No
Insanity	No	No	No	No
Fits	3 cousins	No	No	No
Cause given	----	Sunstroke	----	----
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Nil	Phthisis	Phthisis	Nil
Delusions	Of suspicion & Persecution	Of persecution		
Notes.			Died from Phthisis.	Epilepsy started at 3, but stopped till 15 yrs. old.

Males.

Number.	142.	143.	144.	145.
Age	45	45	24	34
Attack	1st	1st	1st	1st
Suicidal	No	No	No	No
Dangerous	Yes	Yes	Yes	No
Mental Condition	Dementia	Dementia	Mania	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	15	10	14	14
Age, 1st attack of insanity	45	45	24	24
Hereditary History of — Drink	No	No	Father	No
Hereditary History of — Consumption	Maternal uncle	No	Maternal uncle.	Sister
Hereditary History of — Nervous Disease	No	No	No	No
Hereditary History of — Insanity	No	No	No	No
Hereditary History of — Fits	No	No	No	No
Cause given	----	----	----	----
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Nil	Nil	Nil	Nil
Delusions	Of Grandeur.	Of suspicion		
Notes.				

Males.

Number.	146.	147.	148.	149.
Age	22	25	11	33
Attack	1st	1st	1st	1st
Suicidal	No	Yes	No	No
Dangerous	Yes	Yes	Yes	No
Mental Condition	Imbecility	Circular form	Mania	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	15	13	10	7
Age, 1st attack of insanity	Birth	25	28	11
Hereditary History of — Drink	No	No	No	No
Hereditary History of — Consumption	No	No	Father & sister	No
Hereditary History of — Nervous Disease	No	Grandfather and great-grandfather died from "strokes"	No	No
Hereditary History of — Insanity	No	No	No	No
Hereditary History of — Fits	No	No	No	No
Cause given	----	----	----	Fright
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Nil	Nil	Nil	Nil
Delusions		Of suspicion	Of grandeur	
Notes.				Died from pneumonia 4 yrs. after admission.

Males.

Number.	150.	151.	152.	153.
Age	33	28	30	27
Attack	1st	1st	1st	1st
Suicidal	No	No	No	Yes
Dangerous	No	Yes	Yes	Yes
Mental Condition	Mania	Melancholia	Dementia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	26	16	5	13
Age, 1st attack of insanity	33	28	30	27
Hereditary History of: Drink	Father & Mother	No	No	Father
Hereditary History of: Consumption	No	Mother & Father	No	No
Hereditary History of: Nervous Disease	No	No	No	No
Hereditary History of: Insanity	Maternal Uncle	No	Uncle	No
Hereditary History of: Fits	No	No	No	No
Cause given	----	----	----	----
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	----	----	----	----
Delusions	Of grandeur and of a religious nature.	Of persecution		
Notes		Discharged recovered 4 months after admission.		

M a l e s.

Number.	154.	155.	156.	157.
Age	34	48	49	36
Attack	1st	1st	1st	1st
Suicidal	No	No	Yes	Yes
Dangerous	Yes	No	Yes	Yes
Mental Condition	Stupor	Melancholia	Dementia	Imbecility
Bodily Condition	Poor	Fair	Fair	Fair
Epilepsy started	27	35	29	1
Age, 1st attack of insanity	34	48	49	36
Hereditary History of: Drink	No	Both uncles	No	Father
Hereditary History of: Consumption	Mother & Sister	No	No	No
Hereditary History of: Nervous Disease	No	No	No	Father (stroke)
Hereditary History of: Insanity	No	No	No	Cousin
Hereditary History of: Fits	No	No	No	No
Cause given	----	Drink	Fall	----
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	----	----	----	----
Delusions				
Notes	Died from exhaustion on day of admission.			

Males.

Number.	158.	159.	160.	161.
Age	47	17	18	15½
Attack	1st	1st	1st	1st
Suicidal	No	Yes	No	No
Dangerous	Yes	No	Yes	Yes
Mental Condition	Dementia	Melancholia	Dementia	Imbecility
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	46	16	16	1
Age, 1st attack of insanity	47	17	17	1
Hereditary History of — Drink	Father and Mother	No	Father	No
Hereditary History of — Consumption	No	No	No	No
Hereditary History of — Nervous Disease	No	No	No	No
Hereditary History of — Insanity	No	No	No	No
Hereditary History of — Fits	No	No	No	No
Cause given	Alcoholic Epilepsy	----	Self-abuse	Small-pox
Status epilepticus	No	Once (slight attack)	No	No
Chronic Bodily Disorder	----	----	----	Phthisis
Delusions	Of persecution	----	Not ascertained	
Notes.		Discharged recovered 1 yr after admission.	Notes	Died from Phthisis 5 yrs. after admission.

Males.

Number.	162.	163.	164.	165.
Age	49	32	32	34
Attack	1st	1st	1st	1st
Suicidal	Yes	No	No	No
Dangerous	No	No	Yes	Yes
Mental Condition	Mania	Dementia	Melancholia	Imbecile
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	3	21	3	22
Age, 1st attack of insanity	49	21	32	Birth
Hereditary History of — Drink	No	No	Father	No
Hereditary History of — Consumption	No	No	No	No
Hereditary History of — Nervous Disease	No	No	No	No
Hereditary History of — Insanity	No	No	No	No
Hereditary History of — Fits	Mother	No	Maternal Aunt	No
Cause given	Domestic worry	Brain tumour	----	----
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	----	----	----	----
Delusions	On religious subjects.			
Notes.	Fits stopped from three years old to 14 years of age.	Cartilaginous brain tumour in frontal convolution found post mortem.		Died from suppurative arthritis 3 yrs. after admission.

Males.

Number.	166.	167.	168.	169.
Age	67	42	23	32
Attack	1st	1st	1st	1st
Suicidal	Yes	No	Yes	No
Dangerous	No	Yes	No	Yes
Mental Condition	Dementia	Mania	Melancholia	Melancholia
Bodily Condition	Feeble	Fair	Fair	Fair
Epilepsy started	64	39	6	1
Age, 1st attack of insanity	67	42	20	32
Hereditary History of — Drink	No	Father & Aunt	No	No
Hereditary History of — Consumption	Father	No	No	Maternal aunt and uncle.
Hereditary History of — Nervous Disease	No	No	No	No
Hereditary History of — Insanity	No	No	No	No
Hereditary History of — Fits	No	No	No	No
Cause given	----	Drink	Fright	----
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	----	----	Exopthalmus	----
Delusions				Of a religious nature.
Notes.	Died from Epileptic exhaustion 4 months after admission.	Is an Alcoholic epileptic. Discharged recovered 4 months after admission.		Fits stopped between 1 and 21. Fits from fall when young.

Males.

