T H E S I S

on

The Course and Etiology of Epilepsy with Clinical Observations on the Blood and Urine of 100 Cases

For the degree of M.D.,

by

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I N D E X.

P R E F A C E.

I have endeavoured in the introduction to define true idiopathic epilepsy as distinct from hystero-epilepsy and the other so-called epilepsies. Apropos of this the next section deals with the clinical groupings of the disease from the points of view of severity, time, and incidence.

The sections dealing with the examination of the urine and of the blood are preceded by a resumé of the etiology which naturally has an important bearing on the clinical aspect in the light of the theories which associate so positively rickets and kindred conditions with epilepsy.

I have to acknowledge my gratitude to DR. NEIL T. KERR for all his courtesy and for letting me have every facility in my examination while I was his Pathologist at the Lanarkshire District Asylum.

I am also indebted to DR. EDWIN BRAMWELL for his many kind suggestions on the subject of references.

I N T R O D U C T I O N

In spite of the enormous amount of clinical work expended for many years on the study of epilepsy, we have still to acknowledge ourselves practically in the dark with reference to the causation and pathology of this disease.

No harm can be done by adding to the heap of material on the subject a few conceptions, supported by clinical observations, of epilepsy from a series of some 100 patients, some 70 of whom were insane.

The insanity of the subjects does not, in most cases interfere with the value of the observations, the term "epileptic insanity" being misleading in as much as this condition is merely an accidental combination of insanity and epilepsy. If there should be any complication arising from the mental condition, e.g. a gross cerebral lesion or a febrile state in addition to the epilepsy, special reference and allowance will of course be made.

It is only in institutions such as asylums and homes for epileptics that an accurate record of all the seizures grand or petit

can/

can be obtained and, as these records are necessarily a most important factor in considering the course and classification of the disease, it is difficult to see how one can avoid insane cases and at the same time examine large numbers clinically.

Even these statistics may be misleading. There is so often a hysterical tendency in even the most classical cases that such terms as "epileptic hysteria" and "hystero-epilepsy" have for long been current, the latter greatly used by Charcot. (1), is still very commonly used. Gowers (2) prefers the term "hysteroid" - proposed by Roberts (3) - and the term, though convenient, does not get us much further on.

It must be confessed that it is not so easy to separate these convulsions from true epilepsy as might at first appear. They occur time after time in genuine epileptics and present so many of the appearance of true fits, that in many statistics of epilepsy they have been included. Again many epileptics are remarkably responsive to mental suggestion, and in other cases true fits are preceded or followed by hysterical manifestations.

A case in point:-

A boy aged 8 years, father neurotic, endowed with the precocity so

common in epilepsy, and more anxious to conceal than to parade his symptoms, was in the habit of taking major fits two or three times in a month, these were interspersed with rather severe minor attacks and in addition with some that might be characterised as hysteroid. His condition was aggravated by any excitement, the chief of which was seeing visitors (living as he did in the country). If one should see him in the morning before such a visit, and, after listening for a short time to his perfectly normal heart or lungs, tell him definitely that he would have no "turns" that day, he would have no hysteroid attack and any minor one would be modified. Such mental suggestion had no effect on his major attacks.

Another difficulty that presents itself in dealing with epilepsy is the definition of the name. This has been complicated chiefly by the loose attempts to embrace under the term "epilepsy" all sorts of convulsive seizures. Thus we have alcoholic, syphilitic, cortical, cerebral, hemiplegic, saturnine, traumatic, and a host of other epilepsies./

epilepsies. If we adhere to these expressions we must cease to consider epilepsy as a disease and look upon it merely as a symptom. In what follows the cases dealt with, are of the classical type, true idiopathic epilepsies corresponding to those autopsies where there is no definite causative lesion. If a man takes fits because he has some specific lesion in the region of his cortex and if another is convulsed from the pressure of a fracture of the skull in the same area, it seems foolish to categorise both those conditions with cases where we have periodic attacks of the classical epileptic type, where neither during life nor after death can we get - strive as we may - any pathological evidence to refute the term "idiopathic."

Some authorities go the length of discussing other epilepsies most of them evidently visceral, these are such as gastric, intestinal, pleural, uterine and many others.

A man may cough because he has got phthisis and another because he has a crumb in his throat, but, because there is in both cases a reflex action producing the same external manifestation, we cannot go the length of classifying the two complaints together. So it is with the taking of fits. It is generally agreed that fits result from cerebral cortex irritation, and that this may

result/

result from the reflex stimulation of morbid conditions of any part of the body, e.g. convulsions associated with worms, phimosis, teething, pleurisy, gastritis, uterine mischief and many other disorders. If also toxins such as lead and alcohol, to say nothing of certain drugs, can bring about fits, it is surely better to keep "idiopathic epilepsy" as a definite entity. Doing this, the search for the cause, be it toxin or not, will be on definite lines and proportionately the more satisfactory.

It is beyond the scope of this work to criticise and compare the innumerable contradictory results published from time to time regarding the abnormal constituents of nearly every tissue and secretion of the body, many of these, however, will be referred to in their own departments.

COURSE AND FORMS OF EPILEPSY.

It is doutbful whether the course of the disease is more interesting on account of the irregularity of the seizures with regard to their type, time, or frequency, or on account of the regularity in these respects.

It is certain that this irregularity makes classification infinitely harder.

Clinically we have to look for lines on which to classify the disease and we may, in the meantime satisfy ourselves with three:-

A. With regard to type,
   (I) Major.
   (2) Minor.

B. With regard to time of occurrence,
   (I) Nocturnal.
   (2) Diurnal.

C. With regard to frequency,
   (I) Serial.
   (2) Status Epilepticus.
   (3) Attacks at varying periods with years intermitting.

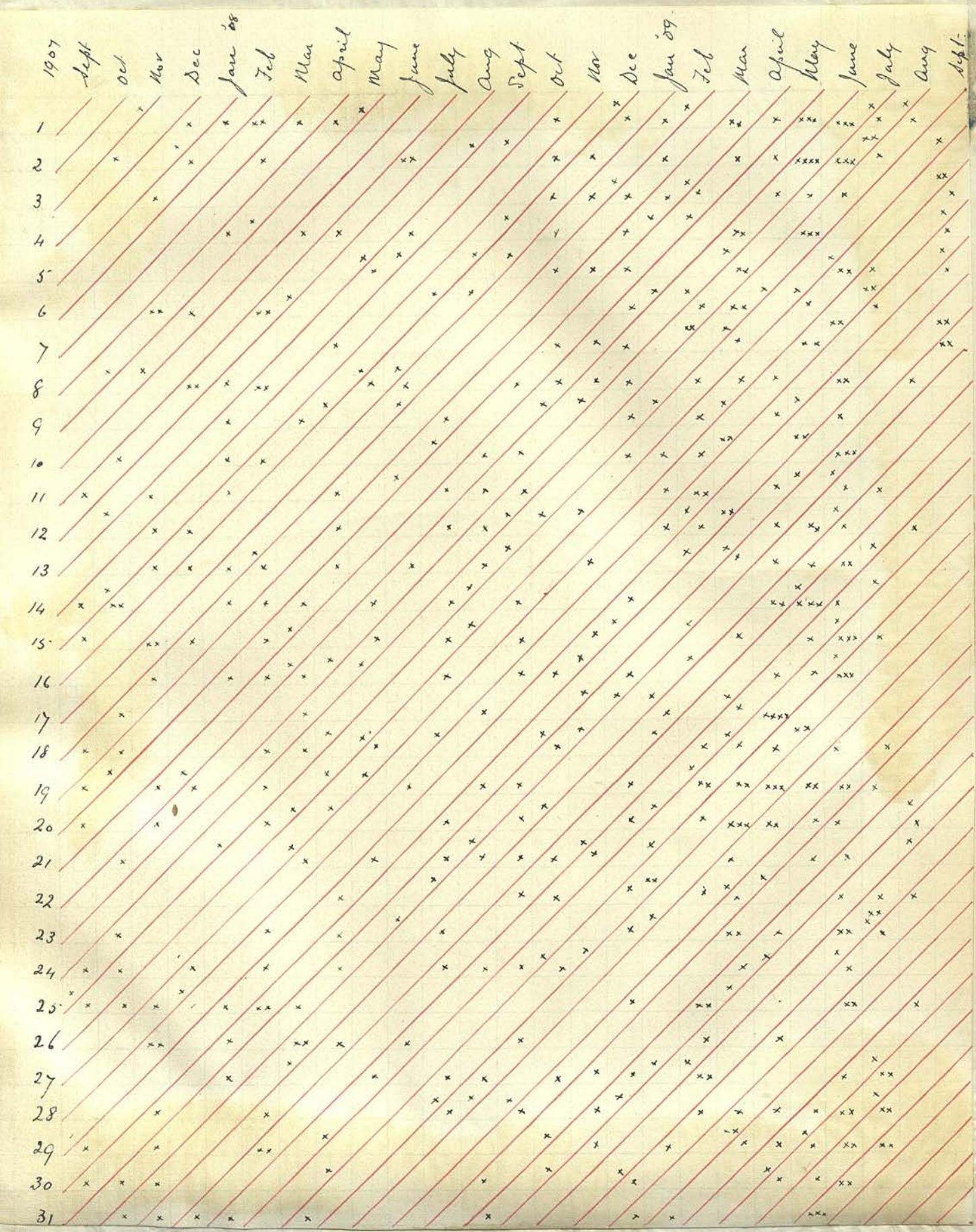
In order to represent these points more graphically than by mere writing, I have drawn up tables of the more typical cases. Each chart is constructed for two periods of twelve months. In some these are consecutive, in others there is an interval between the first and second twelve months. Every twenty-four hours is represented by a square and diagonally through this is a red line, above which the fits occurring between, 7 a.m., and 8 p.m., are individually registered, and below which, those fits occurring while the patient is in bed are similarly recorded. Major fits are represented by a cross, minor ones by a dot.

In spite of our having three bases for clinical classification of epilepsy we are faced with a difficulty in the first two (viz. - type of seizure and time of occurrence) in having only a very limited number of pure cases of each kind, the great majority being of a mixed variety, e.g., major chiefly with a few minor attacks, diurnal cases with some night seizures and vice versa. With regard to the third basis - that of frequency - the periods are in practically every case so irregular that it is more interesting than useful.

Before considering these bases of classification seriatim, an additional value of such charts may be demonstrated. At a glance one follows the progress of the case for better or for worse over an extensive period. Take for example No. (VI):- The increase in the number of fits for the second twelve months over the first is obvious, and on counting up we find that during the year 1902 he had 100 fits (major and minor) whereas during twelve months in 1908-1909, he had no fewer than 240 fits. Now to those who see him regularly there is no obvious change in this patient for the worse. He is still able to do labouring work for most of the day, and though rather deficient mentally I have it on good authority that there is no obvious retrogression in this respect. Physically and mentally, therefore, he shews little or no change, yet we have the certain evidence of statistics to shew us at a glance that his disease is progressing with no uncertain strides.

This is only one of many instances.

Chart I.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A. (I). MAJOR TYPE.

Two charts Nos. I & 2 illustrate this class; No. 2 is also incidentally of the Nocturnal type but that aspect will be referred to under its own heading. It can be seen at once, that No. I. is a severe case, as also that No. 2 is comparatively mild. Both patients are mentally deficient, but by no means demented, it is well to note, however, that in No. I. mental deterioration is progressing far more rapidly.

Perhaps the most interesting feature of this type is that many patients go on for year after year, with no increase in their number of seizures, in chart 2 for example, we even see a certain improvement during the last twelve months. In short, cases appear to be stationary over a great number of years, and we cannot take for granted, as Turner (4.) seems to do, that in every case of the major type the frequency of the attacks increases, and the mental attitude shows signs of further deterioration. I would hold, that there are a certain number of stationary cases where mental deterioration and increased frequency of attacks are not inevitable

Chart II.


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except in as much as decay and death are inevitable to humanity.

There is no type of epilepsy where the mode of termination is so uncertain as in the major type. Look for example at chart 3. This patient aet. 16 was a fairly acute, well nourished lad. His fits occurred in series once or twice a month, but were not specially severe. Just as we thought we were getting the better of the trouble on bromide and general hygienic measures (he had been practically two months without a bout) he took a major fit one morning, a severer one at night, and next evening he went into status epilepticus and died in twenty-four hours.

I have never seen, or seen described, any sudden change for better or for worse in any established case of the major type. It, however, seems pretty certain in many of these cases that the non-incidence or abortion of attacks causes the subsequent attack to be severer than ordinary. Perhaps the fatal termination in case 3 had some connection with this observation which does not seem to be emphasised in the literature. In some cases also where there is a long period between the seizures

Chart III.


