THESIS FOR DEGREE OF M.D.

PUERPERAL ECLAMPSIA

by

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PUERPERAL ECLAMPSIA

A Description of the Disease and Notes of fifty cases occurring under the writer's care in the Glasgow Maternity Hospital and Glasgow Maternity and Women's Hospital

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DESCRIPTION OF THE DISEASE

The subject of puerperal eclampsia still holds one of the foremost places in the field of obstetrics. There is no question which is more urgent, and at the same time obscure, than the above.

Authorities on eclampsia differ on almost every point, and this alone shows that there is a want of precise knowledge in connection with the disease.

The increase in the number of cases was so alarming during my term of office in the Maternity Hospital, that I make no excuse for again introducing this subject.

During the eleven months in which I acted as House Surgeon in the Glasgow Maternity Hospital and the Glasgow Maternity and Women's Hospital I had the opportunity of seeing and of treating, under supervision of the visiting Physicians, fifty cases of this disease.

The cases reported are in the order they entered the Hospital, so as to give a better idea of the variety and sequence of these cases.

No attempt has been made to classify them, as classification in eclampsia is of little value when the types of the disease are so numerous, and the probable cause the same, namely, a toxaemia.

The death rate of the series of cases may be higher than it otherwise might have been, as I have included all cases of puerperal eclampsia admitted to the Hospitals during my term of office. Some of these cases were

admitted practically moribund, and one at least died from a disease other than the one under discussion.

The remarks dealing with the pathology are taken from notes of post-mortem examinations previously made in the Glasgow Maternity Hospital.

In order to make this series of cases more complete, and to emphasise the points of interest raised by them, I have endeavoured to preface these notes with a short description of the disease.

Reference to the reports is made, when they illustrate any of the questions under discussion.

At times, cases of other similar and contrasting diseases occurring under my care are referred to, in order to show similarities or dissimilarities to eclampsia.

I am indebted to the visiting physicians to the Hospitals, Professor Murdoch Cameron, and Drs. Robert Jardine, Munro Kerr, and A. W. Russell for permission to make use of the cases embodied in this article.

DEFINITION: Puerperal Eclampsia is the name given to a pathological condition occurring during pregnancy, labour, and the puerperium, the most manifest symptom of which being one or more convulsions, which are epileptiform in character. The disease is generally associated with some pathological change peculiar to pregnancy in the Kidneys.

A type of this disease, which occurs without convulsions, has been described. The simple term "eclampsia", which means a "bursting out", has been given to

other diseases in the non-pregnant, characterised by convulsions, generally of toxaemic origin.

FREQUENCY: The frequency of eclampsia is about one in three hundred cases. Of the cases admitted to Hospital in my time six per cent were eclamptic. This, however, is a very high percentage, but in addition to the total Hospital cases, the entire number of cases occurring in the Out-door department, and in the practices of medical men who sent their eclamptic patients to Hospital must be included before a correct percentage can be obtained. Esch[1] reports a series of cases in the Olshausen Clinic, Berlin, in which the percentage of eclamptics was 7.45. This is, however, an exceedingly high one. The next largest percentage is that occurring in the Leipzig Clinic, with 2.07 per cent.

PREDISPOSITION: Primiparae are more liable to the disease than multiparae. Elderly primiparae do not seem more prone to the disease than younger women. Munro Kerr[2] from a fifteen years series of cases, found the percentage in primiparae to be seventy-two. In my cases the percentage in primiparae was seventy. Age does not seem to influence the occurrence of the disease.

In the three years 1905, 1906, and 1907, sixty-six per cent of the cases in the Glasgow Maternity Hospital were primiparae over twenty-one years of age, and twenty-eight per cent over twenty-six years. The youngest patient was seventeen, and tne oldest thirty-two.

Of the multiparae, the average age was twenty-nine; the youngest a II para of eighteen years of age; the

oldest a XI para of forty.

In my series, 34.28 per cent of the primiparous cases were under twenty-one years of age; 51.42 per cent were between twenty-one and twenty-five years, and 12.28 per cent were over twenty-five.

The average age of the multiparous eclamptics was 32.2 years. The youngest, a II para, was twenty-two years, and the oldest, a VI para, was forty-six years of age. One of the multiparae, aged forty years, was a XIX para.

Eclampsia is more frequent in twin than in single pregnancies. Twin pregnancies occur once in eighty cases.[3] The statistics of the Rotunda Hospital, Dublin, show that twin pregnancies occur once in 76.62 cases. Olshausen places the frequency of twin pregnancy at 1.25 per cent, and the frequency of twin pregnancy complicated with eclampsia at 8 per cent. Dührssen found multiple pregnancy with eclampsia in 4.5 per cent of his cases, and Kerr at 3.7 per cent.[2] In the fifty cases of puerperal eclampsia reported by me, two cases of twin pregnancy are to be found. This makes a percentage between that of Dührssen and Kerr.

Recurrent attacks of eclampsia in the same patient are said to be of extremely rare occurrence. Olshausen and Dührssen only met with three cases each. Kerr in his analysis of eighty cases reports the occurrence twice.[2] The first case, a V para, had eclampsia in all her pregnancies, and recovered. The second case, also a V para, took eclampsia in the second, third, fourth and fifth pregnancies, and died in this last attack. In my list of cases, two examples (cases XXI and XLV) of

recurrent eclampsia are to be found. The first case, a IV para, had the first and second pregnancies and confinements normally. In the third pregnancy she took eclampsia, and was treated in the Newcastle Infirmary for this disease. The fourth pregnancy was complicated with eclampsia, and is reported below. The second case was a II para, who had eclampsia in her first pregnancy. She was treated in the Glasgow Maternity Hospital, and was dismissed with no albumen in the urine. The second pregnancy was also complicated with eclampsia, and is reported below.

In a certain number of cases of eclampsia it is found that there is a distinct history of mental distress or worry, which acts as a predisposing cause, and it can be readily understood how such conditions in some instances produce a certain loss of balance of the mental equilibrium. The disgrace of illegitimate pregnancy is supposed to predispose to the onset of eclampsia. In only one case (Case XIX) on the list given was there a distinct history of great emotion due to this cause. The ultimate result in this case was melancholia with a suicidal tendency. In many of my cases, however, there was a certain degree of anxiety and mental suffering, of which no definite history could be obtained. The statistics of the Glasgow Maternity Hospital show that eclampsia does not occur in a greater proportion of illegitimate cases. Eighteen women in the accompanying reports were unmarried.

Hydramnios, causing an overdistension of the uterus, is cited as being a predisposing cause of eclampsia. In only one of my cases was the Liquor Amnii over three

pints (Case XV). There were in all sixty-two cases of hydramnios seen by me in the Hospital, and of these only one was complicated with eclampsia.

The increased cerebral irritation to which women are subjected at the end of pregnancy is hardly sufficient to produce convulsions of an epileptiform character, if acting on a healthy brain. If the patient has toxic substances circulating in the blood, is in the pre-eclamptic stage in fact, it can be readily understood how an increased irritation, such as the onset of labour, might be the immediate cause of the seizures. A long and severe labour may cause the onset of convulsions by setting up this reflex irritation in the brain cortex of a patient, who is already in the pre-eclamptic stage (Cases I, XXXII, XXXVII) whereas in healthy persons no such ill effects would ensue from a severe labour. The analogy of this cause and effect is seen also in epileptics, although eclampsia and epilepsy are in no wise similar. An undue stimulus in epilepsy predisposes towards the onset of a fit - which, however, is purely functional in character - much in the same manner as a stimulus in eclampsia is the cause of a convulsion.

It would be reasonable to suppose that in cases where there had been pre-existing renal disease, that pregnancy would be attended by a greater risk of eclampsia, because the Kidneys are already damaged when the pregnancy begins, and hence are not in a good condition to deal with the products of increased metabolism. If the Liver be involved, as we believe it to be in eclampsia, the Kidneys will be further embarras-

sed by an increase of waste products circulating in the system. There seems to be no doubt that nephritis in some cases plays some part in the production of a toxaemia of which eclampsia is the outward manifestation. It is very difficult in most cases to obtain any history of previous Kidney disease in the class of patient brought to Hospital. It must be admitted that practically none of the patients that were sent to Hospital with symptoms of nephritis took eclampsia, but how far active treatment was responsible for this result, it would be impossible to say. It was also noticed that the patient suffering from nephritis who had a great degree of oedema in the sub-cutaneous tissue never developed eclampsia. It seemed as if a quantity of the poison was being kept out of the circulation, and was being stored in the oedema producing fluid.

In Case XXXV, a II para, there is a distinct history of "an acute inflammation of the Kidneys" following on measles in childhood. Her first pregnancy was complicated by a return of the nephritic symptoms, namely, headache, oedema of the face and lower extremities, and albuminuria. In her second (the present) pregnancy, the symptoms of a return of the renal symptoms began to appear, about the thirty-second week. When she left the Hospital against advice the albumen in the urine was one part per thousand as measured in an Esbach tube. In twenty-five per cent of the eclampsia cases coming to Post-mortem examination in the Glasgow Maternity Hospital pre-existing renal disease was found. This, however, was only in a very short series of cases, twelve in all. The effect of preg-

nancy in chronic nephritis is, theoretically at least, an injurious one, because a greater amount of work is thrown upon the already diseased Kidneys. This, however, is very difficult to prove, as the patient, of the Hospital class at least, cannot be kept under observation for a sufficient length of time, that is, a chronic nephritic of this class is seldom seen before the onset of pregnancy, and kept under observation during this time and afterwards.

The fact that the oedema and albuminuria sometimes disappear quickly after delivery does not, in my opinion, afford sufficient proof of a temporary condition (due to pregnancy) present in the Kidney, as the oedema and albuminuria may be signs of a superimposed acuter form of the disease on a chronic type. However true this may be, there is no doubt that a rapid succession of pregnancies is most injurious to the Kidneys, especially if the function of these organs is in any way impaired, because they have not sufficient time to recuperate between the pregnancies.

This condition of affairs is well shown in Case XLI, where the patient, forty years of age, was pregnant for the nineteenth time. There is a history of increasing oedema with each succeeding pregnancy.

Epilepsy has no connection with eclampsia in any way, either as a predisposing or an immediate cause. In none of my fifty cases was there any history of epilepsy. In fifty cases of eclampsia occurring in the years 1900 to 1903, three were complicated with epilepsy. This is a large proportion.

Deformity of the pelvis is cited as a predisposing

cause of eclampsia, but the enormous number of contracted pelves uncomplicated with eclampsia treated in the Glasgow Maternity Hospital make the probability of this being a common cause very slight. It may be that a deformity of the pelvis causes a severe and prolonged labour. and so acts in causing a greater stimulation of the central cortex. This occurring in a patient already in the pre-eclamptic state is sufficient to determine the onset of convulsions. In my cases, there were two in which there was a distinct deformity of the pelvis.

There seems to be no end to the number of causes that are said to predispose to eclampsia. Constipation, distension of the bladder, wearing tight garments, abuse of alcohol, mal positions of the foetus in utero, are some of these mentioned.

TIME OF ONSET OF THE CONVULSIONS: Eclamptic seizures may occur at any time, that is during pregnancy, in any of the stages of labour, or in the puerperium. In my cases, the percentage taking convulsions in pregnancy was forty-eight, in labour thirty-four, and during the puerperium eighteen. I am aware that these figures differ widely from the general statistics given on this point, as most authorities state the majority of cases occur during labour. I am also aware of the difficulty in deciding whether labour or the convulsions commenced first, but I have endeavoured to obtain accurate information, by carefully going into the history of the cases, and by examining them carefully on admission.

In pregnancy the convulsions generally occur dur-

ing the later weeks; from the thirtieth to the fortieth week being the most frequent, although in some of my cases convulsions came on before this time.

In Case L the fits occurred remarkably early, namely in the fourteenth week of pregnancy. This patient had been for some years in a state of unbalanced mental equilibrium, which may have acted as a predisposing cause. This case seems to be one of the earliest recorded examples, as authorities such as Green observed only one case as early as the twenty-first week; Norris one at the fourth month; Olshausen has seen cases at the third and fourth months.[2]

The convulsions in Case XIII occurred at the twenty-third week, and several of the under-mentioned cases started fits about the twenty-seventh and twenty-eighth weeks of pregnancy.

The onset of convulsions generally brings on labour in the cases which take eclampsia in pregnancy, and hence in the great majority of cases delivery is premature.

A large number of the "pregnancy" cases reported by me took convulsions just at full time.

The cases that occur in pregnancy, and in the very commencement of labour are more toxaemic than those that occur later, and it only requires a slight stimulus to set up convulsions in these cases. On the other hand, eclampsia occurring in the second stage of labour, the toxaemia is probably lighter, and it is only the severe reflex irritation of prolonged uterine contractions that are sufficient to overthrow the mental equilibrium. Immediate delivery in such cases

generally lessens the number and severity of the convulsions. Only one of my cases, Case XXXVII, which took convulsions in the second stage of labour, died, and death was due to pulmonary complications.

Immediate delivery in cases occurring during pregnancy or in an early first stage of labour has not the same beneficial influence on the disease.

Post-partum Eclampsia occurred in nine of my cases. The longest period between delivery and the onset of the convulsions was fifty-four hours. Generally, however, the seizures came on within twenty-four hours of delivery. Several cases of long delay in the incidence of eclampsia after delivery have been recorded. Parvin[4] quotes two cases, of Bailly and Simpson, in which the fits occurred twenty-nine and twenty-eight days respectively after delivery. Cases in which the fits are delayed for about seven days seem to be comparatively numerous.

PREMONITORY SYMPTOMS: The pre-eclamptic state is the condition of the patient previous to the onset of the actual convulsions. It is the period in which the poison is circulating in the blood, and affecting the tissues of all the organs. As the poison, and its effects increase, only a determining cause, such as mental shock, or the onset of labour, is necessary to bring on the convulsions which characterise eclampsia. This pre-eclamptic stage varies greatly in its duration.

In a few cases, it is true, there seems to have been no premonitory symptoms present, but those are the exception. The great difficulty in these cases is to get a history previous to the onset of the convulsions.

It will be noticed in some of my cases that no history could be obtained either from the patient herself, or from her relations. A great many patients of the class that come into Hospital are so unobservant as to their symptoms that it requires some severe pain or inconvenience to be present, before the fact that they are ill at all obtrudes itself on their mind.

As a rule, however, this "state" is accompanied by symptoms of varying importance and severity. These symptoms are probably all caused by toxaemia.

The most common, and the most important of all prodromal subjective symptoms are headache and oedema. Headache is very frequently present. In over fifty per cent of the under mentioned cases was it definitely complained of. It is most commonly frontal in situation, although sometimes it may be occipital, and more rarely the pain may be referred to the vertex, or temporal regions.

At first the pain is comparatively slight, and often intermittent in character, but as the intoxication increases it becomes more severe and continuous, until before the onset of the seizures it may be of an excruciating nature.

The length of time that headache precedes the convulsions varies greatly. It may be present slightly and intermittently for weeks, but the severer forms of pain in the head do not generally continue for many hours before the onset of the fits.

Oedema is also present in a fair proportion of cases. It was present in thirty-one of my eclamptics. It varies very greatly in extent, and the amount of

oedema present gives one no idea as to the time of onset or the severity of the convulsions when they do occur.

Oedema may be present for a varying period before the incidence of the fits. It is a sign that there is damage to the Kidneys, although swelling of the lower extremities is present in a great many cases which end normally, and is probably due to intra-abdominal pressure.

The presence of oedema may be entirely forestalled by severe toxaemia, and eclamptic seizures, due to a rapid manufacture and accumulation of toxins in the system. An excessive general anasarca is very rare in eclampsia. Swelling of the lower limbs may be present before that of the face, as is illustrated in Cases I and XXI, and it is possible that many patients notice the swelling of the feet and ankles more readily than a puffiness of the lower eyelids.

It will be seen, that in the great majority of cases reported by me, the oedema of the lower extremities is alone complained of. It has been pointed out that puffiness of the face occurring without any great increase of the exudate into the cellular tissue of the lower extremities is a grave premonitory sign of the onset of convulsions. In one of the reported cases oedema of the face was distinctly noticed five weeks, and in another case three days before the onset of the seizures, and, as in a great many patients no facial oedema occurred, too much importance must not be placed on it as an immediate precursor of convulsions.

A symptom upon which much importance has been placed is the occurrence of epigastric pain.

Kerr[2] only found three instances of this sign in eighty cases. Dührssen is said by him to have had only one case in two hundred. Olshausen remarks that this symptom is "fairly general". Dunlop[5] found epigastric pain in six cases out of eight. He considers that if the history of every case of eclampsia is thoroughly investigated, the presence of this symptom will be invariably found. In every case that came under my observation I made the strictest inquiry into this question, and found this sign to be present in eleven cases out of fifty. Certain it is that when it is present, its significance cannot be too highly appreciated, as this symptom generally makes its appearance a short time before the onset of the eclamptic seizures. In only one of the reported cases (Case XLII) were the convulsions longer than twelve hours in following the commencement of epigastric pain. In this case the epigastric pain was present for two days previous to the onset of the seizures, and gradually passed off before their incidence. The cases, in which this symptom is found, are, generally, of the severer variety of eclampsia. Three out of the eleven cases exhibiting this symptom died, and the others, with perhaps the exception of one case (Case XLIX), were of the deeper toxaemic type of the disease. The pain is usually felt below the lower end of the sternum, and has been described by some of the sufferers, as if they were being caught in a vice. Epigastric pain, as in the case of headache, may be

slight at first, but if it become continuous, and increase in severity, it is a warning of the incidence of eclamptic seizures, which should not be neglected.

Disturbance of Vision, when well marked, is a symptom that should be taken as a guide to the incidence of convulsions. It denotes an advanced state of toxaemia. Like the other premonitory signs, the time of its appearance before the actual onset of convulsions is indefinite. Some writers on eclampsia state that it is fairly frequently present, but in my cases it was only found in four cases (Cases XXVIII; XXIX; XXXVI and XLII). It was therefore a less frequently present symptom than epigastric pain. The type of alteration of vision varies somewhat in character. In many cases it is simply a "blurring of the eyesight". In one of the cases it was a complete loss of vision, the patient having to grope her way to the door to obtain assistance. These disturbances are often due to a poisoning of the cerebral visual centres, and not due to any very gross change in the retina. Meigs[6] mentions a case, in which the patient had a transient hemianopsia, which disappeared after the performance of phlebotomy.

Ramsbotham[7] gives a report of a patient who stated that the room was "studded with diamonds" immediately before taking eclamptic seizures. I had an albuminuric patient who became totally blind before delivery. The gradual onset of the amaurosis (six weeks) and the typical appearances of retinitis gave me the impression that the cause of the blindness was probably entirely due to the changes in the retina. This was, I think, proved by the very gradual and incomplete recovery of

the power of vision. During the pregnancy, however, the case gave me considerable anxiety, and it would not have been surprising if convulsions had ensued.

Sickness and vomiting are preliminary signs that may be present immediately prior to onset of the convulsions. These symptoms are due to the toxaemia. In Case XXXIV there is a history of hyperemesis after five previous confinements, and also in an early period of the present pregnancy. Whether or not the toxin of eclampsia is similar to that which produces hyperemesis it is impossible to say.

Most patients in the pre-eclamptic state suffer from an indefinite sense of feeling ill. Malaise, giddiness and lightheadedness are often complained of. These symptoms in primiparae are generally accounted for as being due to their condition, and consequently neglected. Other sensory phenomena, such as noises in the ears, deafness, hyperaesthesia, and perverted sensations (formication) are not infrequently present.

The patient may be deficient intellectually before the onset of the convulsions. She may be irritable, restless, anxious and excitable. Very frequently a dazed or "numb feeling in the head" is complained of. Stupor may be present for some hours before the commencement of eclampsia. Not infrequently the patient has gone to bed at night apparently well, and after sleeping soundly for some hours, has awakened and commenced to take eclamptic seizures. Very often the patient exhibits none of these preliminary symptoms, and appears to be in her normal condition just before the onset of the convulsions.

THE ATTACK: In many cases premonitary symptoms, such as are mentioned above, are present. The significance of these symptoms should not be underestimated. In looking over the histories of the reported cases, however, it will be seen that in a certain number no preliminary symptoms were noticed.

The onset of the convulsion is usually sudden, although one is able in many cases to predict its occurrence by a minute of so. The patient usually lies quite still and unconscious. The eyes are fixed and staring, and the pupils become dilated. In some cases there are quick convulsive movements of the head and arms. The eyes are turned upwards and to one side, as in the "tonic" stage. Respirations occur in a jerky fashion, and the vessels of the neck beat violently. The face is usually pale and drawn.

After about a minute in this condition the tonic stage commences. Its duration usually lasts from fifteen to twenty seconds.

It is ushered in by no aura or preliminary cry. The eyelids may twitch rapidly; the eyeballs roll slowly, and in many cases become fixed in an upward and outward direction. The pupils generally become contracted. The head is generally fixed to one side or backwards. The teeth become firmly clenched, and respiration ceases. The muscles of the body become rigid, and a position of opisthotonus may be adopted. The fingers are firmly flexed on the palm of the hand, the thumb being innermost. The patient becomes deeply cyanosed, the purple colour being well marked on the mucous membranes. The tongue is frequently caught and lacerated

between the closely clenched teeth during this stage.

The tonic stage now gives way to the clonic. Twitching takes place in the muscles of the face, giving it a horribly distorted appearance. These movements become more violent and extend downward to the neck, body, and limbs.

The whole body shakes as if there were a violent rigor in progress, and in some cases I have seen the buttocks raised for a considerable distance from the bed, owing to the violence of the muscular contractions. The tongue may be protruded between the teeth, and bitten with each spasm.

It has been observed in a few instances that the clonic spasm is very much more marked on one side of the body than the other, and it may be entirely confined to one side. Cases XXII and XXXII illustrate this point, the left side of the body being more affected than the right.

The breath is expired with a hissing sound. There is considerable doubt as to the occurrence of true respirations in this stage. The expulsion of air from the chest is supposed by some to be due to muscular contraction during the clonic spasm. The length of this stage proves that respiration of a certain kind must go on. Many authorities assert that the clonic stage in eclampsia only lasts one or at most two minutes. Galabin[3] states that the clonic stage may last from half a minute to two minutes, most frequently not longer than one minute. Herman[8] states that the duration of a fit is seldom longer than a minute, as fatal

asphyxia must result from interference with respiration for a lengthier period than this. He further asserts that fits lasting many minutes are really a series of convulsions. It is true that in many cases the tonic and clonic spasms occur in quick succession, but one can usually tell when one fit is ended and a fresh one commences. My attention having been called to this point, I made careful observations in many cases, and the duration of each fit noted in the reports is correct. The cases that had series of convulsions are Nos. XXVIII; XXXVI; and XLI. In Case XL the patient's friends reported that the patient had a convulsion lasting one hour and a half. This is probably a mistake, a series of convulsions, and intervening coma, being mistaken for one uninterrupted seizure.

In Case XXXVI there was a series of convulsions of twenty minutes duration. There was no apparent interval between the fits, and the sequence of events was distinct, namely, the tonic spasm preceded the clonic, there being no comatose stage. One of the convulsions in this case lasted six minutes. Chloroform was used in this series of seizures, and had the effect of stopping the fits, showing that the drug must have been inhaled. Natural breathing, of course, cannot take place during the clonic stage, but there must be some interchange between the external air and that in the lungs.

In Case XXIII, a fatal case, the patient had presumably a series of convulsions lasting three quarters of an hour. In this case the sequence of events was not quite so typical, the clonic movements predominat-

ing over the tonic. No chloroform was given in this case, as the patient was practically pulseless. In Case XXVIII, also a fatal case, there was present a continuous twitching of the muscles very like an extremely feeble and exhausted clonic stage. These convulsive movements continued for one hour, but did not interfere with respirations which went on almost naturally, and numbered twenty -five to the minute. It seemed as if it were a similar but more advanced state than was found in Cases XXXVI and XXIII.

After the clonic stage has passed a period of coma ensues, the patient lying livid and motionless. Natural respiration returns, although the patient may remain apnoeaic for twenty seconds after the cessation of the clonic stage. The respirations at first are slow, sighing, and the interval between each is prolonged. The duration of the coma is very variable, depending to some extent on the severity and number of the convulsions. It is often found that after the first few seizures, consciousness quickly returns, but as the number of fits increases the supervening coma becomes deeper and deeper. A single fit, however, may be followed by a very deep and lengthy comatose stage. The number of convulsions taken is extremely variable. Often only a single seizure is taken, although many of the reported cases took over fifteen fits. Case IV had eighty-six convulsions in Hospital, and is said to have taken over one hundred before admission. She made a good recovery. The seizures taken by this patient were all typically eclamptic in character, and the number taken in Hospital was carefully counted. The number of convulsions

taken before admission may or may not be correct, and the statement of "over a hundred fits" was made by a midwife, who was attending the patient. There is no regularity in the time of occurrence of the convulsions, but in a so-called typical case seizures may occur at from twenty to thirty minute intervals. It will be seen, however, that a series of convulsions may occur, one fit immediately succeeding the previous one, In Case IV, already mentioned, the eighty-six convulsions occurred within twenty-four hours. In Case XXIV the intervals between the convulsions must have been extremely long, as the patient had thirty convulsions spread over seven days.

The bladder and rectum are sometimes emptied involuntarily during the convulsions. It is said that the spasm of the involuntary muscles occurs at the same time as contraction of the voluntary ones during a convulsion. The evacuation of the bladder and rectum may be due to spasm of the muscles of those organs, or it may be caused by an increased intra-abdominal pressure from spasm of the abdominal muscles and diaphram. Gowers[9] states that in epilepsy the passage of urine is not due to spasm of the abdominal muscles but to contraction of the involuntary muscles of the bladder, as "the passage of urine is discharged with violence." Again he remarks that the passage of urine during an attack of epilepsy is the result of some peculiarity in the convulsions, as it is invariably found in some patients and never in others.[10] My experience is, that the bladder is practically empty immediately before and

during the convulsion taking period, owing to the cessation of secretion by the Kidneys, hence it is very seldom that any urine is passed involuntarily. Also that extreme constipation is so marked a feature in eclampsia that the bowels are very seldom evacuated involuntarily. It has been stated that the uterus contracts strongly during the fits. I had observed this contraction in several of the reported cases, but whether the convulsions or the uterine contraction occurred first I was unable to say. It is possible that the contraction of the uterine muscle being synchronous with the convulsion is purely accidental.

The superficial reflexes are lost during a convulsion, and in the period of coma that succeeds it. The tendon reflexes, on the other hand, were present during the convulsion, and afterwards, in all the cases that I tested this reaction.

During a fit the pulse is weaker and sometimes almost imperceptible at the end of the seizure. After the convulsion is over the pulse soon regains its former condition, being hard and of high tension. It is only when the fits succeed each other rapidly that the pulse becomes permanently weak, rapid, and of low tension.

The temperature usually rises during a convulsion. It may be, however, very slight, only a fraction of a degree. A number of fits occurring in close sequence has the effect of raising the temperature considerably. In only one of my cases was the temperature normal (Case XIV), and in this case the eclamptic seizures had ceased some time before admission.

It has been suggested[8] that apyrexia is associated with pre-existing nephritis, but the only case reported (Case XXXV) that had a definite history of antecedent renal disease had a temperature of 100° Fahrenheit. The highest temperature recorded was 104.8° Fahrenheit.

It has been seen that violent arterial pulsation, especially in the carotids, is present immediately before the onset of a convulsion. Compression of the carotids has been suggested as a means of suppressing an eclamptic attack. I never tried this method, and therefore I am unable to affirm or deny its efficacy. The vascular tension, in most of my cases, was high, as estimated by digital pressure. This was well shown by the spout of blood that appeared when a vein was opened. In some of the cases that were admitted in a moribund condition the vascular tension was extremely low.

During the convulsion taking period the quantity of urine excreted is reduced to a minimum. In the great majority of cases the urine at this time is solid with albumen, on boiling. It will be noticed that in a few instances this was not the case (Case XL for example). Tube casts and blood are also very constantly present. The excretion of urea is markedly reduced.

In the period between the convulsions the pupils are moderately contracted, but as the onset of another fit approaches the pupils gradually dilate. Contraction of the pupil is found during the attack. It is reported that subconjunctival haemorrhages occur from the violence of the seizures. This happened in none of my cases. This condition is comparable to the same

ecchymosis found after violent attacks of coughing in pertussis.

Retinal haemorrhages may be present in cases of eclampsia before the convulsions have actually occurred. In these cases there is generally antecedent renal disease. Retinal haemorrhages may be present after eclamptic seizures in cases where there is no retinitis or other signs of nephritis. These haemorrhages usually absorb very quickly. In cases VI, XV, and XXV small recent haemorrhages were present. These rapidly disappeared with the commencement of convalescence, and were due to some temporary condition. These cases were all primiparae.

In cases I, XXIV, XXXII and XLII there were in addition to haemorrhages, signs of retinal inflammation, usually slight in nature. These cases were either chronic nephritic ones, or the premonitory stage had probably been more prolonged than in the others. Case XXXV was undoubtedly the subject of chronic nephritis, with well marked retinal changes.

Varying degrees of amaurosis may be found before or after the fits. In only a very few of my cases did the patient distinctly remember of the eyesight being affected, and in those who did notice this symptom the impairment seems to have come on shortly before the onset of the convulsions. In none of the patients was there any permanent loss of vision.

Squint is commonly present during a convulsion, and nystagmus may be also noticed.

In all my cases the blood was very dark in colour,

and contrary to what is the case in normal pregnancy, the specific gravity was raised. The coagulability of the blood was also increased, and this may be the explanation of the small quantity of blood that is lost when the placenta separates, and the evident reluctance of lacerations in the parturient canal to bleed.

When the majority of eclamptic patients are first seen in hospital, convulsions and labour are both in progress, and it is extremely difficult to say whether the onset of labour was the immediate cause of the convulsions, or whether the convulsions were responsible for setting up uterine contractions. In a certain proportion of cases it is possible to say definitely that convulsions are the cause of the onset of labour. My reasons for being certain on this point are:(a) That one sometimes sees patients in convulsions, and on examination one is unable to discover the presence of any uterine contractions, and furthermore the delay of the onset of the first stage of labour proves that no contractions can have been in progress when the patient was first seen. The continuance of the convulsions, however, generally brings on labour sooner or later. (b) That eclamptic seizures occur in patients who are not at the full term of their pregnancy. In these cases premature delivery generally occurs secondarily to the convulsions. It is true that the poisoned state of the blood and the death of the foetus may be sufficient to bring on labour prior to or without the incidence of seizures, but in comparing the number of premature deliveries in the case of eclampsia with that occurring in

albuminurics and patients in the pre-eclamptic state, the bulk of evidence goes to show that convulsions are the usual cause of the onset of labour. It has been stated that occasionally frequent and violent convulsions hasten delivery, owing to the increased muscular contractions of the uterus, the great increase of intra-abdominal pressure, and the lax state of the pelvic and perineal muscles at the end of a convulsion.

I agree with this statement as far as the second stage of labour is concerned. The delivery of the child will be rapid if the convulsions are strong and frequent. With regard to the first stage, however, I do not think that any difference in the duration of this stage was noticed between eclamptics and normal patients. The accompanying table gives the durations of the stages of labour in the unassisted cases, and it will be seen that the length of time of the various stages is much the same as in normal deliveries. Several modifying circumstances must be taken into consideration when giving the duration of stages of labour of any particular case. Some of these are the term of pregnancy, the size of the foetus, the number of the pregnancy, and the general condition of the patient.

No. of Case	No. of Pregnancy	Term of Pregnancy	No. of fits before delivery	Duration of Stages of Labour		Weight of Child
				1st Stage	2nd Stage	
IV	I para	32nd week	179	13 hrs 30 mins	4 hrs 30 mins	4½ lbs
VII	I "	32nd week	8	2 hrs?	2 hrs	4½ "
X	I "	Full time	17	6 hrs 30 mins	2 hrs 45 mins	6¾ "
XIII	III "	23rd week	Numerous	5 hrs	2 hrs 15 mins	½ "
XVIII	I "	25th week	6	(5 hrs 10 mins)		3 "
XIX	I "	36th week	15 in Hospital numerous outside	3 hrs 15 mins	1 hr 15 mins	5 "
XX	I "	Full time	8	8 hrs 20 mins?	4 hrs	8 "
XXVI	I "	27th week	17	(13 hrs)		3½ "
XXXVIII	II "	36th week	6	6 hrs 15 mins	3 hrs 15 mins	5 "
XLI	XIX "	Full time	2	(1 hr 15 mins)		6½ "
XLIV	I "	Full time	Series of fits	7 hrs?	3 hrs 50 mins	6¼ "

Statistics derived from instrumental deliveries are valueless, except in so far as they show that labour is not hastened.

It is said that pregnancy terminates with the convulsions, and rarely continues after the seizures have ceased. In my series of cases this condition was not always found.

Case XIV was delivered of a live premature child,

twenty-five days after the cessation of the convulsions. Case XXI was delivered of a premature macerated foetus ten days after the convulsions ceased. Case XXIV was delivered of a dead premature child seven days after the cessation of the fits. Case XLVI was delivered of a dead premature child twenty-eight days after the symptoms of the disease had subsided. In this case the signs of foetal life were noted, both by the patient and myself on the 15th October 1908. Next day, 16th October 1908, the patient mentioned that she "felt no life", and on examination I failed to detect foetal movements or heart's sounds. She was delivered two days later.

Case XLIX left the hospital, irregularly undelivered, thirteen days after the cessation of the convulsions. The signs of foetal life could be made out on the day of her departure. Unfortunately, no further history of this case could be obtained. In Case L the uterus emptied itself of a four month gestation nineteen days after the cessation of the convulsions. In one or two cases the delivery was delayed over twenty-four hours after the signs of the disease had disappeared. The number of cases of delayed delivery after the symptoms of the eclampsia had subsided may be exceptionally large in this series.

Kerr[2] mentions four similar cases in a series of eighty, the longest time between the cessation of the disease and delivery being two months, and the shortest one month. He quotes a case described by Moran, in which labour came on two months after the disease had been arrested. He also states that Olshausen had five

similar cases, in two hundred cases. Green had two such cases, and Sloan one.

When labour came on in my cases there was no return of the eclampsia, and this seems to have been the experience of others.

Puerperal Eclampsia is a disease that rarely lasts longer than forty-eight hours.

In favourable cases the frequency of the convulsions becomes reduced, and their severity considerably diminished, and ultimately the seizures cease altogether. The depth and duration of the coma lessen, and consciousness and intelligence are recovered. For a varying period, usually over twenty-four hours, after the cessation of the seizures the patient remains dazed, apathetic and somnolent. The memory of events which had occurred immediately before the onset of the convulsions is usually absolutely lost. Sometimes the patient is oblivious of events that happened several weeks before the illness commenced, and at which time she was perfectly conscious, and performed her various duties quite rationally.