Number.	170.	171.	172.	173.
Age	25	55	49	23
Attack	1st	1st	1st	1st
Suicidal	Yes	Yes	Yes	No
Dangerous	No	Yes	No	Yes
Mental Condition	Melancholia	Dementia	Melancholia	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	10	50	44	12
Age, 1st attack of insanity	25	55	49	11
Hereditary History of: Drink	Brother	No	No	No
Consumption	No	No	No	No
Nervous Disease	No	No	No	No
Insanity	No	No	No	No
Fits	No	Brother	No	No
Cause given	----	----	----	----
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	----	Heart Disease	Phthisis	----
Delusions	Of suspicion	Of persecution		
Notes.			Died from Phthisis. 2 yrs. after admission.	Died from pneumonia 8 yrs. after admission.

Males.

Number	174.	175.	176.	177.
Age	20	10	48	22
Attack	1st	1st	1st	1st
Suicidal	Yes	No	No	Yes
Dangerous	No	No	Yes	Yes
Mental Condition	Imbecility	Imbecility	Dementia	Mania
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	4	4	30	13
Age, 1st attack of insanity	1	1	48	22
Hereditary History of: Drink	Father	Father	Mother	Paternal Uncle
Consumption	No	No	No	Paternal Aunt & Maternal Uncle
Nervous Disease	No	No	No	No
Insanity	No	No	No	Paternal Uncle
Fits	Grandmother (Maternal)	Paternal Uncle	No	No
Cause given	----	----	----	Fall
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	----	----	----	----
Delusions			Of persecution	Of persecution
Notes				

Males.

Number.	178.	179.	180.	181.
Age	39	34	62	34
Attack	1st	1st	1st	2nd
Suicidal	No	Yes	No	Yes
Dangerous	Yes	No	No	Yes
Mental Condition	Mania	Delirium	Dementia	Dementia
Bodily Condition	Fair	Poor	Fair	Fair
Epilepsy started	14	20	50	Unknown
Age, 1st attack of insanity	38	34	62	32
Hereditary History of: Drink	No	No	No	No
Hereditary History of: Consumption	No	No	No	No
Hereditary History of: Nervous Disease	No	No	No	No
Hereditary History of: Insanity	No	No	No	Father's Uncle.
Hereditary History of: Fits	No	3 cousins	No	No
Cause given	----	----	----	----
Status epilepticus	No	No	No	Once
Chronic Bodily Disorder	----	----	----	----
Delusions	of Grandeur		Of persecution	
Notes		Died 9 days after admission.		Died in status 3 yrs. after admission.

M a l e s.

Number.	182.	183.	184.	185.
Age	46	21	28	19
Attack	1st	1st	1st	1st
Suicidal	No	No	No	No
Dangerous	Yes	Yes	Yes	Yes
Mental Condition	Mania	Dementia	Mania	Dementia
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	38	11	3½	5½
Age, 1st attack of insanity	46	20	28	15
Hereditary History of: Drink	No	Mother	Both grand-fathers	No
Hereditary History of: Consumption	No	No	No	No
Hereditary History of: Nervous Disease	No	No	No	No
Hereditary History of: Insanity	No	No	No	No
Hereditary History of: Fits	No	No	Cousin	No
Cause given	----	----	----	----
Status epilepticus	No	No	No	Yes
Chronic Bodily Disorder	----	----	----	Died in status
Delusions	Of religious natures			Of religious nature.
Notes.				

Males.

Number.	136.	137.	138.	139.
Age	12	17	52	45
Attack	1st	1st	1st	1st
Suicidal	No	No	No	Yes
Dangerous	No	Yes	No	Yes
Mental Condition	Imbecility	Idiocy	Melancholia	Mania
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	3½	1	52	44
Age, 1st attack of insanity	3½	1	52	45
Hereditary History of — Drink	No	No	No	No
Hereditary History of — Consumption	No	No	No	Father
Hereditary History of — Nervous Disease	No	No	No	Mother (stroke)
Hereditary History of — Insanity	No	No	No	No
Hereditary History of — Fits	No	3 brothers	No	Mother
Cause given	----	----	----	Alcoholic Epilepsy
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	Phthisis	Phthisis	----	----
Delusions	----	----	Of religious nature	Of persecution.
Notes	Died from phthisis 2 yrs. after admission.	Died from Phthisis 2 yrs. after admission.	Discharged recovered in 6 months.	Discharged recovered in 2 months.

Males.

Number.	190.	191.	192.	193.
Age	26	28	30	31
Attack	1st	1st	1st	1st
Suicidal	No	No	No	No
Dangerous	Yes	No	Yes	Yes
Mental Condition	Melancholia	Dementia	Melancholia	Mania
Bodily Condition	Fair	Fair	Fair	Fair
Epilepsy started	21	5	3	27
Age, 1st attack of insanity	26	28	27	31
Hereditary History of: Drink	No	No	Maternal Grandfather	No
Hereditary History of: Consumption	Paternal Uncle	No	No	No
Hereditary History of: Nervous Disease	No	No	No	No
Hereditary History of: Insanity	No	No	No	No
Hereditary History of: Fits	No	No	No	No
Cause given	Business worry	----	----	Head Injury
Status epilepticus	Once	No	No	No
Chronic Bodily Disorder	----	Phthisis	Phthisis	----
Delusions	Of persecution			Of persecution
Notes	Discharged recovered 1 yr. after admission.	Died from Phthisis 8 months after admission.		

Males.

Number.	194.	195.	196.	197.
Age	29	25	74	33
Attack	1st	2nd	1st	1st
Suicidal	No	Yes	No	No
Dangerous	No	Yes	Yes	Yes
Mental Condition	Dementia	Dementia	Dementia	Dementia
Bodily Condition	Fair	Fair	Feeble	Fair
Epilepsy started	13	23	66	15
Age, 1st attack of insanity	29	23	74	33
Hereditary History of: Drink	No	No	No	No
Hereditary History of: Consumption	No	No	No	No
Hereditary History of: Nervous Disease	No	No	No	Mother (stroke)
Hereditary History of: Insanity	No	No	No	No
Hereditary History of: Fits	No	No	No	No
Cause given	Fright	----	----	----
Status epilepticus	No	No	No	No
Chronic Bodily Disorder	----	----	----	----
Delusions	Of identity			
Notes		First attack of insanity came on with the fits and lasted only 24 hours.	Died of senile decay 1 yr. after admission.	

Males.

Number.	198.	199.	200.
Age	27	47	23
Attack	1st	1st	1st
Suicidal	No	No	No
Dangerous	Yes	No	Yes
Mental Condition	Dementia	Mania	Melancholia
Bodily Condition	Fair	Fair	Fair
Epilepsy started	13	45	17
Age, 1st attack of insanity	27	47	23
Hereditary History of: Drink	No	Father & Mother	No
Hereditary History of: Consumption	No	No	No
Hereditary History of: Nervous Disease	No	No	No
Hereditary History of: Insanity	No	No	No
Hereditary History of: Fits	No	No	No
Cause given	----	Grief	Drink and Head injury
Status epilepticus	No	No	No
Chronic Bodily Disorder	Heart Disease	Heart Disease	No
Delusions		Of persecution	
Notes		Discharged recovered 5 months after admission.	Discharged recovered 3 yrs. after admission.