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Death
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these are much more severe than the fits in cases where the intervals are short. The fits of the patient whose case is shown in chart 4, are the most severe individual seizures I have ever seen not accompanied by cardiac or renal disease, he, as will be seen, goes for 5 or 6 months at a time without a seizure.

Chart IV.


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A. (2). MINOR TYPE.

A very small percentage of patients demonstrate this type in its pure form and chart 5. shews the nearest to which our experience goes. As a matter of fact, having seen one attack marked by a cross - as a major fit - I saw that it really was a severe minor fit followed by a sort of rigor without any coma and those few attacks represented by crosses in this case, may, in reality, have likewise been exaggerated minor fits.

Unlike some writers we find the patients in this category more irritable, less attractive, and on the whole less intellectual generally than other epileptics.

The combined type of major and minor epilepsy might be dignified by a class of its own. There are a great number of cases in this class. Chart 6, referred to before as showing gradual cerebral deterioration, is one of many examples.

Chart 9, also shews a very common type in a mild, long standing case. The patient is 59 and has been epileptic since puberty.

Chart V.

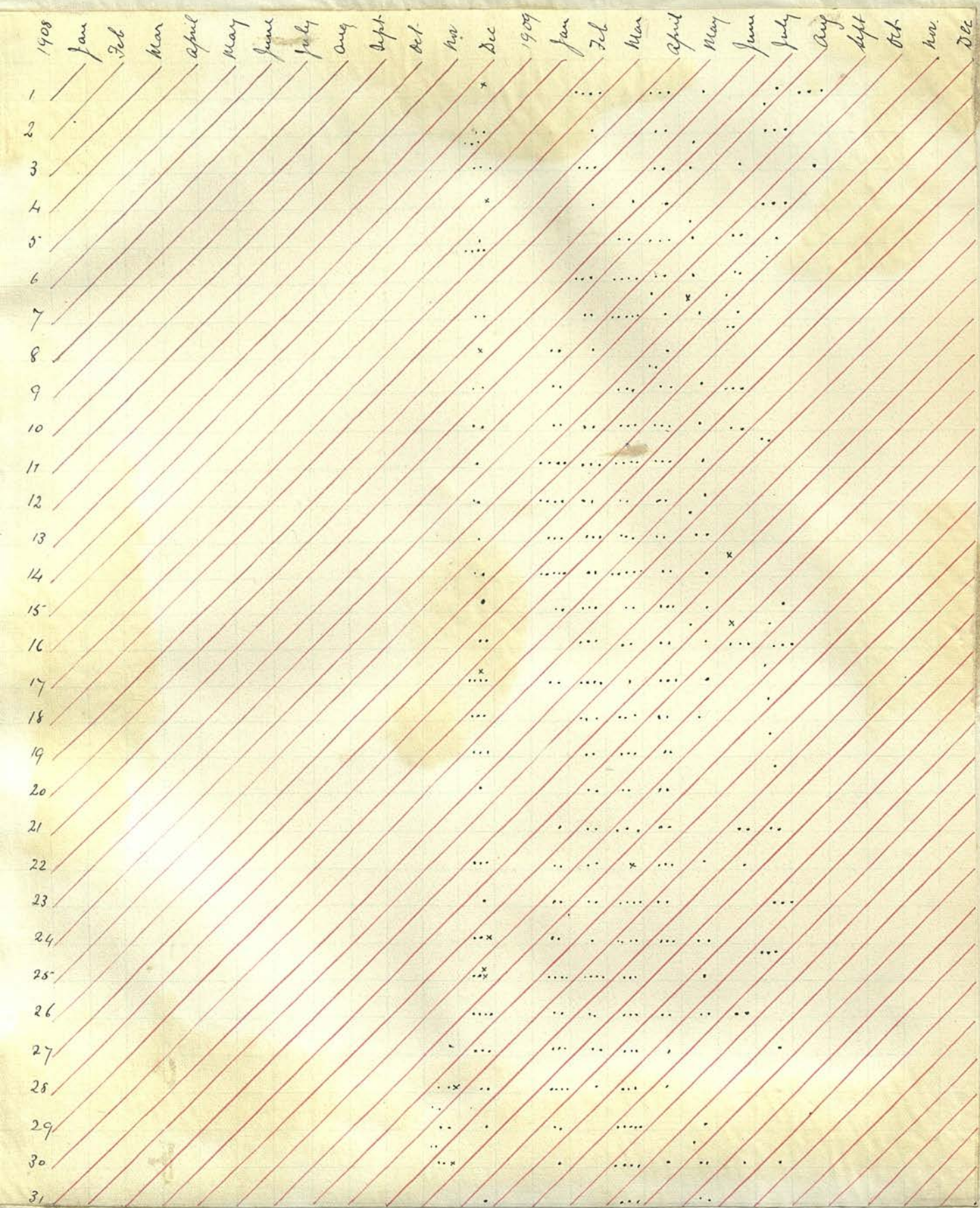
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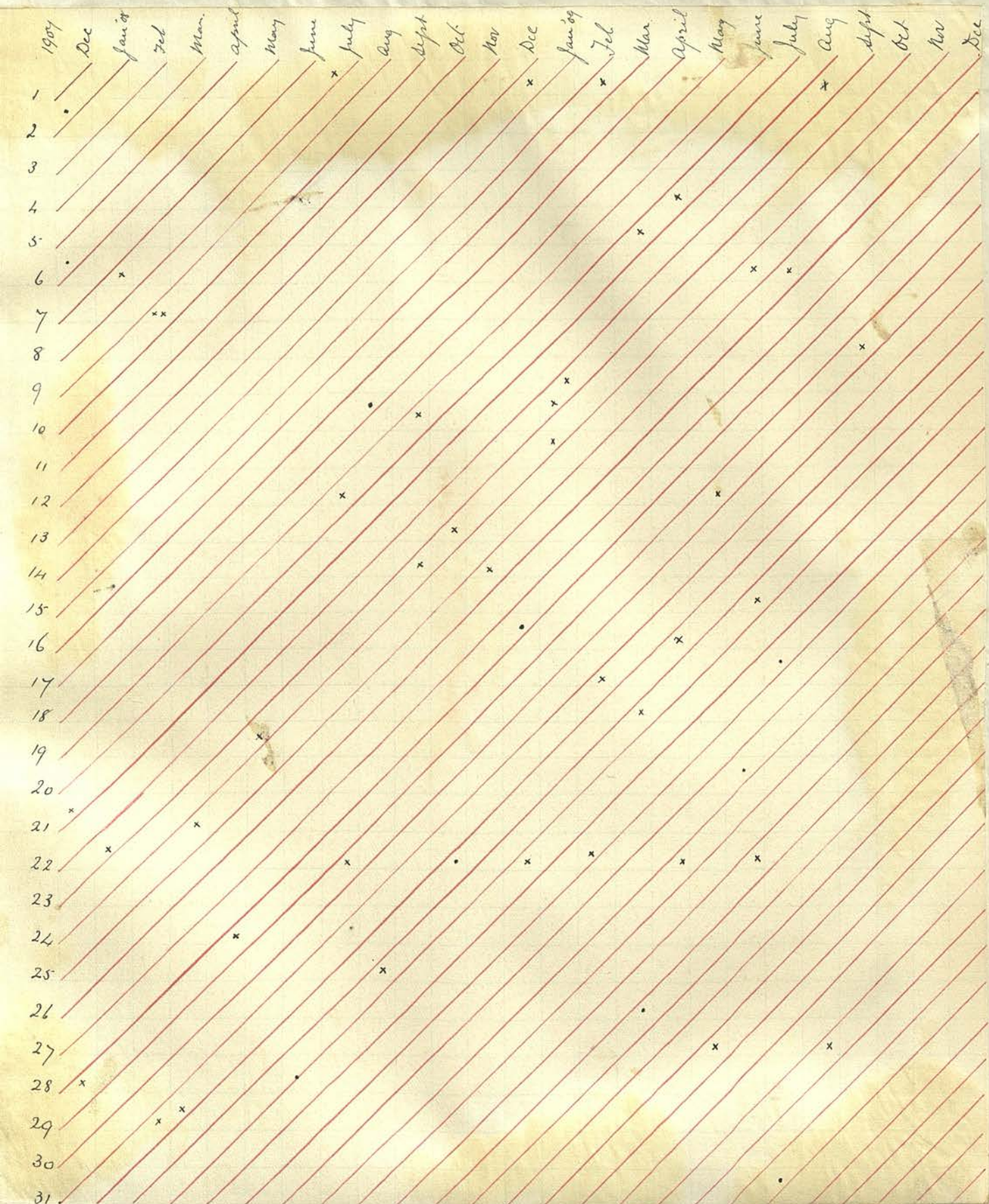
Chart IX.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B. (I). NOCTURNAL TYPE.

Chart 2. is an example of this class as well as being one of the Major Type. There are a very few fits here marked in the day-time but those occurred at times when the patient was confined to bed, and, therefore, do not spoil the nocturnal record. It is exceedingly interesting in this case to note the condition of the early morning urine. It is regularly much reduced in acidity and on boiling a phosphatic deposit is almost invariably thrown down. Such a state of affairs will be referred to later on in connection with the urine.

It is very common for these patients to have a fixed hour for their seizures, in one case 3 a.m., in another I.p.m., and so on.

B. (2). DIURNAL TYPE.

I have had no experience of this type in its pure form.

Chart VI.


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C. (I). SERIAL TYPE.

Under this heading are grouped those cases which from time to time over irregular intervals have a series of fits. Turner(4) describes it as a subacute form of epilepsy. Chart 4. shows a mild case of this type though it may be remembered that in this very case the fits were by no means mild. Chart 8. shows a much more severe case culminating in death in status epilepticus. Both major and minor attacks may be shown in these cases and the type generally merges into the next.

C. (2). STATUS EPILEPTICUS.

This type is usually fatal but there are cases where it becomes periodic, take for example the case represented by chart 7. During the first two months of 1907, it may well be imagined, this patient's condition gave rise to the greatest concern, on several occasions it being exceedingly doubtful that recovery could take place. Since then he has had one or two relapses into this most

Chart VII.


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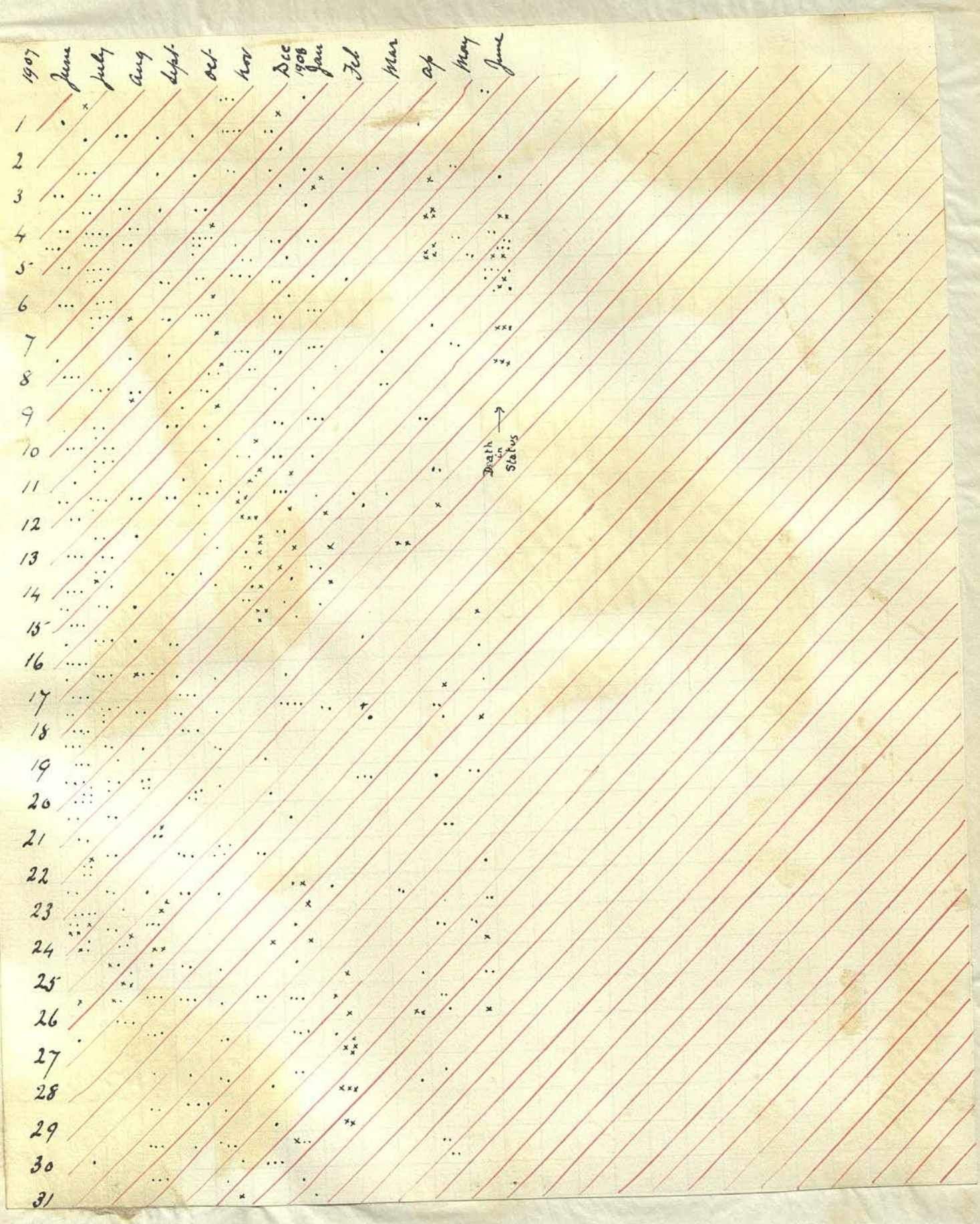
Chart VIII.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Death in Status

serious condition, viz., in March 1907, Dec. 1908, and Jan. 1909. These attacks, however, have not been so prolonged and have been interspersed with serial bouts which seem to act in accordance with the theory suggested above, as safety valves.