Shortly after the cessation of the eclamptic symptoms the secretion of urine usually commences, and is usually in excess of normal within forty-eight hours. Herman[8] states that it takes from two to eight days for the quantity of urine passed to reach that normally secreted. This was not the case in a great many of my cases, and it will be seen from the charts that the diuresis was good in the second twenty-four hours after cessation of the convulsions. If the secretion of urine had not become well established in ten hours after the fits had ceased, in addition to saline transfusions,

sinapisms and hot applications were usually placed over the Kidneys, in order to stimulate diuresis.

Convalescence from uncomplicated puerperal eclampsia is usually rapid. Headache, eye symptoms, and mental derangement may be present for a varying period, usually not more than a few days, after the cessation of the convulsions. At the end of seven days, the case is at the same stage of convalescence as a normal confinement is at the end of the first week of the puerperium.

The rapid disappearance of abnormal constituents from the urine is most striking and constant. Albumen, tube casts and blood usually disappear within the first four days of convalescence.

Involution of the uterus is normal, as a rule. It has been noticed[5] that the involution of the uterus is more rapid in eclamptic cases. I cannot say that in my cases the involution was more rapid than normal, but it certainly was satisfactory. Parvin states that there is a liability to post partum haemorrhage in eclamptic cases. This was not my experience. In only one case (Case XLI) was there any post partum haemorrhage. This patient was 40 years of age, and a XIX para. The uterus was extremely flabby, and the repeated pregnancies had no doubt a considerable influence on the muscles of that organ. Jardine[11] mentions a case of severe post partum haemorrhage in an eclamptic, and adds that he has only seen one other case. Prolonged use of chloroform, he thinks, might be the cause of haemorrhage in many of the cases that exhibit this symptom.

Two cases of eclampsia which were in the hospital when I took charge had the very unusual symptom

of antepartum haemorrhage.

The complications of, and sequelae on, puerperal eclampsia are very important. Permanent damage to the Kidneys, as evidenced by the persistence of albumen, blood, and tube casts in the urine, is met with occasionally. In my cases where this complication occurred the history previously to the pregnancy was indefinite.

Cardiac failure is often present immediately after the cessation of the convulsions. A rapid, feeble, and irregular pulse present at the end of an attack of eclampsia has to be noted, and actively treated. This complication has been recognised in some of the German Clinics, and cardiac stimulation is carried out in a vigorous fashion, namely, by striking the cardiac region with ice-cold cloths, and the administration, usually hypodermically, of cardiac tonics.

One of the most difficult complications to avoid, and to deal with when it occurs, is the pulmonary group. Bronchitis, pneumonia, and oedema of the lungs are often present, and difficult to treat. On looking over the reported cases, it will be seen that there is mention made of lung symptoms in a great many cases. In one eclamptic case not reported in the series, as she was in Hospital when I first went on duty, there was gangrene of one lung following a pneumonia, pus and putrid lung tissue being coughed up in great quantities.

Mental complications, such as melancholia and dementia occasionally occur. Five cases in my fifty showed signs of mental derangement, which was of a temporary character (see under treatment). These mental phenomena are certainly more frequent after eclamp-

sia than after normal confinements. Hemiplegia, due to cerebral haemorrhage, may persist, as a result of convulsions.

In fatal cases the causes of death are as follows:-

Coma: This is the most common cause of death. Coma has been divided into two classes, that with a high temperature, and that with a low temperature, at death. Herman[8] thinks that the patients in the latter class had been the subjects of chronic renal trouble. In the few cases dying comatose in my series I was unable to corroborate this observation.

Cardiac Failure: Quite a large proportion of cases die from this cause. It is a very common termination in cases with oedema of the lungs. This condition in the lungs is caused by a venous engorgement of the right side of the heart in addition to high arterial tension, and general venous congestion. Cardiac failure taking place during a convulsion is rare, and is probably due to a spasmodic muscular action. Death from cardiac failure is said to occur, by those who believe in this action of this drug, when chloroform is given to control the seizures. Cases of eclampsia have certainly collapsed and died during the administration of chloroform, but whether death is due to poisoning of the respiratory centre, or to the toxic action on the heart, is still a disputed point.

Pulmonary diseases are also causes of death. It can be readily understood that, along with a certain amount of pulmonary congestion and cardiac weakness, an eclamptic patient easily becomes a victim to inflammatory pulmonary diseases. The position and unconscious-

ness of the patients also make them liable to diseases of the lungs from decubitus and insufflation. Examination of the reports of fatal cases, given underneath, will show that a number of them suffered from pulmonary complications.

Cerebral haemorrhage has been described as a cause of death.

Prolonged fixation of the chest walls causing asphyxia is a rare form of death, and is described by several writers.

Mechanical obstructions in the upper air passages is also an uncommon cause of death in eclampsia. Herman[8] quotes a case described by Bailly, of a patient who was asphyxiated by the tongue becoming abnormally enlarged, due to laceration. Jardine[12] mentions cases where oedema glottidis was the cause of death.

Eclamptics occasionally die from other intercurrent maladies. Case V died from puerperal septicaemia. It was an example of post partum eclampsia, and had been delivered outside the hospital twenty-five hours before admission. On the temperature rising on the fourth day the uterus was explored, and a piece of decomposed placental tissue removed. Jardine[12] cites a case of eclampsia that died from ruptured duodenal ulcer.

The following table gives the cause of death in the eleven cases in the series.

Number of Case	Cause of Death	Length of stay in Hospital
V	Puerperal Septicaemia	Six days, thirteen hrs, fifty mins
VII	Coma	Fifty-two hrs
XVI	Coma	Fourteen hrs
XXIII	Coma	One hr, forty-five mins
XXVII	Cardiac Failure	Thirty hrs
XXVIII	Coma	One hr, fifteen mins
XXXVI	Cardiac Failure	Twenty hrs
XXXVII	Pneumonia and Cardiac Failure	Five days, two hrs, fifteen mins
XLIII	Coma	Two hrs, forty-five mins
XLIV	Pneumonia and Cardiac Failure	Thirty-one hrs
XLVII	Coma	Twenty-six hrs, fifteen mins

THE URINE

In my experience the urine always shows great changes, both in quantity and in quality. I am only able to find one observation of a case of eclampsia in which albumen, for instance, was absent from the urine.[2]

QUANTITY: In every case of eclampsia that came under my care, the quantity of urine excreted was markedly diminished. In many cases there was almost complete suppression of urine.

The decrease in the output of urine generally pre-

cedes the eclamptic convulsions by a few days.

When the seizures are at their maximum frequency and intensity, there is practically no urine secreted. This is so in spite of any treatment that may be adopted. In favourable cases, however, almost immediately after the cessation of the fits, diuresis recommences, and within twenty-four hours of the occurrence of the last seizure it is usually profuse.

The diuresis is apt to be more tardy, and less satisfactory in cases where the birth of the child is delayed, after the cessation of the convulsions.

If, after the fits have ceased, and delivery has been effected, the flow of urine does not become established, then the outlook for such a patient is exceedingly grave.

Examination of the urine column on the charts accompanying the reported cases will demonstrate these facts.

COLOUR AND CONSISTENCE: The colour of the urine of eclamptics is usually altered to a certain extent. As is to be expected with a diminished urinary output, the colour becomes darker. Abnormal constituents also cause the urine to change its colour, hence there may be a smoky blood , or blackish colour, due to the presence of haemoglobin. The urine may be muddy, due to the presence of blood cells and tube casts. In Case XXXVIII the urine had the colour and consistence of 'hare soup' from these constituents. After the disease has passed over, and as a result of the saline transfusions, the urine is usually milky in appearance owing to urates.

In favourable cases the urine gradually assumes its

normal colour, and with profuse diuresis may become paler than normal.

REACTION: The reaction of the urine is generally acid. A urine that contains a quantity of blood is usually faintly acid, neutral, or even alkaline in reaction. Shortly after a transfusion of saline solution, the urine approaches the alkaline in reaction, but this soon passes off, and the urine again becomes acid.

SPECIFIC GRAVITY: When the output of urine is small the specific gravity is high. With the increased diuresis after the cessation of the convulsions, the specific gravity falls. The rapidity with which the specific gravity falls is often remarkable.

ALBUMEN: According to Lusk,[13] John Lever in 1842 first discovered that the urine of eclamptic patients contained albumen. Herman[8] states that the urine becomes solid with albumen, on boiling. In two of my cases the urine was not so loaded with albumen. In Case I the albumen present up to within one hour of taking a seizure was only 1.5 per thousand as measured in an Esbach's tube. In Case XL the albumen in the urine was 4 per thousand Esbach. This specimen was obtained while the fits were in progress. The urine in the majority of my cases became solid, on boiling, with albumen. It will be noticed, however, that in several of the cases the albumen did not exceed 12 per thousand Esbach. This great degree of albuminuria is usually observed after several convulsions have been taken.

It is almost impossible to give any definite time to the onset of albuminuria in most cases. It is also

impossible to say what relation, if any, the onset of the seizures has to the incidence of the albumen, and vice-versa. Cases[14] have been recorded in which the convulsions had occurred before any albumen appeared in the urine. In Case XXXIII, a fatal case, there was no albumen in the urine five hours before the first fit. Herman[8] cites a case in which there was no albuminuria three and a half hours before the onset of the first seizures, and he knows of "no case of eclampsia where the urine had been examined immediately before the onset of the convulsions."

In Case I, the urine had been examined frequently after admission, and before the onset of the convulsion, and it contained a certain amount of albumen, usually about 1½ per thousand Esbach. This case is hardly a fair test, as the albuminuria had been present for some weeks previous to admission.

The bulk of evidence goes to show that the convulsions and albuminuria commence simultaneously in the majority of cases. Taking this for granted, the probable cause of this phenomenon is the circulation in the blood of a substance poisonous to the brain and the Kidneys, and producing in them an irritation which is evidenced by the convulsions and the albuminuria.

This explanation coincides with the toxaemic theory of the actiology of the disease. The cause of convulsions occurring before a marked albuminuria in some cases seems to be that the more virulent the poison, the sooner the brain cells are affected, and hence it is only after the convulsions have been in progress for some time in these cases that albumen, in any quan-

tity at least, appears in the urine.

It is probable that the convulsions and secondary venous congestion may add to the albumen in the urine. This, however, is difficult to verify, and it may require the renal epithelium to be in a damaged condition before convulsions per se are able to produce sufficient congestion to account for any part of the great albuminuria.

The case of Status Epilepticus which was under my care, and has been reported by Dr Jardine,[15] had, on admission, albumen present in the urine to the extent of 2 per thousand Esbach. After numerous convulsive attacks, the albumen instead of increasing, diminished in amount, and after delivery disappeared entirely. This patient had three hundred and eighteen fits in three days, and after this enormous number of seizures, albumen was absent from the urine.

Herman[8] states that the urine does not become solid with albumen on boiling until after convulsions have occurred in patients who are the subjects of chronic nephritis or the Kidney of pregnancy.

It has been also stated that convulsions themselves cause inflammatory change in the Kidneys. In other diseases characterised by the presence of convulsions, namely epilepsy and cerebral tumour, it is found that little or no albumen is present in the urine after the seizures, and further, if these diseases are present in conjunction with pregnancy, the urine is never loaded with albumen. I had the opportunity of verifying this fact, in the numerous epileptic patients who were

admitted to the Maternity Hospital during my term of office.

After the cessation of the convulsions in favourable cases, the albumen present in the urine diminishes rapidly. Delivery in conjunction with the termination of the disease causes a much quicker diminution in the quantity of the albumen present. Examination of the "Albumen" column on the charts which accompany the reports shows this point very markedly. There is an exception to this fact in Case XIV, where the convulsions occurred on the 29th February 1908, the day of admission, and delivery took place on 27th March 1908. Beyond the albuminuria of the first three days after admission, which registered:- first day, 12 per thousand Esbach; second day, 4 per thousand Esbach; third day, .5 per thousand Esbach; no further albumen was found in the urine. The child in this case was born alive.

I have sought to illustrate this fact further, by taking a case at random. A second case is described under "Urea".

CASE XVIII

Day of Illness	Quantity of Urine	Albumen per Esbach	Remarks
1st	5 ounces	Solid	On admission, after six fits
2nd	6 "	20E	Immediately after delivery and treatment
3rd	12 "	.5E	-
4th	40+ "	Nil	-

The death of the foetus in utero is found to cause a diminution in the quantity of albumen in the urine.

In Case XLVI the foetus was alive, although feeble, on the 15th October 1908. The albumen was 12 per thousand Esbach on that day. The child probably died during the night, as, on the morning of the 16th October 1908, the patient volunteered the statement that she felt "no life", and on examination, the signs of foetal life had disappeared. The albumen in the urine on this day showed a diminution to 6 per thousand Esbach, and next day the quantity of albumen had dropped to 2 per thousand Esbach, and afterwards gradually disappeared.

Case XIV, reported above, shows an exception to this assertion. Here the child remained alive, and the quantity of the albumen steadily diminished.

Some idea of the amount of damage done to the epithelium of the renal tubules may be gathered from the amount of serum albumen present in the urine in comparison with the quantity of paraglobulin serum. Albumen is less diffusible than paraglobulin, and an excess of the first named substance shows that more destruction to the renal epithelium has occurred. The prognostic significance of this fact is much more serious.

UREA: Variation in the quantity of urea present in the urine is an almost constant feature in puerperal eclampsia.

Urea is found usually to be decreased in amount.

I consider that this diminution in the quantity of urea to be a valuable sign in estimating of the onset of eclamptic convulsions. In all my cases where the urea was estimated, the amount present was always greatly

diminished even although the output of urine was extremely small. After the attack of eclampsia had passed, and the quantity of urine was increasing, it was found that the elimination of urea also increased, both relatively and absolutely. It is difficult to tell exactly when the urea begins to decrease, as the estimation of urea is rarely carried out before the patient has taken eclamptic seizures.

Taking Case XLVI as an example, it will be seen that, with diminution in the amount of urine, increase in the amount of albumen, and pre-eclamptic signs, the urea varied accordingly.

CASE XLVI

Date	Quantity of Urine	Albumen	Urea, grs per oz.	Remarks
11/9/08	VI ozs	Solid	3 grs	On admission
12/9/08	XLII "	4 per thousand E.	5 "	
13/9/08	LXXII "	2 "	7 "	
14/9/08	LXXV "	1½ "	8 "	
15/9/08	XXXVIII "	2 "	6 "	
16/9/08	XL "	4 "	5 "	Headache
17/9/08	XXXII "	6 "	4 "	Headache
18/9/08	XXIX "	8 "	3 "	Severe headache, oedema
19/9/08	LV "	4 "	6 "	No headache or oedema
20/9/08	LXX "	3 "	7½ "	" "
21/9/08	Lost	4 "	7 "	
22/9/08	LXXX "	3 "	7 "	
23/9/08	XLII "	2 "	8 "	

In the interval, diuresis was quite satisfactory. Albumen was only present in minute quantities, and the elimination of urea was normal.

Date	Quantity of Urine	Albumen	Urea, grs per oz.	Remarks
14/10/08	XLI ozs	4 per thousand E.	7 grs	Slight headache, high arterial tension
15/10/08	XXX "	12 "	4 "	Increased headache, Oedema of face, epigastric pain
16/10/08	XLV "	6 "	6 "	Above symptoms improved. Foetus died
17/10/08	LXX "	2 "	8 "	
18/10/08	LXXXV "	2 "	8 "	Delivery
19/10/08	LXXIII "	2 "	7½ "	
20/10/08	LXXX "	1 "	8 "	
21/10/08	LXV "	Trace	9 "	
22/10/08	LXX "	Nil	9 "	

The prognosis becomes much worse if after cessation of the fits, the elimination of the urea remains low.

The estimation of urea was made in the reported cases with Gerrard's Ureameter.

BLOOD: Haematuria is present in a great many cases. The quantity is generally greater when the attack of the disease is a severe one. In patients who are in the pre-eclamptic state no blood is present in the urine; it usually appears after the convulsions have commenced. Blood usually disappears very rapidly from the urine in favourable cases. It is generally the first abnormal constituent to go. I have never seen a persistence of blood in eclampsia, such as is sometimes found after an

attack of acute nephritis, in which the albumen has almost disappeared.

TUBE CASTS: As might be expected from the cause of the disease, tube casts are frequently present in the urine of eclamptic patients. They were present in the urine of the great majority of my cases. In a case of threatened eclampsia, reported by Dunlop, no tube casts were found in the urine.[5] Granular casts of the fine and coarse variety are most often present, and they are in excess when there are other varieties present. Epithelial casts were also sometimes found. It was noticed that if the urinary deposit were examined quickly after the urine had been passed, the predominant type of tube cast present was the epithelial. These casts rapidly underwent granular degeneration, granules appearing, and causing the outline of the epithelial cells to become indistinct.

Blood casts are nearly always present when there is a degree of haematuria.

Tube casts also disappear very quickly from the urine, after the attack of eclampsia has ceased.

URATES: Urates are generally abundantly present in the urine during convalescence. Their presence is very marked during the first twenty-four hours after the cessation of the convulsions, and when diuresis has set in.

SUGAR: Sugar is said to be sometimes present in the urine during an attack of eclampsia. The urines of some of my patients reduced the blue colour of Fehling's solution to a coppery or purple colour, but there was no

precipitate present. This is probably due to the presence of an excess of creatinin. Herman[8] points out that sugar may be present in the urine from a re-absorption of sugar from the milk in the breasts.

BILE: Bile may be present accidentally in the urine. It may be produced by prolonged venous congestion in the liver, causing jaundice.

PATHOLOGY

The pathology of puerperal eclampsia is one of the most interesting, and at the same time one of the most difficult problems in the whole field of medicine.

The clinical side of the question is quite familiar to us, but in spite of numerous investigations on the ample material at our disposal, the pathological aspect is still shrouded in darkness.

Indeed, so much in the dark are we that the treatment employed in cases of eclampsia is for the most part theoretical and experimental. Many theories as to the cause of the disease have been advanced, and the majority of these have proved valueless.

The older views were that the convulsions are due to some disturbance of the cerebral circulation, producing anaemia, hyperaemia, and oedema. Experimentally[16] it has been proved that ligation of the carotid and vertebral arteries in animals produces convulsions, but Moxon[17] has since shown that the vaso-constrictor nerves cannot produce a spasm and contraction of the vessels equal to that produced by ligatures.

Epileptiform seizures may be produced by an acute anaemia, after severe haemorrhages, such as severe post partum haemorrhage, and external or concealed ante partum haemorrhage, but in eclampsia there is never found such a degree of anaemia as to be accountable for the convulsions. At post mortem examination, however, it is frequently found that there is a degree of anaemia and oedema of the brain,[14] which may be caused by the convulsions, and not be the cause of them. Traube and Rosenstein advanced the theory of brain hyperaemia with oedema as being an indirect cause of eclampsia. Numerous conditions were supposed to cause this hyperaemia and subsequent serious effusion, namely, a hydraemic condition of the blood during pregnancy, hypertrophy of the heart, and an increased vascular tension.

The increased intra-cranial pressure caused by the effusion was supposed to press on the small cerebral vessels, and to cause an anaemia of an acute character, which in turn produced convulsions.

Objections to this theory were advanced by Playfair,[14] and are as follows:- (a) that "it does not account for those cases which are preceded by well marked precursory symptoms." (b) that "on post mortem examination the brain does not, as a rule, exhibit the oedema, anaemia and flattened convulsions, which this theory assumes." Herman[8] has many objections to this theory. He shows that if this theory were true, serious effusions would go on in other parts of the body more readily than inside a confined area such as the skull, and that we should find the tendency to eclampsia increasing with

the oedema, which is certainly not the case. He also questions the extra hydraemic condition of the blood in eclampsia. The whole theory he finally throws out of court, by explaining that when serous transudation becomes sufficient to cause pressure on the vessels, then it would cease.

Frerich[18] goes on the assumption that eclampsia is due to the circulation in the blood of urea, or ammonium carbonate which has been derived from urea. This theory is now not credited, as it has been shown that these products are present in the same proportion in the blood of healthy persons (Bernard). It is true that with the increase of the toxaemia there is a diminution in the excretion of urea, also that during recovery from eclampsia the elimination of urea steadily rises. The relation between the toxaemia and the excretion of urea is of a very definite character. It has been found by Bouchard that to kill an animal with urea it was necessary that there should be in the blood nineteen times as much urea as is normally excreted during the twenty-four hours.[8]

That puerperal eclampsia is due to a specific bacterium is not generally accepted, as no definite or constant specific organism has been shown. Müller[19] holds that eclampsia is caused by non specific organisms which are present in the uterine mucosa during pregnancy. These micro-organisms in most cases become very active and virulent towards the end of pregnancy, and suddenly flood the system through the blood stream with a large quantity of toxin. The disease only being found in pregnant and puerperal patients

confirms him in the idea that the poison must arise in the uterus. That the foetus, placenta, and membranes play no actual part in the causation of the disease is shown by the fact that the convulsions continue, and sometimes occur after delivery. It may be argued that this is also one of the sources of puerperal fever. He explains the occurrence at one time of eclampsia, and at another of puerperal fever by the fact that in eclampsia the quantity and the rapidity of absorption of the toxins is much greater than in puerperal fever. He asserts that these two diseases have practically the same symptoms, although in different degrees, namely, nervous phenomena, elevation of temperature, and injury to internal organs, principally to the Kidneys and liver. Albert[19] supports this theory, and points out that uterine drainage is imperfect during pregnancy, and that the ever collecting and increasing products of the organisms in the uterus are ultimately poured into the general circulation, and cause the onset of the disease. He mentions cases which died of eclampsia undelivered, and in which the uterine mucosa gave evidence of bacterial infection. He found also that the history of patients who recovered was often that of dysmenorrhoea, endometritis, and leucorrhoea. In the post mortem reports of cases dying of eclampsia in the Maternity Hospitals, Glasgow, no mention is made of the uterus being unhealthy. In only one of my cases (Case V), which was admitted after delivery, was there any evidence of a septic condition in the uterus. The convulsions came on post-partum. A septic portion of placenta was removed from the uterus, some days after

delivery, the patient dying from septicaemia. In no other case was there any evidence of septic infection of the uterus, and in no case could a definite history of dysmenorrhoea, endometritis, or leucorrhoea be obtained. If this view be correct, it is strange that in a great number of normal cases treated in the Maternity Hospitals, there is a distinct history, and often evidence, of some of the conditions mentioned above, and yet no eclampsia supervenes. One would expect to find also that there would be transitional forms between these two diseases, and these are never found.

Uterine inflammations seldom occur in primiparae, yet it is in this class that most of the cases of eclampsia are found.

There seems to be no doubt that there is in many eclamptics a neurotic tendency. The increased irritability of the nerve centres in conjunction with the uterine contractions may in some cases be sufficient to bring on epileptiform seizures, and this has been made the basis of the explanation of cases in which no Kidney lesion has been found.

Acting on the healthy brain, anxiety or severe reflex irritation experienced at the end of pregnancy does not produce convulsions.

It is probable that some other condition, in addition to an unstable mental equilibrium or a reflex irritation, is required before true eclampsia is produced, and these factors mentioned should only be classed as pre-disposing causes. In all the eclamptics I have seen, the urine contained albumen, showing that the Kidneys were in some way damaged.

The views now mostly accepted are those which have for their bases the supposition that the disease is due to a toxaemia. This is much on the same lines as Frerich's theory, but in a more advanced and slightly different form.

In support of the toxaemic theory of eclampsia are many facts. The entire clinical history of eclampsia is in accordance with the above theory. The pre-eclamptic stage, the attacks consisting of convulsive and comatose stages, and the ultimate result, are the logical sequence of events, assuming that the toxins are at the same time being manufactured, and are remaining in the circulation, in consequence of a decreased elimination through the Kidneys. In cases which recover, coincident with improvement, urine is passed in greater quantities, urea is eliminated in greater amount, and the supposition is that the toxins are being passed out of the body in a satisfactory fashion.

In the normal state, as the result of metabolism, there is a constant production of waste material, which if not eliminated by the various excretory organs, remains in the tissues, and acts as a poison.

In pregnancy there is an increased metabolism, and if the action of the excretory organs (Liver and Kidneys) be in any way altered, toxic substances remain in the circulation, producing a toxaemia, and ultimately eclampsia.

It has been thought by some, that the retention in the body of the normal toxins of the urine, due to some damage or failure in function of the Kidneys, is sufficient to produce convulsions and coma. The vari-

ous ingredients of the urine are all supposed to take part in the poisoning. It is a striking fact that in the pre-eclamptic state of the disease there is a diminution in the quantity of urea in the urine (see Case XLVI), and it has been shown that the general poisoning properties of the urine are reduced. On the recovery of an eclamptic, or of a patient presumably in the pre-eclamptic state, it was noticed that the urea, and possibly other toxins were increased. This action might explain the occurrence of eclampsia in patients suffering from renal disease, but it offers no satisfactory explanation of the cause of the disease in cases where no Kidney condition is to be found.

Many experiments have been performed in trying to prove that the urine and blood of eclamptics are toxic.

It was found by the earlier French experimenters that urine of eclamptic patients kills animals, and they tried to standardise this property of the urine by making a "Urotoxic dose". This "dose" was the number of cubic centimeters of the urine per kilogramme of the body weight required to kill the animal.

Stewart[20] himself, and in conjunction with Forchheimer,[21] proved that the above statements are fallacious, and states that the erroneous results obtained by their predecessors were due to insufficient aseptic precautions. These observers found that when fresh unboiled eclamptic urine, at 100° F., was injected into the peritoneal cavity of rabbits under the strictest aseptic precautions, 20 per cent of the animals died. Again, injections of boiled urine was harmless. Urine that had been kept for twenty-four hours without boil-

ing caused death in every instance. Further, Stewart demonstrated that the toxic properties of the urine were due to pyogenic microbes which produced septicaemia.

Tarnier and other writers showed that the blood serum in eclampsia is toxic, and that the poisonous properties of the serum bear an inverse ratio to the toxicity of the urine.[22]

Semb[23] has performed a number of interesting experiments with the sera of eclamptic patients, giving some proof that the blood of such patients is toxic. Under the strictest aseptic precautions he immunised rabbits to normal human serum. This was done to exclude the possibility of a toxic action from normal serum. He then injected the serum of an eclamptic patient into these immunised rabbits. His conclusions were as follows:-

"That rabbits may be immunised to a certain degree "against the toxic action of normal human serum."

"Such immunised rabbits reacted most unfavourably, "usually fatally, to the injected eclamptic serum."

"The organic changes in these animals were in a "general way the same as the pathologic-anatomical "findings in the human eclamptic subjects."

"In a small number of cases there was no reaction "to the eclamptic serum."

The exact nature of the toxin, and the causitive agent, or situation of such, are still quite unknown.

Stumpf[18] believes that the convulsions and coma in eclampsia are produced by an acetone like, nitrogen free substance, which is produced by an "abnormal decomposition" in the mother or child. This poison, in

addition to causing changes in the Kidneys, damages the liver, making it atrophy and necrose. It also helps the liver cells to produce leucin and tyrosin.

This toxin also produced distinctive changes in the blood, and alters its colouring matter.

The placenta has been blamed for the production of a coagulation producing ferment which causes an intoxication, and the formation of thrombi throughout the organs of the body.

Leipmann[23] from observations in the Bumm clinic holds a somewhate similar view. He states that the toxin is derived from the placenta, or rather from the albuminous products of the syncytium, which have undergone insufficient synthesis. This poison is a substance attached to the molecule of albumen obtained from the above source. If the organism is unable to produce a sufficient quantity of antibodies to neutralise this toxin, then convulsions and coma supervene.

Bouchard[8] believes that the formation of the toxins in different cases of eclampsia takes place in different situations, both inside and outside the body. For instance, he states that in some cases of eclampsia the poisons are formed in the tissues or in the secreting glands (principally in the liver and Kidneys), in other cases the formation of toxin goes on in the bowel, by a decomposition of the food causing an autointoxication, or it takes place before the food is eaten, and is ingested with the food. This, however, includes all the tissues and situations that one would expect toxins to be formed in, and does not offer any very definite help in the solution of the problem with which we are

engaged.

As the renal epithelium usually shows signs of rapid degeneration, the cause of eclampsia has been suggested from this fact, and an external poison similar in action to phosphorus has been mentioned in this connection. No such substance has ever been demonstrated, but the possibility of its presence has been hinted at by Hermann.[8]

Bouffe de Sainte Blaise[18] considers that there is some auto-intoxication from the intestines, which produces infarctions in the liver. This liver condition he looks upon as pathognomonic of eclampsia, the convulsions and coma being phenomena of secondary importance. In any liver I have had the opportunity of examining post mortem, or in any of the Maternity Hospital reports of autopsies I have seen, there have been very few cases in which infarction of the liver has been found.

Another theory of Bouchard's which is reasonable, but the findings of which have not been verified, is, that he found two toxic substances excreted in the urine. One of these produced convulsions, the other caused coma.

Another substance has been isolated from the blood of eclamptic patients by Doleris and Butte,[22] which, if injected into rabbits, caused convulsions, and subsequently death. This substance is described by them as "a crystalline inorganic substance". No confirmation, however, of the existence of this salt has been forthcoming.

One of the more recent, and certainly one of the most original theories is that set forth by Nicholson,[24] who states that the primary cause of eclampsia is due to a defective secretion of the thyroid gland. Normally

during pregnancy the thyroid gland enlarges, and produces an increased quantity of "thyroidin", which is required in the process of the augmented tissue metabolism. In the non pregnant a certain amount of this "thyroidin" is required for the ordinary metabolism. Albuminuria has resulted in about eighty per cent of the cases of pregnant women in which no hypertrophy of this gland has been found. If sufficient thyroid secretion is not produced, intermediate toxic bodies are produced, instead of the normal terminal product, namely, urea. "Thyroidin" has been shown to have an antagonistic action, on arterial structures, from the secretion of the suprarenal bodies. The former is a powerful vaso-dilator, and causes a reduction of the arterial blood pressure. The latter is a vaso-constrictor, and produces increased arterial tension. Normally these secretions counterbalance one another. Thus, if the vaso-dilator be deficient, there will be a constriction of arterioles, the renal vessels partaking in the general constriction. Consequently there will be a diminution in the excretion of urine, and a decreased elimination of the imperfect products of metabolism. These poisonous substances circulating in the system set up changes in the various organs, and produce a set of symptoms, which we are familiar with as eclampsia. He sums up the explanation of this theory by saying that "the degree of toxaemia of pregnancy comes to be dependant, directly or indirectly, upon the quantity and activity of the thyroid secretion." The thyroid gland must therefore be given a primary role in the causation of eclampsia, if this theory is correct.

Nicholson's results in a few cases of eclampsia, or patients in the pre-eclamptic state, by treatment with thyroid gland seem to be very satisfactory. This theory undoubtedly helps us in the great primary questions of how the toxins are produced and increased, and of how their elimination is decreased.

If some such theory as the above is not correct and accepted, we are still completely in the dark as regards the formation, and defective elimination of the poison producing eclampsia.

At the end of pregnancy the amount of tissue metabolism is very great, and if from some unknown cause an imperfect product of such a metabolism, or some poison from without, be suddenly introduced into the system, the organs which under ordinary circumstances deal with such toxins, and which at this time are probably working to their greatest extent, may find that the strain of this increased work is beyond them. As a result of this failure of function of these organs, a toxin in increasing amount circulates in the system. This poison causes changes in the various tissues of the body, and produces the set of symptoms found in eclampsia. This vicious cycle, thus set up, quite accounts for the sequence of events found in eclampsia, up to a certain point, namely, that at which in favourable cases improvement occurs. The duration of eclampsia rarely exceeds forty-eight hours. After a varying time there is a sudden cessation of eclamptic symptoms, the urine increases in amount, and the normal functions of the body are resumed, as far as can be ascertained. It is precisely at this time of the sudden disappearance of the symptoms of the disease

that the above theory is defective, and no explanation has been found to fit in with the events that occur. It is true that increased metabolism is brought about by muscular action, and it may be that the convulsions themselves, which consist in very violent muscular action, cause a very greatly increased metabolism which is beneficial to the patient. This method of dealing with tissue change is, of necessity, a dangerous one for the patient, but it is the lesser of the two evils, convulsions and death from coma due to prolonged toxaemia.

If it had been proved that eclampsia was due to the action of organisms, the explanation of the cessation of the symptoms would be that antitoxins were manufactured, and neutralised the action of the causative bacteria. The bacterial theory has such a weak foundation that it cannot be entertained at present.

The fact remains, however, that the aetiology and pathology of eclampsia are still in a very imperfect state, and even the generally accepted toxaemic hypothesis still requires confirmation.

SYNOPSIS OF THE RESULTS OF SIX POST-MORTEM EXAMINATIONS PERFORMED ON PATIENTS DYING FROM PUERPERAL ECLAMPSIA IN THE GLASGOW MATERNITY HOSPITAL

No. of Case	Kidneys	Liver	Spleen	Lungs	Heart	Other Organs
1	Right Kidney:- small; capsule adherent; Cortex small and pale Left Kidney:- the same as above	Normal	Normal	Right Lung:- old pleural adhesions; Emphysema; Oedema Left Lung:- do. do.	Healthy Pericardium fatty	Engorgement of the pia mater. No cerbral haemorrhage or oedema
2	Both Kidneys:- showed parenchymatous changes	Enlarged; passive hyperaemia	Passive hyperaemia	Right Lung:- Voluminous; oedematous Left Lung:- Retracted; signs of old disease; slightly oedematous	Small antemortem clots in right and left ventricles Pericardial fluid = 4 ozs.	
3	Right Kidney:- Large infarction; cortex yellow and anaemic commencing necrosis Left Kidney:- hyperaemic	Liver cells show cloudy swelling changes	Normal	Healthy	Normal in size Right Auricle and both ventricles have ante-mortem clot. Perecardial fluid = 3 ozs.	
4	Kidneys show chronic insterstitial change Right Kidney contains a small cyst	Enlarged; fatty	Sago Spleen	Oedematous adhesions at left base	Hypertrophy of left ventricle	
5						Nothing abnormal found. Brain pale and anaemic
6	Kidneys:- chronic parenchymatous nephritis	Slightly enlarged; Passive hyperaemia (slight) Fatty change (slight)	Slightly hyperaemic	Oedematous	Small fatty. Left ventricle hypertrophied	

PATHOLOGICAL ANATOMY

The pathological findings in cases that have died from eclampsia cannot be said to be constant or uniform. In some cases practically no changes can be found, while in others, morbid changes are quite distinct. As is to be expected from the clinical symptoms, the organs most frequently affected are the Kidneys. The liver also, in a great many cases, shows a certain amount of tissue change. No definite constant lesion has been found that can be considered as pathognomonic.