EPILEPTIC INSANITY.

THE BLOOD OF THE EPILEPTIC.

PART II.

--------ooOoo--------

The Blood of the Epileptic.

It has often ocurred to me that in epilepsy there must be some qualitative change in the blood, possibly associated with some change in the red blood corpuscles.

Among the older writers on the subject of epilepsy some used to say that epilepsy arose from congestion and others from anaemia of the brain. Now many of these authors brought forth experimental proof of the truth of each of these statements. These seemingly incompatible statements become clearer if we consider that in both of these conditions there is a change in the amount of oxygen supplied by the blood to the brain, a decrease in its quantity, if not its quality also. Let us now consider whether there are any known diseases or changes in the blood associated with convulsions. First of all let us remember that in death from haemorrhage convulsions often occur.

Secondly in diseases where the blood contains some poison as in uraemia, and in poisoning by organic compounds as prussic acid and Santonin, there are often convulsions.

Furthermore in such conditions as ptomaine pois-

oning there are muscular twitchings and convulsive flexious of the fingers and hands. In Addison's disease also, where there is much anaemia, we sometimes meet with convulsions. Lastly in severe forms of malarial fever we meet with convulsions, coma, and other nervous manifestations. In this condition rigors are always present. Now rigor according to Victor Horsley, is a form of fit. I would further add that periodicity, which is such a marked symptom in malaria, is common in epilepsy.

It was the consideration of the foregoing facts that led me to examine the blood of my epileptic patients.

I first examined the blood in order to see if the number of red blood corpuscles was decreased. I found this not to be the case however. Nearly every epileptic I examined had 5,000,000 red blood corpuscles per cubic millimetre, several had many more.

Having satisfied myself that there was no definite decrease in the number of corpuscles, I examined the blood in order to find out the percentage of haemoglobin. I found the average percentage to be almost 72% of the normal amount of haemoglobin. I examined 50 cases to obtain this. I shall now give a list of them. They were all females.

The number of fits mentioned in the table is the number of fits during the month of examination.

It will be seen that the amount of haemoglobin does not seem to bear any definite relation to the number of fits.

Name	Age	Weight. Stones	lbs.	Number of fits.	Percentage of Haemoglobin.
E. F.	36	9		6	65
S. D.	24	8	6	14	70
L. R.	30	10	4	6	68
×S. L.	32	8	9	3	65
L. C.	33	7	3	1	80
E. P.	37	9	5	40	80
M. G.	41	9	2	8	70
L. J.	27	7	2	13	65
×E. M.	31	7	8	6	80
H. H.	28	8	3	6	75
G. W.	39	6	9	7	70
L. M.	32	8	12	9	75
M. A.	22	7	6	81	85
E. T.	33	8	12	0	75
M. G.	30	7	11	0	85
×R. A.	37	7	6	16	75
×A. S.	35	7	2	27	90
×G. S.	23	7		8	68
×A. H.	40	7	13	1	70
×J. W.	29	7	4	18	50
×E. F.	35	8	12	6	68
L. F.	25	7		4	30
E. H.	39	7	8	17	85
A. B.	20	8	10	10	80
×I. E.	39	8	2	16	60
×A. G. B.	24	6	13	12	70
B. A.	22	8	2	4	65
E. L.	24	7	8	26	60
E. G.	46	5	9	18	85
E. S.	24	6	7	5	65
M. A.	26	8		11	70
A. B.	24	7	10	28	60

Those patients marked × had a fit on the morning their blood was examined.

Name	Age	Weight Stones	lbs.	Number of fits.	Percentage of Haemoglobin.
✕E. R.	29	6	7	13	75
✕J. B.	43	8	7	28	80
✕M. H.	44	8	11	3	65
✕F. S.	20	7	12	5	75
✕E. R.	36	5	1	8	75
I. H.	21	9		34	60
M. I.	41	6	10	31	75
G. B.	40	8	12	4	85
A. K.	31	8	10	3	82
M. R.	28	9		10	75
M. F.	48	12	4	1	75
M. B.	49	11	12	7	70
M. R.	40	8	6	0	70
M. C.	25	7	13	4	80
M. K.	28	9	4	14	68
M. Mc.	26	12	8	16	70
M. B.	33	8	10	15	70
F. S.	36	9	7	6	85.

Those patients marked ✕ had a fit on the morning their blood was examined.

For a considerable time I have examined the blood of epileptics in order to see what changes, if any, occurred in the red blood in epilepsy. At the same time I examined the blood of non-epileptic patients as contrast cases. I have found certain changes occur in the blood in epilepsy and I shall now describe those changes. In thus examining the blood I have followed three different plans, firstly the examination of fresh unstained blood in a fluid condition, secondly the examination of dried or otherwise fixed blood films unstained, and thirdly the examination of dried stained films.

In all these three methods I have been able to find.

1. That there are changes in the red blood corpuscles.
2. That there are other bodies present in the blood.

I shall now proceed to relate the changes seen in fresh fluid preparations.

Having cleaned the patient's finger in the ordinary way, I make a small incision with a Graefe's knife and touch the drop of blood which escapes with a clean coverglass. Another coverglass is then gently put on the top of this, the blood spreading out in a thin layer, and a ring of vaseline is put round the free edge of the coverglasses. The blood thus prepared remains fluid for a long time.

Proceeding to examine such blood under the high power of the microscope, we find that the changes in the red blood corpuscles are as follows. Firstly, there are alterations in the size of the red corpuscles. Though the majority of the red corpuscles are of normal size there are many as small as blood plates and all sizes between that and the normal are present. Some of the corpuscles are larger than normal.

Secondly, there is often, but not invariably, poikilocytosis present. This change is more marked

in dried films as is usual in conditions were poikilocytosis exists.

Thirdly, the corpuscles are paler than normal and many seem to have a much darker and larger central part than in normal blood. Fourthly, many corpuscles present an appearance like a Saint Ignatius bean - as if they had a dark hollow centre. This may be an exaggeration of the normal concavity.

Fifthly, in or on some of the red corpuscles are seen round clear bodies about one micromillimetre in size. I have not been able to see these alter in shape or move in any way.

Sixthly, there are corpuscles present which have dark spots on them, often arranged round the periphery, but sometimes filling the whole corpuscles. The corpuscles affected are irregular in shape and the bodies themselves when seen under a higher power have the appearance of clean round particles. They are not of a fatty nature, not staining with osmic acid.

These globules have very often a faint blue colour.

Their appearance is shown in Fig. 1.a.

Seventhly some of the red corpuscles have a bright red body of a circular shape in their interior. This is not often seen, however.

Eighthly some of the red corpuscles when seen on side view present the appearance as if there was something occupying their normal concavity on one side as shown in figure 1.b.