C. (3). CASES HAVING A FEW FITS ONLY.

Very many such cases might be cited. Probably these in many instances are not true idiopathic epilepsies, but due to some exciting factor, though an exceptional case might be mentioned. A woman aet. 30, perfectly healthy, married, pregnant at the time, fell down with a loud cry at an entertainment. On examining her we found all the symptoms of grand mal. She had passed urine involuntarily - had also bitten her tongue very badly. She never before had such an attack, in fact never had any fit of any sort previously. Since then, 18 months ago, she has been perfectly well. She is not the least neurotic. This is obviously a reflex seizure, probably pelvic in origin,

E T I O L O G Y

In order that any value at all may be attached to my observations especially those dealing with the differential examination of the blood it is necessary to indicate shortly the thoughts of certain undoubted authorities regarding the etiology of epilepsy. Such theories as those that connect Status Lymphaticus and Rickets with epilepsy must of necessity throw considerable light on deductions drawn from this branch of clinical observation.

Epilepsy being such a general disease, the statistics of different countries with reference to it vary considerably naturally, hence we cannot lay too much stress on its so called relative frequency. From what can be gathered, however, from several writers it is estimated that on an average two out of every thousand of the population are epileptic.

It is a disease essentially of early

life,- 75% of the cases arising under 21 years of age. Out of 1450 cases examined by Gowers(2) 422 developed the disease before the 10th year. And even these figures would be beaten, were it taken into account how many cases of supposed late idiopathic epilepsy may be traced in origin to some injuiry or to some local disease. I have found several such cases, which without close enquiry into the history could not possibly have been described as anything else than late idiopathic epilepsy, where after all, the convulsions were due to some local lesion. If then such suspects were omitted the percentage of cases originating below 21 years would be probably even greater.

Turner(4)says this is a period during which the development of the central nervous system and the maturation of the reproductive organs play an important part. It is during those years that the nervous instability acquired by heredity is most effective, and it is during this period of life also, that causes altogether insignificant or insufficient in stable nervous systems, may light up the tendency to convulsions, which tendency primarily characterises the disease. It is

therefore, clear that no exciting cause is necessary in the majority of cases.

Owing to the extreme reticence displayed by the general public about fits occurring in their own relatives, it is not surprising that I failed to get a neurotic family history in 55 % of my patients. This must, however, not be looked upon as implying non-existence in view of the well-marked stigmata of degeneration in many of these negative cases. Such stigmata are certainly more commonly seen in insane epileptics and are manifested in the face, teeth, palate and ears. They are, incidentally, a great aid in prognosis and in denoting the severity of the cases, especially those beginning in early life.

It is not important from the main point of view of this work to go far into the question of sex-incidence of the disease, it is a point, however, which has led to some variance in statistics. There seems, after all, little doubt that the larger proportion of cases occur in males. Osler(5)states that out of 435 cases there were 232 males and 203 females, i.e. 53·5% were males.

Strangely enough at present in

Lanarkshire District Asylum where much of this work was done, 56% males and 44% females was the proportion. Of the total number of epileptics since the opening of the asylum 14 years ago the percentage of males is 55·5 and that of females is 44·5. The figures given by Gowers(2)from 1450 cases being just intermediate to these observations namely males 55·9%, females 44·1%. This, of course, is merely a coincidence. It should, however, be particularly noticed that this prevalence is a marked feature from infancy to old age and cannot, as is sometimes done, be attributed to the greater strain in the male of adult life.

Alcoholism,especially in the parents, has often been stated as being a cause to which epilepsy could be definitely traced. It is very difficult to give direct support to this theory for this reason:- That although some 70% of my cases had a strong family history of alcoholism (parental very frequently) it is also the case; that an enormous number of non-epileptics of the same class have a like family history of alcoholism, if not more so. It might as well be stated that oral sepsis or caries of the teeth is the main point in the causation of the disease, for one

finds it also extremely prevalent, present in fact in even a larger percentage of cases; but here again we find the mouths of the class of patient from which statistics are procured are usually, even in normal persons, fearfully septic, so much so that one constantly wonders how such a state of affairs is compatible with health.

Again although the deleterious action of alcohol taken to excess on the individual cannot be questioned, yet it would be very hard to shew that habitual drunkards - apart from dipsomaniacs - produce an unproportionately large number of epileptic children. With regard to dipsomaniacs, of course we have the requisite neurotic history and we do not need more than that.

Syphilis has usually been included amongst the immediate causes of epilepsy, but in these cases the fits are due to the direct local irritation, and are naturally not to be considered along with the idiopathic type. Were such cases described here, there would be obvious fallacies in such researches as the examination of the blood and of the cerebro-spinal fluid. This also applies to seizures due to other acute intracranial lesions.

Looking now at the etiology from a broader and more theoretical point of view, it is not to be wondered at, that we should consider the possibility of a convulsive tendency being present in a large number of cases. One must be struck with the fact that the majority of the cases of infantile convulsions one sees resemble in no small degree, the convulsions of epilepsy though in a less complete form, no doubt. Is it not possible that in these cases the seed is often being sown which will bear the evil fruit of the habit of taking fits in the future?

Fits in infancy which are in the great percentage of cases referable to a definite cause, e.g. gastro-intestinal, phimosis, teething, etc. develop in later life into epilepsy or at least into a series of epileptiform seizures for which it is impossible otherwise to account.

This line of thought has been admirably followed out by Olmacher(6)who indeed goes much further into the influence of the infantile state.

His experience which is indisputably great is gained from residence in a colony of epileptics,where there are from eight hundred to a

thousand patients. He has also had 91 autopsies on epileptics. He noted in most of the post mortem examinations on those who died with idiopathic Grand Mal two points viz.

(a) Persistence of the Thymus gland.

(b) A typical morbid lymphatic constitution.

Not only that, but he discovered in the great majority of cases in adults, evidences of early rickets though ante-mortem they had been lost to all appearance. He then works out such a line of thought as the following:-

Persistent Thymus leading to Lymphatic constitution, thence to Laryngismus stridulus and Tetany; from here now the step is short to infantile eclampsia, with rickets as the fundamental basis or "vegetative dysnasia."

In another place he goes the length of saying:-

"But who can say that with the supervision of an intelligent mother (in the full sense of the word intelligent) and an intelligent family physician perhaps there would be no Thymus Asthma, no thymic sudden death, no tetany, no infantile eclampsia and no epilepsy!" (7)

Nor is America alone in this line of thought. The responsibility is also shared by Gowers(2)in this country and Bianchi(8) in Italy

With the former, fully 10% of his cases had a history of late teething, late walking, crooked limbs or some other rickety manifestation. This is indeed remarkable, shewing as it does that even ante mortem the place of rickets in the etiology of epilepsy is obvious, in a fair percentage of cases. Gowers(2)at the same place quotes the case of a child who had no hereditary disposition and who had fits at the age of six months while teething. He had scarlet fever at the age of two years, this was also accompanied by fits. At the age of $4\frac{1}{2}$ years fits accompanied an attack of measles. He became permanently epileptic at the age of $16\frac{1}{2}$ years, this event was brought about by a series of fits accompanying a boil in the neck.

Bianchi(8)says that several of his cases of late epilepsy have suffered from infantile eclampsia. He goes the length of looking at this as a direct continuation, and finds rickets very frequently associated with epileptics.

Again Emmet Holt(9)makes the following

three statements:-

(a) The most frequent cause of infantile convulsions is rickets.

(b) There is little doubt that some cases of epilepsy have their origin in attacks of convulsions.

(c) Not infrequently epilepsy may be traced to attacks of convulsions occurring during infancy and from a strong predisposition to epilepsy, it is easy to see how infantile convulsions (so often connected with rickets) may have been the first of an epileptic series.

Here then we have four excellent authorities agreeing on a most important point in the etiology of epilepsy, namely the intimate association of rickets with disease, and though it is one of the rare instances where authorities do agree in their observation on epilepsy it is not so much from that stand point that I emphasise as for its bearing on what follows here viz. the existence of toxic indication in the examinations of the blood and urine of epileptic patients. Not that for a moment would I revive the theory of an organismal causation of rickets,/

rickets, but the nutritional conditions in this disease are more than likely to have a causation tendency to future intoxications. Hutchison(10) indicates this possibility when he agrees with Jenner in saying that "Rickets is the most common, and, in its indirect results, the most fatal disease which peculiarly affects children".

I had not the opportunity of conducting any post mortem on epileptics but in a certain number of my cases I observed distinctly rickety conditions to be present not only in the insane but also in the mentally acute. Olmacher's (6) observations quoted above from 91 autopsies are also surely quite corroborative.

In addition it might be worth suggesting that the visceral changes so frequently associated with rickets especially those described in the spleen may be accountable for some of the blood changes described later in epileptics, and the frequency of heart complication in epilepsy(in my experience in 25% of the cases) may be assignable to the same cause.

EXAMINATION OF BLOOD.

I. MICROSOPICAL.

The fact that so many confirmed epileptics possess marked tendency to a toxaemic series of symptoms makes the histological examination of the blood not only an important and essential line of study but a most interesting and fascinating one also. It is not too much to say that the preliminary reading of Dr. Lewis Bruce's (11) most suggestive views on this subject is more than an aid, it is an inspiration. He describes a fair percentage of cases where we have the temperature irregular and liable to become febrile at times independently of any outward or bodily symptoms. The pulse is also liable to the same irregularities. The number of leucocytes is also frequently increased with a corresponding increase in the number of polymorphs. Though these symptoms point to a definite intoxication it may be that the toxin at work here is also the author of the insane condition itself. Too much importance therefore, must not be attached to these signs in several cases, especially if the insanity manifests/

manifests itself in a form incompatible with bodily health. To put it more clearly, in some forms of moral insanity the behaviour of the patient does not give the body a fair chance for recuperation, likewise patients who are omniverous or who are filthy in their habits cannot be expected to avoid rises in temperature and even rises in the number of their white blood corpuscles with no definite lesion.assignable. I have often seen the temperatures of masturbators go up to 103°F. unaccompanied by any obvious discomfort and as far as can be judged due to the habit alone. Now I think epilepsy is never propter masturbation but that masturbation is very often post epilepsy, from this reason:- A very large percentage of epileptics of both sexes are persistent masturbators. Many become more and more confirmed in the habit. The sphere of life open to them also is usually so restricted as regards employment,as regards recreation and even as regards domestic comfort, that it is little wonder that so very many, leading as they do a solitary and morose existence, adopt the easiest method of gratifying the body if not at first from the more degenerate standpoint merely pour passer le temps.

We see then that such habits as masturbation/

masturbation and filthy feeding may quite easily vitiate the results of observers regarding the temperature, blood-count and other signs on which one bases the opinion of there being a toxaemic origin to any disease, and specially here to epilepsy.

In order then, to avoid any such fallacy the patient must have very careful supervision and have such bad habits checked by competent and trained attendants.

Apart, however, from such fallacies we can, I think, assume that many of the train of toxic symptoms and signs are essential to the disease, and with this in view it is tempting to subdivide our idiopathatic epilepsies, into two classes based upon this suggestion:-

I. Distinctly toxic; such cases as are described by Dr. Bruce, (1), of which I have one or two to demonstrate here.

2. Cases due to some unrecognised cause also, but at the same time giving no evidence at all of toxaemia.

If such a subdivision should be possible it would undoubtedly prove of the greatest value in the study and treatment of the disease.