KIDNEYS: The Kidneys are the organs which present the most constant changes. Hyperaemia is often present, and varies greatly in degree. Very often there is present an anaemic appearance, which is supposed to be due to spasm of the renal arterioles. The cause of this spasm is thought by some to be caused by direct action of the toxins on the vessel walls; or indirectly by emboli blocking the renal vessels, the emboli being produced by a ferment which causes the blood to coagulate. Others maintain that the contraction of the vessels is due to a reflex irritation set up in the genital tract. Such irritation may be the uterine contractions, or the passage of the foetal head through the os uteri. The renal epithelium in this anaemic condition is infiltrated with fat globules. There are found in a certain proportion of cases changes such as one finds in chronic nephritis either of the parenchymatous or interstitial variety (Cases 1, 2, 4 and 6). In such cases the patient has generally been the victim of nephritis prior to the on-

set of pregnancy. Very rarely one finds a condition present which indicates pressure on the ureters. Dilatation of the ureters, hydro and pyonephrosis, and cystic disease of the Kidney may be found, as resulting from this cause. Some observers found the effects of ureter blocking so frequent that they considered this to be the cause of eclampsia.[18 & 19]

In some instances, infarction and necrosis (Case 3) have been found in the Kidney. This infarction formation may be caused by emboli formed in the manner described above, or they may be formed elsewhere and carried to the Kidneys. Necrosis may be secondary to the infarctions, or may be due to an acute toxin action. The macroscopic appearances may be quite negative, but under the microscope, changes, usually epithelial, are found. The epithelial cells are detached from the basement membrane, and are lying loose in the lumen of the tubules. If the toxin be strong these cells undergo granular changes, but if the poison be less irritant, and the process slower, then fatty changes are most marked. The pathological findings can be corroborated clinically by examination of the urinary sediments. The cortex of the Kidney is much more frequently affected than the medullary portion of the organ. The Kidneys in eclampsia present not the features of a nephritis, but those resulting from the action of a toxin, namely, an acute degeneration.[8]

LIVER: The changes in the liver are similar to those found in acute toxaemic diseases. Haemorrhages, varying considerably in size, are found both deeply and on the sur-

face of the organ.

The colour of the liver is more yellow than is normally found, and the haemorrhages appear studded over its surface, as purplish or dark red spots. The yellow colour is due to fatty degeneration of the liver cells.

Haemorrhages are found round the portal interspaces, and present microscopically a varying set of changes. They may at first simply be enlarged intra-lobular capillaries, or later, these dilated vessels have increased in size, ruptured, and are surrounded by secondarily dilated capillaries. The centre of the area is necrotic, consisting of dead liver cells, blood corpuscles, and other debris. In the last stage these necrotic areas coalesce, and the emboli may be carried off from them to other organs. The cause of these haemorrhages is not yet proved, and depends on what theory of the aetiology of eclampsia is believed in, or on the effects of the convulsions. Hence it may be due to poisons, chemical or bacterial, or the result of emboli coming from the syncytium, or due to rupture of blood vessels due to the violence of the seizures.[18]

SPLEEN: In two of the cases mentioned (Cases 2 and 6) the spleen is described as being hyperaemic. This condition was probably terminal. In one case (No. 4) the "sago spleen" was found.

LUNGS: The lungs are found to be congested, especially at the bases. Oedema is also very generally present. Bronchitis and pneumonic areas are found in a great many cases. In Cases 1 and 4 there were pleural adhes-

ions, which were probably of long standing. Ecchymoses are found under the pleura, and in the lung substance. Areas of necrosis may also be present due to a septic pneumonic condition caused by insufflation, or emboli.

HEART: Pericardial effusion was found in Cases 2 and 3. Ante-mortem clot was found in two cases. With chronic renal disease the left ventricle is considerably hypertrophied. Haemorrhages under the pericardium have been described.

BRAIN: As in the other organs, haemorrhages, often very minute, may be present in the brain tissue. Oedema, with slight flattening of the convolutions, anaemia and hyperaemia are the other conditions found in the brain.

THE FOETUS AND PLACENTA: The liver and Kidneys of the foetus often show changes such as are found in the mother. The placenta may exhibit white infarctions.

DIAGNOSIS

The diagnosis of eclampsia is usually easy. There are, however, one or two conditions found in the puerperal state which might be confounded with this disease. These are epilepsy, hysteria, brain lesions, and the rare condition of Status Epilepticus.

Most text books point out the possibility of confusing the seizures in puerperal eclampsia with the convulsions of epilepsy, hysterical attacks, apoplexy and brain tumours.

In epilepsy there is usually a history of the patient having taken fits previously to the onset of pregnancy. The coma of epilepsy is never so profound or so lasting as that of eclampsia. Urinary changes are never found in uncomplicated epilepsy. The involuntary passage of urine and faeces is seldom experienced in eclampsia. In eclampsia there is no preliminary cry, as in epilepsy. Aura are seldom found in puerperal eclampsia. In a few cases of epilepsy, albumen is found in the urine, probably due to a co-existing renal disease, and it is in these cases that difficulty in diagnosis is apt to arise. The history of previous fits, and perhaps pre-existing nephritis will help in making a diagnosis, although it must not be forgotten that all these conditions may be present at the same time. It can be well understood that with an unstable mental equilibrium, such as is present in epilepsy, the presence of even a small quantity of toxin circulating through the brain may be quite sufficient to overthrow the mental balance, and produce true eclamptic convulsions.

In hysteria the manifestations are different from those of eclampsia. During a convulsion in hysteria the patient is never so completely unconscious as she is in eclampsia. Hysterical seizures are usually present in the earlier months of pregnancy. The urine of a hysterical patient never contains albumen. In hysteria the onset of a convulsion is usually gradual, and the patient may scream during the fit. There is no regular sequence of events in hysteria, struggling and throwing about of the limbs alternating with quiescent periods. In hysteria the patient seldom bites

the tongue. Cold douching is often effective in terminating a hysterical seizure. There is no comatose stage after a hysterical attack. A patient was admitted to the Maternity Hospital with supposed eclampsia. Shortly after admission the woman took a seizure, which was not typically eclamptic. She cried out, and began to struggle violently. The urine contained no albumen. Cold water was applied to the face, and the patient quickly recovered. It was discovered afterwards that this patient had fallen on the sacral region whilst descending a stair. The nervous seizures had occurred secondarily to the accident.

Convulsions from brain lesions:- A gross cerebral condition such as a brain tumour has a probable history of previous convulsions, localising symptoms, pain over the localised area of the skull, and optic neuritis. Unconsciousness in a cerebral condition arising from a haemorrhage or an embolus is sometimes difficult to distinguish from the coma of eclampsia. The history of the occurrence of the coma, lack of history with regard to premonitory symptoms, and localised paralysis generally help in making a diagnosis. Diminution in the excretion of urine is not so pronounced in apoplexy, but there may be marked albuminuria.

Eclampsia and apoplexy are apt to occur in patients with much the same conditions present, namely, renal change and arterio-sclerosis. Apoplexy may occur, however, during an eclamptic seizure, and the definite symptoms of either condition may be masked. The presence of a cardiac valvular lesion may help in the diagnosis of embolus.

The most important time to make a diagnosis of eclampsia is before the convulsions supervene, that is, in the pre-eclamptic stage. In hospital practice, however, this is not always possible, as the great majority of eclamptics are sent in, in the convulsive stage. In every case of pregnancy that comes under one's care, frequent routine examination of the urine should be made. If albumen is found to be present in increasing amount, and if the excretion of urea becomes markedly diminished, one should be forewarned that the patient is in danger of eclampsia. If, in addition, there be signs of toxaemia, such as headache, oedema, epigastric pain, and eye symptoms, then convulsions will certainly supervene, unless active treatment is immediately carried out.

The differentiation of the status epilepticus in the pregnant from eclampsia is a matter of some difficulty. An attack of status epilepticus at its commencement simulates eclampsia very closely. In the only case of this disease that came under my care[15] the appearance of the early convulsions was extremely like that of eclampsia. There was also a small quantity of albumen in the urine. As the fits became more numerous, however, the albumen gradually disappeared from the urine, the quantity of which was never markedly diminished. The seizures themselves changed in character, becoming shorter in duration, more unilateral, and having practically no comatose stage. These facts showed that the case was not one of true eclampsia.

PROGNOSIS

The prognosis in eclampsia is always grave. The maternal death rate is a high one, from 20 to 30 per cent. In my series of cases the mortality was 22 per cent.

The death rate in multiparae is said to be much higher than that of primiparae. In a series of cases collected by Jellett,[18] 25 per cent of the deaths occurred in multiparae, and 17.5 per cent in primiparae. Schauta[8] in 306 cases found the death rate in multiparae to be 44.9 per cent, and in primiparae 37.3 per cent.

Kerr found that the greatest mortality occurred in primiparae. In his 80 cases, 23 per cent were multiparae, and 42 were primiparae. He gives Duhrssen's and Goldberg's figures, which are respectively 19 and 21 per cent in primiparae, and 28 and 45 per cent in multiparae. Olshausen found the mortality in multiparae and primiparae about the same.[2]

In my series of cases I found that the mortality in primiparae was greater than that of multiparae. Of the fatal cases, 72.7 per cent were primiparae, and 27.2 per cent were multiparae.

The death rate does not seem to be affected by the age of the patient, although the average age in my fatal cases was 27 years, which is rather high.

Generally speaking, the greater the number of convulsions the higher the death rate. Authorities seem agreed on this point. Herman[8] states that if the fits exceed twenty in number, 50 per cent of the patients die, and if the fits exceed fifty, almost all the patients die.

Bidder[2] found that where the fits were more than sixteen then 43 per cent of the patients died, while in those with less than sixteen, the death rate was 7.5 per cent. In fifty cases of eclampsia occurring in the Glasgow Maternity Hospital, collected by Dunlop, the average number of fits taken by the fatal cases was twelve, and the average number taken in favourable cases was nine.

In my series of cases I am unable to give definite figures as to the number of fits taken, as uncounted convulsions occurred before admission to Hospital in some cases, and in others the seizures were continuous, and could not be satisfactorily distinguished from one another. The greatest number of fits taken by any one of my fatal cases was twenty-five, and the greatest number taken by any one of the favourable cases was over one hundred and eighty-two (Case IV).

I consider much more important points in giving a prognosis to be the severity of the convulsions, the depth and duration of the coma, and the incidence of complications. In Case XXVIII the patient had taken only four convulsions, which were very severe and lengthy. The coma following them was also very deep, and was never entirely gone. The patient died three and a quarter hours after the onset of the first seizure. When she was admitted to Hospital there was little difficulty in giving the gravest possible prognosis. Case XLIII died five hours after the onset of the first convulsion. She only took two seizures. They were very severe, and of long duration, and there seems to have been no return of consciousness between the first and second fits, and the second fit and death. In the other

fatal cases in which death occurred from the poison of eclampsia, and not from complications, it will also be seen that the seizures were severe in all three stages. In cases such as the two above mentioned, where the convulsions were few in number, but the coma deep and of long duration, the cause of the fatal termination is due in all probability to the quantity and quality of the toxin present in the body. Hence a fairly accurate prognosis can be made by paying careful attention to the two factors described, the characters of the convulsions and of the coma. In a case with a long severe seizure and a deep lengthy coma, and an interval between the fits taken up by total unconsciousness, the prognosis is of the worst. A less gloomy outlook may be taken if the coma is short (especially after the last fit) and consciousness returns between the paroxysms, even although the fits appear severe and are of long duration. The most hopeful cases are those in which the seizures are few in number, short in length, mild in character, and the supervening coma light and transitory. I do not think the prognosis is greatly altered in the few cases where an isolated fit, or series of fits, occurs some hours after the supposed termination of the disease. In the intervening time between the first and second attacks the patient is semi-conscious. Two such cases are mentioned in the Glasgow Hospital Reports. The first terminated fatally. The patient, a primipara, aged 28 years, took ten convulsions before delivery (accouchement forcé). On the fourth and fifth days after the delivery she had seven fits, after which she died. The second case ended in recovery, two seizures occurring

eighteen hours after the first convulsive period had passed. Three of my cases had this condition of what might be called delayed convulsions. They all terminated in recovery. In Case IV, nine hours elapsed between the cessation of the first attack and the three fits which constituted the second. In the period between these attacks the patient was semi-conscious. The second attack seemed to have been stimulated by abdominal palpation. In Case XIII, after thirty-three hours an isolated convulsion occurred. In Case XIX, five and a half hours elapsed between the main attack and two isolated convulsions.

Eclamptic convulsions that commence before or at the onset of labour have a much graver prognosis than those that occur later.

Of my fatal cases which took convulsions in pregnancy, the percentage was 45.4 per cent. Those cases that commenced labour before the onset of the seizures had a mortality of 36.3 per cent. In the post-partum cases the death rate was 18 per cent. It must be remembered that one of the puerperal cases died from septicaemia, which was an accidental circumstance, and bore no relation to the eclamptic condition.

The following table[2] gives an idea of the death rates, found by different authorities, in the various epochs of the puerperal state:-

	Pregnancy	Labour	Puerperium
Olshausen	25 per cent	25 per cent	25 per cent
Zweifel	17 " "	14 " "	10 " "
Duhrssen	38 " "	19 " "	12 " "
Kerr	43 " "	28 " "	0 " "

It has been stated that the cases which take convulsions early in pregnancy have a slightly less favourable outlook than those in which the convulsions occur at full time. In my series of cases, however, I found that the most fatal period was towards the end of the gestation, and at the commencement of labour. By observers of great numbers of cases it has been noticed that the later in labour a patient is, the more favourable the prognosis. It was also discovered that cases, in which the convulsions ceased, or were of much less severity after the birth of the child, were more favourable. It may be mentioned here, one cannot be sure that convulsions will abate after the birth of the child, therefore it is unwise to bring about forcible delivery of the child before other means of treatment have been tried, and have failed. Eclampsia occurring after the birth of the child is certainly the most hopeful. In my list of cases two post-partum cases ended fatally. One of these, as has already been mentioned, died of septicaemia, and can therefore be disregarded. The other succumbed from the effects of the eclamptic seizures. Late post-partum eclampsia, that is, eclampsia which occurs more than twelve hours after delivery, is supposed to be less favourable than eclampsia occurring immediately after delivery.

The following table shows the time of onset of the eclampsia in relation to labour, and the ultimate result, in the reported cases:-

	Recovery	Death
Pregnancy	19	5
1st Stage of Labour	8	3
2nd Stage of Labour	5	1
Puerperium	7	2

It is said that death of the foetus in utero has a favourable influence on the disease, both in regard to decreasing the number and severity of the convulsions, and diminishing the albuminuria. This was not the case in my experience. In Case XXI the child died, presumably during the attack before admission. About fourteen hours afterwards she had a second attack of convulsions, there being seven seizures which were severe. The albumen in the urine was also abundant. In Cases XIV and XLVI the child remained alive, after the cessation of the fits, and the albuminuria diminished considerably.

Of necessity it is very difficult in most cases to say whether the death of the child does or does not improve the condition, as in my experience the foetus in utero usually dies after several seizures have occurred. and just prior to the period when improvement might be expected. I think it would be erroneous to conclude that the death of the child was the cause of the improvement, as in the greater proportion of the fatal cases it will be seen that the foetus also died in utero before the cessation of the convulsions. Further, it will be noticed that from the three cases cited above the convulsions can recur after the death of the foetus, and also the cessation of the fits may take place while the foetus remains alive.

To draw any exact conclusions with regard to the influence of the foetal death on the albuminuria is almost impossible, because treatment must be adopted at once which renders any observations made in this connection valueless.

The quantity of urine secreted is one of the most important factors to be considered in giving a prognosis in any case. The smaller the diuresis, especially after delivery and treatment, the graver the outlook. With complete suppression of urine the prognosis is very bad indeed. If, after the convulsions cease, the urinary flow commences and gradually increases in quantity, then the prognosis is good.

It must be remembered, however, that a long delayed increase in the amount of urinary secretion, especially in cases where there has been almost total suppression, does not alter the very grave prognosis given in such cases.

The quantity of albumen in the urine during the convulsion taking period may influence the outlook somewhat, but I do not think it does so to any extent. Nearly all the reported cases had albumen present in great abundance. The same may be said of blood and tube casts.

In Case XXXVIII the urine when drawn off at the time of taking convulsions was very much like hare soup in colour and consistence. On the urine standing for some time, the sediment occupied more than one half of an ordinary urine specimen glass. Microscopically the sediment consisted almost entirely of tube casts, of all descriptions. This urine was the worst in this respect I have ever seen. The patient made a good re-

covery, leaving the Hospital without a sign of albumen or tube casts in the urine.

The temperature does not offer much help in considering the outlook of a patient suffering from eclampsia. In some cases it will be seen that the patient dies with a very high temperature, while in others the temperature is normal or sub-normal.

The pulse also is an uncertain guide per se in giving a prognosis. It is true that a rapid, feeble, irregular, and low tension pulse is of serious import, but this condition may be greatly improved by drugs, and saline transfusions. The prognosis is thus entirely altered.

The prognosis for the child in eclampsia is very bad. There are several reasons for this high mortality. There is no doubt that the maternal convulsions are the cause of the foetal death in most cases. When accouchement forcé is adopted in cases of eclampsia, occurring in pregnancy or an early first stage of labour, or forceps applied in cases in the second stage of labour, the death rate of the children is markedly reduced.

The circulation of toxins in the foetal body probably make it less able to withstand the convulsive seizures.

Again, a certain proportion of the children are under, or just at the viable age, so their power to resist undue strain is practically nil. In multiple pregnancies also the children are generally smaller, and their strength is less. Dunlop[5] found in one of his eight cases that the foetal heart could be heard after the patient had twenty-six seizures. The child, however,

was still-born. He explains this occurrence as being due to haemorrhages, and a partial separation of the placenta. And he thinks that these placental apoplexies may be the cause of foetal death in many cases. In view of this explanation, I examined the placentae in most of my cases, and on comparing them with the placentae of normal cases, I was unable to say that placental haemorrhages occurred more frequently in eclampsia.

The foetal statistics of my cases are as under:-

Occurrence of Eclampsia	No. of Cases	Children Alive	Children Dead
Pregnancy	24	2	22
Labour	17	8[x]	11[x]
Puerperium	9	8	1

[x] One case of twins occurred in these columns.

In the four cases that were dismissed undelivered the children are included in the "dead" column, as the foetal heart could not be heard, or the foetal movements felt, on dismissal of the patient.

Twenty-three viable children died. In seven cases the children were non-viable. In four cases the viable children were undelivered.

54 per cent of the children that died in my cases had reached the viable age.

This compares with[2]

77	per	cent	Winckel
67	"	"	Green
57	"	"	Kerr
49	"	"	Duhrssen
34	"	"	Zweifel
28	"	"	Olshausen
23	"	"	Bidder

If the children are forcibly delivered at the commencement of the convulsive seizures then the death rate

is reduced.

It is well known that the child of an eclamptic patient when born alive frequently suffers from convulsive attacks. Fitzgerald[25] cites a case in which the child had thirty-nine fits in the first few days after delivery, and made a good recovery. Jardine[12] mentions two cases in which the children were born dead. The first child had its arms flexed and rigid, and the second was in a state of complete rigidity. He states further that after ten maternal fits there is little chance for the child. If it be born alive, death may occur from convulsions or debility. Only about 40 per cent of these children ultimately survive.

The children of eclamptics, born alive, in my cases seemed healthy and likely to survive.

TREATMENT

The treatment of puerperal eclampsia at the present time is for the most part empirical, and it must of necessity be so, as the aetiology and pathology of the disease are still so little known. The treatment that is employed may not be rational or scientific, in as much as it may not influence the real cause of the disease, but it has nevertheless the effect of benefitting certain morbid conditions, which, if not removed or ameliorated, produce a state of matters which ends in eclampsia.

It is known that suppression of urine, total or partial, occurs in puerperal eclampsia, and a persistence of such a condition usually ends in death, hence a line of treatment is indicated, which will cause the

Kidneys themselves to act vigorously, or will stimulate other excretory organs to greater efforts. To some extent, therefore, this method of treatment can be called rational, and it is certainly scientific.

It is true we have no specific for this disease, yet recovery is materially aided by certain lines of treatment which have been adopted. Treatment of eclampsia may be divided into prophylactic and curative.

PROPHYLAXIS: There is no doubt that in a great many patients exhibiting symptoms of the pre-eclamptic state, active treatment has the result of warding off the actual seizures of eclampsia, and of bringing pregnancy to a successful termination. The efforts of prophylactic treatment are: (Firstly) To reduce to minimum the metabolism going on in the tissues. By so doing, the already damaged organs of elimination are relieved of the duty of trying to throw off an ever increasing quantity of waste material. Rest in bed is beneficial in bringing about the first indication. By placing the patient on a bland fluid diet the production of waste material is also reduced. (Secondly) Diluent fluids such as milk and imperial drink are given in as large quantities as possible, so that the poisons may be quickly washed out of the system. It is unlikely at this stage of the disease that there will be any obstruction to the passage of water through the renal tubules, owing to inflammatory changes.

Quantities of fluid given by the mouth, or in severer cases transfused, have the effect of diluting the toxic agents circulating in the blood, hence the renal

epithelium does not suffer to so great an extent as it would by being brought in contact with a more concentrated poison.

One must not neglect the other organs of elimination, namely, the bowels and the skin. The bowels should be freely moved daily by purgatives which produce watery evacuations. Salines are preferable, although hydragogue cathartics may be exhibited.

It is usually sufficient to keep the skin acting gently by keeping the body surface at a warm equable temperature. As a rule this treatment is all that is necessary to cause improvement of the symptoms of the pre-eclamptic stage. If, however, the symptoms persist and increase, further treatment is indicated. This usually consists in making further calls on the eliminative organs, on the skin by means of hot wet packs and steam baths; on the Kidneys by saline transfusions, hot applications, and cupping; on the bowels by drastic purgatives. In several cases admitted to the hospital while I was in charge, pre-eclamptic symptoms were present. These symptoms consisted of headache, oedema, and albuminuria in most of the cases, and epigastric pain was felt in one case. Under a course of treatment such as is described above, these cases improved and terminated successfully.

The question of emptying the uterus now comes to be discussed in event of failure of these remedies.

Improvement in the pre-eclamptic state usually follows evacuation of the uterus, although when convulsions have actually occurred, a similar result cannot in all cases be expected. Induction of labour by

Krause's method is probably the best one to employ. If the foetus be viable and likely to survive, then there need be no delay in carrying out the induction of labour. If the child be not viable, or so premature as to almost certainly die, then the operation should be delayed as long as possible, in the hope that either the toxaemia will disappear, or that it will not increase, and allow of the child reaching an age at which its life will be tolerably secure. It must not be forgotten, however, that to delay this treatment until the onset of the convulsions is to prejudice the life of the child very greatly, as only a small proportion of the children born after the incidence of the fits survive.

In some cases in which the symptoms of toxaemia came on early in pregnancy, or in which the foetus is dead, it may be necessary to induce labour at once in order to avoid the risk of the patient taking eclampsia.

In one of my cases the pre-eclamptic stage was well marked, there being present headache, increasing oedema of the face and lower extremities, increasing albuminuria (latterly 8 parts per thousand per Esbach's tube), and a decreasing quantity of urea. Optic neuritis and retinitis were so marked that the patient became totally blind. The patient was in the 36th week of pregnancy, and the child was alive. Induction of labour was performed by Krause's method. The child was born alive, and ultimately became strong and healthy. The symptoms of the preliminary stage of eclampsia abated, and although there was always a distinct trace of albumen in the urine afterwards, the patient enjoyed good health. It took four months for the eyesight to recover suffi-

ciently for the patient to be able to read.

Lavage of the bowel has been recommended as an adjunct to prophylactic treatment.[19]

The beneficial use of thyroid extract in this connection will be alluded to elsewhere.

CURATIVE TREATMENT: The methods of treatment of puerperal eclampsia are numerous, and the advocates of each line of treatment seem to be equally convinced that the particular method adopted by them has succeeded in lowering the death rate very markedly. This condition of affairs is to be expected, where the cause of the disease is so much a matter of controversy and speculation.

In my opinion, treatment divides itself into three main issues, namely: (1) The arresting of the convulsions. (2) The termination of pregnancy. (3) The prevention of complications. As the treatment of this third point often overlaps that of the first division it will be discussed when convenient with it.

Numerous methods and drugs have been employed in endeavours to arrest and diminish the number and violence of the convulsions.

CHLOROFORM: The use of chloroform is recognised as beneficial in controlling the convulsions of eclampsia.

Personally I have not had much experience of this drug in this disease. When any operation such as catheterisation, transfusion or examination was to be performed, chloroform was often given in my cases. I observed in these cases that no convulsions occurred for some hours after the administration of the drug, only to re-appear later. This temporary sedative action

was due in all probability to the anaesthetic. It seemed to me that it tended to increase the pulmonary irritation in many cases. It also has a depressing effect on the heart, and it was for these reasons that I did not administer the anaesthetic oftener. Many writers advise that the drug should be given just before the fit commences, but in my experience this is too late to arrest the impending seizure. The signs I took to indicate the approach of a fit being increase in the number of the respirations, pallor of the face, and dilatation of the pupil. In Case XXXVI chloroform was given to control the convulsions, and perform phlebotomy and transfusion, and although a series of convulsions was cut short the pulse became very weak and irregular. In a case of Status Epilepticus, which was sent into Hospital as eclampsia, I gave chloroform continuously for several hours on several occasions. If the patient were placed very deeply under the anaesthetic, convulsions were controlled, but the pulse became very weak, and the respirations threatened to cease. If the drug were given so that the patient was moderately anaesthetised, the convulsions continued, although in a modified degree. The lungs tending to silt up, the retention of the mucus in the trachea and bronchi, and the increasing tendency to cardiac and respiratory failure were contra-indications against the continuous use of chloroform in this case. She had 318 convulsions in three and a half days.

Playfair[14] advocates chloroform in the following words:- "The great indication in the management of "eclampsia is the controlling of convulsive action by

"sedatives. Foremost amongst them must be placed the "inhalation of chloroform."

Galabin[3] says of the action of chloroform:- "The "administration of chloroform is the most valuable rem- "edy of all. It has a great influence in preventing the "recurrence of fits. The arterial tension is lowered, "and the pulse at the same time becomes slower. Rest- "lessness is diminished, contraction of the pupil passes "off, and usually the breathing becomes less stertorous, "and the venous congestion of the face diminishes."

"The seizures are best controlled by chloroform, "which should be given freely to begin with, and again "every time the convulsions occur." (Fothergill[26])

Trousseau[27] says that chloroform holds the first place amongst the anti-spasmodics which are indicated in the treatment of eclampsia.

"As fits are provoked by peripheral stimuli it "would seem good practice so to treat the patient as to "protect her nervous system from such stimuli. This "can be done by keeping her anaesthetised by chloro- "form." (Herman[8])

CHLORAL HYDRATE: In almost all my cases was chloral given with beneficial results. The action obtained is similar to that of chloroform, but is more uniform, and is without the pulmonary irritation. Large dose must be given in order to obtain the desired result. The objection to chloral hydrate is its depressing effect on the heart. It has been noticed in some cases that after severe seizures have been controlled by the drug, the heart's action becomes feeble and irregular. Chloral is said to have the advantage over chloroform in not

requiring the continuous presence of a physician during its use. It has been my experience in puerperal eclampsia that the medical attendant cannot be absent for any prolonged interval, as the disease is acute and ever changing.

Several obstetricians give accounts of splendid results obtained by the use of chloral hydrate. Winckel had only seven deaths in ninety-two cases.

In most of my cases the drug was given per rectum.

POTASSIUM BROMIDE is usually given in conjunction with chloral as a cerebral sedative.

MORPHIA: The use of morphia in eclampsia has given rise to much controversy, and advocates both for and against its use seem to be equally strong in its favour and in its condemnation.

The German school of Obstetricians use morphia freely in the treatment of eclampsia. Veit[28] records a series of sixty cases of the disease, treated with this drug, in which the death rate was 3.3 per cent. His method of administering the drug was by giving an initial dose of morphia grain $\frac{1}{2}$ hypodermically, and grain $\frac{1}{4}$ two hourly until the convulsions ceased. Not more than 3 grains of morphia, however, were to be given in the twenty-four hours. Bidder, and other German physicians, also recommend the drug, but in smaller doses than Veit used, as they noticed on several occasions that symptoms of morphia poisoning occurred after its administration in large quantities.

The Dublin Rotunda Hospital physicians are most strongly in favour of this treatment, having a death

rate of 25 per cent, which is the same percentage as Olshausen found using this drug. It is the practice in the Olshausen clinic to empty the uterus as quickly as possible, in addition to the morphia treatment.

In America also, the drug is largely used, and large doses are given.[3] As much as 12 grains hypodermically have been given in four days.

Morphia is said to check the convulsions as effectually as chloroform, and to be less of a cardiac depressant. In addition, it is supposed to relax the arterial tension, through the nervous system, hence the renal arterioles are dilated, and diuresis results.

The administration of the drug is also regarded by some as a temporary hindrance to metabolic processes in the body. The drug is usually given hypodermically. These advantageous properties, however, are for the most part purely theoretical, if not fallacious.

Jardine[12] states that after giving morphia a fair trial, he has entirely abandoned its use, as he is convinced that it decreases diuresis. For controlling the convulsions he believes that the other drugs at our disposal are as efficient, and much less harmful. Personally, in the treatment of all my cases morphia was never once given. One or two cases had morphia administered before admission. Case XXIV was admitted with obvious signs of being under the influence of morphia. In this case it was with the greatest difficulty that the excretion of urine could be raised. Jardine[12] mentions a case that died after the giving of morphia, the pupils being contracted to the "pin point" size, and the urine being totally suppressed.

VERATRUM VIRIDE has been used in Great Britain and America in the treatment of eclampsia. It is used hypodermically in 10 to 20 minim doses of the liquid extract. It reduces arterial tension, reduces the pulse rate, reduces the temperature, and produces diaphoresis. Hirst[29] uses this drug in nearly every case. Mangiagalli[30] writes an interesting article advocating the use of this drug. He states that the drug is contra-indicated when "the "pulse is rapid and small, and the arterial tension but "slightly elevated." It is precisely in this class of case that a drug is required which will act as a specific in curing the disease. It is comparatively easy to treat an eclamptic patient, with a high tension pulse and one in which the skin is not acting, by other and more efficacious methods. The statistics published by him are slightly misleading, as he excludes moribund and cerebral haemorrhage cases. In the Glasgow Maternity Hospital, Veratrum Viride was freely used without any apparent increased benefit.

PILOCARPIN is mentioned in the treatment of eclampsia. It causes diaphoresis, and a reduction in the arterial tension.

I have no personal experience of this drug. Other safer methods of reducing the tension in the arteries should be adopted, as pilocarpin causes an increased secretion from the bronchial tubes and oedema of the lungs - conditions that occur only too frequently in eclampsia. For these reasons I consider pilocarpin undesirable, and not to be recommended in the treatment of this disease. Herman[8] states that pilocarpin causes

a sweating of the bronchial tubes as well as of the skin. Galabin[3] writes that it is dangerous where deep coma exists, for if profuse bronchial secretion occur, the patient will be asphyxiated with this secretion.

THYROID EXTRACT: Nicholson of Edinburgh[24] recommends the administration of thyroid extract, both in the prophylaxis and curative treatment of the disease. As a prophylactic dose he recommends five grains of the extract night and morning. When convulsions are present he gives full doses of the extract hypodermically, one or two hourly, until the signs of eclampsia subside.

If his theory of thyroid deficiency be correct, then this line of treatment is a sound one, but more evidence on this subject is required.

BLEEDING is employed at the present day with advantage in certain cases. It is not the custom now to perform venesection in every case of eclampsia, although it was practised in nearly all my cases. In cases where there is engorgement of the right side of the heart, and oedema of the lungs, it is of service. With high arterial tension, phlebotomy is distinctly of advantage. Its action in this connection is very transitory, but it allows of time for other methods of treatment to act. It has the effect of quietening the patient, and perhaps reducing the number and severity of the convulsions. Perhaps is the word used advisedly in the above sentence, for one generally gives a saline transfusion, or some drug, at a short interval of time from the withdrawal of blood. Thus it is difficult to definitely assert that the bleeding alone has had the effect of reducing the number or

severity of the seizures. It is known, however, that in similar uraemic conditions the cerebral activity is much reduced by blood letting, and it is reasonable to suppose that the same action takes place here. It has been argued against phlebotomy that its beneficial action is very transient, that the quality of the blood is reduced, and that it has not yet been proved that high arterial tension is a disadvantage. The cases in which I performed the operation seemed to be distinctly benefitted. In these cases there was generally high arterial tension, and venous engorgement, as was shown by the cyanosis, enlargement of the right heart, oedema of the lungs, or subcutaneous oedema. Plethoric patients can lose a pint of blood with advantage.

One of the objections to phlebotomy states that it reduces the quality of the blood. This may be so, but at the same time the toxin in the blood is also reduced. Therefore the cerebral centres are supplied with blood, which contains less poisonous material, and there will be a tendency towards the reduction of the number of the convulsions and the depth of the coma. This, in my opinion, is the great benefit to be derived from this practice. There is little risk in withdrawing 16 to 20 ounces of blood from the majority of patients. Most women can conveniently stand this amount being lost post partum. It has been the custom in the Glasgow Maternity Hospitals to encourage a certain degree of post partum haemorrhage in eclampsia, but unfortunately it seldom occurs.

In Case I there was slight ante-partum haemorrhage, but it was too trifling to be of much service in the

reduction of the toxins. In two cases of eclampsia which were in Hospital when I came on duty there had been accidental haemorrhages, which is a very rare condition indeed. It will be noticed that in certain cases (Cases XVI, XXIII, XXVIII, XLIII, XLIV) venesection was not performed, as the patients were too ill to attempt such a procedure.

SALINE TRANSFUSION OR INJECTION: There is no question that the transfusion of saline solution has greatly aided us in the treatment of puerperal eclampsia.

Dr. Jardine is responsible for bringing this method of treatment before the medical profession in this country. It is now used by the common consent of the obstetrical staff of the hospital in almost every case of eclampsia. Jardine[12] has no hesitation in saying that the death rate has been reduced 50 per cent by this line of treatment. The action of saline solution introduced into the body is as follows:- (a) It causes the Kidneys to act. (b) It dilutes the poison in the circulation. (c) It causes mild diaphoresis. (d) It is a cardiac stimulant.

The most important action is the diuretic one. "The method is based on the assumption that there is a "toxin circulating in the blood. We do not know what "this substance is, and therefore cannot administer an "antidote. Our efforts must, in the meantime, be limit-"ed to freeing the system from it, as quickly as possi-"ble."[12]

Herman[8] states that the urinary secretion is only re-established in from six to twenty-four hours after

the convulsions cease, and that three or four days elapse before the secretion again reaches the normal amount.

The diuretic effect of saline solution can be proved by comparing the above statement with the records of the urinary output on the charts, and in the reports of the cases given. Take, for example, Case XIII in the twenty-four hours following transfusion and cessation of the fits. Fifty-two ounces of urine were excreted, and in the next twenty-four hours 94 ounces were eliminated. Again, in Case XXXI the diuresis on the first day was 11 ounces; on the second day, 72 ounces; on the third day, 99 ounces; and on the fourth day 88 ounces. Jardine[11] in an analysis of urines of cases reported by him shows that "there is marked increase in the excretion of urea "and uric acid, and considering that the urine is tre- "mendously increased after the injection, the amount of "urea and uric acid expelled from the system must be very "great."