Proceeding to the other bodies present in the blood we see that there are small round bodies of a blueish colour, which I do not believe to be blood plates, which occasionally are seen to be moving when every other body present is still. This, of course, may be due to the fluid currents present not being strong enough to move a more bulky body. Again there are round bodies occasionally to be seen, about one micromillimetre in diameter, presenting a dark spot in their centre as shown in figure 1.y. Sometimes these have a red or even a bright red colour.

If we take a fresh specimen of epileptic blood which has been allowed to dry (or even has been dried by heat) and then fixed in a mixture of equal parts alcohol and chloroform, we will see under the low power that some of the corpuscles have something in their centres, some have a dark spot others a clear spot.

If we now examine with the high power we find the corpuscles of a good yellow colour. Many of them

present a cup-like appearance and look like a St. Ignatius bean. Now in the centre of this cup-like appearance is sometimes a small pinkish refractile body. This body may exist without the cup-like appearance of the corpuscle, simply being fixed on the corpuscle. This pink body in other corpuscles is larger and has a dark spot in it. Other corpuscles show in their centres bodies with a more or less rounded shape some with defined outlines, others with ill-defined irregular outlines. Some of these are white bodies, others pinkish. Some of them present a sort of indistinct convoluted appearance. Others have dark spots like pigment in their interior. Some have a central spot seemingly of another nature, possibly a nucleus.

In proceeding to consider the changes seen in stained blood let me first say that I made a coverglass preparations in the usual way, and either fixed by gentle heat or in equal parts of alcohol and chloroform. I gave up the method of fixing by heat in order to note if the changes were present when the blood was fixed by other methods. I found that the changes seen were identical.

Before passing on to study the various stains

used in detail let me enumerate some of the changes I found by this method.

1. Many stains when used alone showed that the corpuscles presented clear centres. These clear centres by a combination of staining agents could themselves be stained.

2. Some corpuscles presented a granularity from small globule like bodies inside them.

3. Some corpuscles presented the appearance as if bodies were being discharged from them.

4. Some stains showed bodies of an irregular or sometimes sickle shape, and often with a differently coloured spot on them, apparently attached to the red blood corpuscles.

5. There were free bodies in the blood which stained in various ways.

6. In status epilepticus the red corpuscles present a degenerated appearance. I shall now proceed to describe the actions of the various stains I used. I shall describe actual specimens I possess.

One of the first stains I used was a saturated solution of Eosin. Having prepared the coverglass preparation I left it overnight in this solution.

On examining such a specimen with the low power the normal red corpuscles are seen stained red throughout. Other red corpuscles are seen with clear white centres. Sucn a condition is shown in Microphotograph 1. On examining with the high power many red corpuscles are seen breaking down. Others are seen with clear unstained parts in them. The unstained part as a rule occupies the centre of the corpuscle and is round. Sometimes it is sickle shaped at others it is half-moon shaped. Its outline is not always clearly defined.

Another appearance is shown in Fig. 2 No. 4.

Microphotograph No. 2 is from this specimen.

I next stained specimens with Erlich's Triacid Stain (Simon's Diagnosis page 69).

I used a rather strong solution, staining over night, and found that the red corpuscles were stained blackish, had clean spots of various shapes, very often had a central dark spot surrounded or not by a clear rim, and showed poikilocytosis.

Many of the red cells were breaking down. Some had granules in them.

If the specimen is not stained deeply and only for a few minutes the red cells stains greenish brown

and show central spots and sickles of a pink colour. The granules in the corpuscles also stain pink.

In one deeply stained specimen I noted reddish bodies apparently attached to the red corpuscles. These were round, oblong, half-moon or sickle shaped. Such corpuscles as the above are shown in Figs. 3,4, and 5, and in Microphotograph 4.

I next proceeded to stain the blood with Chenzinsky-Plehn's Solution (Simon's Diagnosis page 69).

Some of the red corpuscles thus stained showed blue centres which did not seem to be nuclei. Some of the corpuscles had a double outline as if there were something inside them. In their centres were often granules. These granules in some cases took the form of globules. The granules or globules were either clean, pink, or darkish in colour.

Many of the red blood corpuscles stained much more deeply in their centres than at their edges.

The second specimen I examined by the Chenzinsky Plehn's method happened to be from a case of Status Epilepticus and presented a remarkable appearance. It shows that in that condition many of the red blood corpuscles degenerate. On looking at this specimen with the lower power, the degenerated corpuscles appear

black and in this specimen seem in about equal number to the normal ones, which are stained pink.

On looking at these degenerated corpuscles with the high power they are seen to be of a very refractile coppery pink colour and are very granular. Many of the corpuscles which looked normal under the low power are also granular the granules appearing red on a pink ground.

The above conditions are shown in Figure 2 No. 5 and in Microphotograph No. 3.

I did not as a rule get satisfactory results with this stain so I combined it with other stains. I first combined it with equal parts of Loeffler's Methelene Blue. When thus stained for some hours the red blood corpuscles appeared either yellowish or greenish and had a differently coloured centre. The centre was either purplish or brownish. The appearance presented is shown in Figure 6.

I afterwards added to the Chenzinsky-Plehn solution more than the same quanity of Methelene blue. The result was that the red corpuscles were of a greenish colour and the irregular shaped centres were generally of a deep blue colour. Sometimes, however, they were pinkish or became so on drying.

Such a condition is shown in Fig. 7.

Microphotograph No. 5 is taken from this specimen.

I next combined Chenzinsky-Plehn's stain with equal parts of Carbolic Fuchsin solution.

All the red corpuscles stained a deep red. The affected corpuscles showed a refractile pink body in their centres or laterally.

This could be seen even with the low power.

The appearance presented is shown in Fig. 8.

I next used Malachowski's stain (The Parasites of Malarial Fever, New Sydenham Society 1894).

With this stain I got some very pretty specimens. The corpuscles stained blue and the central body pink, purple, or brownish.

Such an appearance is shown in Fig. 9.

I now come to one of the best stains and one which acts quickly. This stain is Picronigrosine in strong solution. If such a solution be put on the film side of a prepared coverglass and then after a minute or two quickly dried on blotting paper a beautiful result is obtained. The red corpuscles stain a fine yellow or greenish yellow. In their substance are seen the different shaped bodies before mentioned. These stain

brown, black, or in some cases remain clear. They often become coloured after the specimen has been kept a day or so.

This method of staining shows the various shapes assumed by the central masses very well. These are found to be round, oblong, sickle-shaped, half-moon shaped, or ring shaped in different corpuscles. The ring form is especially well marked and is very evident.

It was in a specimen stained with Picronigrosine that I came across a most curious and suggestive appearance. So suggestive was it that I dared to call it spore formation.