Unfortunately Dr. A.S.M. Peebles (12) seems

to include in this toxic category all cases of idiopathic epilepsy, and though this would be more convenient than ever, were it feasible, it is unfortunate that the statistics for the great percentage of cases I have worked at, do not support the theory in its entirety by any means. He goes the length of saying that:-

Every case of epilepsy has shewn a hyperleucocytosis, not only during the periods when the patients were suffering from seizures, but even in the intervals when the patient was quite free from attacks. The most marked hyperleucocytosis succeeds a fit or occurs during the period when the patient suffers from a series of seizures.

It is not only on my own work that I base any contradiction of this, because directly in apposition to Dr. Peebles, we have Kauffmann, (13) stating that he can find no changes in the blood on miscroscopical examination. In view of this one is forced - for one's private satisfaction even, to try and discover the discrepancy.

I began with a set of 16 confirmed epileptics, again these were chosen at random. To save space all my original figures will not be given but

only an average of three successive examinations on each patient. The blood was always taken in the morning about four hours after food, and was taken irrespective of fits or any other symptoms. Twelve of those cases, whose figures follow, shewed practically no departure from the normal save in having an increased number of eosinophiles and a slight tendency to an anaemic state of the blood.

This tendency to an eosinophilia is also noted by Pugh (14) who also finds a leucocytosis occurring during fits and following them. What these eosinophiles signify I cannot pretend to explain, having only met with the condition previously in cases of parisitic infection and in an isolated case of lymphadenoma.

The average counts then of these 12 cases are as follows:-

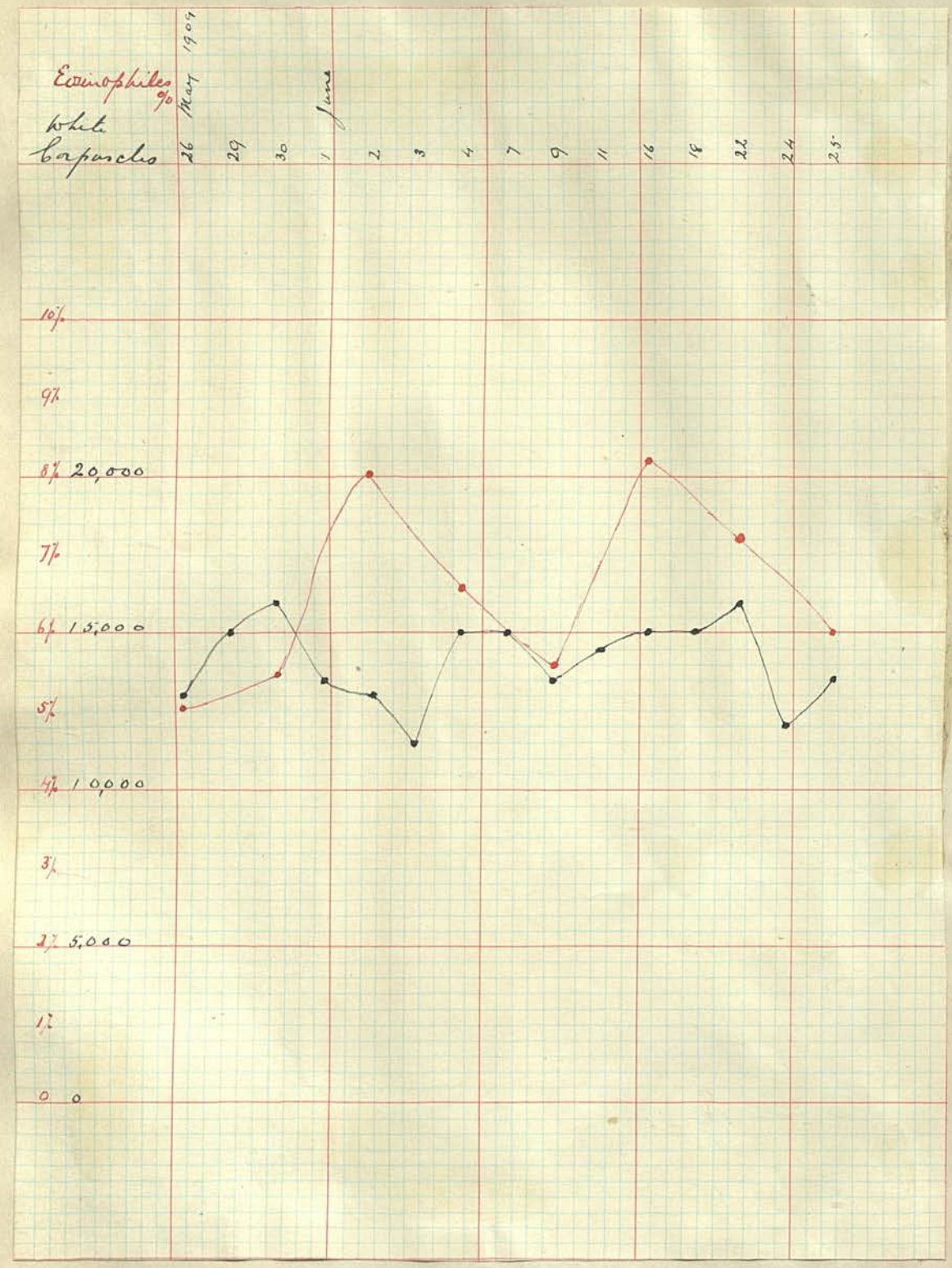
The average of 3 successive blood counts on the 12 epileptics out of the original 16.

These shew little deviation from normal save in the eosinophil count.

Patient	Red blood. Corpuscles.	White blood Corpuscles.	Hb.	Poly.	L.L.	S.L.	Eos.	Mast.
FAIRLIE	4700000	4800	85%	74%	12%	13%	1%	-
FLYNN, THOS.	4600000	5700	90%	70%	10%	16%	4%	-
SHANNON. JAS.	5200000	6000	95%	72%	12%	12%	3%	1%
FERGUSON W.	4800000	4600	100%	73%	12%	14%	·5%	·5%
MACMAIL H.	5000000	7000	100%	75%	10%	12%	3%	-
WILSON ED.	4200000	5200	75%	67%	12%	18%	2%	1%
MACKELLOR, ARCH.	4600000	5800	80%	70%	11%	16%	3%	-
WILKINSON JN.	5000000	7800	95%	69%	13%	15·5%	2%	·5%
CLEGHORN R.	4800000	6500	90%	73%	17%	10%	-	-
BAXTER R.	4800000	6000	95%	75%	15%	9%	1%	-
GIBBONS, WM.	4200000	7800	80%	72%	15%	11%	1·5%	·5%
COUTTS, RBT.	4400000	8600	75%	80%	10·6%	5·3%	3·3%	·8%

Chart A.

DAVID BURT, permanent leucosytosis and high eosinphile count.



It is remarkable that in the case of Coutts a patient referred to before, who suffers from recurrent attacks of status epilepticus, there should be so little deviation one way or another in the microscopic character of the blood. It was examined on several occasions, before and after severe seizures and to all intents was the same.

The other four cases, i.e. 25% of the original 16, were again about normal with regard to the number of red blood corpuscles and to the percentage of haemoglobin, but in each there was a varying leucocytosis. I then made further examinations of the blood of these four and with regard to them came to the following conclusions:

I. In David Burt's case the whites shewed a constant increase always between 12000 and 16000, his small lymphocytes were usually about 20%, his eosinophiles often reached as high as 8·8% but this last only in the proximity of the attacks. He is a congenital epileptic imbecile, rickety, aet. 20. He has no bad habits, works outside, eats well and is otherwise healthy. The following is a typical count:-

Reds-4000000, whites-13600, Hb. 80%, P-60% L.L.-14% S.L.-20% Eos-6%.

The accompanying chart (A) will, however, give/

gave a far better idea of the state of affairs than can a solitary description. It is strange that in spite of his leucocytosis and high eosinophile count he is able to get over a considerable amount of laborious work every day. It is also to be noted particularly that the rise in the number of eosinophiles is absolute and in no way dependent on a corresponding rise in the number of the rest of the white cells.

2. Anderson has great variety in his white blood counts, this variety seeming to follow no definite lines. The table annexed will give some idea of what occurs:-

June 9th.	1909 Leucocytes =	15,000
" 10th.		14,800
" 11th.		14,000
" 12th.		13,600
" 13th.		9,000
" 14th.		9,600
" 16th.		14,600
" 18th.		16,800
" 20th.		12,600
" 21st.		8,800
" 23rd.		8,600

Here then we have a sort of regular swing but on referring to the other charts of this patient we find no reasonable connection between the swing and the other symptoms, fits, or other bodily state. There was a very steady differential count nearly always about:- Polys.-76% L.L.-7·5% S.L.-12% Eos.-4·5%.

He was weak physically, and confined to

Chart B.

HENRY ANDERSON'S Whites. These variations were unaccompanied by any other clinical sign; the eosinphile remained about 4·5%.

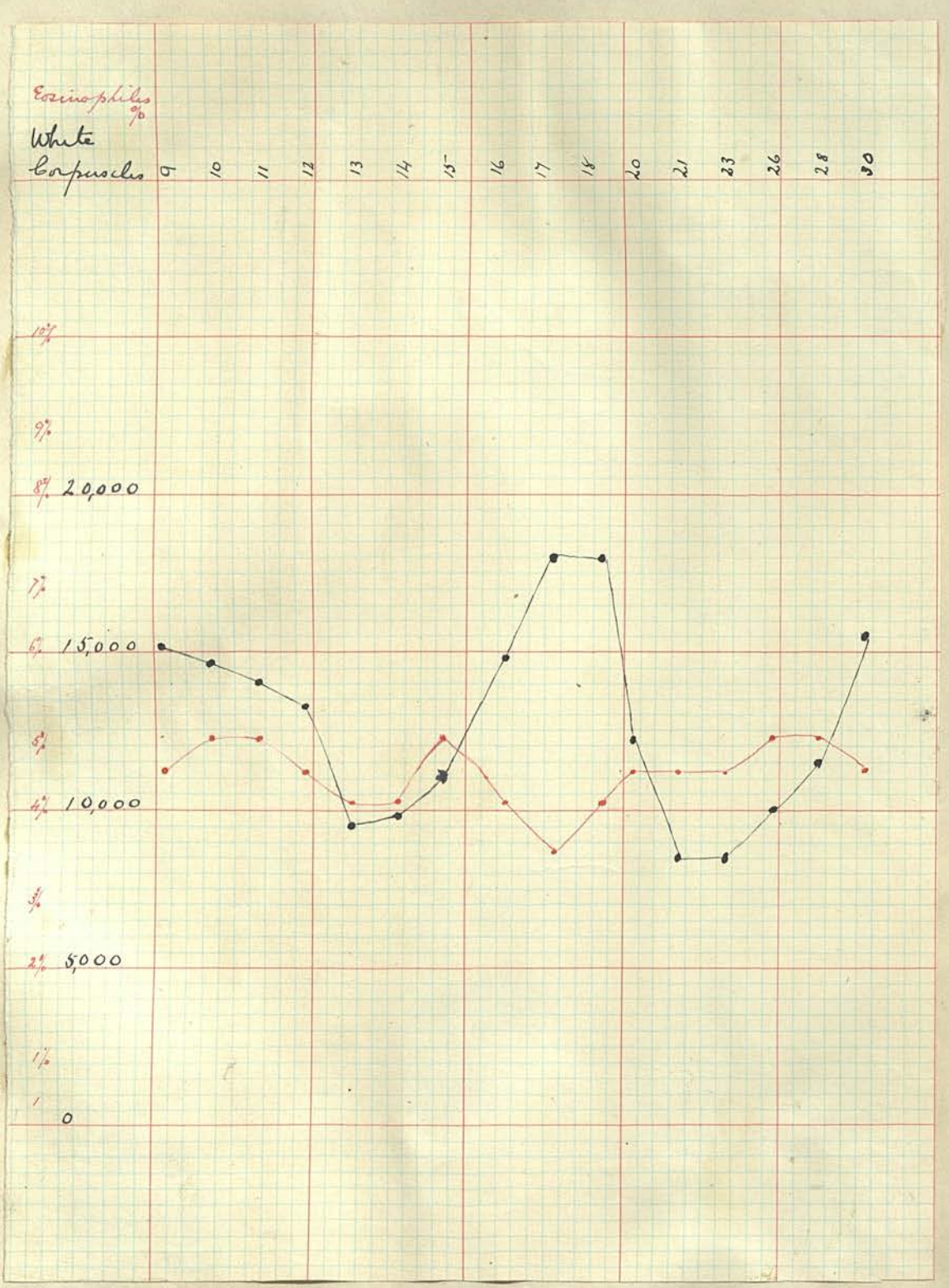
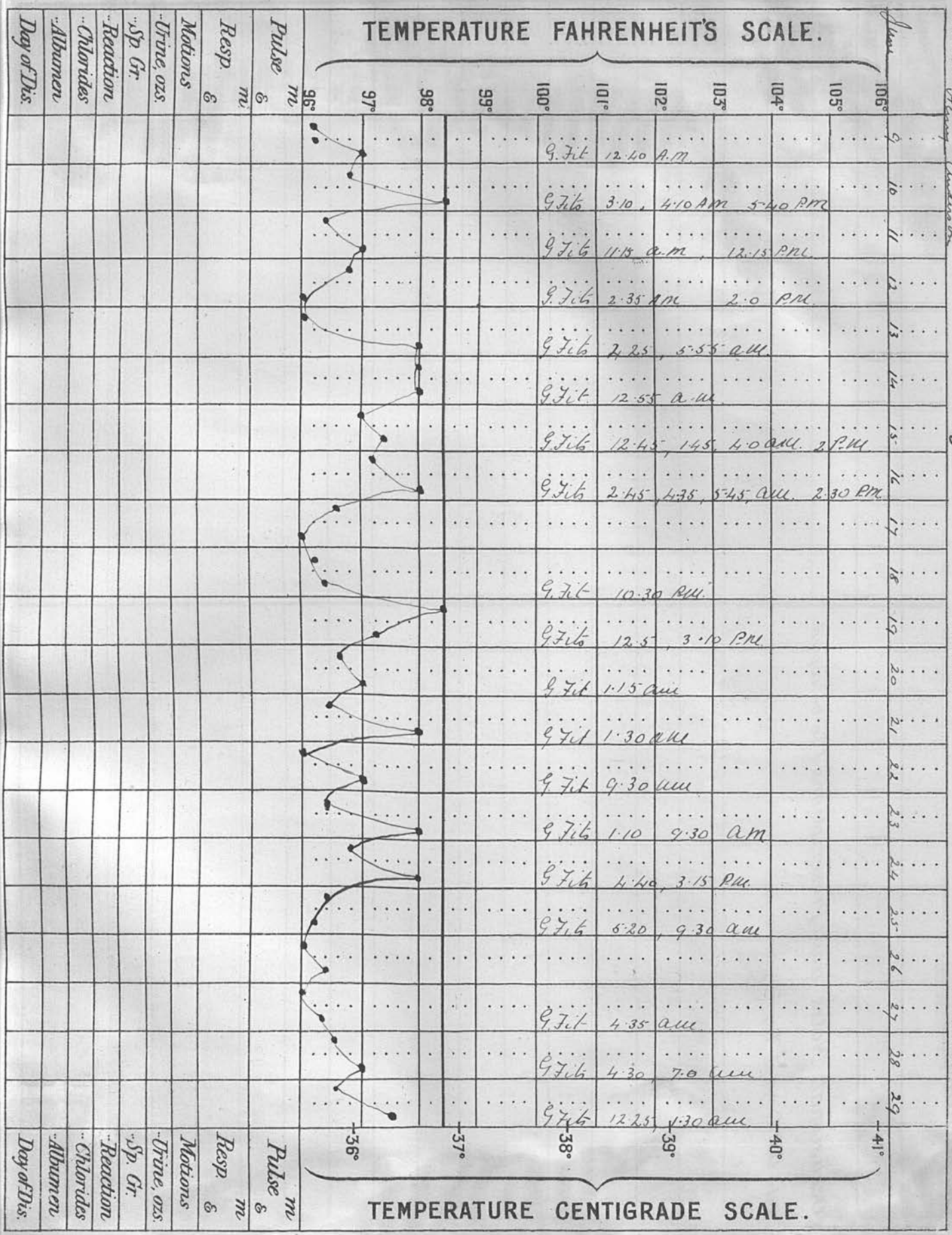