The second action of saline solution introduced into the circulation is to dilute the poison. The length of time that the extra fluid remains in the circulation and tissues is rather transitory, as a diuresis is set up in most cases almost immediately. In the meanwhile, however, the toxins are diluted, mixed with this solution, and carried off by the increased action of the Kidneys. This action is well seen in uraemic patients who are suffering from a first acute attack of nephritis. The effects of the diluting fluid in the circulation are observed in, as much as before diuresis sets in, the convulsions become less frequent, the patient becomes quieter, and the tendency is towards consciousness.

The diaphoretic action of the saline solution is often noticed, a patient entering the Hospital with a dry, harsh skin, after transfusion breaks out in a gentle perspiration. This may be due in some measure, but not entirely, to the cardiac stimulation. Of the stimulating effect of saline solution on the heart there can be no doubt. In collapsed conditions after severe convulsions, or after delivery in eclampsia, the advantage following saline transfusion is most marked. There is no doubt that saline solution given indiscriminately, as regards time, and without relation to the symptoms present, is distinctly adverse to the patient. With great pulmonary oedema, general oedema or engorgement of the right side of the heart, the introduction of large quantities of fluid into the venous system only tends to embarrass the circulation still further, and causes an increase of the oedema. If there be any of the above signs present, blood letting is generally performed before the transfusion, and never more than two pints of saline solution are introduced into the body at one time. In the cases transfused by me, without taking off some blood previously, there were never any marked signs of the right side of the heart being unduly embarrassed.

In Case XXXII, before the second saline transfusion was given, the only evidence of engorgement of the venous system was a few moist rales in the lungs. There was no cyanosis or engorgement of the heart. As the patient was continuing to have convulsions, and was becoming weaker, I deemed it necessary to supplement the stimulant given by a second saline transfusion, with the result that a slight general oedema followed. The

immediate effect was that of a cardiac stimulant, and the patient had only one very slight convulsion after this second transfusion.

With undue venous engorgement, bleeding alone is indicated, and personally I have never seen this condition in eclampsia, although in a case of cardiac disease there was a sudden venous engorgement and embarrassment of the heart after delivery, which was entirely relieved, and the patient's life undoubtedly saved, by the immediate removal of a quantity of blood.

It was pointed out to me by an authority on insanity that saline transfusion tended to cause a state of dementia in patients suffering from eclampsia. Taking the fifty cases reported by me, and analysing them, we find that five cases exhibited symptoms of mania or dementia. Two of these cases had mental derangement before the saline transfusion was given. Only two cases were removed for asylum treatment, and ultimately recovered. The other three cases recovered in a few days after the cessation of the convulsions. Considering the nature of the disease under review, and its close connection with the nervous system, it is to be expected that some forms of mental derangement will occasionally ensue, but to lay the blame of this complication on the saline transfusion is absurd.

The saline solution used for introducing into the system is a combination of the salts of acetate of soda and chloride of soda, one drachm of each to the pint of sterile water. Dr. Jardine used the Potash salts at first, but although he never saw any ill effects from them, the feeling was so much against their use on ac-

count of their action on the blood and heart that he substituted the soda salts.

The salts mentioned are dissolved in sterilised water which is at the temperature of 100° F. This solution is passed into the cellular tissue or a vein by means of a sterilised cannula. Jardine recommends the cellular tissue being used, on account of the slower absorption of the saline fluid, and hence the more prolonged action. If the patient is being bled, it is more convenient to inject the solution into the same vein as the blood has been taken from. The mammary region is generally the most convenient before delivery. The breast and surrounding areas are first sterilised. The mammary tissue is grasped in one hand, and drawn away from the chest wall, the cannula is then introduced into the tissues under the gland with the other hand. If this operation be properly performed the flow of the solution into the tissues is almost as quick as when done intravenously, and the pain is very slight. Sub-mammary tissue usually holds two pints without difficulty. If the gland tissue be struck with the cannula, the passage of fluid stops, or is very slow, and the operation is accompanied by much pain.

The lax tissues of the abdominal wall are very convenient for transfusion after delivery.

Saline rectal injections may be given, but the bowel usually rejects the fluid, as the quantity to be given is large.

PURGATION: It is our custom to give a purge to all cases of puerperal eclampsia. The bowels are usually obstinately

constipated, and large doses of purgative medicine are required.

Magnesium Sulphate was used almost exclusively, and it was given in doses of from two to four ounces. If the patient is unconscious, and is unable to swallow naturally, the drug has to be given by the stomach tube. The action of the saline is often greatly aided by the administration of an enema a few hours after the giving of the salts.

Croton Oil is much favoured by certain authorities as a purgative in eclampsia. Its action, however, is uncertain, and cases of Oedema Glottidis have been reported as occurring after its use.[12]

Jalap has also been used with advantage.

The bowel is another channel through which the toxins may be removed from the body, hence if free evacuation is produced, the poison in the body is reduced.

Herman[8] is of opinion that free purgation produces a reflex irritation, and is liable to be a cause of convulsions. He also states that this line of treatment is disadvantageous in that it reduces the quantity of urine, and so interferes with a means of prognosis. I admit that the administration of large doses of salts is likely to produce convulsions by its irritant action on the stomach and intestines, and this was proved in some of my cases by the vomiting of the salts, and the occurrence of convulsions after their ingestion. At the same time the bowel is a most important eliminant organ and connot be neglected.

Smaller doses of Epsom Salts, followed by large enemata, would be perhaps less irritating and just as

effective.

HOT PACKS, BATHS, ETC.: With the same object in view, namely, that of increasing the elimination of toxin, the skin should be made to act freely. This is best obtained by hot wet packs, hot air treatment, and hot baths. In the Maternity Hospital hot wet packs were used, and their action was usually most satisfactory. During the packing process the patient has to be carefully watched, as the pulse is apt to become rapid, feeble, and irregular. From twenty to thirty minutes is ample time in which to produce an efficient result. Hot packs, however, are only accessories in the general scheme of treatment. It would be very difficult, for example, to demonstrate how this treatment alone affects the death rate in eclampsia. In only one Case (No. XLVI) did I try placing the patient in a hot bath, and after fifteen minutes the pulse became so weak and rapid that it was deemed advisable to remove the patient from the bath. The bath had an excellent effect on the skin, and also had a distinctly sedative effect on the patient's nervous system. Repeated 'packing' is useful, if restlessness continue.

Hot applications, or dry cupping, over the region of the Kidneys are useful in promoting a quicker and greater action of these organs, and I think their action was beneficial in the cases in which I used this treatment.

These applications were also used to stimulate the lungs in cases where there was oedema and congestion.

Cold sponging was used in all cases where there was a continued pyrexia.

Various operative measures have been tried in the treatment of eclampsia, and two of the more important are Kidney decapsulation, and Lumbar puncture.

KIDNEY DECAPSULATION has been recommended and performed as a curative method for eclampsia.

Polano[31] performed this operation unsuccessfully in one case of eclampsia, and he attributed his failure to the operation being delayed too long. Johnston[32] gives an interesting account of a successful case. After taking numerous fits, the patient had the right Kidney decapsulated. Diuresis certainly occurred after the operation, although the patient took fourteen convulsions after this procedure. In this case, however, every other method of treatment was attempted before, and particularly after the operation. Morphia was given frequently. Twenty pints of saline solution were transfused after the operation. Chloroform was administered; diuretic and diaphoretic methods were employed. The bowels were thoroughly evacuated. I do not think in such a case that one would be justified in giving the decapsulation of one Kidney credit for the ultimate recovery of the patient.

LUMBAR PUNCTURE: This method of treatment has been tried, but with scant success. Thies[33] performed lumbar puncture in fifteen cases. In undelivered cases this method of treatment was not alone relied on, as phlebotomy was done, a saline solution was transfused, and forcible delivery was also performed. Summing up the results of this treatment, he states that in seven cases the number of convulsions diminished; in only one case did they subside entirely; and in the remainder no benefit resulted. In

several cases, symptoms of irritation were observed some time after the operations. Diuresis was not increased. Seven of the cases died.

I performed lumbar puncture and injected Potassium Bromide into the spinal canal once in a case of Status Epilepticus reported by Dr. Jardine.[15] The cerebro-spinal fluid was ejected under considerable pressure, showing that there was considerable tension in the skull and spinal canal. The result in this case was not a fortunate one.

NURSING

A most important part of the treatment of eclampsia is the general management and nursing of the patient. In the Glasgow Maternity Hospital special nurses are appointed to take charge of the eclamptic case.

Constant and careful nursing is absolutely essential to the ultimate success of treatment in this disease.

At the first suggestion of the onset of a fresh convulsion, a soft resistant material should be placed between the upper and lower jaws, so that the tongue may not be lacerated. A tough indiarubber wedge is used in the Maternity Hospital. One of those instruments is placed in a conspicuous position in each of the various Wards, so that no time is lost in procuring this article when required.

During the seizure, and more especially after the comatose stage has passed, the patient may be violent, and may require to be restrained.

The pharynx must be cleared repeatedly, as froth, mucus and blood frequently collect in this situation, and are likely to impede respiration, and be insufflated into the lungs.

If the patient be able to swallow, she should be encouraged to take large quantities of fluid, milk and imperial drink being most commonly used. If the patient be unable to swallow naturally, it is necessary for the nourishment to be given by a nasal tube.

If the temperature be continuously elevated, the patient should be sponged with ice cold water.

If perspiration be profuse, tepid sponging may be carried out with advantage.

The emptying of the urinary bladder must be carefully attended to.

Frequent change of position is necessary, when there is any tendency towards oedema or hypostasis in the lungs.

In addition, the ordinary careful attention must be given to an eclamptic patient as is given to a normal puerperal case.

A nurse should note the number and character of the convulsions, and the condition of the patient between and after the fits, whether unconscious, or in a state of stupor, delirium, mania or restlessness.

The amount of natural sleep, the quantity of urine passed, the number and character of the motions, the condition of the skin, and the amount of nourishment taken, must be all observed. Any change in the condition of the patient should be noted.

I am of opinion that during the fit taking period,

and provided that the patient is able to stand chloroform, it is better to perform small operations such as phlebotomy, transfusion, passing the stomach tube, passing the catheter, and even examinations abdominally or per vaginam, with the aid of an anaesthetic. In Cases IV and XIX it was very noticeable that after a long period free from convulsions, palpation brought on three fits in the first case, and two in the second. No further seizures occurred in either of those cases.

OBSTETRICAL TREATMENT

The obstetrical treatment of puerperal eclampsia is still a question of much controversy.

There are three important views on this subject, which are worth considering. The first view is that the physician's whole attention should be directed towards the treatment of the convulsions, labour being allowed to come on, and to terminate naturally. The second is that the uterus should be emptied immediately in every case; and thirdly, that a middle course should be taken, namely, of delivering artificially when the parturient canal is sufficiently dilated to enable the physician to apply forceps. It is in very rare cases that the upholders of this view advocate accouchment forcé.

The supporters of the first opinion hold that any manipulations or operations, even when performed under anaesthetics, cause increased irritation, and convulsions. They contend that an eclamptic patient bears such operations very badly. Further, they argue that no bene-

fit accrues from the delivery of the child.

Advocates of the second view point out that uterine contractions cause reflex irritation, and encourage convulsions. They aver also that immediate delivery of the child is beneficial to the patient in causing the fits to cease, or at least in lessening their number and severity.

The second argument in favour of immediate delivery does not hold good in cases of post partum eclampsia.

The third or moderate view I am inclined to favour, as the reported cases were treated by me more or less in accordance with this belief, with good results, and a low mortality.

The cases which may make the advocates of this last opinion perform accouchement forcé are those in which the convulsions are extremely numerous and severe, after all methods of palliative treatment have failed, and in which labour has not commenced, or is extremely slow.

On reviewing the first and second methods of treatment, there seems to be a certain amount of truth on both sides, and it is very difficult indeed to decide which method of treatment is the better.

That peripheral irritation sets up convulsions is agreed on by all, and a method of treatment should be adopted that will least irritate the nervous system of the patient. Whether the natural irritation of uterine contractions, or the artificial stimulation from obstetrical operations is the worse, it would be impossible to say. Statistics are made up in favour of both arguments.

The treatment of eclampsia coming on before labour

has set in, or at the beginning of the first stage of labour, is where the divergence of opinion is mostly seen, as convulsions coming on when the os uteri is fully dilated offer little difficulty in treatment. Delivery in these cases is soon effected naturally or by means of forceps.

Large quantities of statistics have been compiled showing cases where convulsions had ceased with the delivery of the child.

Such statistics are apt to be misleading, and one would like to ascertain the length of time that has elapsed from the cessation of the fits to the completion of labour in each case, and also whether or not the convulsions were decreasing in number and severity before delivery. These observations are absolutely necessary, in order that the emptying of the uterus may not be given credit for an improvement in the condition which had begun antepartum. The cases of delayed delivery which I have already mentioned might be included in such a list of statistics, as the convulsions all occurred antepartum, but there would be no mention of the time elapsing between the cessation of the convulsions and confinements which occurred many days after the disappearance of the disease.

In the Olshausen clinic, a series of thirty-six cases of rapid termination of pregnancy in puerperal eclampsia showed a mortality of 22.2 per cent, a percentage which is certainly not lower than that obtained by less active methods of treatment.[34]

Herman[35] gives a summary of statistics gleaned from many writers, and he finds that in 56 per cent of cases,

convulsions cease with delivery of the child, while in the remaining 44 per cent they continue. In 50 per cent of those 44 per cent the fits were much less severe after delivery. He states that the tendency of convulsions in eclampsia is to become less severe in any case, and also that the disease rarely lasts longer than forty-eight hours. In a case where the convulsions occur in pregnancy or early labour, one would expect a cessation or a diminution in the severity and number of the fits within a reasonable time, whether the patient is delivered or not.

If my cases were analysed for the purpose of statistics with regard to the effect of delivery on the fits, the findings would be erroneous, as in many cases there were no further convulsions after delivery, although delivery had nothing to do with the cessation of the fits.

I will endeavour to take each case individually and describe briefly the relation of delivery to the convulsions.

CASE

I One fit when the head of the child was on the perineum. None after delivery.

II Antepartum, ten fits; Postpartum, three fits (slight).

III " three " " five " All severe.

IV " one hundred and eighty-two fits; Postpartum, none.

V Postpartum Eclampsia.

VI Postpartum Eclampsia.

VII Antepartum, eight fits; Postpartum, none.

VIII Postpartum Eclampsia.

IX Antepartum, thirteen fits; Postpartum, none.

CASE

X Antepartum, seventeen fits; Postpartum, none.

In Cases IV, VII, IX and X the patients had recovered from the eclampsia before delivery was effected. Consciousness had returned before delivery took place in those cases.

XI Antepartum, one fit; Postpartum, numerous fits.

XII Postpartum Eclampsia.

XIII Antepartum, numerous fits; Postpartum, one fit (severe).

XIV " three " " none. Twenty-six days elapsed between fits and delivery.

XV Antepartum, nine fits; Postpartum none. The last fit was at 2 p.m. Delivery took place at 11 p.m. on the same day. In the interval the patient had become semi-conscious.

XVI Postpartem Eclampsia.

XVII Postpartum Eclampsia.

XVIII Antepartum, six fits; Postpartum, none. No fits in hospital.

XIX Antepartum, numerous fits; Postpartum, none. At 10.30 a.m. the last fit occurred. Delivery took place at 4.15 p.m. on the same day.

XX Antepartum, eight fits; Postpartum, none. Over twelve hours occurred between the last fit and delivery.

XXI Antepartum, eighteen fits; Postpartum, none. Eleven days elapsed between the last fit and delivery.

XXII Antepartum, twelve fits; Postpartum, none.

XXIII Admitted moribund. Died undelivered.

XXIV Antepartum, thirty fits; Postpartum, none. Six days elapsed between last seizure and delivery.

XXV Antepartum, seven fits; Postpartum twelve fits.

CASE

XXVI Antepartum, twenty-seven fits; Postpartum, none.

XXVII " " " " " three fits.

XXVIII Admitted moribund. Died undelivered.

XXIX Antepartum, twenty-three fits; Postpartum, none. The severity of the fits became very distinctly less after transfusion.

XXX Antepartum, five fits; Postpartum, none.

XXXI " twenty-seven fits; Postpartum, none. Two days elapsed from the time of the last fit until delivery. Delivery was effected by induction of labour by Krause's method. In this case the patient was 'in extremis' on admission, and I felt sure that any violent operative means taken to empty the uterus would end fatally. I decided to employ a slower method of emptying the uterus, and in the meanwhile to concentrate all my efforts in allaying the convulsions, and in improving the general condition of of the patient. An anaesthetic was not used when introducing the bougies, or during the delivery, on account of the condition of the patient. Forceps were not used in delivery, but the foetal head was assisted manually. The head was very soft, and was hydrocephalic. It could be easily moulded and assisted by the hands.

XXXII Antepartum, one fit; Postpartum, thirteen fits.

XXXIII " numerous fits; Postpartum, none. (The severity of the last seven fits was of the slightest).

XXXIV Postpartum Eclampsia.

XXXV Antepartum, numerous fits; Postpartum, none. There were no fits after 8.10 p.m., 30th June 1908. Delivery

CASE

took place at 12 noon, 1st July 1908.

XXXVI Antepartum, numerous fits; Postpartum, seven fits.

XXXVII " one fit; " twenty-seven fits.

XXXVIII " six fits; " none. Last fit 6.40 p.m., Delivery 11.30 p.m.

XXXIX Postpartum Eclampsia.

XL Antepartum, numerous fits; Postpartum, none.

XLI " two " " one fit.

XLII " four " " ten fits. Severity equal.

XLIII Admitted moribund.

XLIV Antepartum, numerous fits; Postpartum, none. Last fit 3.30 p.m., 31st August 1908. Delivery 12.50 a.m., 1st Sept. 1908.

XLV Antepartum, seven fits; Postpartum, one fit.

XLVI " numerous fits; " none. Thirty-six days elapsed from cessation of fits until delivery.

XLVII Antepartum, six fits; Postpartum, nine fits.

XLVIII Postpartum Eclampsia.

XLIX Antepartum, three fits. As the patient left the Hospital irregularly and undelivered, no after history can be given. There were no fits from 24th September 1908 until dismissal on 6th October 1908.

L Antepartum, several fits; Postpartum, none. The last fit occurred on 24th October 1908. Delivery took place on 11th November 1908.

This brief summary shows that the majority of cases are uninfluenced by the birth of the child.

In a report[5] of seven cases occurring previously in the Glasgow Maternity Hospital, accouchement forcé brought

about cessation of the fits, but six of the patients subsequently died.

It is true that the foetal mortality is much greater when expectant treatment is adopted than when accouchement forcé is performed. In the case of eclampsia, the mother's life is of paramount importance, and as early obstetrical interference causes a greater shock, and hence a graver risk to the mother, this line of treatment must be discarded in favour of a less severe one, even although the child's life is endangered, a life which in any case is likely to be insecure.

The physicians of the Glasgow Maternity Hospital recommend a method of treatment which is a sound one. It consists in making every effort to control the seizures, and bring about an elimination of the toxin. If this fails, and the fits continue to be severe and numerous, delivery of the child is resorted to.

In order to treat puerperal eclampsia successfully it is necessary for the physician to be unbiased by the many methods of treatment formulated by others. A course should be adopted that gives the best results, and which coincides with his views of the aetiology and pathology of the disease.

It is only by doing so that he will be successful in the treatment of the disease.

The physician should also keep himself informed of any new work done on this subject, and try to advance our present knowledge by carefully observing the effects of new treatment he may consider beneficial to the patient.

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C A S E I

DISEASE.

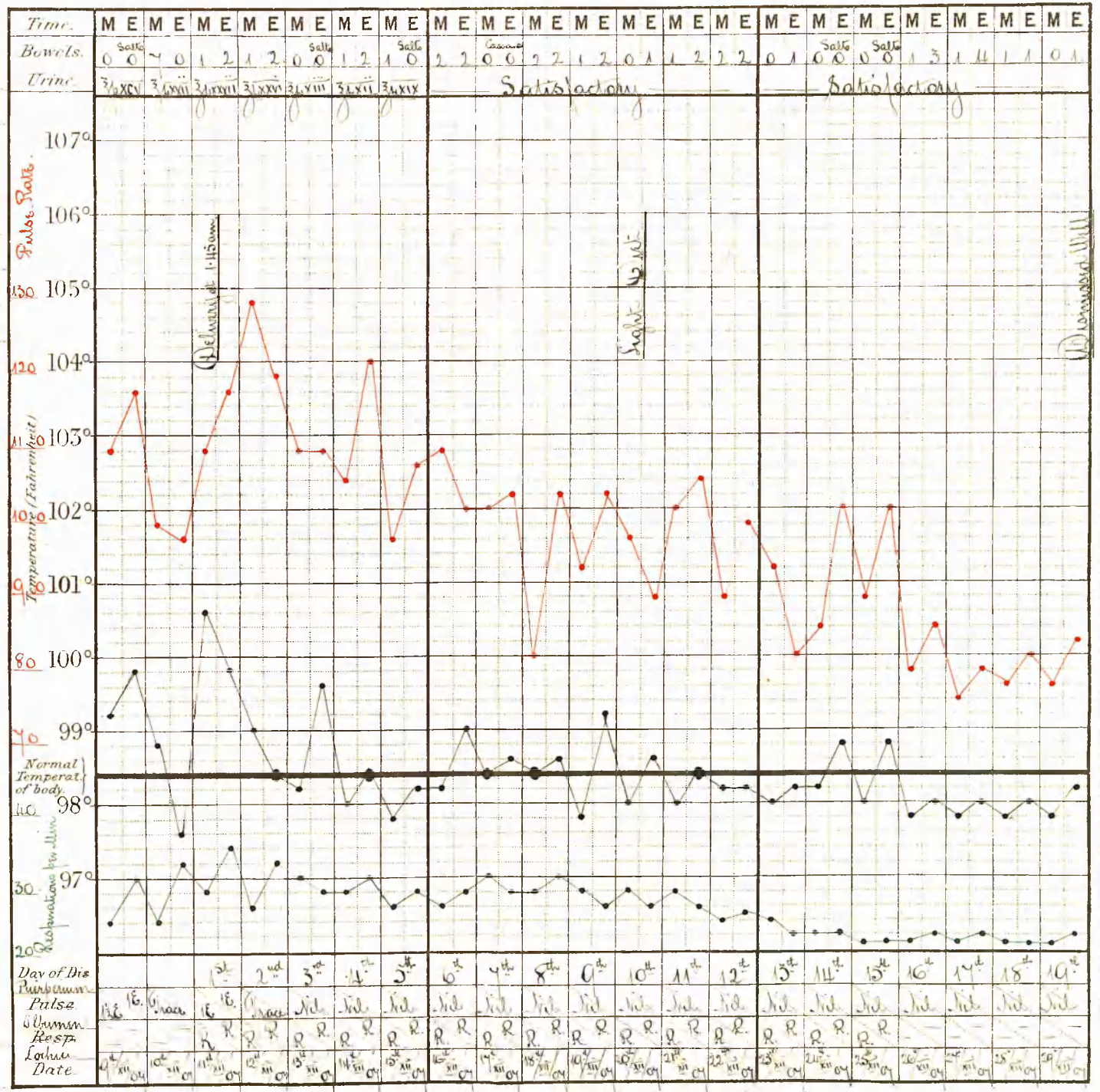
Gambia

Notes of Case.

Name: Keller
Fraser
Age: 25 Years
Sex: Male
Case Book No: 1

1 Para

Date of admission
6th December 1904
Result Recovery



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Gould's Clinical Chart

CASE I NELLIE FRASER, primipara, aet. 28 years. Full time. Recovery.

Admitted on 9th December 1907 at 7.30 a.m.

Delivered on 11th December 1907 at 1.45 a.m.

Presentation vertex.

Child, Female alive, 11¼ lbs. weight.

HISTORY: Beyond the illnesses of childhood the Patient had never had any disease that she was aware of. When 7 months pregnant she noticed that her feet and ankles became swollen on standing for any length of time. These symptoms became gradually worse, and in addition she observed that her eyelids were swollen in the morning during the last five weeks. She had occasional slight headache, but not worse than she had before the commencement of pregnancy. She attended the Glasgow Maternity Hospital as an out-patient and her condition improved under suitable treatment (diuretics and diet). The albumen in the urine coming down from 2 per thousand Esbach to "a trace". Constipation had been present for six days before admission. She was admitted to the Hospital on December 9th 1907 on account of slight uterine haemorrhage. The fundus of each eye had been examined by my predecessor, and he found blurring of the retinal discs.

PRESENT CONDITION: Patient is a large well nourished woman. She has no headache. Temperature is 99.2°F. Pulse is 108 per minute, regular and of fair tension. There is considerable oedema of feet and legs. The vulva is also swollen. Oedema is also present in the abdominal wall.

External abdominal palpation shows the child to be large. The head presents in the first obstetrical position. The foetal heart's sounds and movements can be readily heard and felt. Slight uterine contractions can be felt. Examination per vaginam confirms the above points. There is some slight haemorrhage from the uterus, but no placental margin can be felt. There appears to be abundant urine passed.

Urine. Quantity 98 ozs.; pale straw colour; acid; Specific Gravity 1015; Albumen 1½ per thousand per Esbach; no blood and no tube casts.

Treatment. Milk diet. Imperial drink, Calomel grs. followed in three hours by Magnes. Sulph. 3 ozs. At 10 a.m. hot pack with satisfactory results. Diuretic mixture.

10th December 1907: The slight haemorrhage has now ceased. The os uteri is almost fully dilated. The uterine contractions are fairly strong. This morning it was found necessary to pass the catheter as it was noticed that there was some bladder distension. Even after the voluntary act of passing urine 32 ozs. were withdrawn. Four hours later the catheter was again passed and 30 ozs. withdrawn. The membranes ruptured at 8 p.m. The catheter was passed at 11 p.m. and 20 ozs. withdrawn. About 11.30 p.m. Patient took a typical eclamptic seizure of three minutes duration. The right retina exhibits a slight recent haemorrhage to the nasal side of the macula. There is perhaps a slight indefiniteness of the retinal margin in both eyes.

Treatment. Milk diet. Imperial drink. After the convulsion, 2 (pints) Saline solution was injected into the right mammary region.

Urine. Quantity 72 ozs. Milky (urates), faintly alkaline, Specific Gravity 1012. Albumen trace. No blood or tube casts.

11th December 1907: As the os was fully dilated the patient was anaesthetised with chloroform and forceps were applied to the child's head and child was delivered at 1.45 a.m. Considerable difficulty was experienced with the shoulders which were very broad. The child's right arm was paralysed.

Later. Patient is now comparatively well. There have been no further convulsions. Bladder is atonic. Catheterisation every four hours.

Treatment. Milk diet. Imperial drink.

Urine. Quantity 82 ozs.; pale amber; acid; Specific Gravity 1015. Albumen 1 per thousand per Esbach tube. No blood or tube casts.

12th December 1907: Patient's condition is satisfactory. No further convulsions have occurred. Haemorrhage on right retina has cleared up.

Treatment. As above.

Urine. Quantity 76 ozs.; amber; acid; Specific Gravity 1018. Albumen trace.

14th December 1907: Improvement continues. There is now no Albumen in the urine. The bladder has regained its tonicity. Catheterisation is stopped.

16th December 1907: Recovery uninterrupted.

20th December 1907: Light diet given.

29th December 1907: Patient dismissed well. Child's arm has improved slightly.

C A S E II

DISEASE.

Eczema

Notes of Case.

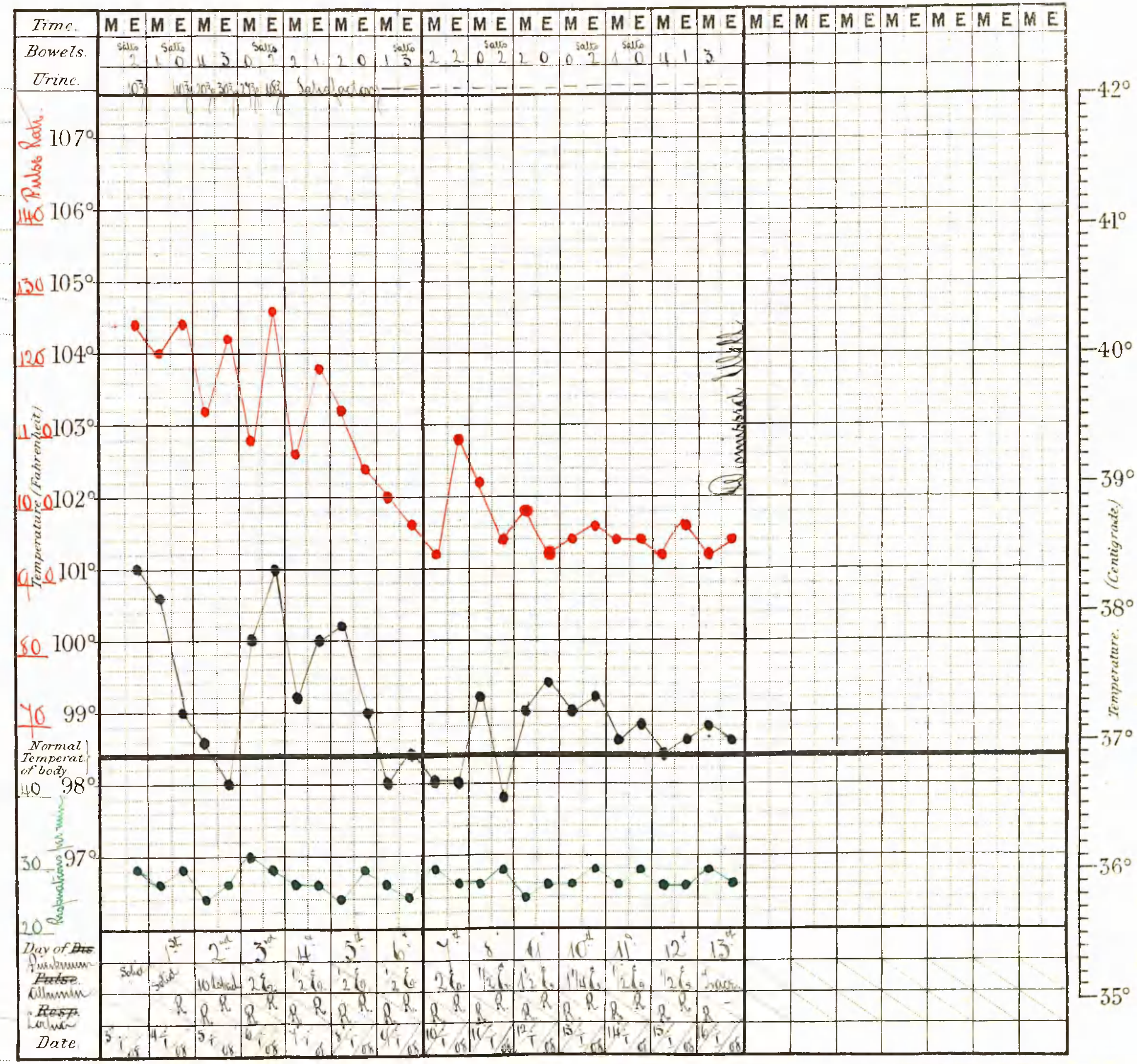
Name: James Barlow

Age: 14 years

Diet: White

Case ~~Book~~ No 11

i Para



Date of admission. 3rd January 1908.

Result Recovery

CASE II AGNES BARBOUR, primipara, aet. 17 years. Full time.
Recovery.
Admitted on January 3rd 1908 at 4.15 p.m.
Delivered on January 4th 1908 at 11.55 a.m
Presentation Vertex
Child, Male dead, 8 lbs. weight.

HISTORY: As far as can be ascertained patient has never been ill previously.

On the 1st January 1908 she complained of headache which continued up to time of the onset of the convulsions. Neither she nor her relations had noticed any oedema on any part of the body. The quantity of urine did not seem to be diminished. Between 12 o'clock noon and 4.15 p.m. (the time of admission) on 3rd January 1908 patient had taken three convulsions.

PRESENT CONDITION: Patient is rather ill-nourished, slightly cyanosed, and quite unconscious. Temperature is 101°F. Pulse is 130 per minute regular and of moderate tension. Respirations are 28 per minute. They are rather stertorous in character. She has several lacerations on the tongue. There is slight oedema over the ankles. Palpation of the abdomen fails to reveal the presence of uterine contractions or of foetal movements. The foetal heart's sounds can be heard on auscutation over the uterine area. Per vaginam the os uteri is found to be closed and the cervix is fully taken up. The foetal head presents, and is well down in the pelvic cavity.

Urine (Catheter Specimen) quantity 10 ozs., dark in colour. Acid specific gravity 1030. Albumen almost solid on boiling, and with Esbach's reagent. Urea 6 grs.

per oz. Granular tube casts. No blood or sugar.

Shortly after admission she took two eclamptic seizures in quick succession. Each fit lasted three minutes. Between 7.20 p.m. 3rd January 1908 and 12.35 a.m. 4th January 1908 patient had eight convulsions, the average duration of each attack being two minutes. No further convulsions occurred.

Treatment. Milk diet, Imperial drink Magnes. Sulp. 3 ozs were given per nasal tube. At 6 p.m. 2 pts saline solution was transfused under the right mammary region. At 7 p.m. she was placed in a hot pack.

4th January 1908: About 12.30 a.m. uterine contractions commenced. After the cessation of the convulsions patient was very restless, and she remained in a state of semi-consciousness. At 11 a.m. as the os was fully dilated the patient was anaesthetised, the membranes were ruptured, forceps were applied, and the child delivered.

During the remainder of the day patient lay in a semi-conscious condition.

Urine (Catheter Specimen) quantity 40 ozs., milky, faintly alkaline. Specific Gravity 1035. Albumen almost solid. Urea 7 grains per ounce. (A few granular tube casts, No blood or sugar)

Treatment. Milk diet. Imperial drinks. Hot applications on Kidney region.

5th January 1908: The patient is still semi-conscious. She can be aroused. The oedema has completely disappeared.

Urine (Catheter Specimen) quantity 60 ozs., pale amber colour; acid; Specific Gravity 1020; Albumen 10 per

thousand per Esbach tube; Urea 8 grains per oz.; Granular casts (scanty); no blood or sugar.

Treatment. Milk diet. Imperial drink.

6th January 1908: Patient is still dazed and gives incoherent answers. She is however much more conscious than before. Engorgement of breasts took place.

Urine quantity 75 ozs.; pale straw colour; acid; Specific Gravity 1015. Albumen 2 per thousand per Esbach tube. Urea 8½ grains per ounce. No casts, blood, or sugar.

7th January 1908: Patient's condition has distinctly improved. She is fully conscious.

Urine is satisfactory in amount. Albumen is ½ per thousand per Esbach.

8th January 1908: Improvement continues; Albumen is ½ per thousand per Esbach.

9th	"	"	"	"	"	½	"	"
10th	"	"	"	"	"	2	"	"
11th	"	"	"	"	"	½	"	"
12th	"	"	"	"	"	1½	"	"
13th	"	"	"	"	"	1½	"	"
14th	"	"	"	"	"	½	"	"
15th	"	"	"	"	"	½	"	"
16th	"	"	"	"	"	Trace	"	"

Patient was allowed to go at her own request comparatively well.