The red corpuscle is much enlarged. It stains yellowish-green but round its periphery and at other times over its surface or sometimes in both these places are white round or egg-shaped masses. Besides this appearance there were other egg-shaped bodies present. These were about 2 micromillimetres long and had a yellowish or brown colour quite distinct from the colour of the corpuscle. They were either free, scattered round these affected corpuscles or in some cases they seemed to be either in or on the corpuscles themselves.

This appearance is shown in Fig. 12.

Other examples of the action of this stain are seen in Figs. 10 and 11.

I now come to an excellent stain namely, a 1% watery solution of Aniline Blue Black.

A specimen stained with this stain for a few minutes shows all the changes shown by the other stains. It is perhaps the most convincing of all the stains. This arises from the fact that the bodies are so well seen. The corpuscles stain blueish black and on them exceedingly well shown are these sickle-shaped, round, or half-moon shaped bodies which take on a bright red or reddish brown colour and are somewhat refractile. They stand out extremely well from the Dark corpuscle. Some of these bodies have a blue spot on them which looks like a small nucleus.

In a specimen stained like this I noticed that some of the red corpuscles had the shape of a kidney and at the hilum there seemed to be a faintly slate coloured mass leaving the corpuscle.
The appearances given by this stain are shown in Figs. 13 and 14.

I next come to a stain I made up for myself. In it I combine Dablia, Safranin, Aurantia, Ether, alcohol, and water. I made it up haphazard one day and did

not take the exact quantities used so I cannot give them. However, it resulted in a very good stain. If the specimen be stained overnight in this the red corpuscles become purplish and the bodies orange brown. The result is very pretty. If the specimen be stained for only a few minutes, washed, and dried on blotting paper the result obtained is different. The corpuscles stain a brownish green and the bodies a pinky-white colour. The bodies are, perhaps, better seen when the specimen is thus prepared.
The appearances thus presented are seen in Figs 15. and 16.

I now wish to point out a peculiar appearance which results from the use of a 1% watery solution of Safranin. Where this is used to stain epileptic blood the red corpuscles lose their colour and become white but scattered over their surface are pink or red granules and in their centres a dark red mass. Such appearances is seen in Figs. 17 and 18.

I shall now give extracts from notes I made on the blood of some of my patients.

E. L., Epileptic maniac, aged 22. At present suffering from a Gastric Epileptic attack.

Examination of ~~the~~ Blood by Erlich's Method.

Low Power. Leucocytes abundant. Red blood corpuscles breaking up.

High Power and Oil Immersion. Red Blood corpuscles of normal size as a rule but some smaller ones are present. Poikilocytosis is not marked. A few nucleated (?) red corpuscles are seen. Red corpuscles with clear centres in fair amount. Of the Leucocytes only two in the whole specimen were Eosinophiles. Neutrophiles are present, some of exceedingly large size. No Basophiles. Lymphocytes are in excessive number. Very few blood plates are seen.

M. E. S., aged 17. Blood taken while in Status Epilepticus.

I. Chenzinsky-Plehn's Solution.

Low Power.

Two sorts of corpuscles are seen - the normal corpuscle stained pink and a corpuscle stained brownish-black. Some of these latter seem to have a dark spot in the centre surrounded by a clear ring. Others have a clear centre. Leucocytes are present but not abundant.

High Power.

The dark cells are seen to be altered red corpuscles. They are now seen to be stained a sort of coppery pink, some being nearly yellow. They seem to have darkish particles in them, which make them look black on focussing up. A sort of network may be seen in some of them.

Some have a clean refractile granule occupying almost the whole cell. There is marked crenation and poikilocytosis present. In other parts of this specimen there are red corpuscles stained pink with these darkish granules in them, and you can see them in all stages up to that where the cell looks blackish-brown altogether.

By oil immersion these corpuscles are found to be granular, dark black granules appearing. On focussing down we find that these dark granules become clear globules. The corpuscle is simply full of them at the worst, but in some corpuscles you can see the process beginning at one side of the cell.

Very few white corpuscles or blood plates were seen in this specimen.

2. Erlich's Method.

Examined with oil immersion leus. Marked poikilocy-

tosis is seen, also crenation. The corpuscles suffering thus have nearly all pale centres.

The granularity of the red cells is not so marked but is to be seen.

As regards the white corpuscles there are more neutrophiles present than lymphocytes. No other forms were seen.

J. P., aged 36. A case of epileptic stupor with few fits.

1. Erlich's Method.

Low Power.

Small red corpuscles with clear centres seen. They are somewhat amaller than normal. Normal red corpuscles present quite unstained throughout.

Average number of leucocytes present. Some large neutrophiles are present.

High Power.

Red corpuscles seem smaller than normal. Clear centred corpuscles seen. There are some corpuscles darker and larger which seem to represent the granular corpuscles seen in the last case. Poikilocytosis and crenation are not marked. The only whites present are lymphocytes and neutrophiles and there are few of these.

E. C., aged 22. Blood taken while in a state of epileptic mania occurring during dementia.

1. Chenzinsky-Plehn's Method.

Low Power.

Leucocytes in excessive numbers. Lymphocytes also in large numbers. Red corpuscles darker than usual.

High Power.

Size of the red corpuscles is average but some of the granular ones are larger than normal. Poikilocytosis is not marked. Granularity of the red corpuscles is general. In some there are dark granules round the periphery. Some of the red corpuscles have clear globules in them. There are some double outlined forms with a central dark spot. Some of the leucocytes have dark granules in them. Eosinophiles present in more than usual numbers.

S. A. H., aet 43. Epileptic dement.

1. Chenzinsky-Plehn's Method.

High Power.

Size of the red corpuscles is larger than normal. There is little or no poikilocytosis.

The red corpuscles are granular. Some are pale and not granular, or only slightly so and are almost filled with a faintly blue round body with a dark spot

in its centre. This leaves a clear rim at the edge of the cell. There is a double ring form with a pale blue centre.

Some of the red corpuscles are very pale and finely granular. There is one corpuscle with a tri-radiate star in its centre. There are a few red corpuscles which seem to have a blue mass extending.

There are small fine refractile bodies about one micromillimetre in size.

A. W. B., aet 23. Epileptic dement.

1. Erlich's Method.

Low Power.

Most of the red corpuscles have clear centres some have not. There are few leucocytes.

High Power.

Some of the red corpuscles have dark outlines, then a clear ring with a bright red body in it. Some of the red corpuscles have something extruding. One is seen with a red body extruding (or intruding?).

There are some pale red corpuscles with clear bodies in them. Some have two clear bodies. Many corpuscles have black granules.

There is marked poikilocytosis. In this specimen the effected cells are larger than the normal ones.

2. Chenzinsky-Plehn's Solution.

Low Power.

Red blood corpuscles more or less clear centred.

Leucocytes very few.

High Power.

Red corpuscles show pink granules, one or two in each.

3. Carbolic Gentian Violet.

High Power.

Many of the red cells show central darkly stained spot, some two.

4. Safranin.

Low Power.