Chart B. (Continued)



bed no lesion could be made out to account for his leucocytosis, he was not phthisical and did not suffer from any parasitic disease that might have accounted for his eosinophiles. His temperature also occasionally rose unaccountably at times without any obvious connection with fits or anything physical. Masturbation, a common cause of temperatures in Asylums, was never detected. His bowels were well regulated and he was on Pot. Brom. grxxx twice daily. Age 24, an early, but not a congenital, epileptic. The charts (B)of this case are very interesting and are attached here.

3 & 4 These two patients, Wilson and Smillie, certainly conformed to a definite rule, and their cases go far to support the theory that there is a toxic group of epileptics amongst the idiopathic patients. In both cases I have given in addition to the usual blood-count charts,(C & D)temperature charts as well. These shew definitely a distinct rise in temperature about the time of the seizures. They must be compared along side of the blood-count charts in order to fully appreciate their importance. In the case of Wilson the number of eosinophiles was increased and varied a good deal in amount, this variation however did not correspond to the occurrence of the fits. The eosinophile count in the case of Smillie was only taken once on which occasion they only registered 1·5%.

48.

Chart C.

WILSON. White count corresponding to fits; eosinophiles which corresponds to nothing at all

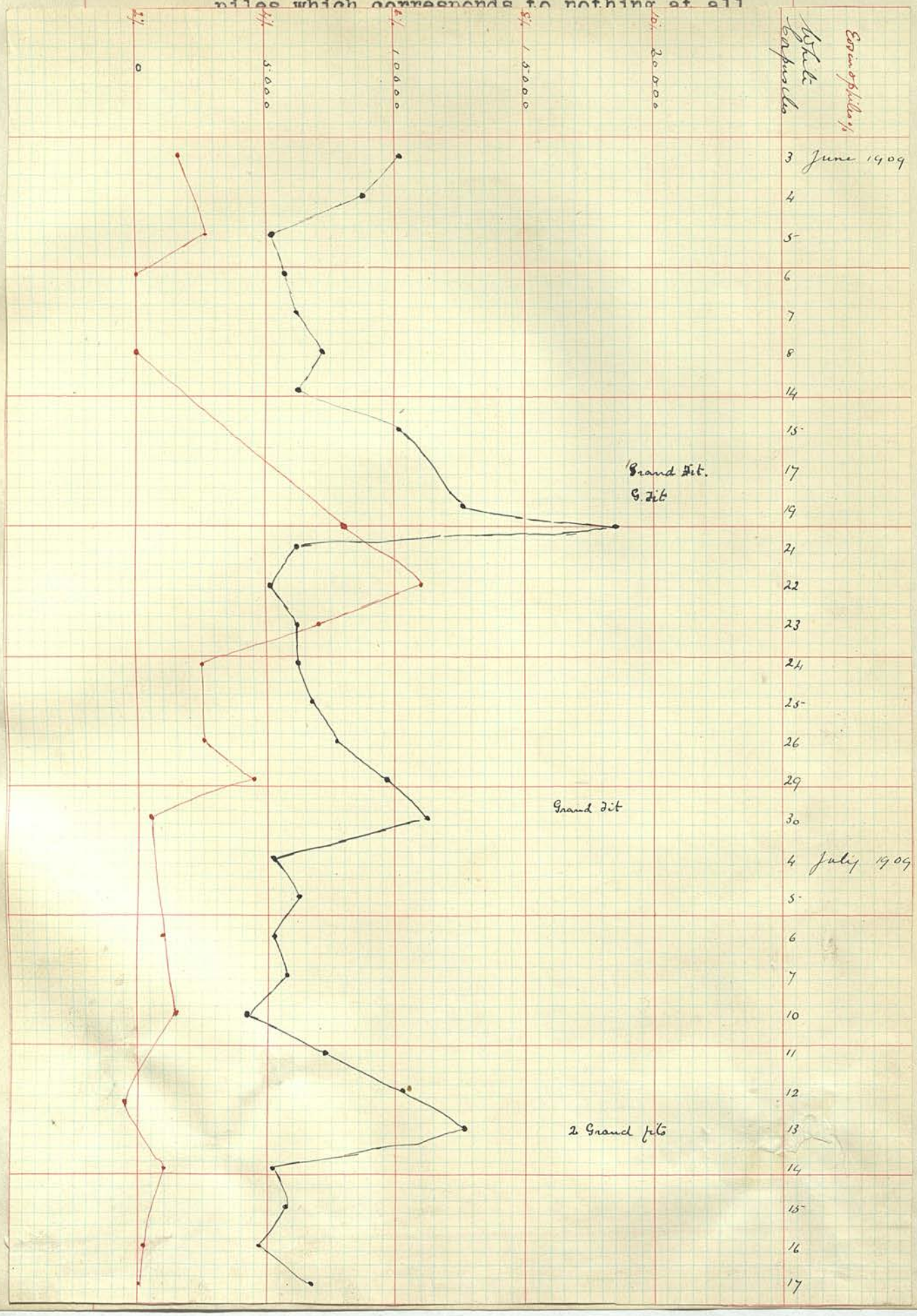


Chart C. (Continued)

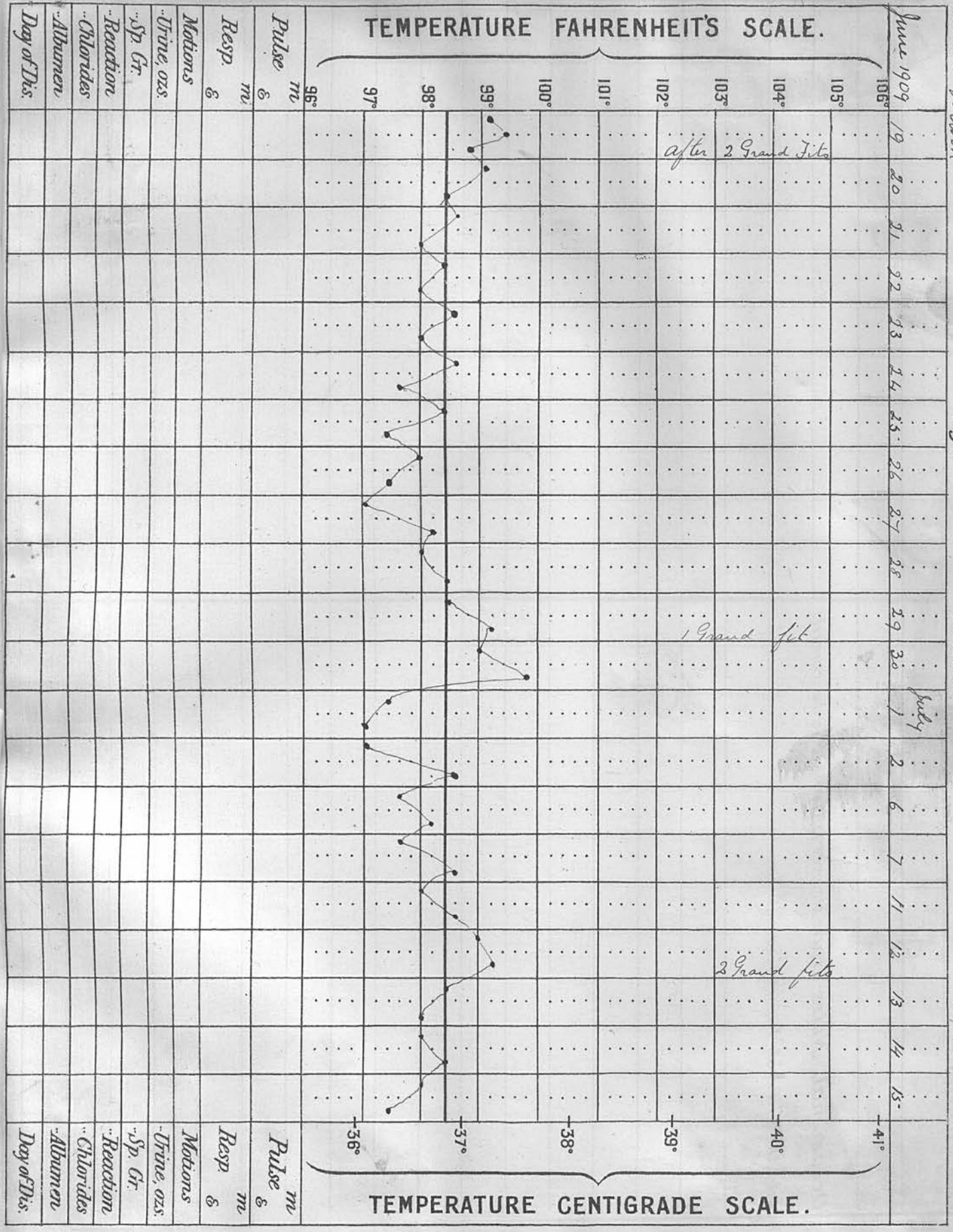


Chart D.