C A S E III

DISEASE.

Plumbosa

Notes of Case.

Mrs.

Walgal

21 Years

Wife

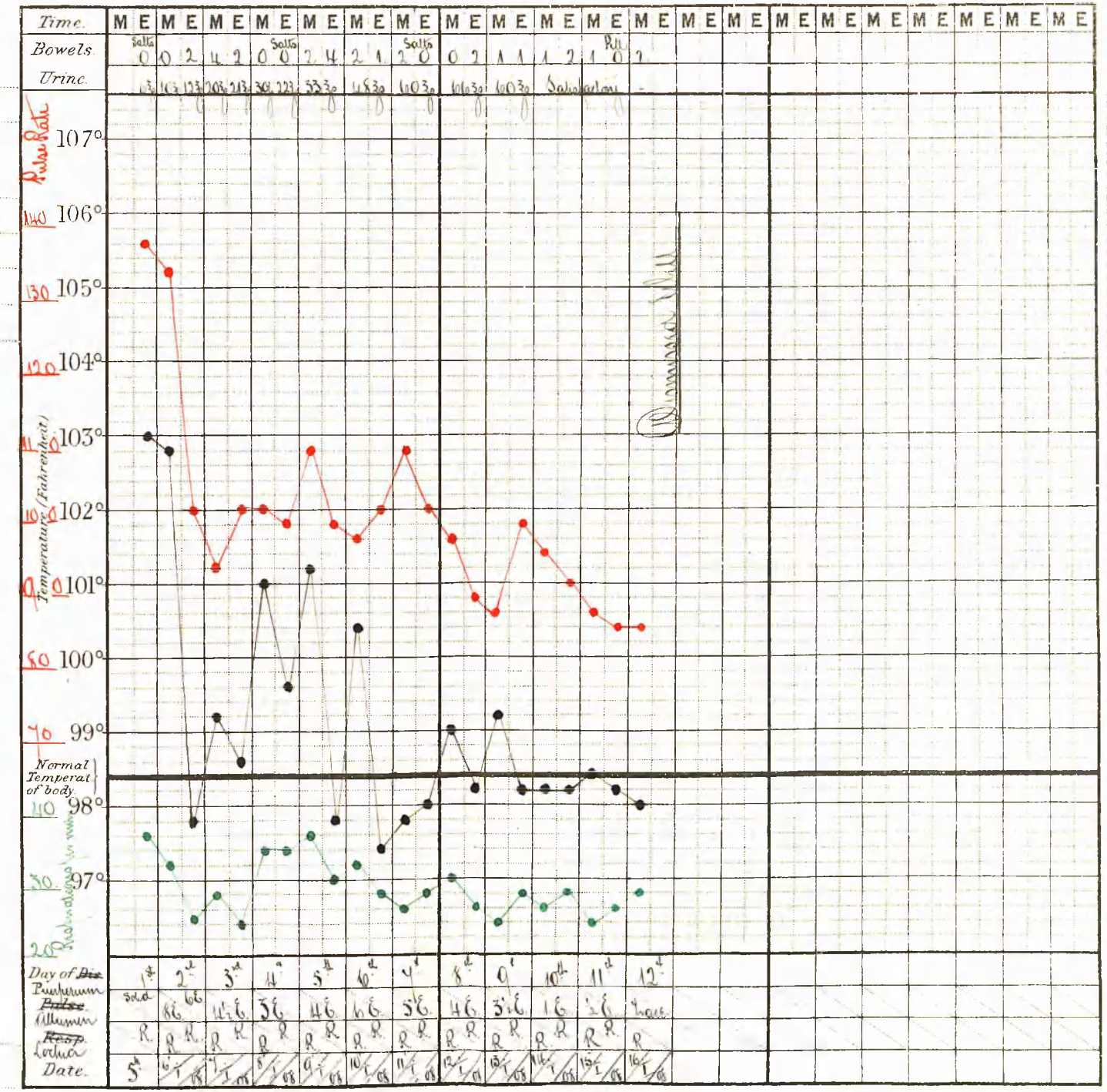
Book No. III

1 Para

Date of admission.

5th January 1888

ult Recovery



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CASE III MRS DALZIEL, aet. 21 years, primipara. Full time.
Recovery.
Admitted on 5th January 1908.
Delivered on 5th January 1908, before admission.
Child, Female dead.

HISTORY: No history of previous illness can be obtained. An outdoor nurse attended the patient during the afternoon of the 5th January (day of admission). As the patient did not seem to be in labour the nurse returned to Hospital. At that time there seems to have been no evidence of disease of any kind. There was no headache, epigastric pain, or eye symptoms complained of nor did the nurse observe oedema on any part of the body. About 8.30 p.m. a message was sent to the Hospital reporting that the woman was taking convulsions, and the nurse again visited her. While waiting on the ambulance to convey the patient to the Hospital she was delivered of a dead female child. Four convulsions were taken before admission.

PRESENT CONDITION: The Patient is unconscious. Pupils are contracted, and do not react to stimuli. The face is puffy and cyanosed and the tongue is lacerated. Temperature is 103° F. Pulse is 130 beats per minute regular and of fairly high tension. The Respirations number 36 per minute and are rather stertorous. There is blood tinged froth about the mouth. The heart and lungs are normal.

Palpation of the abdomen shows the uterus well retracted and about one inch above the umbilicus. Later, the patient is now semi-conscious and can swallow.

Urine (Catheter Specimen) dark colour; acid; Specific Gravity 1025. Albumen 12 per thousand Esbach. Tube casts granular and hyaline. Trace of blood. No sugar. Urea 5 grains per ounce.

Treatment. Milk diet. Saline purge (Magnes Sulp. 3 ozs. by the mouth. Hot pack for ¾ hour.

6th January 1908: At 3 a.m. she had four eclamptic convulsions in rapid succession. Each fit lasted from two to four minutes. There were no further convulsions. Later, the patient is semi-conscious and can be roused sufficiently to enable her to swallow.

Urine (Catheter Specimen) dark; acid; Specific Gravity 1030. Albumen 8 per thousand Esbach. A few granular tube casts. Trace of blood. Urea, 5 grs. per oz.

Treatment. After the succession of convulsions a saline transfusion (2 pints) was given intracellularly under the right breast, otherwise the treatment was as before.

7th January 1908: Patient is much improved. She is only slightly dazed. Albumen milky (urates); acid; Specific Gravity 1020. Albumen 4½ per thousand Esbach. No tube casts. No blood. Urea, 7 grs. per oz.

Treatment as above.

In the evening she complained of pain at the base of the left lung behind and of cough. There is an impairment of the percussion note at the extreme base. Auscultation reveals presence of fine crepitant rale. Temperature, puls and respirations are raised. Hot applications over this region relieved the pain. A stimulant expectorant mixture was also given.

8th January 1908: Pain has completely disappeared from the affected lung region, but the other signs are still present. The mental condition is now normal.

Urine (Catheter Specimen) quantity satisfactory; pale amber; acid; Specific Gravity 1022. Albumen 3 per thousand Esbach. Chlorides not diminished. No blood or tube casts. Urea.

9th January 1908: Patient's condition is much the same as yesterday. The dulness at the base of the left lung is decreasing.

Urine contains 4 per thousand Esbach.

10th January 1908: Urine contains 6 per thousand Esbach.

11th January 1908: Dulness has gone from over the left base of the lung. A few fine rales are still heard on auscultation. Urine has Albumen 5 per thousand Esbach.

12th January 1908: Urine contains Albumen = 4 per thousand Esbach.

13th January 1908: Urine contains Albumen = $3\frac{1}{2}$ per thousand Esbach.

14th January 1908: The Rale has gone from lung. Urine contains Albumen = 4 per thousand Esbach.

15th January 1908: Urine contains Albumen = $\frac{1}{2}$ per thousand Esbach.

16th January 1908: Urine contains trace Albumen.

Patient dismissed well.

C A S E IV

DISEASE.

Eclampsia

Notes of Case.

Name: Mrs. ... Intyre

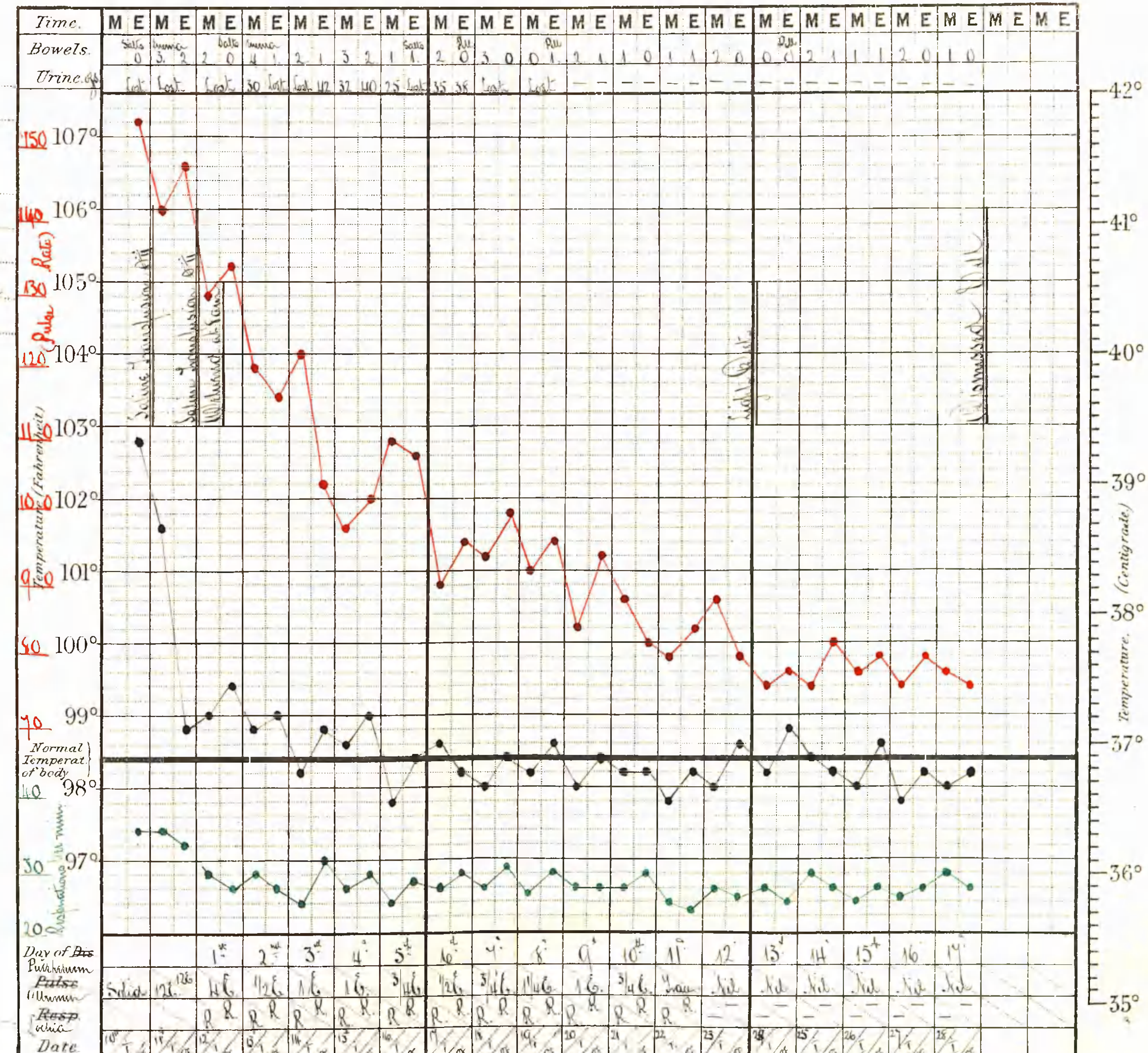
Age: 21 years

Diet: Milk — light diet

Case Book No: IV

Date of admission. 10th January 1908

Result: Recovery



CASE 1V MRS MACINTYRE, aet 21 years, primipara. 32 weeks pregnant. Recovery.

Admitted on 10th January 1908 at 5.10 p.m.

Delivered on 12th January 1908 at 8 a.m.

Child, Male dead, premature, 4½ lbs. weight.

HISTORY: There is no history of any previous disease. The pregnancy seems to have been quite normal up to the onset of the convulsions. Headache, loss of vision, scantiness of the urine, or oedema of any part of the body had never been noticed or complained of. During the evening of January 8th 1908 the patient began to take fits. The convulsions continued with increasing frequency until day of admission. It is known that the patient had at least one hundred convulsions before admission.

PRESENT CONDITION: The patient is quite unconscious. The face is puffy and cyanosed. The pupils are dilated, and do not react to stimuli. The tongue is much swollen and lacerated. There is a blood stained froth round the mouth. Temperature is 102.8°. Pulse numbers 152 per minute. It is feeble, irregular and of poor tension. Respirations are 34 per minute. Abdominal palpations show the head to present. It has not engaged the brim of the pelvis. No foetal movements can be made out. No foetal heart's sounds can be heard on auscultation. Per vaginam. The cervix uteri is not fully taken up. The os admits one finger. There are no uterine contractions. 8 ozs. urine were drawn off by catheter.

Urine. Dark muddy colour, faintly acid, Specific Gravity 1038. Albumen solid on boiling and per Esbach.

Tube casts (granular) are abundant. Blood is present. Urea, 3 grs. per oz.

Four convulsions occurred before any treatment could be adopted.

Treatment. Under chloroform Saline 2 pints were transfused under the right mamma, 3 ozs. Magnes. Sulp. were given per nasal tube, and their action was aided afterwards by a large soap and water enema. A hot pack was also given, and the skin acted well. Nourishment and stimulant was given by a tube two hourly.

Later. Patient is still taking convulsions and their frequency is diminishing. The convulsions are all of the eclamptic character, and the duration of each is from 2-3 minutes.

11th January 1908; From 5.30 last evening until 5.30 a.m. the patient had seventy-nine convulsions. From 5 a.m. until 2 p.m. she remained quite unconscious and had no convulsions. The pulse had improved somewhat, becoming more regular and stronger. The temperature was falling. At 2 p.m. immediately after palpation of the abdomen three convulsions occurred in rapid succession. After these attacks, which were the last, she gradually became conscious, and restlessness set in, which was increased by the onset of uterine contractions.

Urine. The quantity appears to be abundant but it cannot be measured, as it is passed unconsciously. It is dark in colour, Specific Gravity 1022, acid in reaction. Albumen 12 per thousand Esbach. Granular tube casts and blood are present. Urea, 5 grs. per oz.

Treatment. Milk and stimulant were given at regular intervals. She was able to swallow after 6 p.m. After

the three attacks at 2 p.m. one pint of saline solution was injected into each breast under chloroform.

12th January 1908: The membranes ruptured at 3.30 a.m. At 8 a.m. patient was delivered naturally of a premature, dead, male child. She is now almost quite conscious, and has had no convulsions since the previous afternoon. Temperature 99° F., and the pulse has improved.

Urine. Quantity satisfactory, milky, Specific Gravity 1020, faintly acid. Albumen 4 per thousand Esbach. A few tube casts. No blood. Urea 7 grains per ounce.

13th January 1908: Patient's general condition is improving. Albumen is ½ per thousand Esbach.

14th January 1908: She is very restless and inclined to be restless. Albumen is 1 per thousand Esbach. Chloral and Bromide ā ā 20 grs. were given and repeated in six hours.

15th January 1908: Patient is still restless but quite composmentis. Albumen 1 per thousand Esbach.

16th January 1908: One dose of Chloral and Bromide was given with a satisfactory result. Patient is much improved today The restlessness has quite subsided. Albumen is ¾ per thousand Esbach.

17th January 1908: Convalescence established. Albumen is ½ per thousand Esbach.

18th January 1908: " " " ¾

19th January 1908: " " " 1¼

20th January 1908: " " " 1

21st January 1908: " " " ¾

22nd January 1908: " " " Trace

23rd January 1908: Convalescence established. Albumen is Nil. Light diet.

24th January 1908: " " " " "

25th January 1908: " " " " "

26th January 1908 " " " " "

27th January 1908: " " " " "

28th January 1908: Dismissed well.

C A S E V

DISEASE.

Eclampsia (Post Partum)
(Broncho Pneumonia
Septicaemia)

Notes of Case.

Name Mrs Dobson

Age 28 Years.

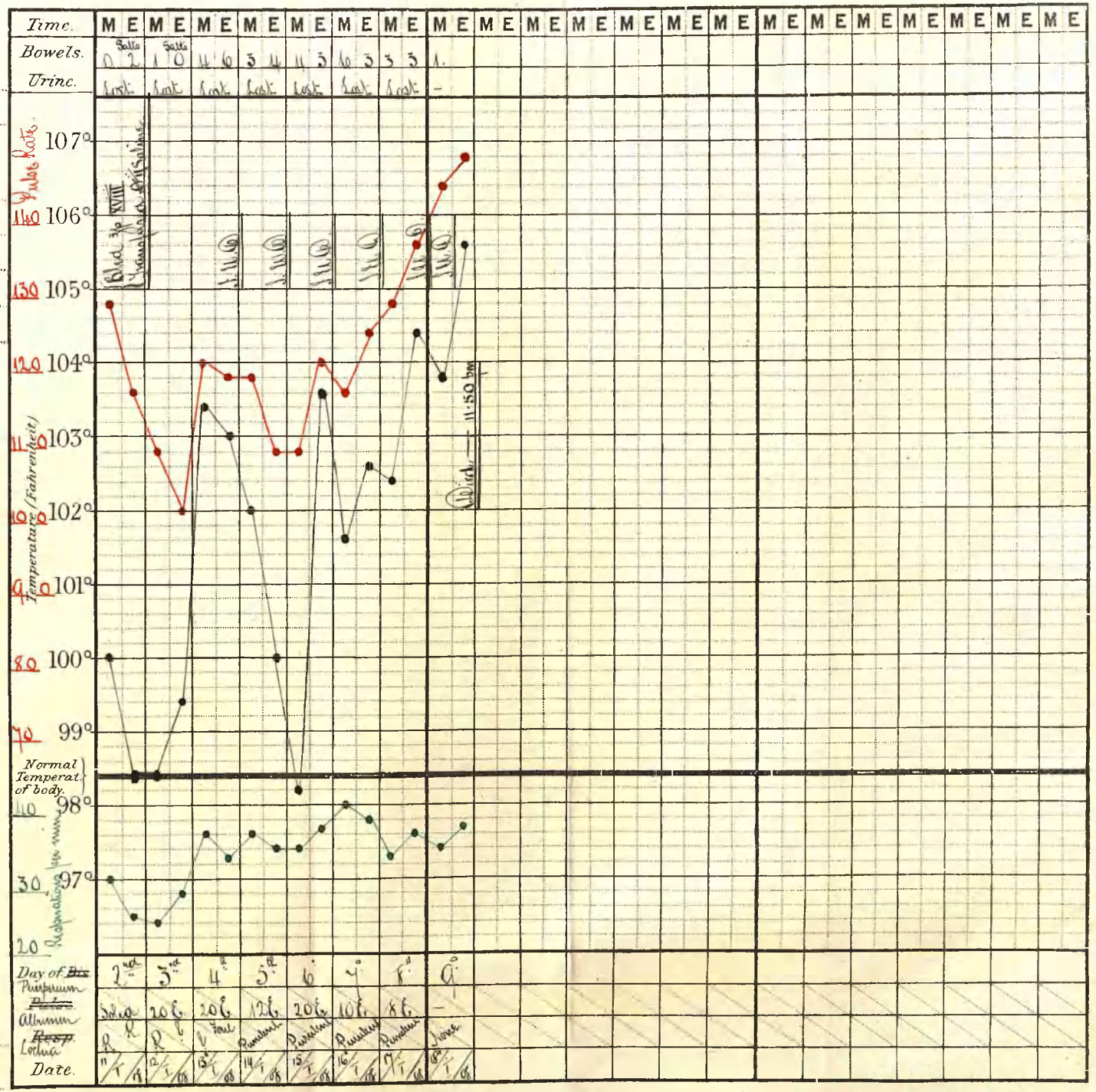
Diet Milk

Case Book No V

I Para

Date of admission.
11th January 1908

Result Death



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CASE V MRS. PATERSON, aet. 28 years, primipara. Full time.
Death. Postpartum Eclampsia. Broncho-pneumonia.
Septicaemia.
Admitted on 11th January 1908 at 10 a.m.
Delivered on 10th January 1908 at 9 a.m.
Child, Male alive.

HISTORY: The patient had been treated for some weeks before her confinement for headache, oedema, and albuminuria. The confinement was said to have been quite normal. The convulsions commenced twelve hours after the delivery, and she had seven fits before admission.

PRESENT CONDITION: Patient is unconscious. The pupils are moderately contracted and react to stimuli. There is oedema and slight cyanosis of the face. The lower abdomen, sacral region, and lower extremities are slightly oedematous. Temperature is 100°F. Pulse 128 regular and of rather high tension. Respirations number 30 per minute. There are signs of pulmonary oedema. The uterus is six inches above the symphysis pubis. While the patient was being prepared for admission to the Ward she took three severe eclamptic seizures in succession. Each fit lasting from five to eight minutes. After these attacks patient lay quiet and unconscious for some hours, when she became very restless. No further convulsions occurred. The lochia was normal.

Urine. (Catheter Specimen) 8 ozs; acid; dark porter colour; Specific Gravity 1040; Albumen solid; granular tube casts abundant; small quantity of blood; Urea, 5 grs.

Treatment. Immediately after the convulsions the median basilic vein was opened, and 18 ozs. of blood were withdrawn. 2 pints saline solution were transfused, 2 ozs.

Magnes. Sulph. were given per nasal tube. Later, as the skin was dry, a hot pack was given with good result. For the restlessness Chloral Hydrate 20 grs. and Potassium Bromide 30 grs. were given per rectum. Milk was given at intervals during the day by a nasal tube.

12th January 1908: No further convulsions have occurred. The patient is semi-conscious, and she can swallow without difficulty. In the evening the lochia had a somewhat heavy odour although the colour remained normal, and the quantity was not diminished.

Urine. Acid; milky; Specific Gravity 1025; Albumen 20 per thousand Esbach; Urea; a few granular tube casts; no blood.

Treatment. Milk. Imperial drink. In the evening the head of the bed was raised on blocks on account of the character of the lochia.

13th January 1908: The patient looks very ill this morning. The temperature, pulse, and respirations are all raised. She is fairly conscious. There is slight tenderness over the lower abdomen. The breasts remain flaccid, and the lochia is somewhat diminished and has still a heavy odour. No further convulsions have occurred.

Treatment. Milk Imperial drink. Intra-uterine douche (Hydrate Biniodid 1-2000, Sterile water, Lysol solution 1%) Stimulants.

14th January 1908: Patient is in much the same condition as yesterday. The temperature and pulse are inclined to come down, but the respirations still remain rather rapid. No dulness

can be discovered over the lung area but at places fine crepitant rale can be heard on auscultation. Albumen = 12 Esbach.

Treatment is as above.

15th January 1908: The patient is inclined to be delirious. The uterus was explored and a piece of decomposed placenta removed. The lochia is scanty and purulent. The patient has now a troublesome cough and there is some muco-purulent expectoration. Percussion over the lungs behind reveals circumscribed areas of impaired note. Auscultation over these areas shows that there is distinct prolongation of the expiration and that there is abundant moist rale. There is still albumen in the urine 2.0 per Esbach.

Treatment is as before. (The stimulant was increased 4 ozs. brandy, also strychnine $\frac{1}{60}$ gr. was given hypodermically four hourly.)

16th January 1908: The signs of broncho-pneumonic patches are now quite distinct. The patient's condition is not so good. She seems very much weaker. Albumen = 10 Esbach.

Treatment is as before. Patient was sponged frequently with iced water.

17th January 1908: The patient is almost unconscious. The temperature and pulse have risen considerably. The pulse at times is running in character. The broncho-pneumonic patches are increasing in size. Albumen 8 per Esbach.

Treatment as above.

18th January 1908: The patient is quite unconscious. The pulse

is running in character. The lungs were not examined on account of the weakness of the patient. She gradually sank and died at 11.50 p.m.

Treatment as above.

C A S E VI

Chart I.

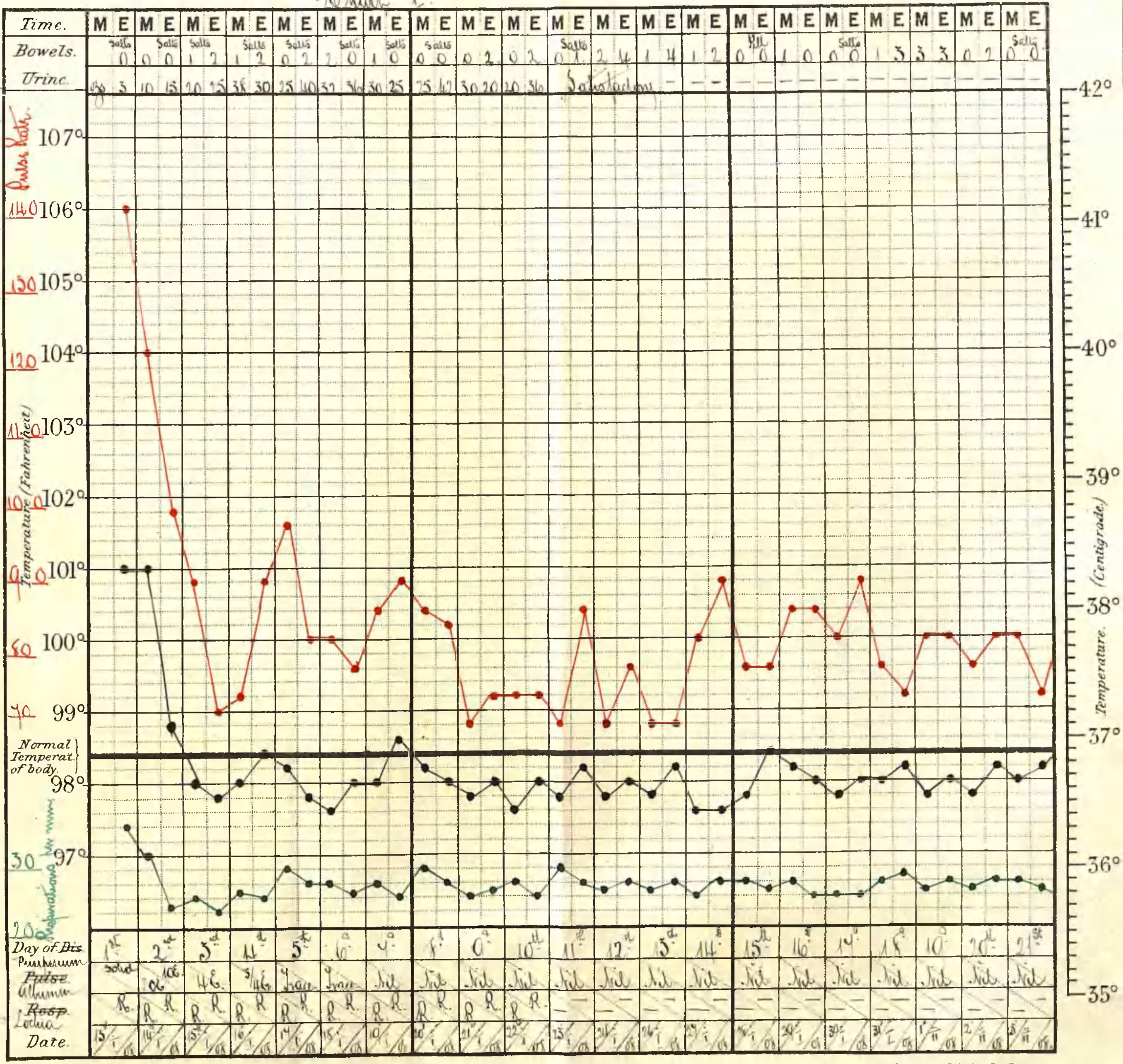
DISEASE.

Eclampsia
Post Partum

Notes of Case.

Name: Lizzie Ivory
Age: 18 Years
...rt: Milk &c.
Case Book No. VI.

I. Para



Date of admission. 13 January 1908

Result Recovery

Chart II

DISEASE.

Adamsaca

Post Partum

Notes of Case.

Name Tiffin Ivory

Age 18 Years.

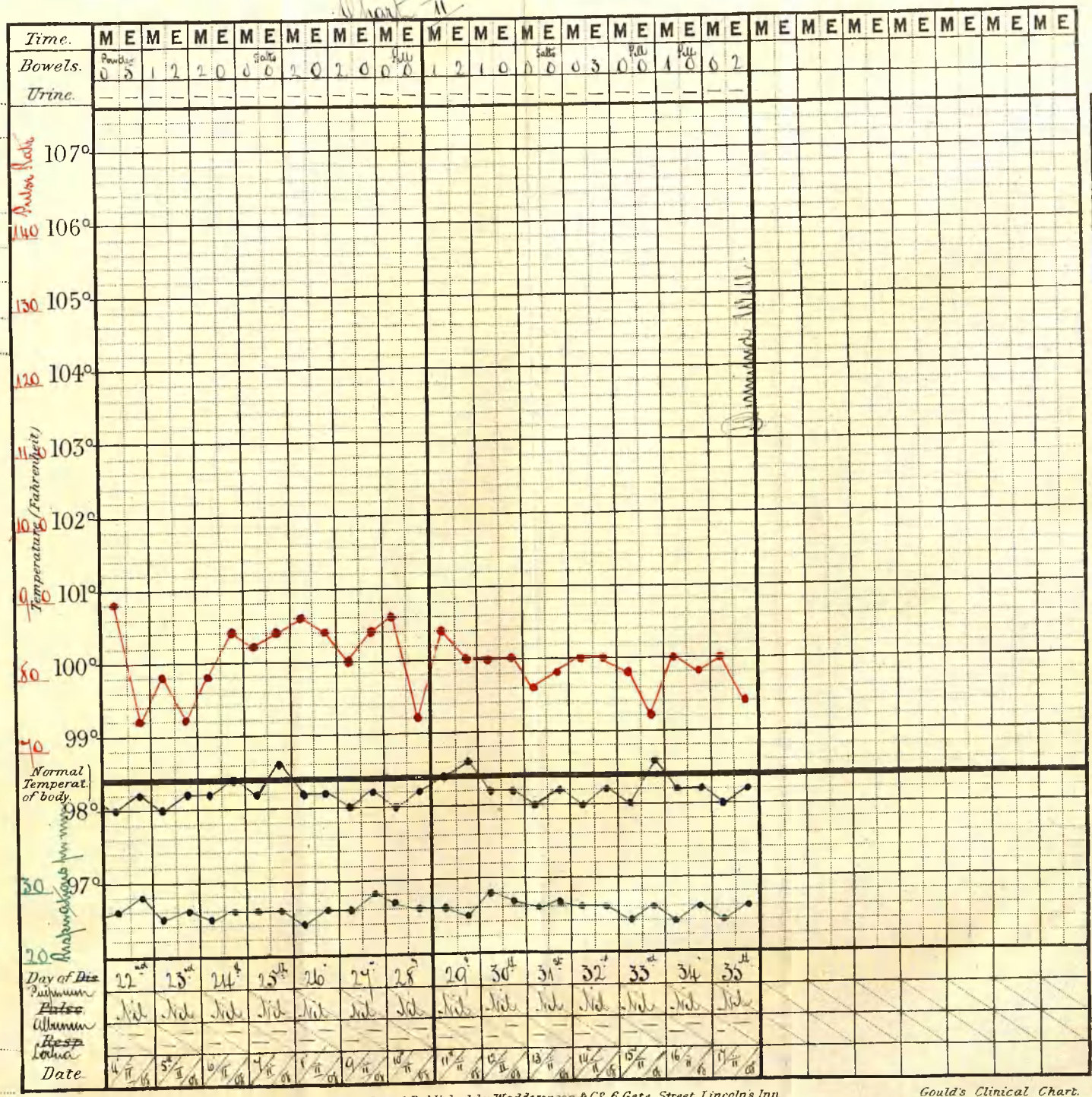
Diet Milk etc

Case Book No. VI

1 Para

Date of admission. 15th January 1908.

Result Recovery



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CASE VI LIZZIE IVORY, aet. 18 years, primipara. 34 weeks.

Recovery.

Postpartum Eclampsia.

Admitted on 13th January 1908 at 10 p.m.

Delivered on 13th January 1908 at 6 a.m.

Child premature Male dead.

HISTORY: During the pregnancy the patient had not complained of any symptoms, such as headache, oedema, etc. Beyond the illnesses of childhood she has never had any disease. The confinement was apparently normal, neither unduly prolonged nor difficult. Eight hours after the completion of the delivery she was suddenly seized with a convulsion lasting about five minutes. She had six more convulsions before admission.

PRESENT CONDITION: The patient is a fullblooded and well nourished girl. She is quite unconscious and very much cyanosed. The tongue is considerably lacerated. Temperature is 101° F. Pulse is 140 per minute. The tension is high. There is no oedema of the face but the extremities are slightly swollen. The uterus is about half an inch above the umbilicus.

Convulsions were very frequent after her admission, and she had six in the first hour and a half of her stay in hospital. She had four further convulsions afterwards, the last being at 7 a.m. 14th January 1908. The attacks were typically eclamptic in character and the average duration was three minutes. Between the fits the patient was very restless and semi-conscious. When the pupils were dilated after a convulsion the retinaewere examined and a few small

recent haemorrhages were found in each.

Urine. (3 ozs. per Catheter) Dark in colour; acid; Specific Gravity 1045; Albumen solid; tube casts and blood; Urea, 4 grains per ounce.

Treatment. Milk per nasal tube. Saline solution 1 pint transfused under the right breast. She was bled 1 pint and 1 pint transfused intravenously. 3 ozs. Magnes. Sulph. was given per nasal tube. Later, hot wet pack, Chloral Hydrate and Potass Bromide ā ā 25 grs. were given per recturm, and this was repeated once on account of the restlessness.

14th January 1908: Patient is improved today. She has had no further convulsions since 7 a.m. As the day advanced she became more conscious and slept at intervals. The skin has acted well but the kidneys and bowels are not satisfactory. Swallowing can now be performed without difficulty.

Ten hours after transfusion, Urine (Catheter specimen) milky; acid (faintly); Specific Gravity 1020; few casts; no blood; Albumen 10 per thousand Esbach

Treatment. Milk diet. Imperial drink. Magnes. Sulph. 3 ozs.

15th January 1908: The patient is quite conscious today. Skin, kidneys and bowels are now acting satisfactorily.

Urine (Catheter specimen) amber; acid; Specific Gravity 1015; Albumen 4 per thousand Esbach; no casts; no blood. Treatment as above.

16th January 1908: Improvement continues.

Urine (Catheter specimen) pale straw; acid; Specific Gravity 1015; Albumen $\frac{3}{4}$ Esbach.

Treatment as above.

18th January 1908: Improvement continues.

Urine (Catheter specimen) straw; acid; Specific Gravity 1018; Albumen trace.

19th January 1908: Albumen has gone from the urine. Patient gradually convalesced and was dismissed well on 17th February 1908.