Most of the red corpuscles have clear outlines and dark centres. Many have a central darkly stained part.

High Power.

Red corpuscles very granular. Red granules on clear cells which have dark centres are seen. There is some poikilocytosis. In some of the cells with dark centres is a still darker central part.

There are some curious white conglomerate bodies like raspberries, made up of clear round bodies each about one micromillimetre in size. In some of these little bodies are dark central spots.

In some of the red cells are bodies shaped like fig leaves or like sycamore leaves.

There are many dark brown blood crystals in this specimen.

E. T., aet. 26. Epileptic dement.

1. Blood stained with Bismark brown (Alcoholic) plus Aurantia (Alcoholic). Afterwards washed in saturated watery eosin for one or two minutes, and immediately washed in water.

Low Power.

Very indistinct. Some corpuscles stained darker than others. Eosinophiles seen with large deeply stained nuclei.

High Power.

There are some refractile bodies staining orange brown or remaining unstained, about one micromillimetre or more in size. There are some faintly pink red corpuscles with small brown central spots.

Sometimes these brown bodies appear round the edges of the corpuscles. Some of the corpuscles show a large central orange brown body.

There are red corpuscles (or bodies of the same size and shape) pale in colour and full of granules. Poikilocytosis is not marked in this specimen.

2. Safranin.

Low Power

Red corpuscles seen with black spots in them.

There are many bright pink leucocytes with black spots in them

High Power.

Red corpuscles are clear, refractile, granular, and full of globules, white, pink, or black. These become brighter on focussing up. There is often a larger pink body in the centre. Many fine bodies are seen both black and clear.

3. Erlich's Method.

Low Power.

Most of the corpuscles are dark centred some are clear centred.

High Power.

Most corpuscles seen are normally dark throughout Some have a clean white centre. Some show dark spots, not generally central. On careful focussing this often turns into a globule with a pink tinge. There are some pale kidney-shaped corpuscles with pink globules at the hilum. There are a few corpuscles with clear leaf-shaped bodies in them.

Many of the corpuscles have a double outline.

Some of the pink central parts have dark spots on them.

Some corpuscles show half-moon or sickle-shaped pink or colourless masses in them.

4. Chenzinsky-plehn's Method.

High Power.

Corpuscles are seen to be pink, granular, refractile. The granules are clear pink or dark. Some of the red corpuscles are pale blueish in colour.

5. Aniline Oil Gentian Violet.

High Power

The red corpuscles are granular and have dark spots on them.

M. R., aet 45. Epileptic dement.

I. Loeffler's Methelene Blue.

High Power.

Very good specimen. Red corpuscles stain faintly blue and in their centres are seen convoluted bodies and granules. There are many blood platelets present staining blue. There are also clear refractile bodies about the same size, unstained. This is one of the best specimens I have seen of bodies in the centre of the corpuscles.

2. Special Stain.

High Power.

Corpuscles all have dark granules in them. Some corpuscles have granular bodies almost filling them. There seem to be many clean bodies present about the size of red corpuscles.

3. Melachowski's Stain.

High Power.

Corpuscles stained blue with pink central masses. Some clear bodies are seen free. Some of these stain somewhat pink.

G. M. S., aet 25. Epileptic Circular Insanity.

I. Picronigrosin.

Corpuscles of a larger size than normal. Poikilocytosis marked. Red corpuscles show crescentic or dumb-bell or round bodies inside, generally central. These have a pinky white mother-of-pearl-like look. On focussing up many become smoky-black. White corpuscles with nuclei of a greenish tint are beautifully shown.

2. Chenzinsky-Plehn's Method.

Corpuscles greenish. Many show a cupped bean like appearance. Some have a pink round refractile body attached(?). Some have a round paler spot.

M. G., aet 24. Epileptic dement.

Chenzinsky-Plehn's Solution plus Methylene Blue.

Nearly all the corpuscles show changes. The red corpuscles stain brownish and the bodies have a purple colour. The white corpuscles stain bright blue. The blood plates stain blueish-green. As a rule we see the purple bodies in or on the brownish corpuscle, but in a deeply stained specimen the corpuscles are green and the central body deep purple. The effect is very pretty. Some of the bodies are seen in the form of a ring. There is one body seemingly leaving the red corpuscle.

Finally what is the meaning of all these changes? Are they the cause or the result of the epilepsy or are they accidental? Are they due to changes in the blood after it leaves the body?

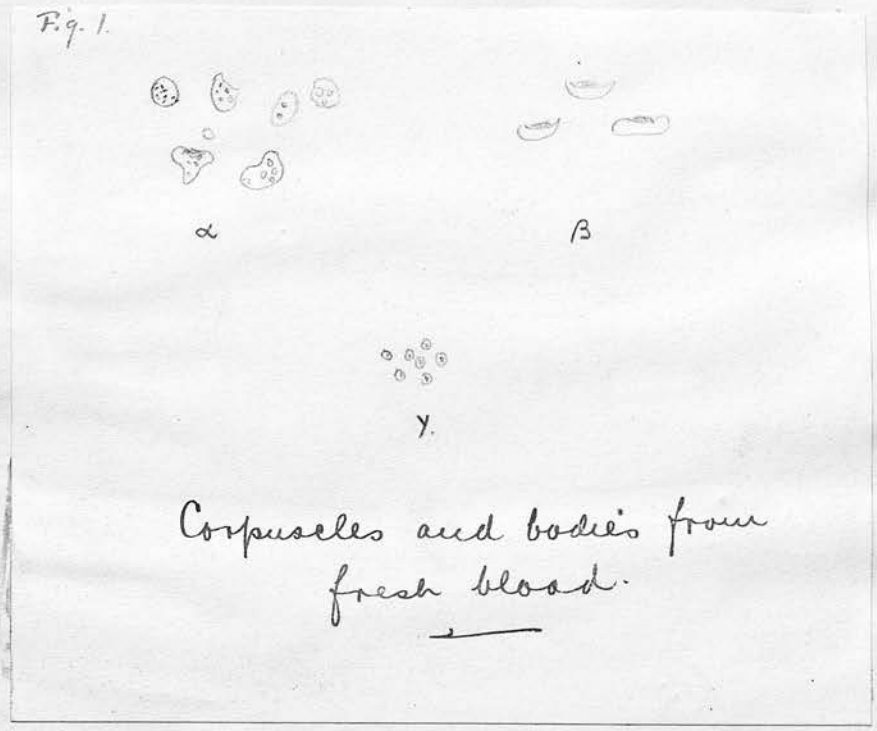
Though some of the changes mentioned may possibly be due to changes in the blood after it leaves the body, or to faulty manipulation, or to the action of the staining reagents, I do not think it possible that they all are so caused.

As to whether they are the cause or result of epilepsy it will require time and experiment to show. But the peculiar changes seem quite compatible with

the former theory. The fact that I did not observe these changes in any of the test contrast cases seems to show that they are not accidental.