R. SMILLIE, definite rise in white blood corpuscles corresponding to fits.

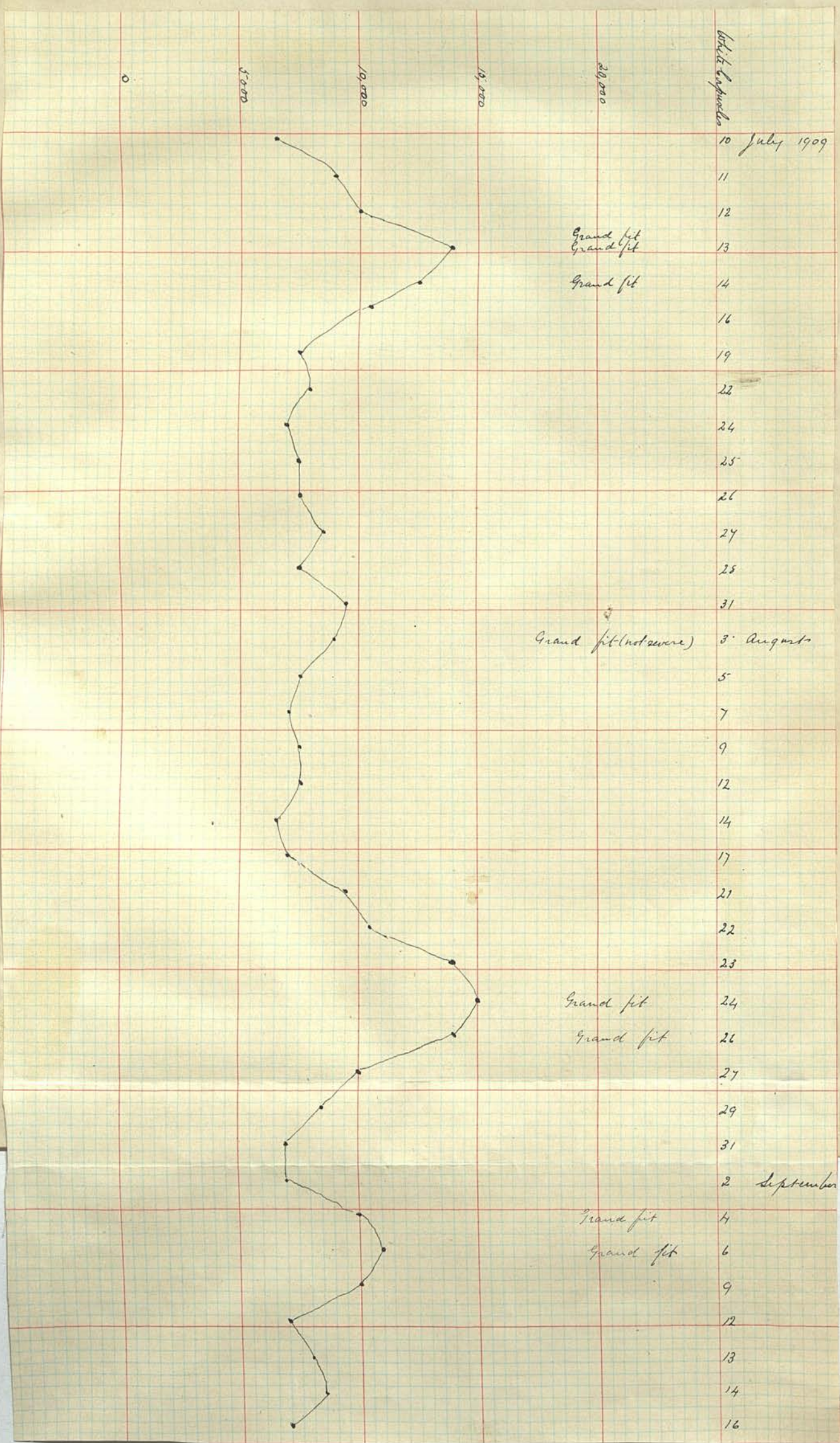


Chart D. (Continued)

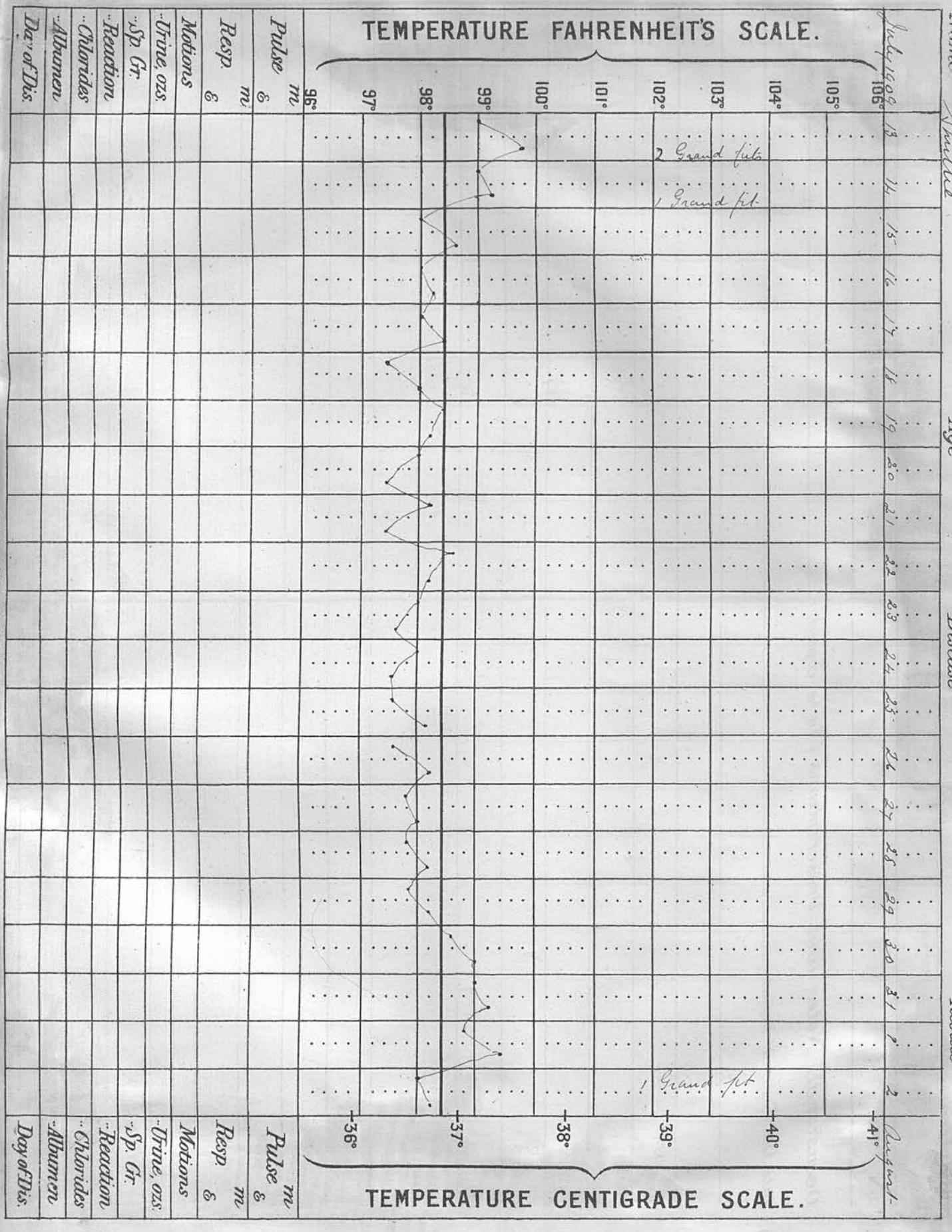


Chart D. (Continued)

Name Lucille (continued) Age Disease Result

TEMPERATURE FAHRENHEIT'S SCALE.

106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°

August 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 September

1 Grand fit

1 Grand fit

1 Grand fit

41° 40° 39° 38° 37° 36°

TEMPERATURE CENTIGRADE SCALE.

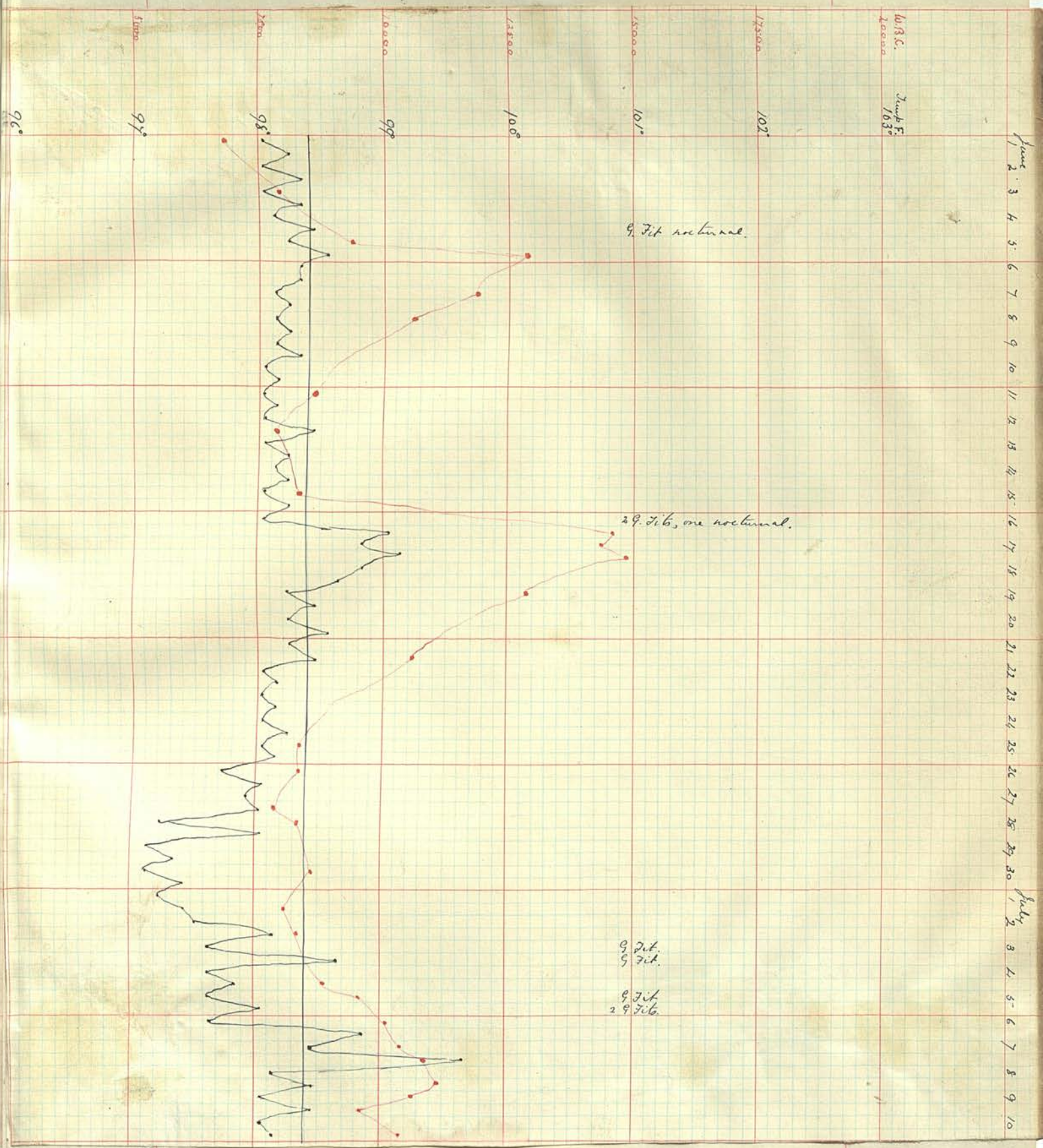
Pulse & m
Resp. & m
Motions
Urine, ozs.
Sp. Gr.
Reaction
Chlorides
Albumen
Day of Dis.

R. MITCHELL & SONS, LITHO 80 CLYDE STREET, EDINBURGH.

After getting these two excellent cases out of my original 16 (making quite a good percentage provided it is not merely a coincidence), I took the blood of several new cases, examining in them the number of the white corpuscles only. I only took note of those whose white count shewed a definite rise in amount. I next took these and examined them periodically in paroxysmal and interparoxysmal periods to see if they supported the toxic theory. As a result I got other four cases all more or less typical of this state.

The chart (E) of one of these - John Scott - is appended. It shews the temperature and leucocyte swing in the neighbourhood of the seizures. The other 3 do not shew these points so definitly, but are yet quite distinctive.

54.
Chart E.
JOHN SCOTT.



2. COAGULABILITY.

In view of the more recent theory regarding the pathology of epilepsy which has been brought forward by Turner and which is a revival to some extent of a theory advanced in 1864 by Hughlings Jackson, I spent considerable time in ascertaining the Coagulation rate of the blood in a large number of the patients.