C A S E VII

DISEASE.

Eclampsia

Notes of Case.

Jeanie Mason

19 Years.

Milk

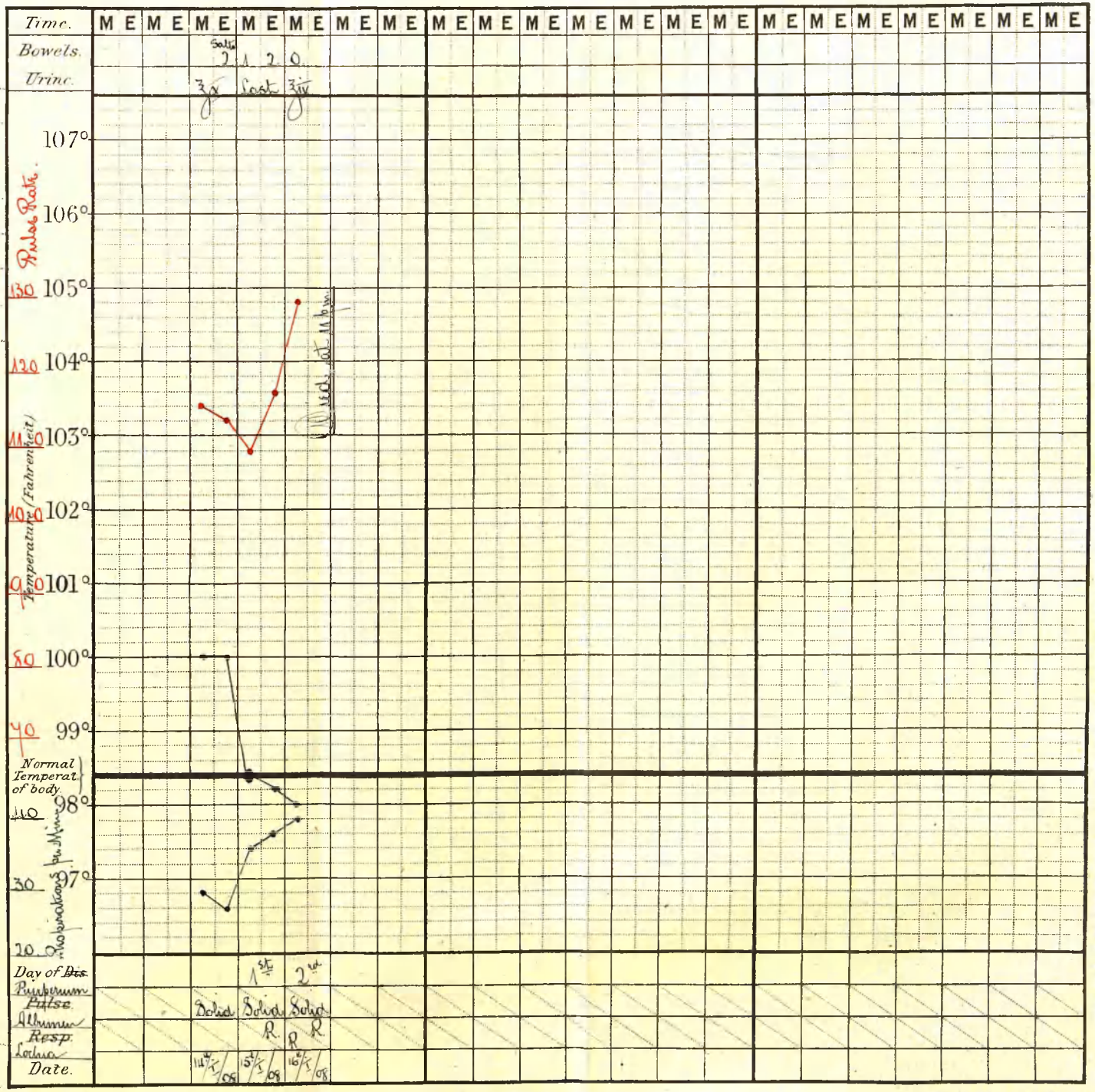
Book No VII

1 Para

Date of admission.

14th January 1908

Result Death



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CASE VII JEANIE MASON, aet. 19 years, primipara. 32 weeks. Death.

Admitted on 14th January 1908 at 12 noon.

Delivered on 15th January 1908 at 10 a.m.

Child Male premature dead, 4½ lbs. weight.

HISTORY: For three weeks before onset of her illness the patient suffered from headache and swelling of the feet and ankles. She had had five fits which came on eight hours before admission.

PRESENT CONDITION: The patient is quite unconscious and cannot be roused. The pupils are contracted and do not react to stimuli. There is a slight oedema of the whole surface of the body, more marked however over the ankles and the feet. Cyanosis of the face is present. The breathing is embarrassed by mucus. Temperature is 100° F. Pulse numbers 124 per minute and is of high tension. Respirations are 28 per minute.

Abdominal palpation shows that the head is presenting. There are no uterine contractions. There are no foetal movements nor can any foetal heart's sounds be heard. There is no history as to when the movements ceased. On vaginal examination the os uteri is found to be closed, and the cervix is not fully taken up. Examination of the lungs. The percussion note is slightly impaired at both bases behind. Auscultation over the bases reveals the presence of moist crepitant rale.

Shortly after admission the patient had two eclamptic convulsions. There was a third slight fit after transfusion. Late in the evening she became semi-conscious and exceedingly restless. The mucus in the throat continued to cause her considerable inconvenience.

Urine. Dark; acid; Specific Gravity 1030; Albumen 20 per thousand Esbach; abundant granular casts; faint trace of blood; epethelial debris; Urea, 3 grains per ounce.

Treatment. Bleeding 1 pint. Saline transfusion 2 pints. Magnes. Sulph. 3 ozs. per nasal tube. Hot pack. Frequent change of position. Later, Chloral Hydrate and Potassium Bromide ā ā 25 grs. per rectum.

15th January 1908: The patient is still semi-conscious and extremely restless. There have been no further convulsions. At 7 a.m. uterine contractions were first noticed. Membrane ruptured at 9 a.m. and she was delivered naturally of a premature child at 10 a.m.

There is now marked dulness over both bases, and there is some tubularity in the breath sounds. Moist rale is heard over the whole lung area.

Urine contains abundant Albumen casts and a trace of blood.

Treatment. Milk diet. Strychnine $\frac{1}{60}$ gr. hypodermically, and Brandy 2 ozs. four hourly.

16th January 1908: Patient is unconscious. Temperature is rather sub-normal. The pulse and breathing are rapid. She is much cyanosed. In spite of vigorous stimulation patient gradually sank and died at 4 p.m.

Urine is loaded with Albumen and contains casts and a trace of blood.

Treatment as above. Strychnine increased from $\frac{1}{60}$ gr. to $\frac{1}{30}$.

C A S E VIII

DISEASE.

Eclampsia (Post Partum)

Notes of Case.

Name: Mrs Graham

Age: 25 Years

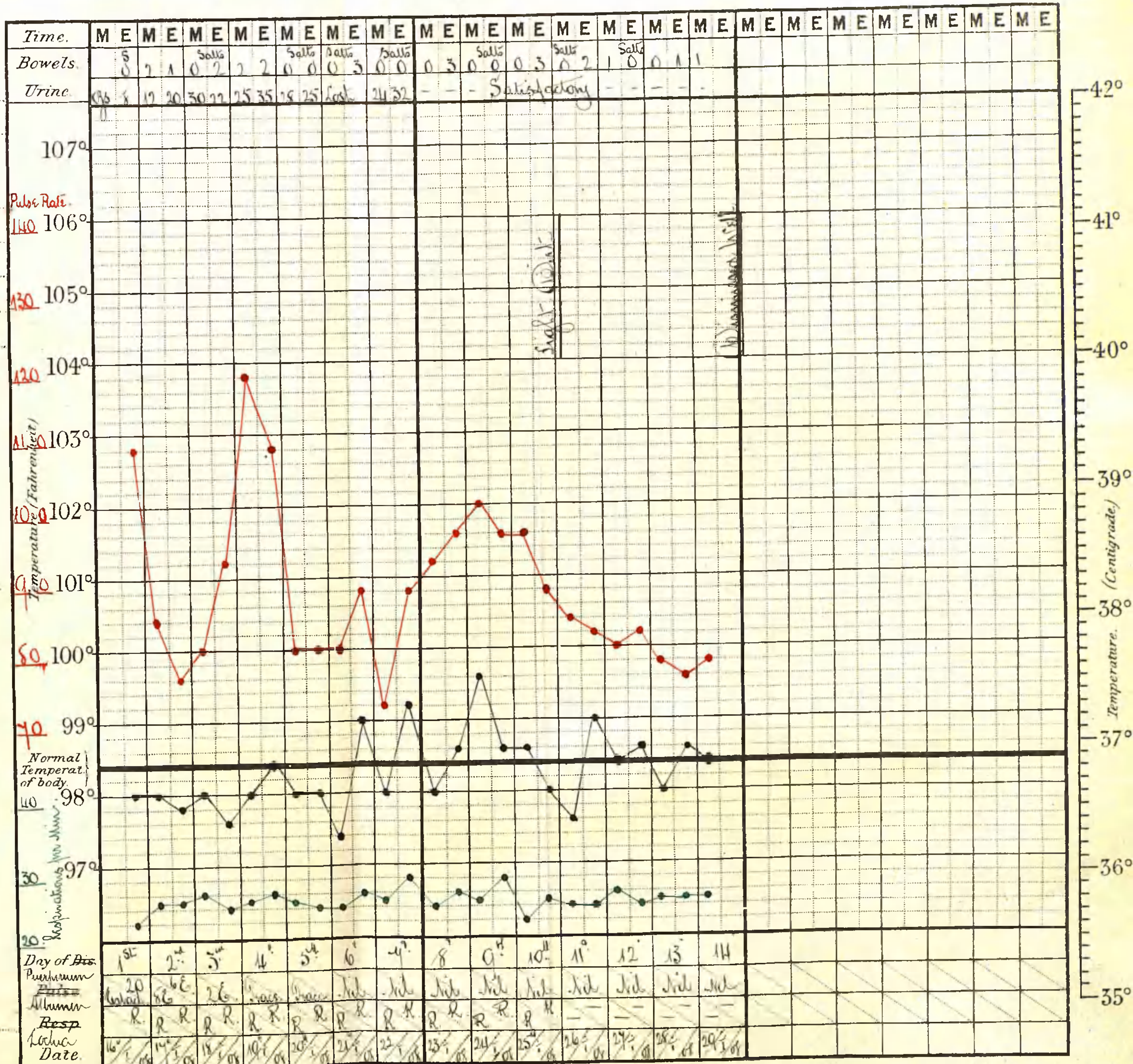
Diet: Milk – Light

Case Book No: VIII

1 Para

Date of admission. 16th January 1908

Result: Recovery



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CASE VIII MRS GRAHAM, aet 25 years, primipara. Full time.

Recovery.

Post Partum Eclampsia.

Admitted on 16th January 1908 at 6.30 p.m.

Delivered on 16th January 1908 at 6 a.m. before admission.

Child Female alive.

HISTORY: During the pregnancy there seems to have been no notice taken of headache, oedema, etc. Labour was as far as can be ascertained quite normal. Half an hour after delivery at 6.30 a.m. she began to take convulsions, and from that time until the time of admission at 6.30 p.m. she had had ten convulsions.

PRESENT CONDITION: The patient was semi-conscious, and she can be roused to consciousness. The face is slightly cyanosed, and the tongue has some small lacerations. There is no oedema on any part of the body.

Abdominal palpation shows the uterus six inches from the symphysis pubis.

Urine. Dark amber; acid; Specific Gravity 1028; Albumen 20 per thousand Esbach; granular and hyaline tube casts; no blood.

While being prepared for admission she took a single eclamptic seizure lasting seven minutes. It was not however a severe convulsion, although of considerable duration. The patient could swallow two hours after convulsion.

Treatment Salts 2 ozs. Milk Pot. Imp. After the fit she was transfused 1½ pints Saline into the right mammary region.

17th January 1908: Patient had a good night having slept well. She is in a slightly dazed condition, but can answer questions intelligently when roused. There have been no further convulsions. Temperature and pulse are normal.

Urine. Amber; acid; Specific Gravity 1020; Albumen 8 per thousand Esbach; no casts.

Treatment. Milk diet, Pot. Imperial.

18th January 1908: Improvement has continued. Albumen 2 per thousand Esbach

19th January 1908: Albumen - trace.

21st January 1908: Albumen - nil. Slight turgidity of breasts, which were treated with massage and a firm binder. Salts. This condition ultimately disappeared on the 24th January 1908.

25th January 1908: Light diet.

29th January 1908: Patient dismissed well.

C A S E IX

DISEASE.

ambsea

Mania

Notes of Case.

Mr.

Abraham

38 Years.

M.K.

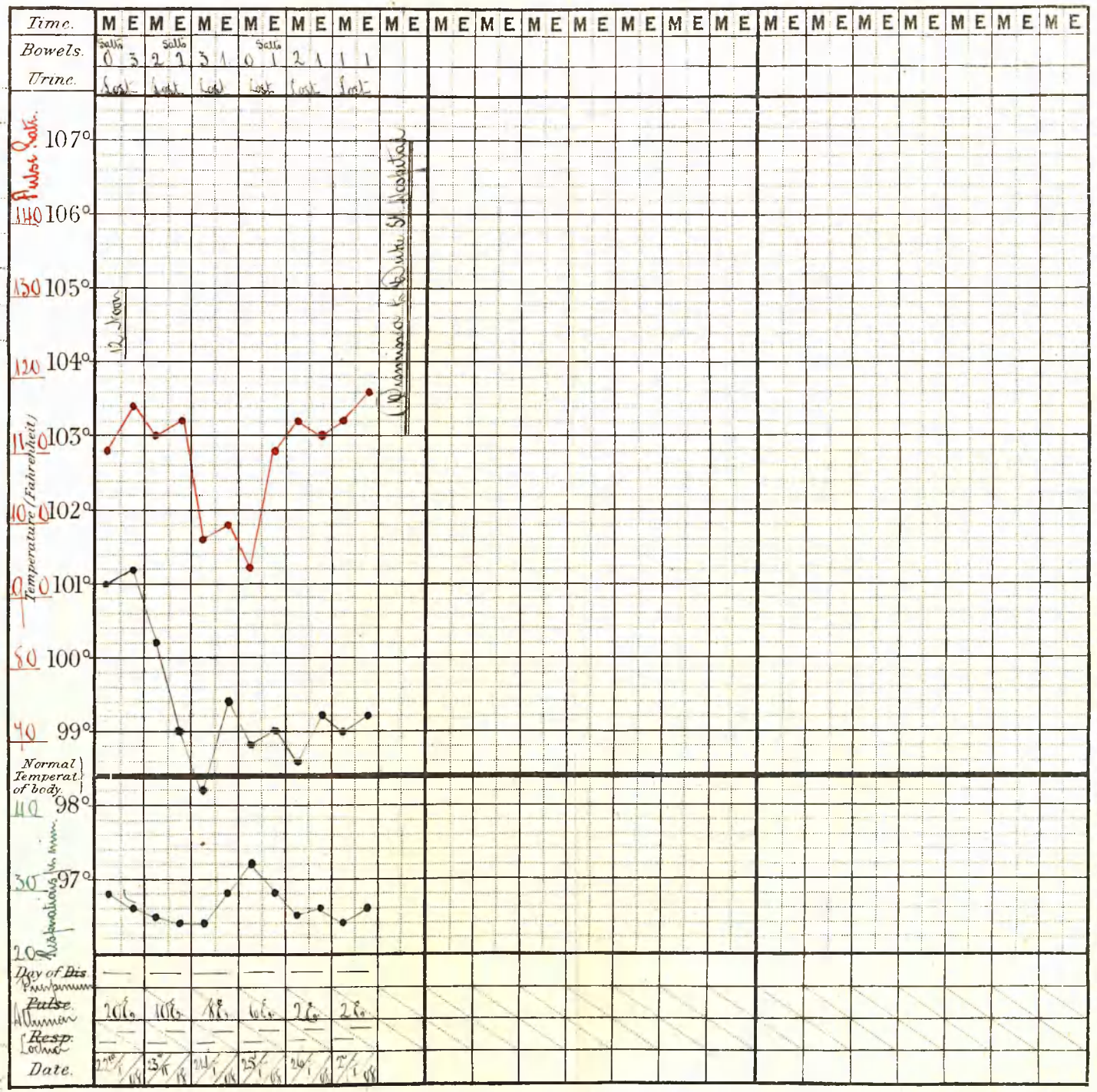
Book No. IX

Dora

Date of admission.

2nd January 1908

Removed to Asylum



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CASE IX MRS GRAHAM, aet 38 years, VI para. Twenty-eight weeks pregnant.

Admitted on 22nd January 1908 at 12 noon.

Dismissed undelivered.

HISTORY: No exact history as to the patient's present pregnancy or her previous pregnancies or confinements can be obtained. The patient had had three fits before admission.

PRESENT CONDITION: The patient is a very large, stout, and powerful woman. She is quite unconscious and slightly cyanosed. There are some small lacerations on the tongue. The pupils are very small and do not react to stimuli. Temperature is 101° F. Pulse 108, regular and of high tension. Respirations number 28 per minute. Abdominal palpation reveals the uterus enlarged, its upper border being slightly above the umbilicus. On vaginal examination the cervix is found to be much lacerated, and to be of considerable length. The os uteri is closed. Auseultation over the uterus reveals the presence of foetal heart's sounds, but no movements can be found.

Urine (Catheter specimen) dark in colour; acid; Specific Gravity 1040; Albumen 20 per thousand Esbach; granular and hyaline casts; trace of blood; Urea 4 grains per oz.

Treatment. Magnes. Sulph. 2 ozs. given per nasal tube. At 2 p.m. she was bled to the extent of 1 pint and transfused 2 pints Saline into the median basilic vein. Later, a hot wet pack was given. Chloral Hydrate and Potassium Bromide a a 30 grs. were given per rectum. Diet, milk per nasal tube.

Later, The patient had ten convulsions between time

of admission and midnight. Four fits occurred before the giving of the Saline, and six afterwards. The last three convulsions were not so severe as those preceding. Between the convulsions she was very restless.

23rd January 1908: There have been no further convulsions. The patient slept for short periods and was extremely restless in the intervals. She is not compos mentis.

Urine (the quantity is indefinite on account of the number of motions, and the involuntary passage of the urine). Dark; thick; acid; Specific Gravity 1032; Albumen 10 per thousand Esbach; Urea 6½ grs. per oz.; very few casts; no blood.

Treatment. Milk diet. Pot. Imp. (Patient can now swallow small quantities of fluid). Chloral and Bromide a a 20 grs. per rectum, four hourly.

24th January 1908: The patient is still very restless and is in a state of semi-consciousness. She sleeps for short periods. Urine. The Albumen is 8 per thousand per Esbach. Urea 6 grs. per oz. Treatment is as above.

25th January 1908: The patient is much better today, being conscious and sensible. She is drinking large quantities of fluids, and passing great quantities of urine.

Urine contains 6 per thousand Esbach. Urea 8 grs. per oz. Treatment. Chloral and Bromide stopped early.

26th January 1908: Early this morning she became very restless and later in the day violently maniacal. She was also very noisy.

Urine contains Albumen to the extent of 2 per thousand

Esbach and Urea 8 grs. per oz.

Treatment. Chloral and Bromide a a 20 grs. were given repeatedly.

27th January 1908: Patient had an extremely restless night, and was only kept within bounds by pushing the narcotics. She is still violently maniacal, and noisy, and as the Hospital is not suitable for the care of such patients she was sent to the observation wards in Duke Street Hospital.

C A S E X

DISEASE.

Eclampsia

Notes of Case.

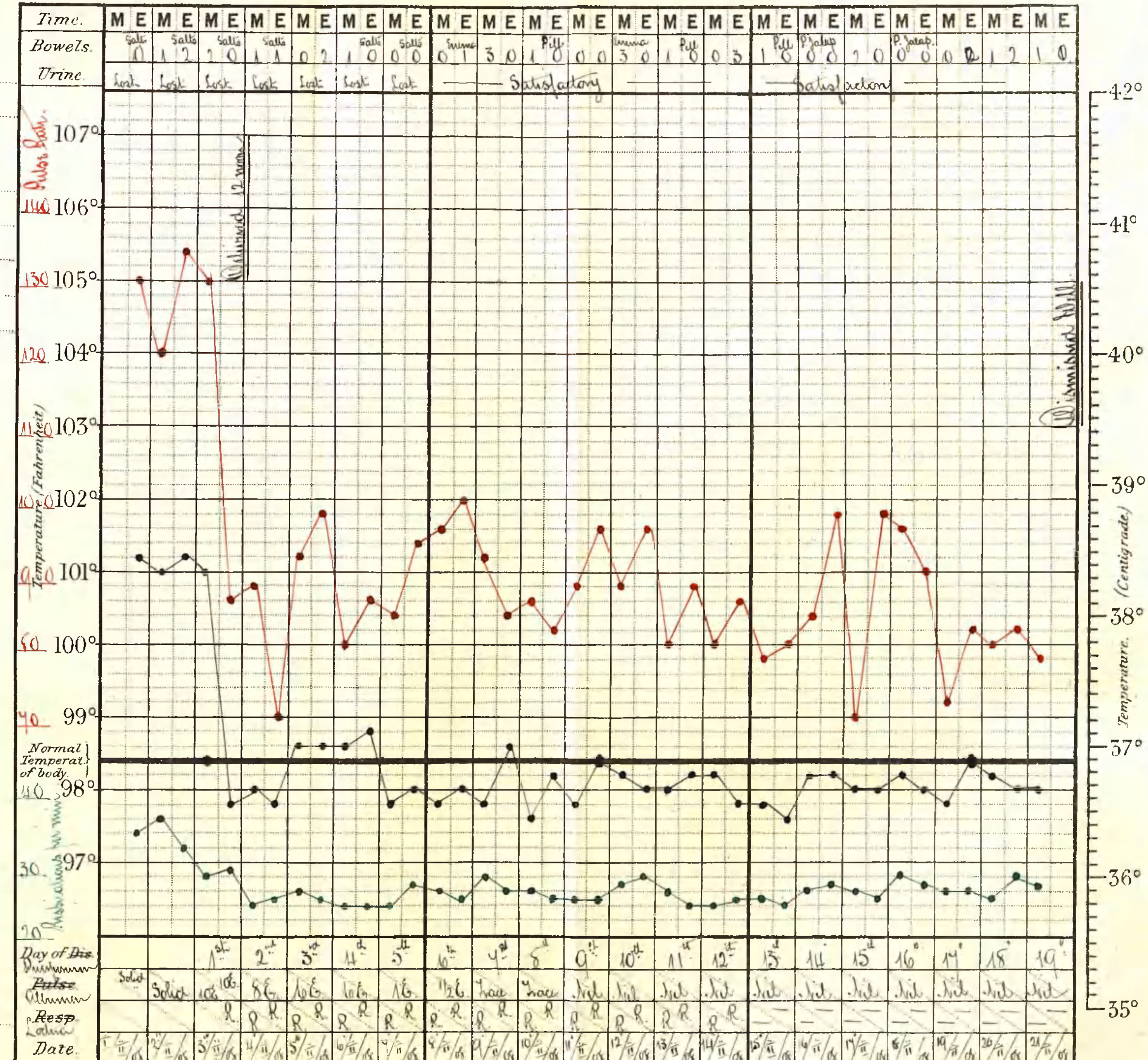
Name: Mrs Morrison

Age: 21 Years

Occupation: Mill — Night

Case Book No. X

1 Para



Date of admission. 1st February 1908

Result: Recovery

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CASE X MRS MORRISON, aet 21 years, primipara. Full time.
Recovery.
Admitted on 1st February 1908 at 9,15 p.m.
Delivered on 3rd February 1908 at 12 noon.
Child Female dead, $6\frac{3}{4}$ lbs. weight.

HISTORY: There is no history of any disease connected with the urinary system previous to her pregnancy.

During the last three months of the pregnancy there had been slight but increasing swelling of the feet and ankles. She had never noticed any swelling of the face. Life had not been felt for the week preceding admission. On the 30th and 31st January 1908 she had suffered for the first time from headache. On the day of admission she had had a number of fits ("over ten"), the first of which was heralded, in addition to the headache, by a severe epigastric pain.

PRESENT CONDITION: Patient is quite unconscious. The face is puffy and slightly cyanosed. There is slight oedema of the lower extremities. The pupils are moderately contracted and do not react. Temperature is 101.2° F. Pulse is 130 per minute, regular and of high tension. Respirations number 34 per minute and are slightly stertorous. The abdomen is distended to the size of that of a full time pregnancy. Auscultation and palpation over the uterus fail to show any signs of foetal life. The vertex was found to present. Per vaginam the os uteri was quite closed, and the cervix was wholly taken up. Labour had not commenced. She is taking frequent eclamptic convulsions.

Urine (Catheter specimen). Dark, opaque; acid; Specific Gravity 1032; albumen solid; granular and hyaline casts (few); no blood.

Treatment. Magnes. Sulph. 3 ozs. were given per nasal tube.

15 ozs. blood were withdrawn from the median basilic vein and 2 pints Saline were transfused. Later, as convulsions were still continuing, Chloral and Bromide a a 1 oz. were given per rectum. A hot wet pack was also given

2nd February 1908: The patient is semi-conscious and restless. There have been no further convulsions. The skin, bowels, and kidneys are acting satisfactorily. She has had 17 convulsions from admission at 9.15 p.m. yesterday until 10 a.m. today. Seven were taken before the saline transfusion. She is able to swallow and had taken a fair quantity of fluid. Milk & Imperial Drink.

Urine. Milky, neutral; Specific Gravity 1025; Albumen solid; a few tube casts; and a trace of blood.

Treatment. Fluid diet. Chloral Hydrate 15 grs. Potassium Bromide 20 grs. twice at a six hourly interval.

3rd February 1908: There have been no further convulsions. Early this morning at 2.45 a.m. labour commenced. The membranes ruptured at 9.15 a.m. and she was delivered naturally of a slightly macerated female child at 12 noon. After delivery the restlessness subsided.

Urine. Albumen - 10 per Esbach; a few tube casts; no blood.

Treatment. Fluid Diet.

4th February 1908: Patient is again restless and is still semi-conscious. She can swallow without difficulty.

Urine contains Albumen to the extent of 8 per Esbach; few tube casts, no blood.

Treatment. Fluid diet. Chloral and Bromide four hourly.

5th February 1908: She slept at short intervals during the night, but the general condition is the same as yesterday.

6th February 1908: The patient is much better today. Consciousness has now fully returned. She does not remember anything of what has happened since she had the first fit.

Urine. Albumen 6 per thousand Esbach; no tube casts; no blood.

Treatment. Fluid diet. Chloral and Bromide stopped.

7th February 1908: Improvement continues. Albumen 1 per thousand Esbach.

10th February 1908: Improvement continues. No albumen.

21st February 1908: Dismissed well.

C A S E XI

DISEASE.

Pneumonia

Notes of Case.

Name { Emma Van Wyck

Age 16 Years

Sex Female

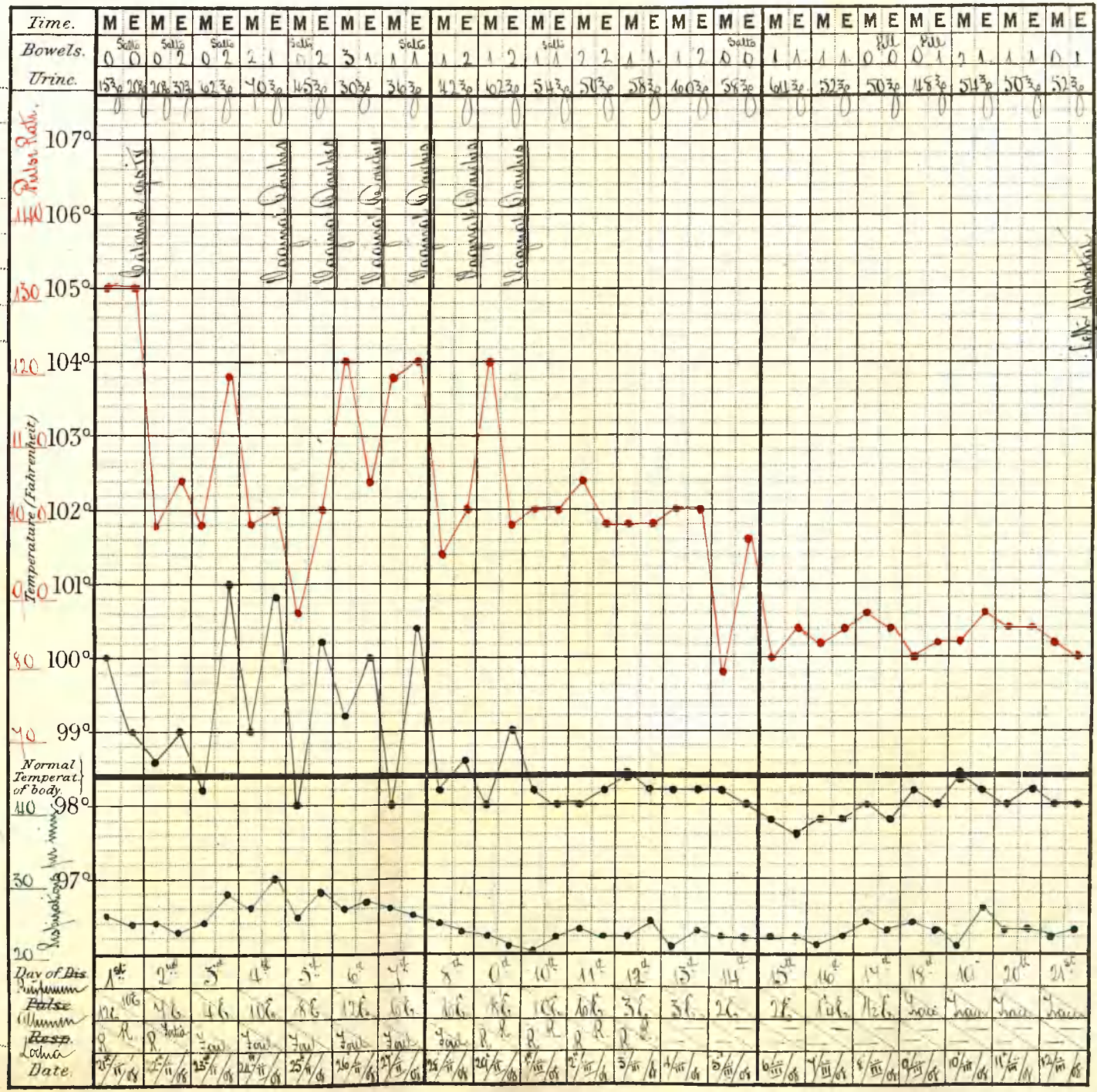
Case Book N° XI

1 Para

Date of admission.

21st February

Result Recovery



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CASE XI EMMA MacGHIE, aet 16 years, primipara. 36 weeks.

Recovery.

Admitted on 21st February 1908 at 11.15 a.m.

Delivered on 21st February 1908 at 3 a.m. before admission.

Children twin Females alive.

HISTORY: Previous to her pregnancy the patient had had no symptoms of any renal disease. The first symptom that she noticed was a slight oedema of the feet and ankles. This occurred about one month ago. Two weeks later she became troubled with a persistent headache, and the oedema in the lower extremities became worse. The face was never swollen and there is no history of eye symptoms or epigastric pain. At 3 a.m. on 21st February 1908 she was delivered of twins, and had had one convulsion just prior to delivery. Numerous convulsions occurred between time of delivery and that of admission. The exact number is unknown.

PRESENT CONDITION: The patient is unconscious and much cyanosed, but she had had a convulsion in the ambulance waggon just before admission. The pupils are contracted and do not react to stimuli. The tongue was severely lacerated and there is a blood stained froth at the mouth. Temperature is 100°F. Pulse 130 beats per minute, regular and of high tension. Respirations number 25 per minute. The uterus is $2\frac{1}{2}$ inches above the umbilicus. There is oedema over the sacrum and of the lower extremities.

While being prepared for admission to the Wards she had two typical eclamptic seizures, which were the last.

Urine. Dark; acid; Specific Gravity 1030; Albumen is 12 per thousand Esbach; few casts (granular and hyaline); trace of blood.

Treatment. She was bled 1 pint and transfused intravenously with 2 pints Saline solution. Later, as she could swallow, Calomel 4 grs. followed by Magnes. Sulph. were given. Milk and Pot. Imp.

22nd February 1908: Patient passed a quiet night and is semi-conscious today. She can swallow without difficulty.

Urine is abundant, and contains 12 per thousand Albumen.

23rd February 1908: Patient is conscious today, but inclined to be drowsy. Temperature rose in the evening. The lochia is slightly foul, probably from the vaginal lacerations, and the head of the bed was raised to promote efficient drainage.

Urine abundant. Albumen 11 per thousand Esbach.

24th February 1908: The lochia is septic but abundant this morning and the lacerations in the vagina show a tendency towards sloughing. Vaginal douches were given night and morning. Albumen 10 per thousand Esbach.

25th February 1908: Albumen 8 per thousand. Vaginal Douche.
26th February 1908: " 12 " " " "
27th February 1908: " 6 " " " "
28th February 1908: " 6 " " " "
29th February 1908: " 8 " " " " Vagina healthy. Lochia sweet.

1st March 1908: " 10 " "
2nd March 1908: " 6 " "

3rd March 1908: Albumen 3 per thousand.

4th March 1908: " 3 " "

5th March 1908: " 2 " "

6th March 1908: " 2 " "

7th March 1908: " 1¼ " "

8th March 1908: " ½ " "

9th March 1908: " Trace

10th March 1908: " "

11th March 1908: " "

12th March 1908: " "

Patient left of her own accord.

Mrs Macmillan, [illegible] years. [illegible]

Mercury.

Post Partum Eclampsia.

Admitted 2nd February 1899 at

Delivered on 22nd February 1899 at

midnight.

Child [illegible] alive.

C A S E XII

DISEASE.