Can it be possible that these changes partake of the nature of a blood parasite? Whatever be the cause, nature, and result of these changes, I believe that they have some definite connection with the neurosis epilepsy.

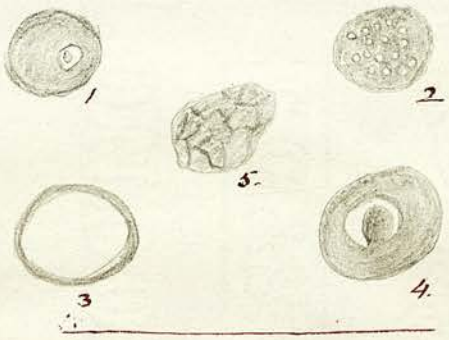
Fig. 1.



Corpuscles and bodies from fresh blood.

Fig. 2.

Fig. 2.



1. Clear body with central spot attached to corpuscle
2. Vacuolation of corpuscle
3. Typical look of a corpuscle from a case of Epilepsy
4. Form commonly seen
5. Degenerated form from a case of Status Epilepticus

Fig. 3.

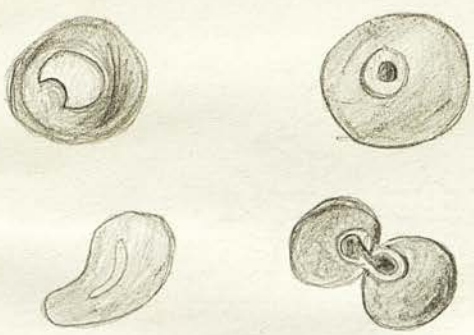
Fig. 3.

Red Cells from a case of Sporadic Epilepsy
Stained with Ehrlich's Triple Stain.

1. Red cells breaking down showing a, Shadow cell.
2. The ring form of red corpuscles.
3. Granular red corpuscles.

Fig. 4.

Fig 4.



From a case of Epileptic Imbecility.

Altered red cells showing central mass stained pink and the rest of the corpuscle greenish-brown.
Stained with weak Ehrlich Triple Stain.

Fig. 5.

Epileptic Blood deeply stained with Ehrlich's Triple stain. One of the reddish bodies attached to the corpuscles has a blue spot on it.

Fig. 5.

Fig. 6.

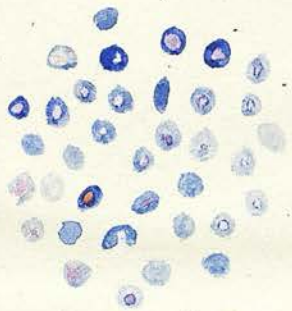
Fig. 6.

Epilepsie — Blood.

Stained with Chenzynsky-Plehn solution plus an equal quantity Loeffler's Methelene Blue.

Fig. 7.

Fig. 9.



Epileptic Blood.

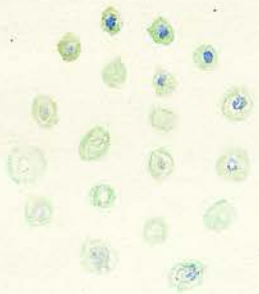
Malachowski's stain.

Fig. 8.

Epileptic Blood stained with Cheuzinski-Plehn's Solution + equal part Carbolic Fuchsin Solution.

Fig. 9.

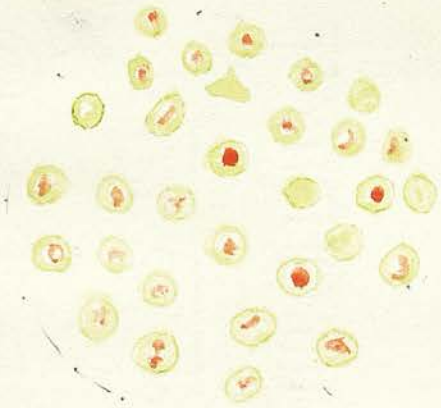
Fig 9.



Epileptic blood stained with Cleungmaky-
-Pleh's solution + more than ½ some
quantity Loeffler's methylene Blue.

Fig. 10.

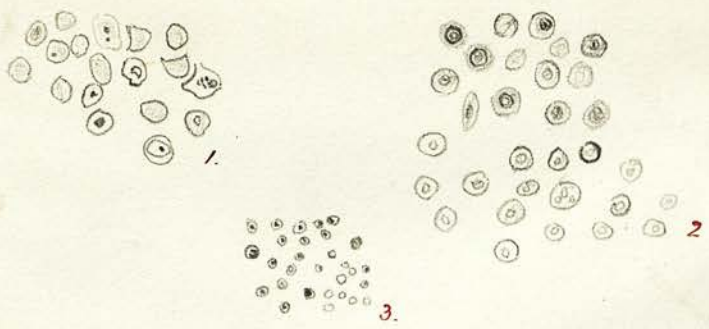
Fig. 10.



Epileptic Blood from a specimen stained with picronigrosin.

Fig. 11.

Fig. 11.

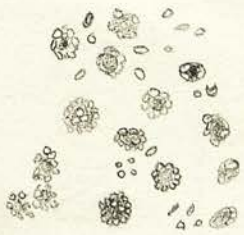


Specimen stained with Picronigrosin

1. Yellow stained corpuscles showing black spots.
2. Advanced stage: Larger bodies inside
3. Low power view of the Smear.

Fig. 12.

Fig. 12.



Red Corpuscles from a case of Epileptic Dementia

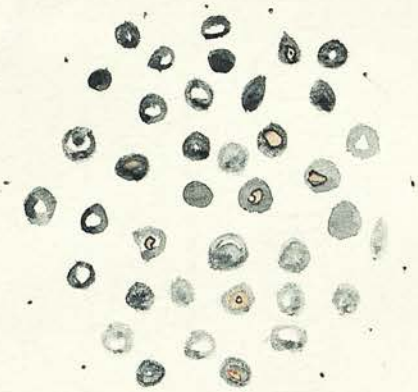
Spore Formation in Red Corpuscles

Free bodies are seen between the corpuscles.

Stained with Picro-nigrosin.

Fig. 13.

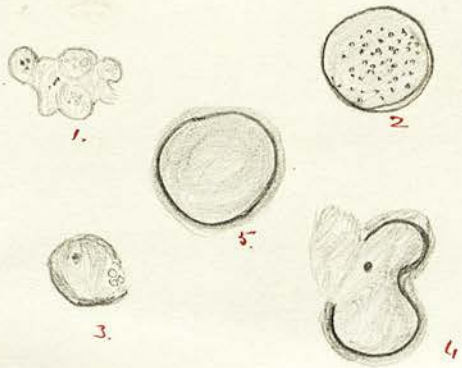
Fig 13.



Blood of an Epileptic Dement stained with Aniline Blue Black showing bodies attached to corpuscles

Fig. 14.

Fig. 14.