The theory roughly is that epileptic fits are the result of intravascular thrombiplus a brain hereditarily and structurally predisposed to instability and convulsion.

He divides these thrombi into four groups and thinks that the immediate cause of the fit is obstruction to the blood supply causing cortical stasis. He suggests that just before a fit the blood-plates cohere forming granular or hyaline debris, which either blocks the vessels where it arises or is carried by the circulation into smaller vessels where it becomes impacted. The larger masses met with are probably the result of a more intense coagulation process, a supposition which is borne out by the fact that they are more commonly met with, in cases with status epilepticus. Further a fit may not occur merely as the result of interference with the blood supply. Other factors such

as the area where the interference occurs must be taken into account. He believes that the epileptic equivalents, which so often replace the fit, may be due to plugging of the vessels in a non-explosive area of the cortex. He lays special stress on the importance of defective development as a necessary factor, and explains the failure of Kussmaul and Tenner in four cases to elicit epileptic attacks by compression of the carotids, by the absence of this factor, whereas in two individuals with feeble intellect epileptiform attacks were produced. (15)

The method I adopted was that suggested by McGowan (16). This consists of taking the blood in capillary tubes all blown to the same calibre 1·5 mm., laying these down on a table beside a thermometer and noting the temperature. The tube is then filed through partially and carefully snapped across at a different part of the column of blood. The moment of coagulation is indicated by a thin thread of fibrin uniting the broken ends, this is carefully noted along with the final temperature of the room.

According to the same authority the variations in the temperature of the room make very little or no difference within limits of 15°C. to 20°C. If this is so there is no need of a uniform temperature chamber for rougher work. He also notes that coagulation rate is much slower in the morning than at night; I therefore took all these observations about 12 a.m. or as near as possible to that hour.

I present some observations on 31 cases. The coagulation rate varied between 6 min. and 18 min. both extremes in solitary cases and not occurring again even in the same individuals. Twenty of these cases shew the state of the blood with reference to seizures, the other 11 had no fits during the periods of observation.

Of these twenty only twelve or 60% support the idea that the blood is more easily coagulated in the proximity of the fits than in the interparoxysmal periods. These would naturally support also the theory of epilepsy being thrombic in origin or at lease in causation. Eight cases on the other hand shew the opposite or are indefinite.

With regard to the temperature of the room this varied between 14° and 19° ., and certainly did not seem to interfere with the results in any appreciable way. Examples of this may be seen in the

following cases:-

I. Cleghorn, No. 16; In this case the temperature varied between 16 and 14 C., and the time of coagulation remained practically the same.

II. Scott, No. 22; Here the temperature varied between 19 and 14·5 C., and the coagulation rate remained constant at 7 min.

III. Humm, No. 30; In this case the temperature varied between 17 and 14·5 C., and only at the latter figure did the rate become 10 instead of 9.

IV. In the case of a non-epileptic where I made repeated observations with the temperature varying from 14 to 19 C. and where the result was as follows:-

Temp.	Rate.
17.	11.
19.	11.
18.	10.
14.	11.
15·5.	11.
16.	12.

It seems perhaps rather futile to dogmatise with so little evidence before one, but certainly 60% is not a sufficient majority with which to

support such a theory, and on the other hand, 40% makes a very strong opposition to it.. One of the negative cases, viz., that of Smillie, No. 21. is a negative case worth several confirmatory cases. On Sept. 27th. I took his blood during a fit. It took 11 min., to coagulate at a Temp. of 14·5 C., five days later, without any intervening fit, the rate was 9 min. at the same temperature. Two days later, again without any further seizure the coagulation rate was 8 min. On the 8th. Nov. or four days later the patient had a major fit and immediately after this his rate was 11 min., the temperature this time being 15·5 C.

Judging, then, by this and one or two less marked cases I found it impossible to support the statement that "in the proximity of the seizures the time of coagulation falls, thus making the patient more liable to thrombi". Not that I for a moment would contradict any such statement, but one would like to see more evidence brought forward on both sides with regard to diet, habits, occupation and mental condition of the patients. Such fallacies are bound to complicate results at any time, and may have complicated my own, although I did my best to eliminate them.

TABLE showing the time of coagulation of the blood of various epileptics with the room temperature at the time and notes regarding the vicinity of seizure.

NAME	DATE	TEMP	TIME OF COAG	NOTES.
			min.	
1. GUNN	4.10.09.	18.5 C.	9½.	
	9.10.09.	16	8	after a fit.
2. HIGGINS	4.10.09	18.5	7	
	13.10.09	16	9	after a fit.
	16.10.09	15	9	no other fit.
3. HERON	5.10.09	18	9	
	7.10.09	15	8	after a fit.
4. HENDERSON	5.10.09.	18.5	9	
	7.10.09.	17	7	after a fit.
	10.10.09	15	8	no other fit.
5. WILSON W.	27. 9.09	17	9	after a fit.
	30. 9.09	16	9	no other fit.
	2.10.09	17	9	after a fit.
6. BAXTER	27. 9.09	17	14	
	5.10.09	18.5	11	after a fit.
7. KIRK-PATRICK	27. 9.09	17	11	
	4.10.09	15	13	no fit at all.
8. GIBBONS	27. 9.09	16	12	
	5.10.09	18.5	10	after a fit.
9. PRYCE	26. 9.09	17.5	11	
	29. 9.09	17	11	after a fit.
	3.10.09	15	13	no other fit.
	6.10.09	16	11	after a fit.
10. HARPER	26. 9.09	17.5	15	
	7.10.09	15.5	14	
	10.10.09	14	15	no fit at all

NAME	DATE	TEMP	TIME OF COAG.	NOTES.
			min.	
11. SMITH	26.9.09	17	10	
	30.9.09	16	8	after a fit.
12. COUTTS	28. 9.09	15	18	case of re-
	2.10.09	16	16	current status.
	6.10.09	16	17	
13. MORRISON	28. 9.09	16	11	after a fit
	9.10.09	14	13	
	12.10.09	15	13	no other fit.
14. GRAINGER	28. 9.09	16.5 c.	7.	
	1.10.09	15	7.	no fits.
15. McMAIL	28. 9.09	14	16	
	2.10.09	15.5	11	after 2 G. fits
	6.10.09	16	14	no more fits.
16. CLEGHORN	28. 9.09	14	15	
	2.10.09	14	14	
	6.10.09	15	14	
	10.10.09	16	14	no fits.
17. SHANNON	28. 9.09	14	16	
	1.10.09	15	12	just before G. fit.
18. FERGUSON	28. 9.09	14	18	
	1.10.09	15	16	no fits
19. FLYNN	28. 9.09	14	13	
	6.10.09	16	10	during a G. fit.
	8.10.09	16	11	no other fit.
20. McCONNELL	27. 9.09	14.5	9	no fits
	1.10.09	15.5	7	
21. SMILLIE	27. 9.09	14.5	11	during a fit.
	2.10.09	14.5	9	no other fit.
	4.10.09	16	8	no other fit.
	8.10.09	15.5	11	after a g. fit.

NAME	DATE	TEMP	TIME OF COAG	NOTES
			min.	
22. SCOTT	4.10.09	14.5	7	
	4.10.09	19	7	no fits.
	13.10.09	16	7	
23. JACK	27. 9.09	14.5	11	
	13.10.09	16	10	no fits.
24. BURT	27. 9 09	14.5	9	
	13.10.09	16	7	after a fit.
	15.10.09	14	11	after a fit.
25. TRAQUAIR	27. 9.09	14.5	8	
	13.10.09	16	9	after a fit.
26. FAIRLIE	26. 9.09	16.5	8	
	2.10.09	14	8	no fits.
27. McDOUGAL	25. 9.09	16	9	
	30. 9.09	15	7	
	12.10.09	14.5	10	just before a fit.
28. BOSWELL	25. 9.09	17.5 C.	6	
	30. 9.09	14.5	13	
	12.10.09	14	9	after severe
	14.10.09	15	12	fit. and no other fit.
29. ANDERSON	25. 9.09	16.5	10	
	30. 9.09	15.5	9	no fits.
	12.10.09	15	11	
30. HUMM	24. 9.09	17	9	
	25. 9.09	15	9	
	26. 9.09	15	9	
	27. 9.09	14.5	10	
	28. 9.09	17	9	before a G. fit.
	28. 9.09	16.5	9	after a G. fit.
31. MACLAUCH-LAN	30. 9.09	15	13	
	12.10.09	17	11	no fits.

T O X I C I T Y.

Although I did no work personally on this point, this part would be incomplete if some reference was not made to the toxicity of the blood.

The results I got from consulting certain works were most confusing and disappointing and can only be shortly repeated for what they are worth.

Bra (17) discovers in his epileptics an organism in the blood which he calls the monococcus and which he holds responsible for the trouble.

Besta (18) as well as Tivelli and Brossa (19) deny this organism and put its existence down to faulty methods or defective technique.

Herter (20) says the blood of epileptics is normal as regards toxicity whereas Mairet and Vires (21) say it is less toxic then usual, and Krainsky (22) holds that the toxicity is distinctly increased.

CONCLUSIONS FROM EXAMINATION OF THE BLOOD OF EPILEPTICS.

In the large percentage of cases, perhaps 75%, there is no histological change except in the differential count where there is an eosinophilia this occurs in 90% of all cases examined.

In about 20% of cases there is a leucocytosis not constant but varying with the proximity of the seizures. These support the idea of a toxic type of idiopathic epilepsy.

In a few cases there is an irregular leucocytosis not obviously referable to fits or physical changes, this increase may be due to the toxin which is responsible for the mental condition, and possibly can be neglected.

There are practically no grounds for supporting the Thrombic-causative theory from the evidence of the changes in the coagulability of the blood, 40% of cases examined were either neutral or distinctly negative.

URINE EXAMINATION

It is strange how very contradictory is much of the evidence published regarding the condition of the urine of epileptics, and it is difficult to find a sufficient excuse for this variance amongst authorities, unless it be in the fact that several of the abnormalities present in a great many cases,are of an exceedingly transitory nature, and that in the intervals the urine is often practically normal. That this may be the case, is seen in some of the examples described later. It is generally agreed, however, that the colour and quantity passed per diem are normal while the reaction is generally acid.

Strangely enough when we come to such a definite and important point as that of albuminuria we have two diametrically opposed opinions. Voison and Peron (23) saying that postparoxysmal albuminuria is common, though variable in its intensity and duration, this variation depending on the degree of renal vasodilation present, the latter being indicated by the amount of facial cyanosis present during the seizure. Hence it is always present in status epilepticus, serial epilepsy, and occasional attacks, never with petit mal.

Again Lannois and Mairet (24) support these results, also finding temporary albumen present only in these cases which were cyanosed during the attack.

The findings of Reynolds Sala Rossi(25) & Krainsky(26) do not in any way support these results.

With regard to the specific gravity of the urines of epileptics, it is generally stated to be on the whole, high, this is presumably based on

observations in the vicinity of fits.

I have chosen 36 epileptics at random for the purpose of urine examination, taking a comparatively small number in order to be able to repeat the observations more frequently. All these 36 are males to avoid the complication of menstrual urine and the other fallacies so common to female bladders.

A L B U M E N.

In only two of those cases was found any albumen. In the rest not a trace.

The first of these, A.C. - a congenital epileptic imbecile, aged 24 - shewed constantly, both during paroxysmal and interparoxysmal periods, a trace of albumen in his urine. The further evidence of the microscope, however, demonstrated hyaline and granular casts, thus accounting for the albumen by the presence of chronic renal disease. It is a noteworthy fact that in this case the amount of albumen present, was always greater after a major fit, also that here the facial cyanosis was often very slight.

The second case that of J.T. aged 21, epileptic since puberty, shewed albumen on only

one occasion and that was a mere trace. This was certainly after a major fit, but it never occurred again either in the paroxysmal or interparoxysmal periods. There was no further evidence of renal disease, and may have been attributable to a seminal discharge, though I failed to find any spermatozooa microscopically in the centrifugalised deposit.

In the majority of these 36 cases, also, there was distinct cyanosis during the attacks and in all probability a corresponding renal vasodilation.

These observations must then very definitely support the position of those who hold that there is no albuminuria necessarily associated with epileptic seizures.

U R E A.

I found the output of urea in these cases averaged about the normal amount daily. Little stress must, however, be laid on this, considering the various diets of the patients as well as the fact, that many of them working outside, it was difficult to estimate in any way correctly, the

total amount passed. The different occupations of the patients also make for variety in skin excretion and this also vitiates conclusions regarding the amount of urea in the urine.