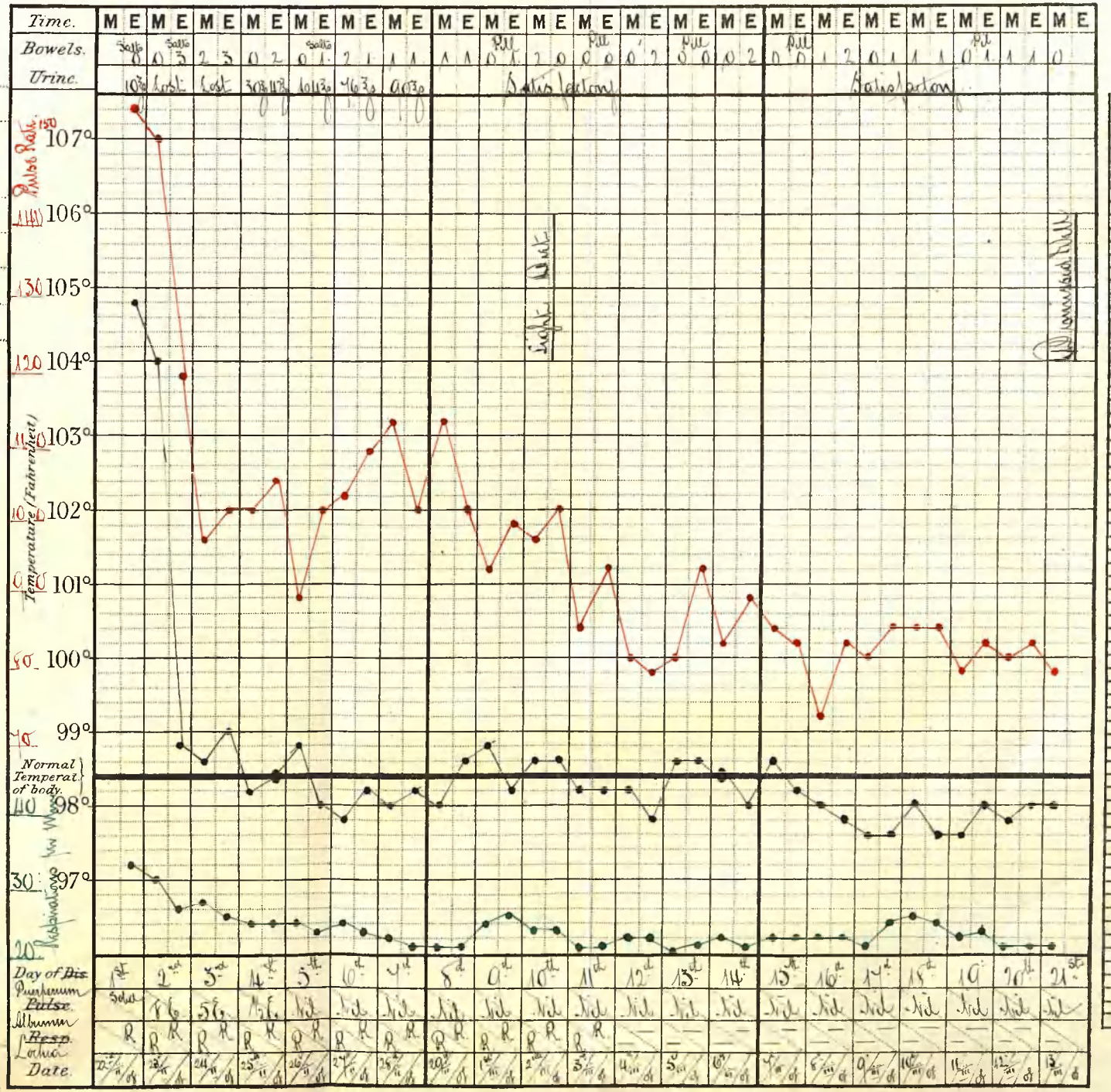
Delirium
Post Partum

Notes of Case.

Age
Mrs Hallum
24 Years
Milk Light
Case Book N° XII

II Para

Date of admission.
22nd February 1908
Result Recovery



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CASE XII MRS MacCALLUM, aet 27 years, II para. Full time.

Recovery.

Post Partum Eclampsia.

Admitted on 22nd February 1908 at 10.15 p.m.

Delivered on 22nd February 1908 at 10 a.m., before admission.

Child alive.

HISTORY: Her previous health had always been satisfactory. The first pregnancy and confinement had been quite normal.

In the last month there had been slight oedema round the ankles, after standing for some time. No headache had been complained of. She was delivered of a live child at 10 a.m. on 22nd February 1908 after an apparently normal labour. The first convulsion occurred quite suddenly at 2 p.m. on 22nd February 1908, and she went from one into another until time of admission at 10.15 p.m., 22nd February 1908.

PRESENT CONDITION: The patient is quite unconscious. The face is cyanosed. The tongue has been severely bitten and there is a quantity of blood tinged froth round the mouth. The pupils are dilated and do not react to stimuli. Temperature 104.8° F. Pulse numbers 154 per minute, thready and irregular. Respirations are 32 per minute. Before 12 midnight she had six eclamptic seizures, each of which lasted from two to three minutes.

Urine (Catheter specimen). Very dark; acid; Specific Gravity 1030; Albumen solid; numerous tube casts; no blood.

Treatment. Magnes. Sulph. 2 ozs. per nasal tube. Saline transfusion (under breast) 2 pints. Chloral 10 grs.

and Bromide 15 grs. twice at three hours interval.

23rd February 1908: From 12 midnight until 10 a.m. the patient had taken six further convulsions. The first four of these occurred between 12 midnight and 3 a.m..The others at long intervals. They were of moderate severity. From 10 a.m. she began to improve and fell into a quiet sleep, waking up in the late afternoon in a dazed but conscious condition. The execretory organs have acted satisfactorily. Urine contains Albumen 8 per thousand per Esbach.

Treatment. During the early morning two doses Chloral and Bromide 20 grs. were given. Stimulant (Brandy 2 ozs.) was given twice per rectum. Diet milk.

24th February 1908: The patient is now quite conscious but suffers from slight dementia.

The quantity of urine is satisfactory. Albumen 5 per thousand per Esbach. No tube casts.

25th February 1908: The dementia of yesterday has completely passed off and the patient is much improved.

Urine contains Albumen ½ per thousand per Esbach, and no casts.

26th February 1908: Marked improvement. Albumen nil.

Convalescence uninterrupted.

13th March 1908: Dismissed well.

C A S E XIII

DISEASE.

Malaria

Notes of Case.

Name Mr. Jermant

Age 30 Years

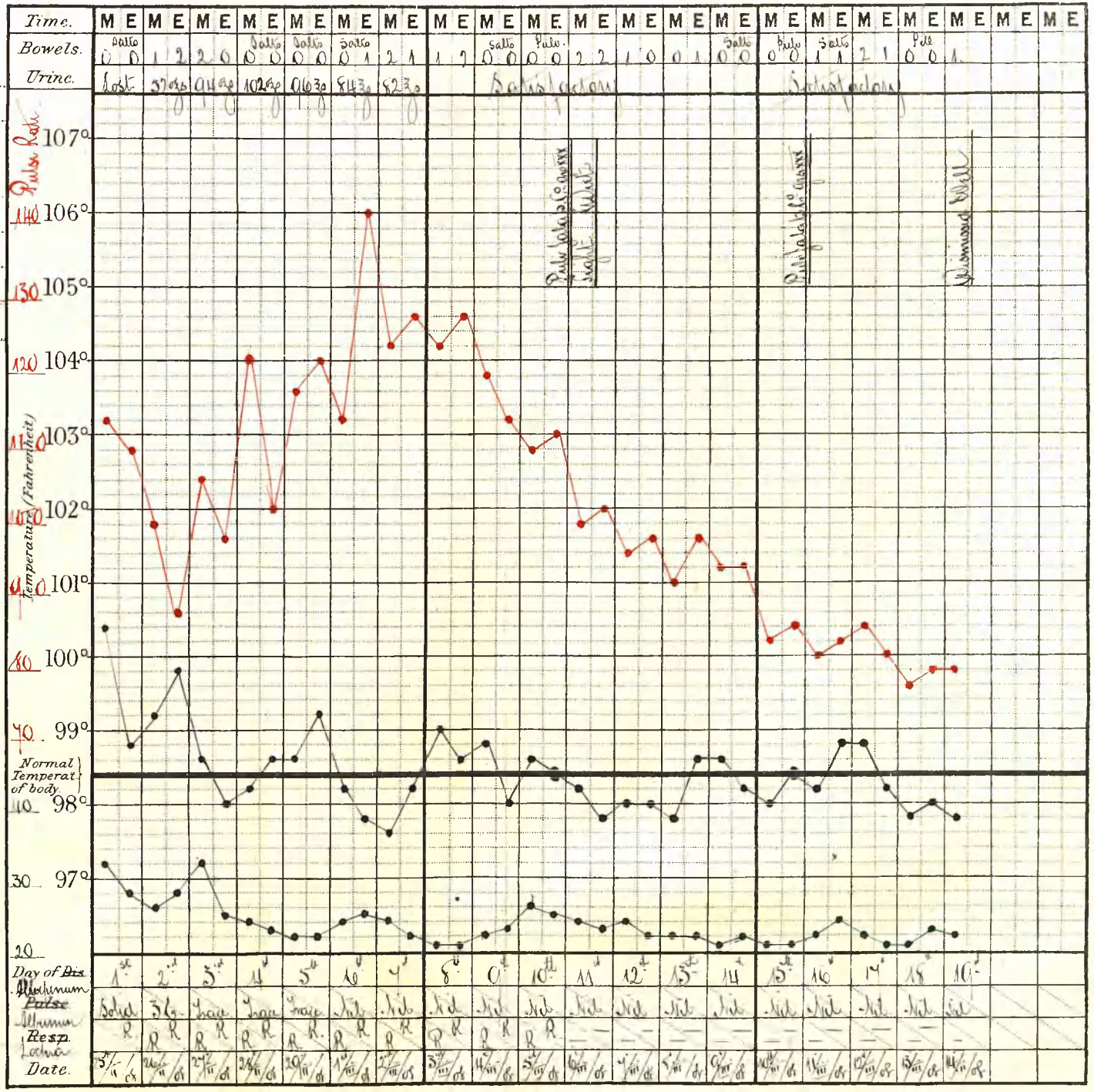
Sex Male Light

Case Book No. XIII

Para

Date of admission. 25 January 1908

Result Recovery



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CASE XIII MRS TENNANT, aet 39 years, III para. 23 weeks.
Recovery.
Admitted on 25th February 1908 at 4.30 a.m.
Delivered on 25th February 1908 at 11.45 a.m.
Macerated Foetus.

HISTORY: The previous pregnancies and confinements had been quite normal, so far as can be ascertained.

During the present pregnancy she noticed a slight oedema of the feet and ankles when about 16 weeks pregnant. This swelling increased slightly as pregnancy advanced. There was no other symptom, however, which obtruded itself upon her notice. On the evening before admission she felt dazed and shortly afterwards "took fits" which continued until time of admission. The exact number of convulsions taken previous to her admission to hospital is unknown. "Life" had never been felt in the foetus.

PRESENT CONDITION: The patient is semi-conscious, extremely restless, and almost maniacal at times. The tongue is greatly lacerated. Temperature is 100.4° F. Pulse numbers 112 per minute, regular and of high tension. Respirations are 25 per minute. The uterus is at the level of the umbilicus and is in a state of active contraction. The os uteri is dilating, and admits one finger. The presentation is vertex in the first position

Urine (Catheter specimen), acid dark muddy; acid; Specific Gravity 1032; Albumen solid; finely and coarsely granular tube casts and blood; Urea, 4 grs. per oz.

Treatment. Salts. Bled ¾ pint. Transfused intravenously 2 pints Saline solution. Chloral and Bromide a a 25 grs. at four hourly intervals. Milk and Pot. Imp.

The os was fully dilated at 9.30 a.m. and the membranes ruptured. At 11.45 a.m. she was delivered of a macerated foetus which weighed ½ lb. An intrauterine douche was given after the expulsion of the uterine contents.

26th February 1908: The patient passed a very restless night and slept very little. She is still very restless but the maniacal condition seems to have passed off. At 2 p.m. she had a single eclamptic seizure which lasted for three minutes.

Urine 52 ozs; milky; neutral; Specific Gravity 1028; Albumen 3 per thousand Esbach; no blood or tube casts can be found; Urea, 6 grs. per oz.

Treatment. Milk. Imperial Drink. Chloral 15 grs. and Bromide 20 grs. were given four hourly.

27th February 1908: The restlessness has now quite passed off, but there is still a condition of slight dementia remaining.

Urine, abundant; Albumen, trace; no blood; no casts; Urea, 8 grs. per oz.

28th and 29th February 1908; Slight dementia persists. Albumen, trace.

1st March 1908: Improvement has now commenced and it is only for short intervals during the day that the dementia returns. No Albumen.

3rd March 1908: Mental condition is now normal. No Albumen.

Convalescence was uninterrupted, and she was dismissed well on 14th March 1908.

C A S E XIV

DISEASE.

Adenoids

Notes of Case.

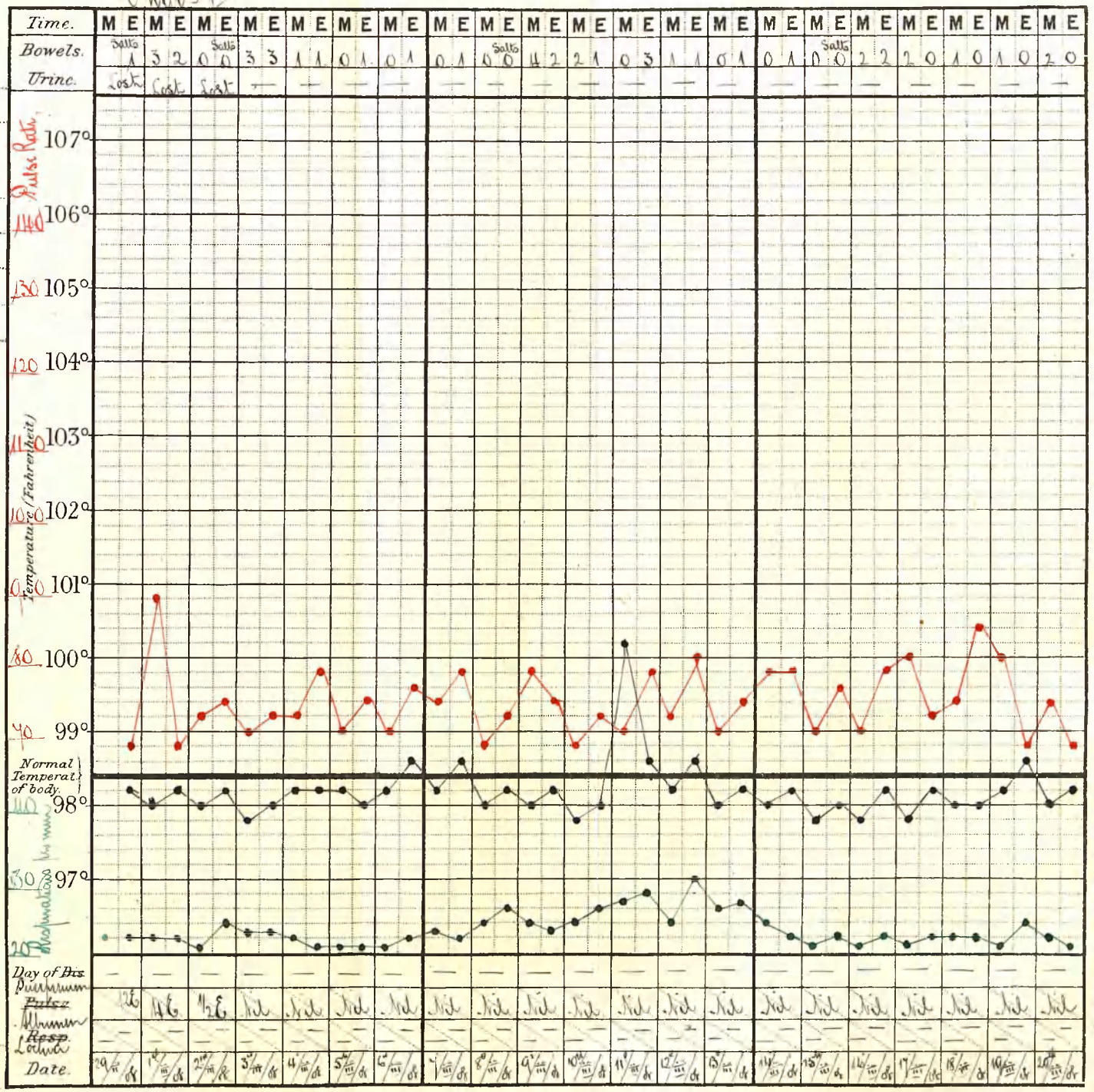
Mrs
Wood
18 Years
Milk - Scales

Book No. XIV

L. Para

Date of admission.
20th January 1908

Result Recovery



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DISEASE.

Dumpsina

Notes of Case.

No

1 Wood

18 Grass

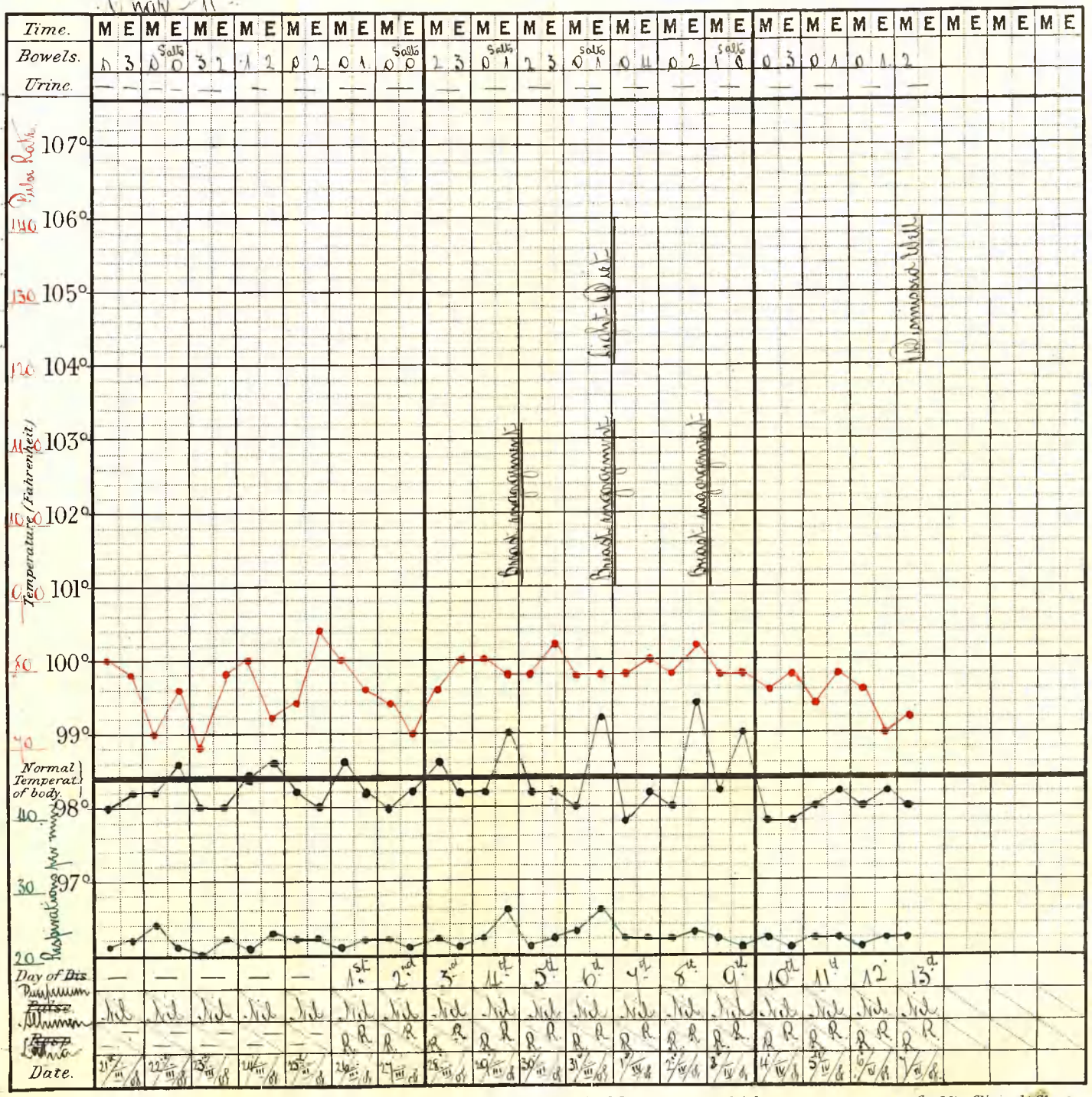
Milk Soup

Book No XIV

Para

Date of admission.

Recovery



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CASE XIV MRS WOOD, aet. 18 years, primipara. 32 weeks.

Recovery.

Admitted on 29th February 1908 at 3 p.m.

Delivered on 26th March 1908 at 2.30 a.m.

Child Female alive, 5 lbs. weight.

HISTORY: For one month previous to the onset of the fits the patient had had swelling of the feet and ankles. Severe frontal headache had been felt for several days before admission. Just before admission she had taken three convulsions, which, from the description given these seizures, appeared to have been eclamptic in character.

PRESENT CONDITION: The patient is quite unconscious and slightly cyanosed. The tongue is lacerated. Temperature, pulse, and respirations are quite normal. Abdominal inspection shows that the abdomen is about the size of a thirty-second week pregnancy. The head presents. No uterine contractions can be felt. Foetal heart's sound and movements can be distinctly heard and felt.

Later, the patient's condition is much as on admission. The cyanosis has passed off. Restlessness comes on at intervals.

Urine. Quantity 20 ozs; dark, opaque; Specific Gravity 1020; Albumen 12 per thousand per Esbach; tube casts scanty; no blood; Urea 6 grs. per 1 oz.

Treatment. Magnes. Sulp. 3 ozs. per tube. Saline transfusion 2 pints. Chloral 15 grs. and Bromide 20 grs. per rectum, four hourly.

1st March 1908: She is more conscious and can swallow fluids today, and the restlessness has increased. There have been no

convulsions. Albumen 4 per thousand per Esbach. Urea 8 grs. per 1 oz.

Treatment. Milk diet. Pot. Imp. Chloral and Bromide stopped.

2nd March 1908: Apart from a slightly dazed condition the patient is conscious. Albumen ½ per thousand per Esbach. Urea 8½ grs. per 1 oz.

3rd March 1908: Complete consciousness has now been restored. There is no Albumen present. Urea 9½ grs. per 1 oz.

4th March 1908: Improvement continues. The signs of foetal life are still present. There are no signs of commencing labour.

As the patient has a wretched home, she was kept in Hospital until her confinement. The diet was always fluid. The urine showed no further trace of Albumen.

26th March 1908: The patient was delivered of a live female child which weighed 5 lbs. There was no return of the convulsions or the Albumen.

Puerperium normal except for swelling of the mammae.

7th April 1908: Dismissed both well.

C A S E XV

DISEASE.

lampsia

Notes of Case.

No.

Name Mac Intyre

22 Years

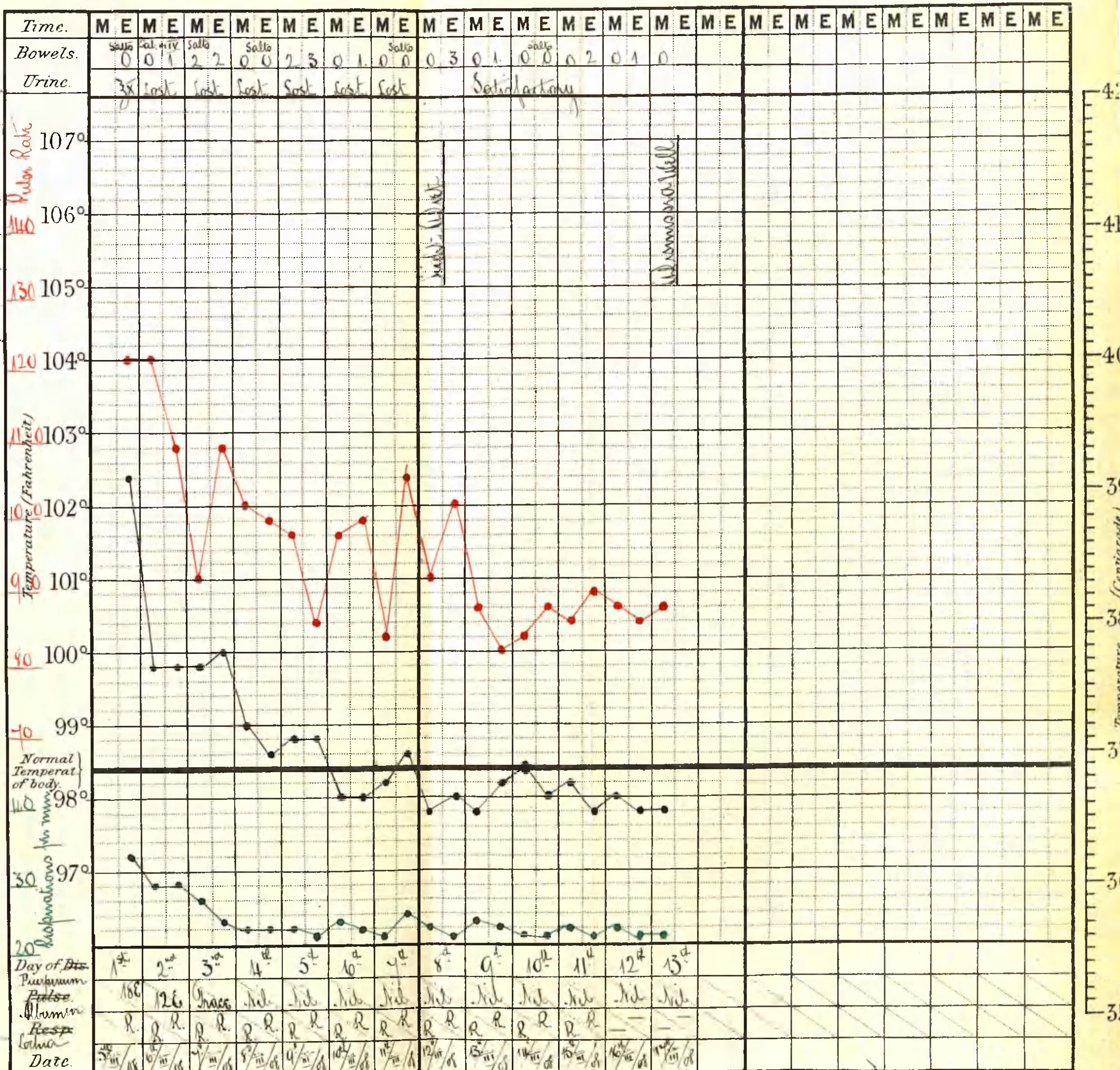
Milk Light

Book No. XV

T. Dara

Date of admission. 5th April 1908

Result Recovery



CASE XV MRS MacINTYRE, aet 22 years, primipara. 27 weeks pregnant. Recovery.

Admitted on 5th March 1908 at 1.30 p.m.

Delivered on 5th March 1908 at 11 p.m.

Child Female, premature, dead, 2½ lbs. weight.

HISTORY: The only prodromal symptom present was a slight oedema of the ankles of about two weeks' duration.

At 3 a.m. on the day of admission she awakened in a "curious dazed condition" and shortly afterwards took a "fit". She had eight convulsions before admission.

PRESENT CONDITION: The patient is quite unconscious and cyanosed. One eclamptic convulsion occurred while she was being washed. Temperature is 102.4° F. Pulse is 120 per minute, full, regular, and of high tension. Abdominal examination showed the abdomen to be more distended from that of a 27 week pregnancy. The shape of the abdomen is distinctly globular, and no foetus can be discovered on palpation. No signs of foetal life can be made out. The uterine contractions had commenced. The os uteri admits one finger. Ballottement can be performed on vaginal examination. Ophthalmoscopic examination reveals the presence of a small haemorrhage on the left retina.

Urine, dark amber; acid; Specific Gravity 1032; Albumen 18 per thousand per Esbach; tube casts; trace, blood.

Treatment. Magnes. Sulph. 3 ozs. per tube. Bled ½ pint, transfusion intravenously Saline 2 pints. Later, hot wet pack. Milk diet.

Later, as labour was not making much progress in spite of vigorous uterine contractions, a canula was

introduced through the os uteri and through the membranes 5 pints of amniotic fluid were drained off in this manner. When the uterus was freed from the excess of liquor amnii labour made rapid progress. The patient was delivered of a premature female child, at 11 p.m. It weighed 2½ lbs.

6th March 1908: She is conscious at intervals. The restlessness has passed off. Urine is fairly abundant. Albumen is 12 per thousand Esbach. Milk and Pot. Imp. were given freely. Calomel 4 grains were given at night.

7th March 1908: The patient is now quite conscious.

Urine - abundant. Albumen - trace.

Treatment. Milk, Imperial drink. Pulv. Jalap Co. 30 grains given in evening.

8th March 1908: Improvement continues. No albumen is present.

15th March 1908: Retinal haemorrhage disappeared. No albumen. Convalescence uninterrupted.

16th March 1908: Dismissed well.

10th October 1908: Patient again pregnant (14 weeks). No premonitory symptoms. No albumen. Retina clear.

C A S E XVI

CASE XVI ELLEN LUKE, aet 21 years, primipara. Full time.

Death.

Post Partum Eclampsia.

Admitted on 12th March 1908 at 11 a.m.

Delivered on 11th March 1908 about 6.30 p.m. (before admission)

Child alive.

HISTORY: No definite history can be obtained previous to the pregnancy. Oedema of the feet and ankles had been present for one month previous to the onset of the convulsions. Headache had also been complained of for this period. She was delivered naturally of a live child on the 11th March 1908 about 6.30 p.m. Three or four hours later she began to take convulsions. These seizures occurred at intervals of from fifteen to twenty minutes, the patient being quite unconscious between the attacks.

PRESENT CONDITION: The patient is completely unconscious and greatly cyanosed. The pupils are dilated and do not react to stimuli. Temperature is 101.4° F. Pulse 145 beats per minute, irregular and of poor tension. Respirations number 30 per minute. The tongue is much lacerated and there is a blood stained froth round the mouth. In the trachea and bronchi there seems to be a considerable quantity of mucus. Percussion of the pulmonary area shows that there is marked impairment of the note behind, (the vocal fremitus is slightly increased). On auscultation moist crepitant rales are heard over the entire lung area. These rales are most abundant at the bases. There is considerable anasarca over the entire body. The oedema is most marked on the feet and ankles,

and over the sacrum. She had five convulsions from time of admission until time of death at 1 a.m. 13th March 1908. Each attack was very severe and lasted for about ten minutes. The comatose phase was particularly long and deep.

Urine. Scanty; high coloured; Specific Gravity 1042; Albumen solid; abundant hyaline and granular casts; Blood abundant; Urea, 2 grs. per oz.

Treatment; Magnes. Sulph 2 ozs. per nasal tube. Saline transfusion intravenously. Stimulants: Strychnine $\frac{1}{60}$ grain four hourly, Brandy 2 ozs. per rectum four hourly, Chloral Hydrate and Potass. Bromide a a 10 grains per nasal tube twice. Hot poulticing of Lung bases and Kidneys.

Later, in spite of treatment which caused the skin, bowels, and kidneys to act freely, the patient became gradually worse and died at 1 a.m. 13th March 1908. The lung condition had become more marked. Temperature, pulse and respirations rose considerably before death.

C A S E XVII

DISEASE.

Anamissia

Postpartum

Notes of Case.

Mrs

Mrs Shaw

24 Years

Nitr - Right

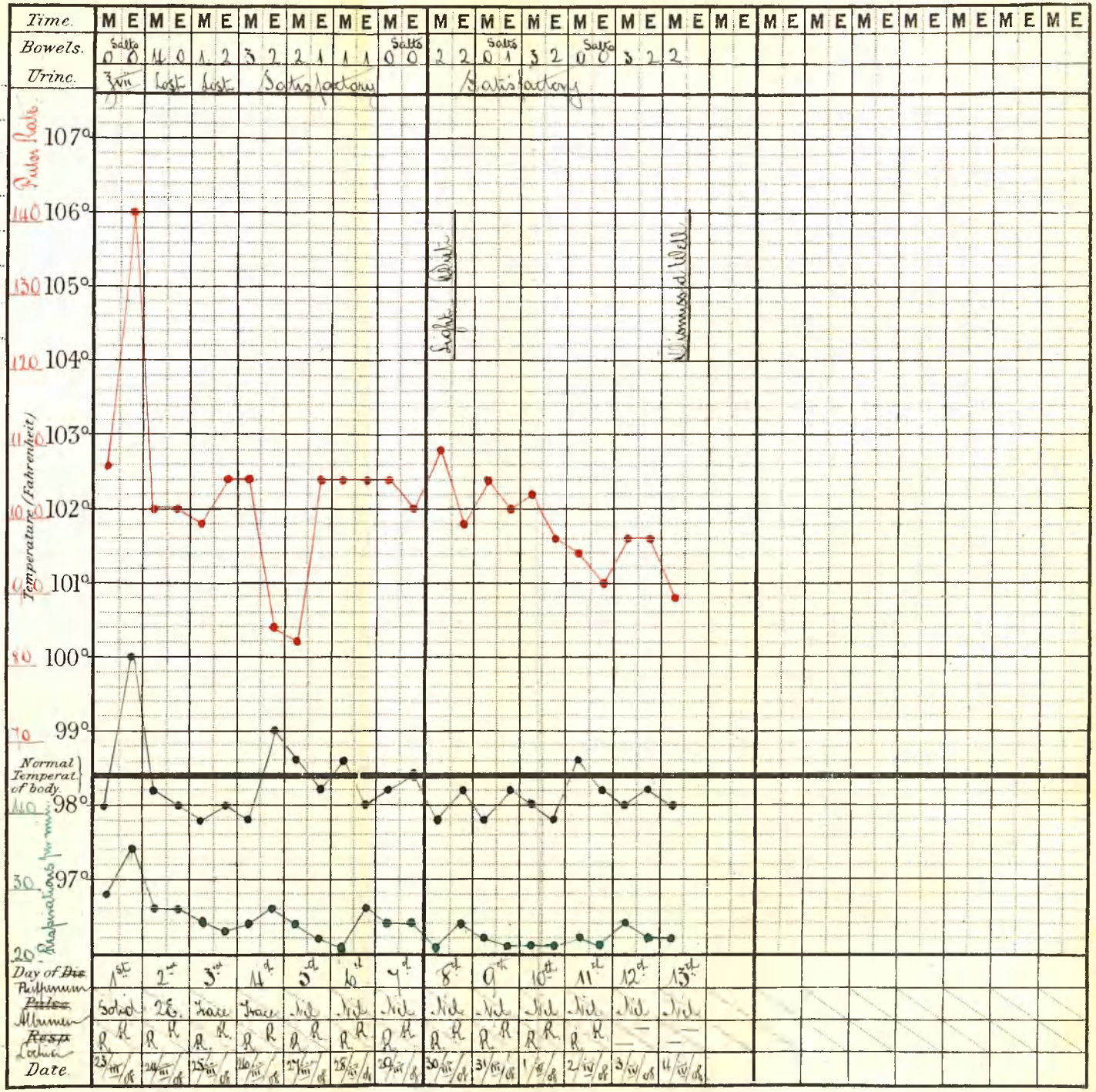
~~Book~~ N° XVII.

Para

Date of admission.

15th March 1908

Result Recovery



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CASE XVII MRS MacPHEE, aet 27 years, IV para. Full time.
Recovery.
Post Partum Eclampsia.
Admitted on 23rd March 1908 at 2 a.m.
Delivered on 22nd March 1908 at 1 p.m. (before admission).
Child Male alive.