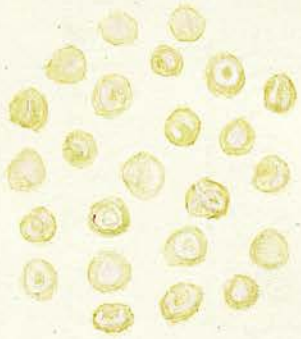


Red cells from a case of Stuporous Epilepsy
Stained with Aniline Blue Black 1%.

1. Massed fused red cells with bodies inside.
2. Red cell presenting granular appearance.
3. Clear bodies in cell on the point of being absorbed.
4. Burst cell.
5. Double outlined red corpuscle.

Fig. 15.

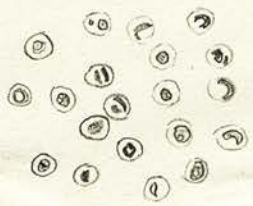
15.
Fig.



Epileptic Blood stained with a weak solution of my special stain.

Fig. 16.

16. Fig.

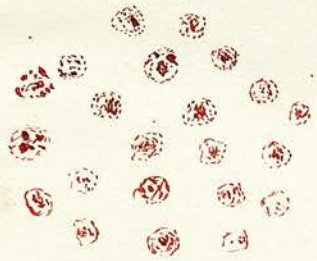


The Parasite of Epilepsy

From a specimen stained with my Special Stain for 5 minutes. Washed + dried on blotting paper. The parasite stains purplish on yellow corpuscle.

Fig. 17.

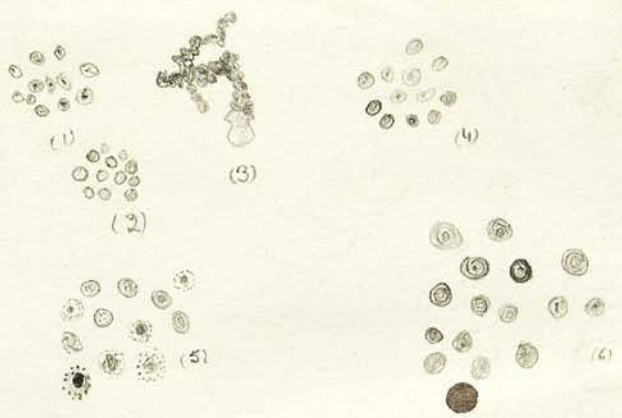
Fig. 17.



Curious appearance shown by epileptic blood stained with Safranin.

Fig. 18.

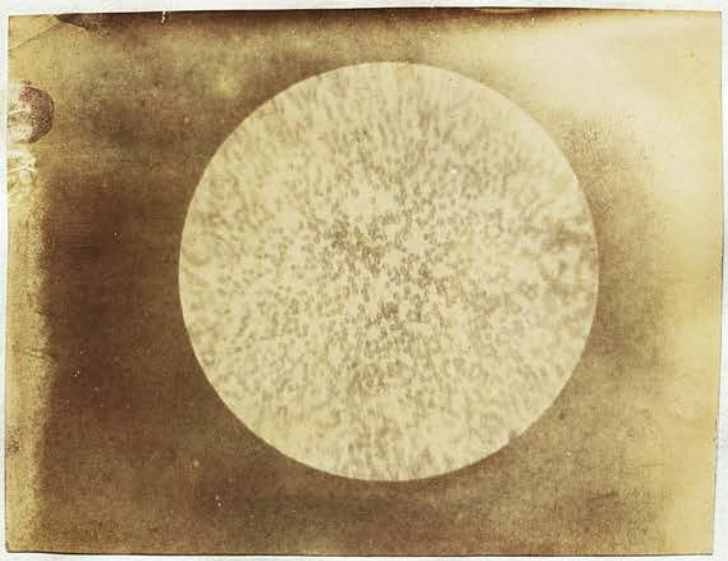
Fig. 18.



Safranin Staining

1. Epileptic corpuscles seen by low power.
2. Normal corpuscles from the same specimen.
3. Fusion of corpuscles from the same specimen.
4. Normal blood under safranin.
5. Epileptic blood high power.
6. Normal blood high power.

Microphotograph No. 1.



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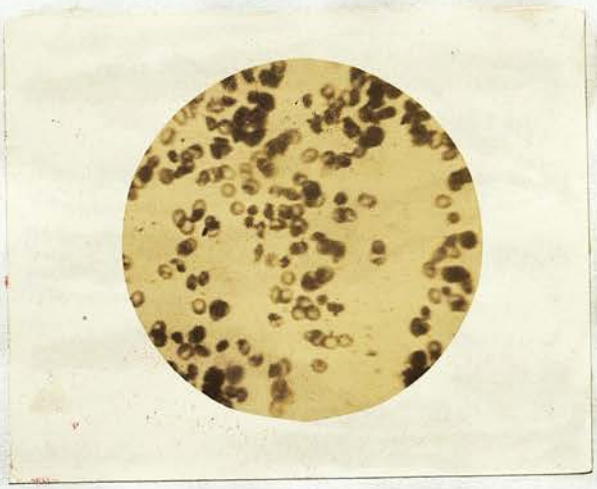
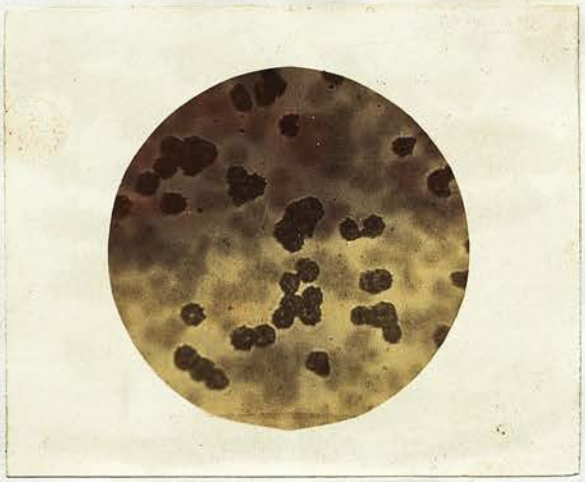


Microphotograph No. 3.



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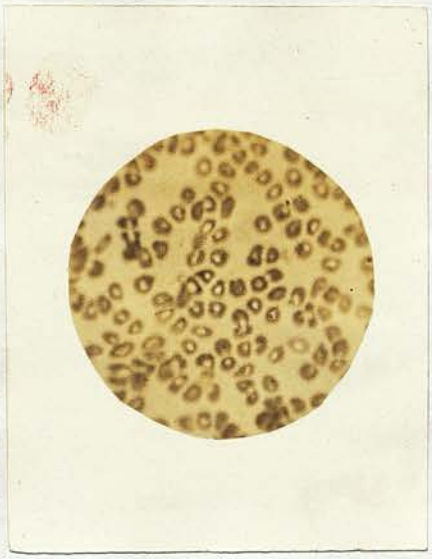

Microphotograph No. 5.