Here again there is a difference amongst authorities, Alessi and Pierri (27) stating that the daily output of urea is lessened; Sala and Rossi (28) on the other hand stating that the daily urea is average in amount.

The number of fallacies which obstruct the path to the correct estimation of the daily output of urea keep us from laying so much importance on this branch, as on the amount of indican present in the urine which will be considered later.

SPECIFIC GRAVITY AND INDICAN

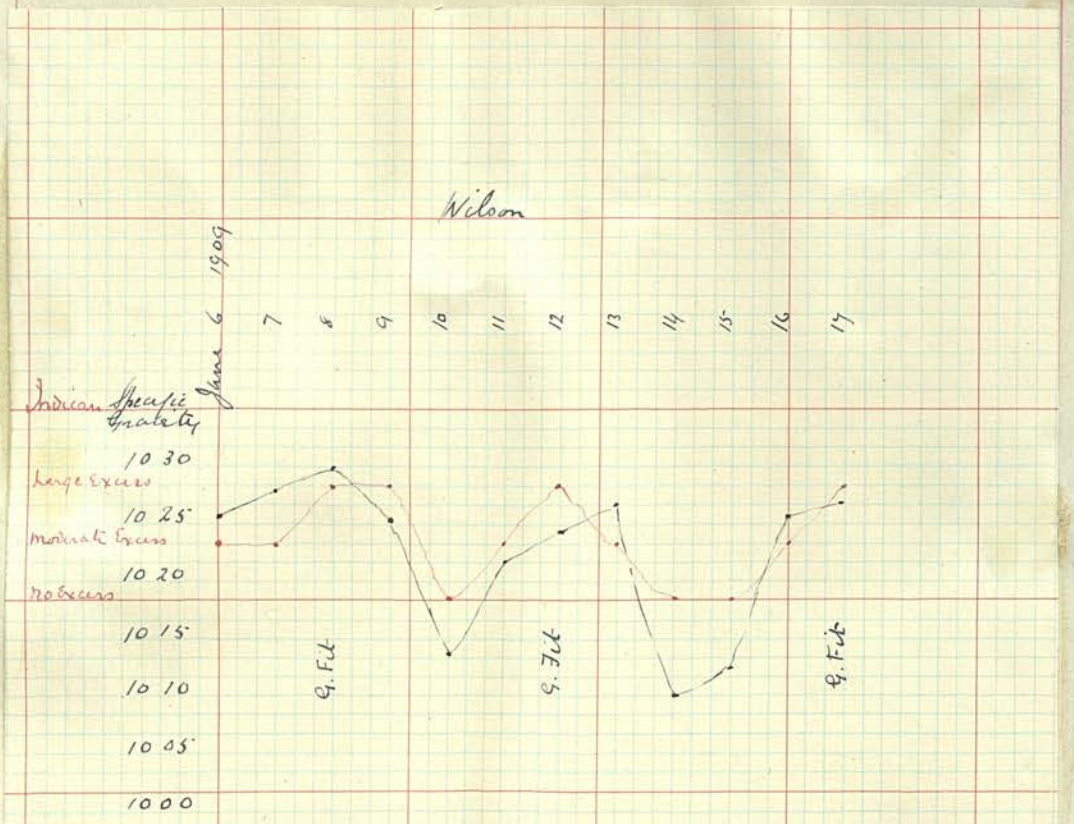
As stated before the high specific gravity of epileptics' urine mentioned by many is probably based on the examination in the proximity of a fit. And with regard to my own

results gathered without any reference to the proximity or otherwise of seizures, the figures shewed a disappointing variety from 1005 to 1030. This, however, was only disappointing at first sight, and after comparing the individual figures with the corresponding amount of indoxyl potassium sulphate (indican) present, it became evident that in a large percentage of cases there was a relationship between the two. In practically every case where I found an excess of indican the specific gravity was not lower that 1020 and in the majority of cases was nearer 1030.

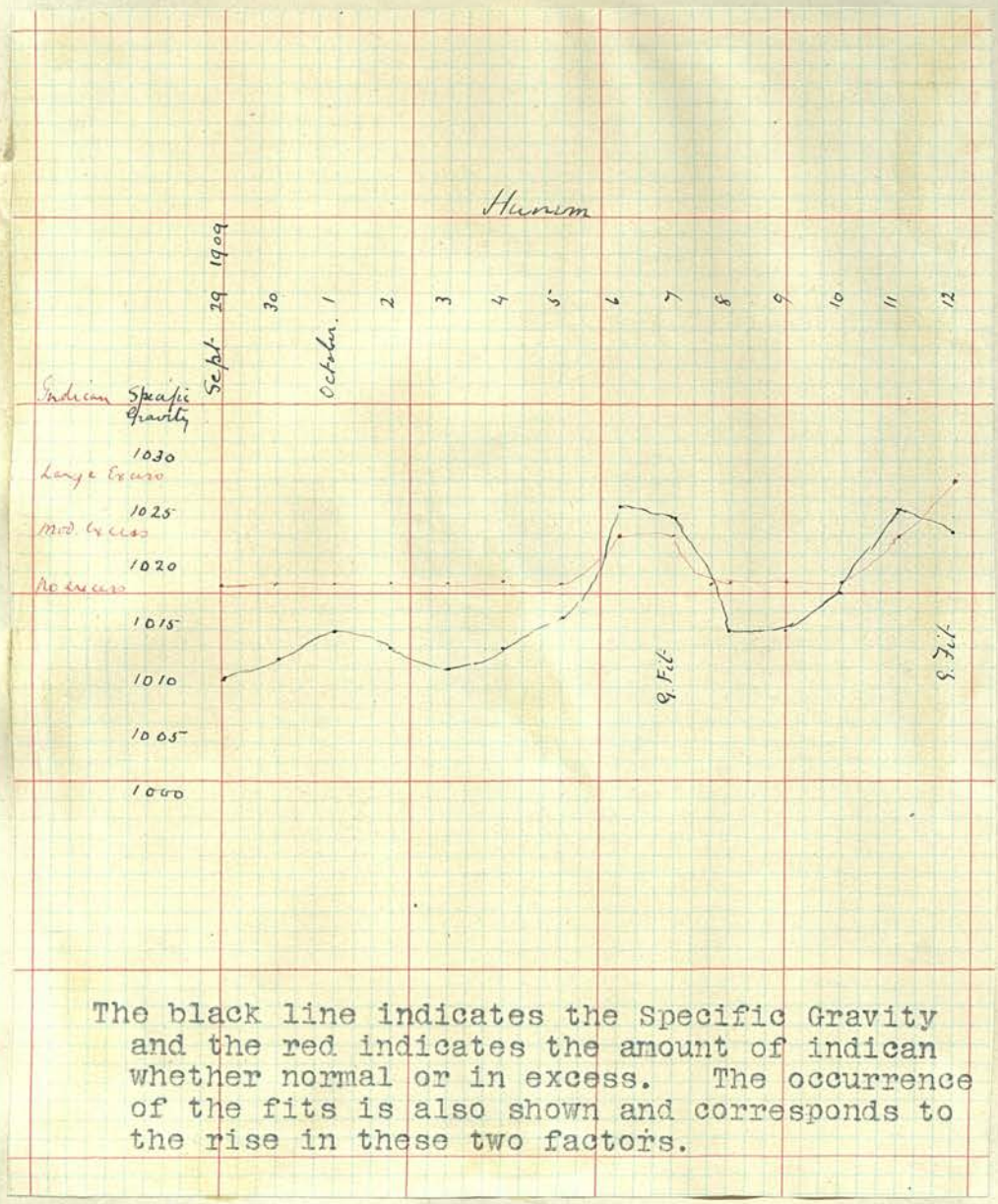
To this there were five exceptions in some hundreds of observations, these were:-

sp. gr.	indican.
1018	moderate excess.
1019	"
1018	"
1015	"
1014	"

These were all in different patients and occurred only once. The first three are so near 1020 that they need not be remarked on.



The black line indicates the Specific Gravity and the red indicates the amount of indican whether normal or in excess. The occurrence of the fits is also shown and corresponds to the rise in these two factors.



The black line indicates the Specific Gravity and the red indicates the amount of indican whether normal or in excess. The occurrence of the fits is also shown and corresponds to the rise in these two factors.

The fourth, i.e. 1015, was taken immediately after admission and may be discounted as unworthy from dietetic and other obvious reasons. The fifth, 1014, had constantly a tendency to a phosphatic urine, but there seemed to be no other abnormality on that examination, on other occasions his urine conformed to the lines indicated above. To such an extent was this striking, that I began to consider a high specific gravity plus any excess of indican as almost pathognomic of a seizure, in fact one often correctly prophesied an attack of some sort on these grounds. This is shewn by the accompanying charts of Wilson and Humm.

The method used for the estimation of indican is only roughly quantative not accurately so, it is recommended by Jaffe (29) and is described by A.B. Townsend (30). Shortly modified it is as follows:-

> Add to the urine in a test-tube an equal amount of strong hydrochloric acid and then drop by drop

a weak solution of bleaching powder, shaking all the while. In normal urine there is a faint trace of indican and this gives a faint blue tinge changing to violet. For convenience Jaffe(29) uses three grades:-

1. Faint trace, i.e. no excess.
2. Moderate excess.
3. Large excess.

Care must be taken to use in all cases the same amounts of urine and hydrochloric acid, and to note the number of drops of the bleaching powder solution - 5% was the strength I used - required till the liquid in the tube becomes darker and then suddenly ceases to do so.

As regards other opinions on the amount of indican in the urine of epileptics; Kauffmann (31) states that there is usually a great increase in the output, it may even rise as

high as one grain daily. He holds that this is nervous in origin and not intestinal. G. Guidi (32) says that in all his cases the excretion ofammoniacal products in the urine was above the normal even in patients on different diets. He blames faulty hepatic metabolism for this state of affairs.

In the cases I examined a considerable number had an excess of indican present in their urine. Counting all the examinations in fact there were 52% shewing this. As stated before, practically all these had a specific gravity of over 1020. Though the distinction between moderate and large excess must have clinical importance it has no bearing on the relative severity of the seizures, in fact, out of those who shewed an excess, 65% were only "moderate", while only 35% were "large". It must also be specially noted that the converse of the rule stated above (viz. that a high specific gravity plus an excess of indican indicated the proximity of a fit) does not hold,a large number of patients having fits

or series of fits without any excess of indican or any increase of specific gravity, the patient, whose chart was shewn before as representing repeated status epilepticus, has never at any time had an excess of indican. I must mention a notable exception to the above rule, that is the case of D.H. aged 30, an outdoor worker, on ordinary diet, whose bowels were carefully regulated. On October 16th his urine showed sp.gr. 1012 with no excess of indican: three hours after this passage he had a severe major fit. Two days previously his urine shewed sp.gr. 1016 and no excess of indican, he himself being meanwhile very well. Three days before (October 11th) sp.gr. was 1010 with a moderate excess of indican present, this was taken <u>immediately after a major fit</u>.

It holds then, that the average epileptic passes an amount of indican in his urine considerably above the normal, whatever be the cause of this increased output, it is highly significant that it occurred in those patients irrespective of employment, diet or constipation.

Some were engaged in outdoor labour, others at indoor work, while a few were confined to bed; some were kept for a time on milk diet, others on a light diet, while others were on ordinary diet; most of them had the strictest attention paid to the condition of their bowels while a few were allowed to remain constipated for a time. Under all these conditions the output of indican in the vicinity of an attack hardly varied, so it seems that the indican is not, altogether at least, intestinal in origin.

CONCLUSIONS FROM THE EXAMINATION OF THE URINE OF EPILEPTICS.

The amount passed per diem and the colour are practically normal.

Urea excretion is probably normal in amount.

There is no essential albuminuria associated with the seizures but in cases with a pre-existing albuminuria this may be transitorily increased.

In the majority of cases we have an increase in the amount of Indican excreted and in these cases a correspondingly high specific gravity, especially in the vicinity of a fit.

The Urine of Epileptics who take fits only in the night time is usually alkaline and phosphatic on the morning examination.

Though the clinical observations preceding only touch the fringe of the subject it is evident that the regular examination of the blood day after day and a similar regular examination of the urine of epileptics is the only way to acquire accurate facts to base theories on.

It is, therefore, to my diagrams and charts that I would draw more attention than to my text as they really indicate by far the greater part of my work on the subject.

Much harm may be done, and has been done, by the haphazard examination of epileptics at varying intervals, thus confusing statistics and giving perhaps altogether erroneous statistics, which the consecutive daily examination over some weeks would probably negative entirely.

A comparatively small number of such results are really of more value than a great quantity of promiscuous statistics.

It will be seen that in my cases several did not correspond to any definite rule but that on the other hand, others certainly go to justify us in concluding that many cases of idiopathic epilepsy are toxic.

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