HISTORY: Her first three confinements had been normal. During each pregnancy there had been a slight swelling at the ankles. This oedema was more marked after standing for some time, and it was not worse than might be expected during pregnancy. During the fourth pregnancy the swelling of the feet and ankles returned in the later months but it had been at no time excessive. She had also had slight headache from time to time. The eyesight had always been normal. The confinement had apparently been normal. About 9 p.m. on the evening of the delivery she took the first convulsion. This was followed by a second at 12 midnight, and a third occurred at 2.15 a.m., 23rd March 1908. A doctor saw her then, and sent her into hospital.

PRESENT CONDITION: The patient is slightly dazed. She could be aroused and answer questions intelligently. Soon after admission she began to be restless, and had convulsions at 3.55 a.m.; 8.10 a.m.; 9.40 a.m. and 11 a.m. These convulsions were eclamptic in character and were all fairly severe, lasting two to three minutes each. The coma after the attacks lasted about ten minutes. There was a final fit at 1.35 p.m. Between the fits, during

the remainder of the day, and during the next night there was great restlessness. Temperature 97° F. Pulse 106 beats per minute. Respirations 28 per minute. The patient is able to swallow without difficulty.

Urine (Catheter specimen). Quantity 7 ozs; dark amber; acid; Specific Gravity 1040; numerous finely and coarsely granular tube casts; blood distinct; Albumen solid; Urea. Fehling's Solution discoloured but not reduced.

Treatment. Milk diet. Imperial drink. Purge (Magnes. Sulp. 2 ozs.) Hot wet pack. Chloral Hydrate and Potass. Bromide a a 20 grains per rectum every three or four hours. At noon 23rd March 1908 she was bled 1 pint, and transfused Saline 2 pints intravenously.

24th March 1908: Today the patient is much improved. She is conscious and quiet, the restlessness having passed off during the night.

Urine. Quantity greatly increased; milky; tube casts scanty; no blood; Albumen 2 per thousand Esbach.

Treatment. Milk diet. Imperial drink. Chloral and Bromide stopped during the night.

25th March 1908: Improvement continued. Urine abundant, Albumen - trace.

26th March 1908: Improvement continued. Urine abundant, Albumen - trace.

28th March 1908: Convalescence established. No Albumen.

4th April 1908: Dismissed well.

C A S E XVIII

DISEASE.

Eclampsia

Notes of Case.

Mrs. Hunt

23 Years

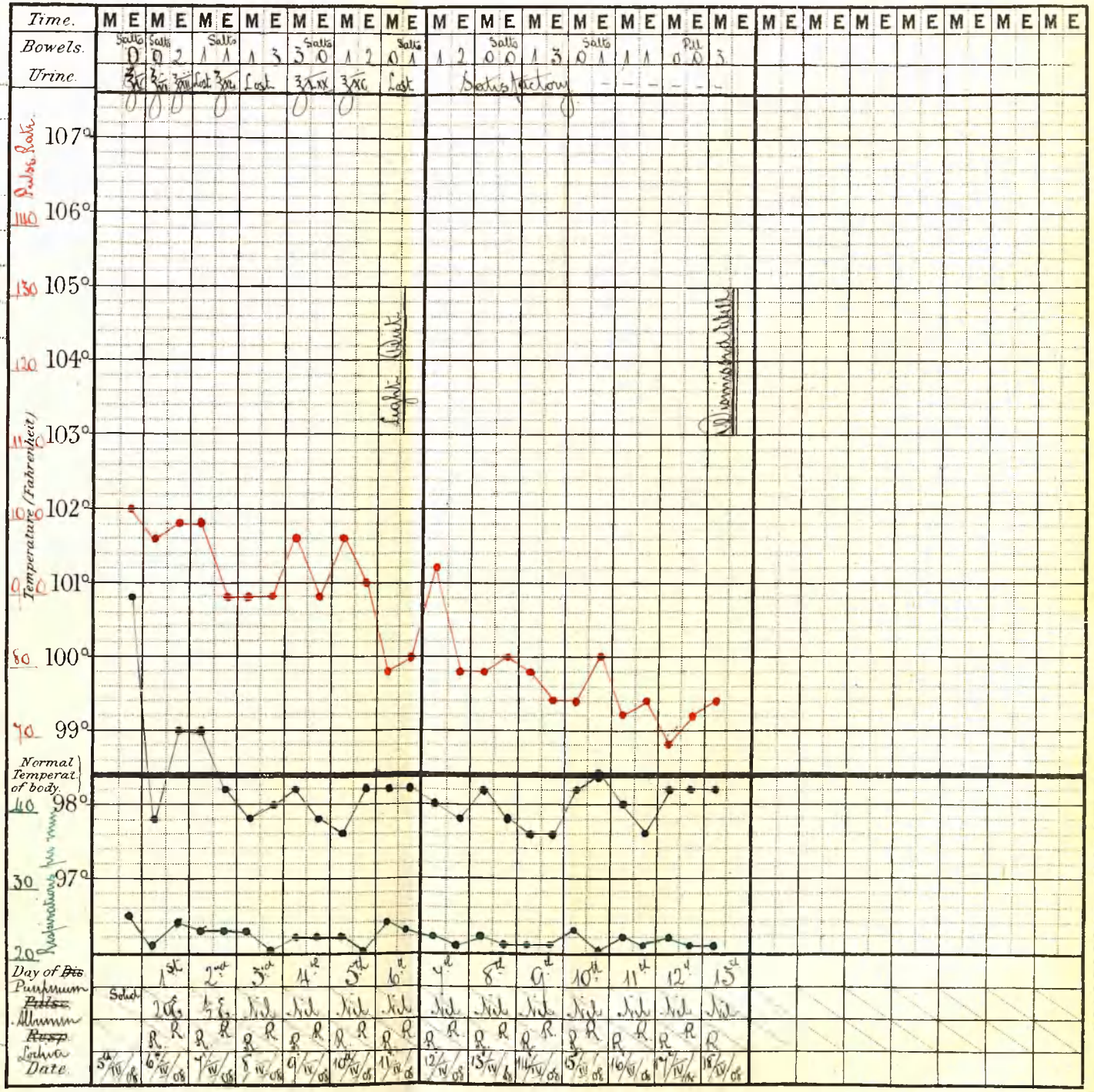
Milk Light

Case Book No. XVIII

L. Para

Date of admission. 3rd April 1908.

Result Recovery



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CASE XVIII MRS FLINT, aet 23 years, primipara. 25 weeks.

Recovery.

Admitted on 5th April 1908 at 5.50 p.m.

Delivered on 5th April 1908 at 11.15 p.m.

HISTORY: There is no history of the patient having had any previous illness. For two months before admission she had had swelling of the feet and ankles, but no oedema of the face was noticed. Frontal headache had also been complained of, and on the day of admission she had had severe epigastric pain. She had had six convulsions in the twelve hours before admission, the last being at 2 p.m.

PRESENT CONDITION: The patient is conscious and sensible. The face is pale and puffy, especially under the eyes. The tongue is lacerated. Temperature is 100.8° F. Pulse 96 beats per minute, and Respirations 25 per minute. Abdominal inspection shows that the uterus is about the level of the unbilicus. There are slight uterine contractions. Per vaginam the os uteri is found to admit the tip of a finger. The contractions of the uterus gradually became stronger and the contents of the uterus were expelled intact at 11.15 p.m. She had no convulsions in Hospital.

Urine. (Catheter specimen) 5 ozs.; dark; acid; Specific Gravity 1040; Albumen solid; tube casts; no blood; Urea, 4 grs. per oz.

Treatment. Milk diet. Imperial drink. Magnes. Sulph. 2 ozs. At 7 p.m. 18 ozs. blood withdrawn from median basilic vein and 2 pints Saline transfused.

6th April 1908: The oedema of the face has now subsided,

otherwise the condition of the patient is much the same as yesterday.

Urine. Scanty; dark; acid; Specific Gravity 1032; Albumen 20 per thousand Esbach; no blood; Urea, 6 grs. per oz

Treatment. Milk diet, Imperial drink. In the evening hot wet pack given, and later hot poultice applied to the Kidney region.

7th April 1908: Patient's condition is greatly improved. The urinary output has risen to 98 ozs. Albumen is ½ per thousand Esbach. Urea, 8 grs. per oz.

8th April 1908: Albumen - nil. Large diuresis.

Convalescence uninterrupted.

18th April 1908: Dismissed well.

C A S E XIX

DISEASE.

Melancholia

Notes of Case.

Name: Mary Crichton

Age: 21 Years

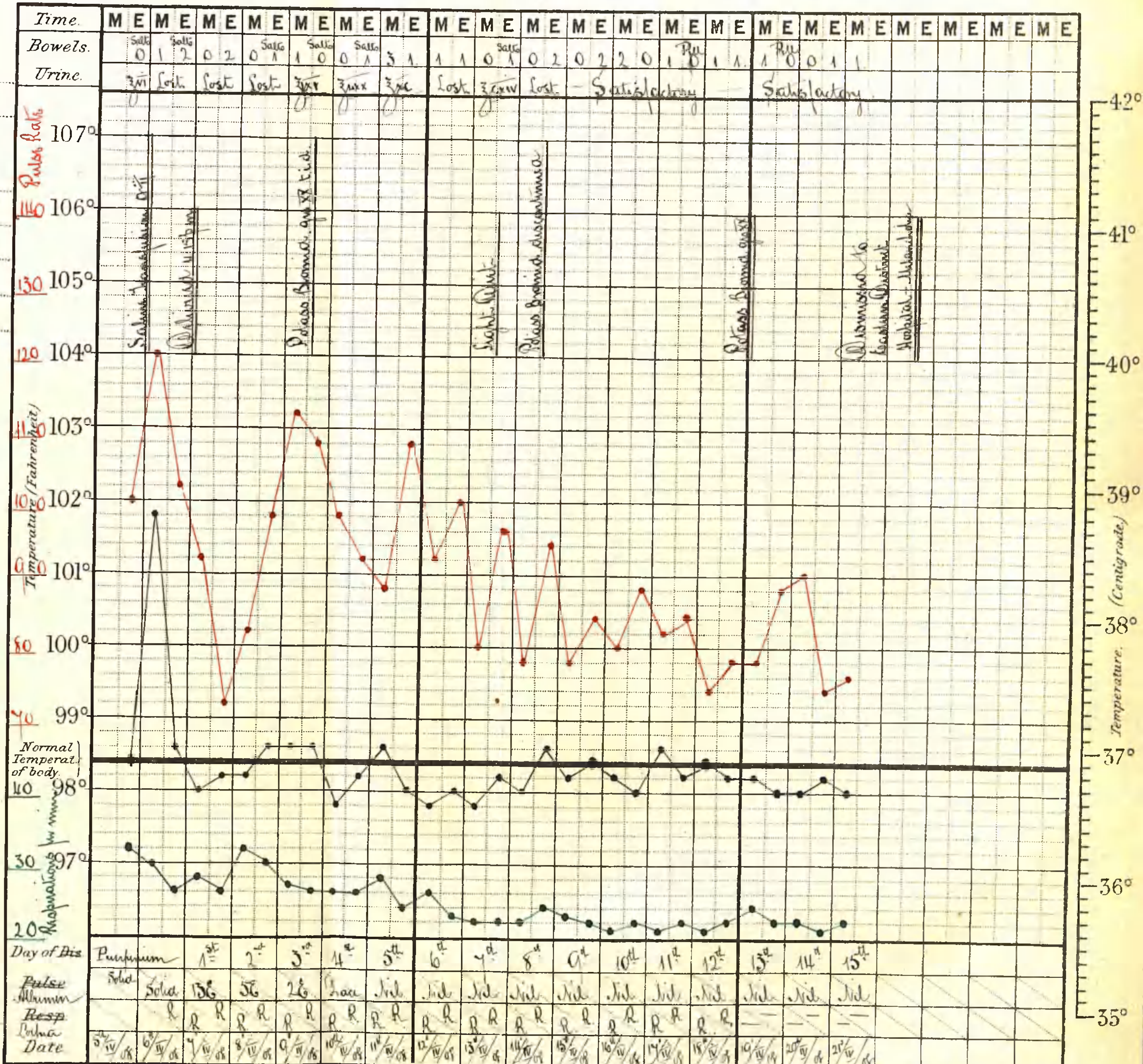
Diet: Milk - Light

Case Book No. XIX

I. Para

Date of admission 5th April 1908

Result Recovery



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CASE XIX MARY CRICHTON, aet 21 years, primipara. 36 weeks.

Recovery.

Admitted on 5th April 1908 at 6.40 p.m.

Delivered on 6th April 1908 at 4.15 p.m.

Child Female alive, 5 lbs. weight.

HISTORY: Patient had had always good health. Her mother stated that she had been greatly affected mentally during her pregnancy, especially during the last three months. The convulsions commenced about eight hours before admission. She had had numerous fits, the exact number is not known. There had evidently been no warning of the onset of the attacks. For beyond a slight oedema of the dorsal aspects of the feet there had been no symptoms.

PRESENT CONDITION: The patient is quite unconscious. The pupils are moderately contracted and react feebly to stimulation. There is slight oedema of the feet but none elsewhere. The tongue is lacerated. Temperature is normal. Pulse 100 beats per minute, it is regular and the tension is moderately high. Abdominal examination shews the uterus to reach almost to the ensiform cartilage. The foetal heart can be heard but no movements of the child can be discovered. The vertex presents and is in the left occipito-anterior position. Per vaginam the cervix is short, and the os uteri is closed. No uterine contractions can be discovered.

Shortly after admission the patient took a typical eclamptic convulsion which lasted two minutes, and in the first hour in the Hospital she had six similar attacks. She had further convulsions at 8.20 p.m., 8.50 p.m.,

10 p.m., 3.45 a.m., 4.15 a.m., and 5 a.m. Between each attack she remained in a more or less comatose condition. The length of the these convulsions varied from one and a half to three minutes.

Urine. Slightly acid; dark, muddy; Specific Gravity 1030; Albumen solid; granular tube casts; faint trace of blood; Urea, 6 grs. per oz.

Treatment; Magnes. Sulph. 2 ozs. per nasal tube. She was bled 1 pint, and transfused 2 pints Saline (This was during the first hour after admission. She had 4 fits in Hospital before transfusion, and 11 afterwards) About 9.30 p.m. a hot wet pack was given. Chloral Hydrate and Potass. Bromide a a 25 grains were given three or four hourly per rectum.

6th April 1908: The patient is semi-conscious today, and inclined to be restless. The restlessness increased as the day advanced. In addition to the oedema of the feet there is a puffiness of the face. The skin, kidneys, and bowels are acting satisfactorily today. Swallowing can be performed without much difficulty. At 10.30 a.m. when palpating the abdomen she took two convulsions in quick succession. These were the last. The uterus was found to be contracting and the os dilating. The membranes ruptured at 1.45 p.m., and she delivered herself naturally of a live female child, at 4.15 p.m.

Urine. Exact quantity passed could not be measured, as quantities were passed involuntarily and at stool, but there seemed to be a fair diuresis; acid; Specific Gravity 1025; Albumen solid; casts; blood - a trace; Urea, 5 grs.

Treatment. Milk, Pot. Imperial. Chloral and Bromide

stopped.

7th April 1908: The patient was quiet but in a state of dementia. Albumen 12 per thousand Esbach. Few casts. No blood. Urea, 6 grs. per oz.

8th April 1908: She is much depressed, and wept frequently. Albumen 5 per thousand Esbach. Urea, 6 grs. per oz.

10th April 1908: The patient is still in a melancholic state, with occasional hysterical outbursts. Albumen - trace. No casts or blood. Urea, 8 grs. per oz.

Potass. Bromide 20 grains given four hourly.

13th April 1908: She is much in the same condition as when last reported. Albumen nil. Urea. 9½grs. per oz.

16th April 1908: A slight suicidal tendency developed, and she has to be closely watched. Albumen nil. Urea, 8 grs. per oz.

21st April 1908: As there is no marked improvement in the patient's mental condition she was sent to Duke Street Hospital for observation. She did not nurse the child, which was taken home the day following the birth.

C A S E XX

DISEASE.

...ampsia

...neumonia

...otes of Case.

Thomasina

McIsaac

20 Years

Wife

Book No. XX

1 Para

...ate of admission.

22nd April 1908

...lt Recovery

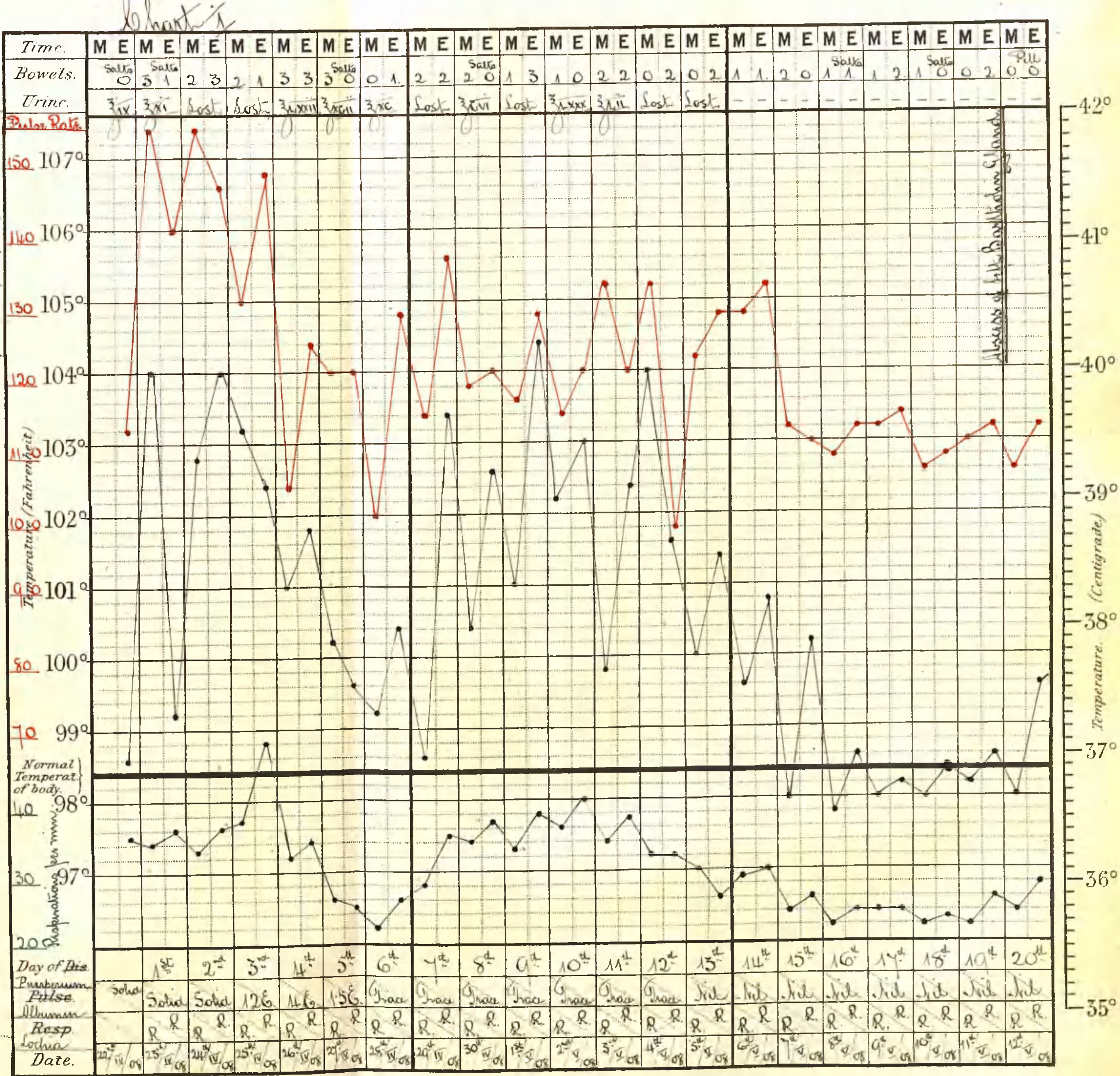

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Chart No 11

DISEASE.

Eclampsia
Pneumonia

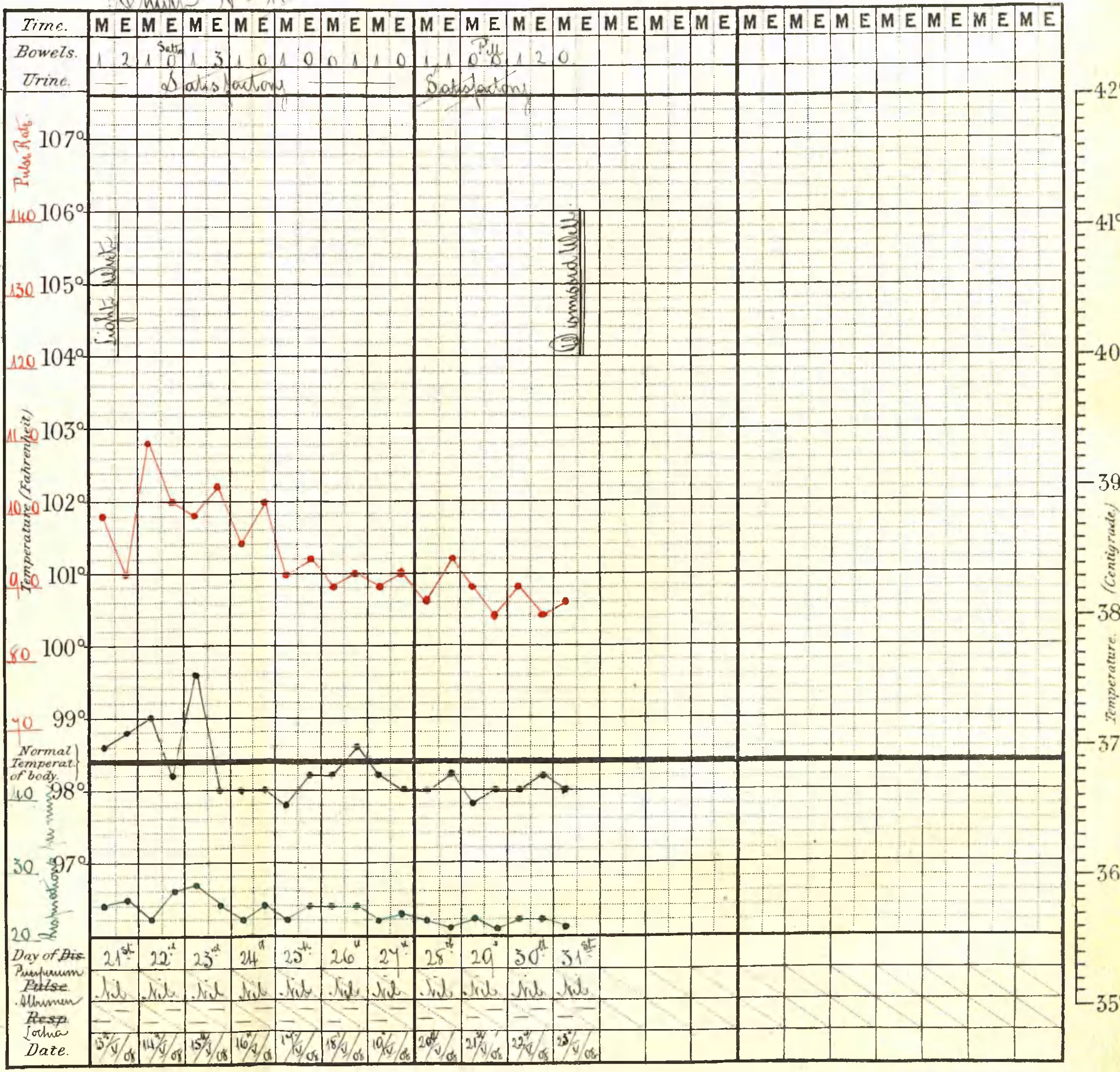
Notes of Case.

Thomasina
Mrs Isaac
20 Years
Milk
Book No XX

1 Para

Date of admission.
22nd April 1908

Result Recovery



CASE XX THOMASINA MacISAAC, aet 20 years, primipara. Full time. Recovery.

Admitted on 22nd April 1908 at 6.40 p.m.

Delivered on 23rd April 1908 at 7 a.m.

Child Male dead, 8 lbs. weight.

HISTORY: About one month ago the patient first noticed a swelling of the feet and ankles. This was observed at first only on standing or on allowing the lower extremities to remain in a dependant position. The swelling afterwards became permanent. No oedema of the face was noticed. Intermittent headache was felt and only became extreme on the morning of admission. At this time also she complained of a severe epigastric pain. She also vomited several times. The first convulsion came on at 2 p.m., and between that time and the hour of admission (6.40 p.m.) she had had eight attacks.

PRESENT CONDITION: The patient is quite unconscious. She is cyanosed, puffy about the face, and there is some oedema on the lower extremities. The pupils are small and react to stimuli. The tongue and buccal part of the cheeks are badly lacerated. Temperature is normal. Pulse numbers 112 per minute, is regular and of moderately high tension. Respirations number 35 per minute. There are moist rales heard over the whole pulmonary region, most marked however at the bases behind, where the percussion note is somewhat impaired. The uterus is equal in size to that of a full time pregnancy. The vertex presents. Auscultation fails to reveal foetal heart's sounds. No foetal

movements can be discovered. There are slight uterine contractions to be felt. Per vaginam the cervix is found to be fully taken up, and the os uteri admits one finger.

Urine. Quantity, per catheter 9 ozs.; dark amber coloured; acid; Specific Gravity 1035; Albumen solid; granular casts; no blood.

Treatment. Purge (Magnes. Sulph. 1 oz.) Bled 10 ozs. Saline transfused intravenously 2 pints. Chloral and Bromide a a 30 grains per rectum.

23rd April 1908: Uterine contractions were strong, and she was very restless during the night. Membranes ruptured at 3 a.m. At 7 a.m. she was delivered naturally of a dead male child. The temperature rose to 104° F., and the pulse was uncountable.

Later, she is still restless and semi-conscious. The oedema of the upper parts of the lungs has disappeared.

Urine 10 ozs.; dark coloured; acid; Specific Gravity 1040; Albumen solid; tube casts; no blood.

Treatment. Chloral and Bromide a a 20 grains were given twice per rectum in the early morning. A hot wet pack was given. When the pulse became very feeble Saline 1½ pints was transfused under the right breast. Alcohol ½ oz was given four hourly.

24th April 1908: Patient is very ill, is now quite quiet, and semi-conscious. She has a high temperature, and a feeble pulse. The urine is still solid with Albumen. In addition to the alcohol Strychnine $\frac{1}{60}$ grain and Digitalin $\frac{1}{100}$ grain were given four hourly.

25th April 1908: The patient is much in the same condition as

yesterday. Temperature 103.2° F. Pulse 130 beats per minute. Respirations number 48 per minute. At the base of the left lung behind there is crepitant rale to be heard. Percussion, vocal fremitus and resonance are not appreciably altered. In the morning the pulse became very weak and irregular. Strychnine was increased to $\frac{1}{30}$ grain and Saline 1½ pints was transfused under the right breast. There was marked improvement in the pulse after this treatment. It numbered 120 beats per minute, and was more regular.

Urine. Albumen 12 per thousand Esbach.

26th April 1908: She is still semi-conscious. Temperature and pulse have fallen somewhat. Lung condition is as yesterday. Lochia is scanty and has a little heavy odour.

Urine. Albumen 12 per thousand Esbach.

Treatment as before.

27th April 1908: The patient is now conscious, and there is a further improvement in the temperature and pulse. Lochia is still less than usual in amount, but the colour and odour are again normal. Albumen 4 per thousand Esbach.

28th April 1908: The condition of the patient is much the same as before. Albumen 1¼ per thousand Esbach.

29th April 1908: Cough is present today. Respirations are more rapid, 35 per minute. The pulse and temperature rose considerably. Examination of the base of the left lung shows that there is an impaired percussion. Fine rale is present in abundance and there is a suspicion of increased vocal resonance.

Urine. Albumen - trace.

30th April 1908: The condition of the left lung has become more marked. The dulness is deeper and has extended upwards to the mid scapular line and outwards to the mid axillary line. Tubularity is present over this area, and there is increased vocal fremitus and resonance. The patient also complains of pain in this region on drawing a deep breath or on coughing. The sputum is gelatinous, rusty, and contains pneumococci. Herpes is appearing on the lips.

Urine. Albumen - trace. Chlorides diminished.

1st May 1908: The physical condition of the left lung is much the same as yesterday. Stimulants still given as before.

Urine. Albumen - trace. Chlorides diminished.

2nd, 3rd, and 4th May 1908: There is no change in the condition of the patient. The temperature gradually fell to normal and reached this level on the 8th May 1908. The condition of the left lung also resolved, a few days later, and gradually disappeared. Convalescence was uninterrupted.

11th May 1908: Small abscess formation in Left Bartholin Gland whichwas opened two days later, and healed quickly.

She was dismissed on May 23rd 1908 well. The Albumen had completely disappeared from the urine some days previously.

C A S E XXI

DISEASE.

Malaria

Notes of Case.

Name { Mrs. Shields

Age 23 years.

Diet Milk

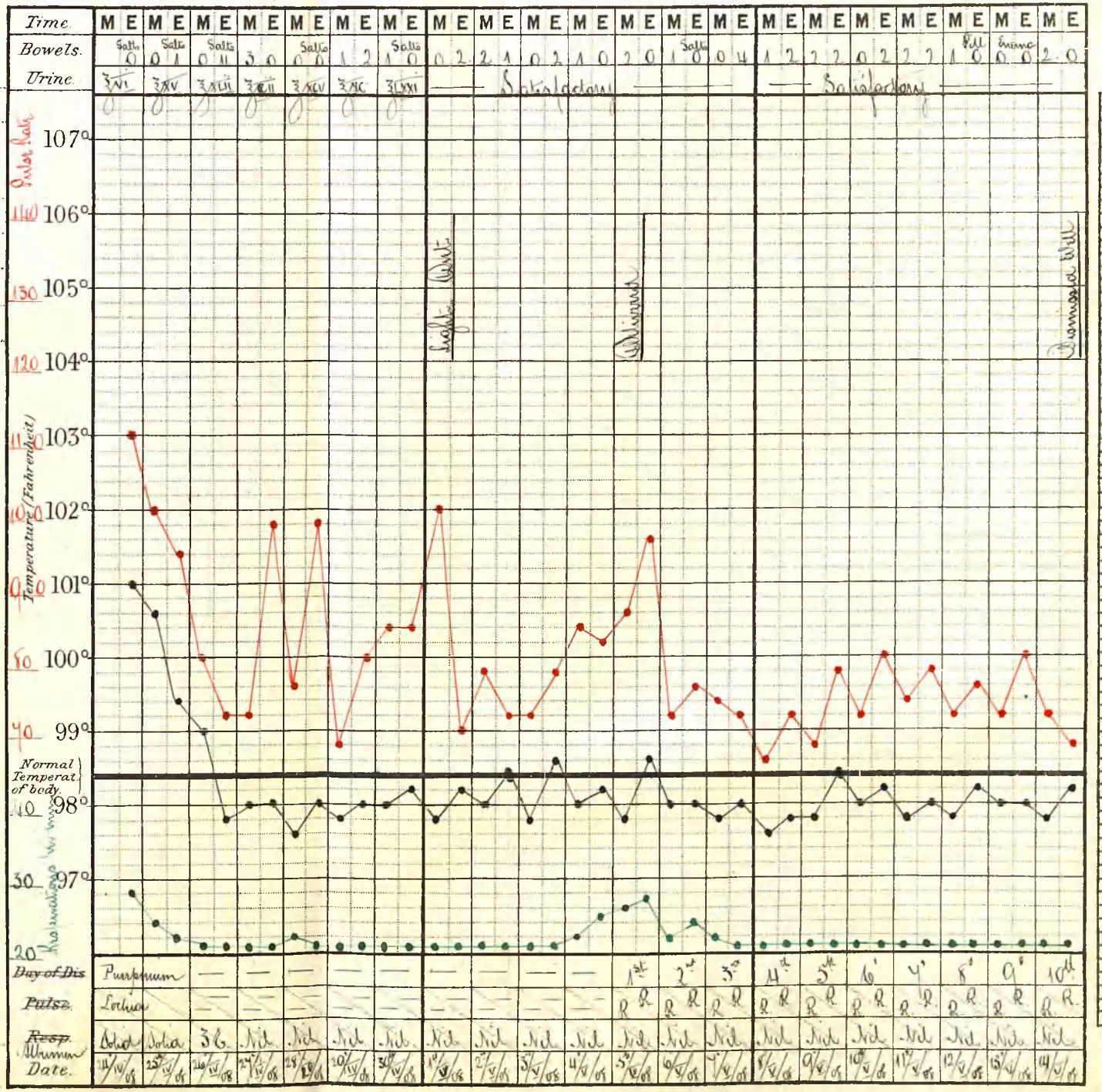
Case Book No. XXI

IV Para

Date of admission.

24th April 1908

Result Discharged



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CASE XXI MRS SHIELDS, aet 23 years, IV para. 34 weeks pregnant.
Recovery.
Admitted on 24th April 1908 at 4.25 p.m.
Delivered on 5th May 1908.
Child Female dead (macerated) 3 lbs. weight.

HISTORY: The patient had had no illnesses previous to her first pregnancy. Three years ago, during the first pregnancy, she had swollen feet and puffiness of the face. She was in the Newcastle Infirmary suffering from eclampsia at her first confinement, which came on at the seventh month. The second and third pregnancies each terminated between the third and fourth months. There did not seem to have been any albuminuric symptoms on those occasions. In her present pregnancy she had had oedema of the feet and ankles of about two months duration, and puffiness of the face of three days duration. From time to time she had had severe headache. About noon on the 24th April 1908, after suffering from severe headache for some hours she began to take convulsions and had had eight before admission.

PRESENT ILLNESS: The patient is quite unconscious. The pupils are dilated and the reflexes are gone. The face is cyanosed, and the entire body is slightly oedematous. The tongue is lacerated, and there is a blood-stained froth about the mouth. The breathing is stertorous in character. Temperature is 101° F. Pulse numbers 110 per minute, is full and of high tension. Respirations are 28 per minute. The retina show signs of inflammation and there are old haemorrhages present. The fundus of the

uterus reaches almost to the umbilicus. No uterine contractions are found. Foetal movements cannot be felt, nor can the foetal heart's sounds be heard. On vaginal examination the os uteri only admits the tip of a finger, and the cervix is not wholly taken up. The fundi oculi are normal.

Urine. Quantity scanty, 6 ozs. withdrawn per catheter; dark amber coloured; Specific Gravity 1020; acid; Albumen abundant; granular casts; no blood; Urea 4 grains per oz.

Treatment. Magnes. Sulph. 3 ozs. was given per stomach tube. The patient was then bled 1 pint and transfused with 2 pints Saline solution. Chloral Hydrate and Potass Bromide a a 30 grains were given twice per rectum at a three hourly interval.

Later. Between admission and transfusion one hour later she had two typical eclamptic seizures. After transfusion she had one convulsion.

25th April 1908: The patient has had an extremely restless night, and is semi-conscious. At 6 a.m. she again began to take convulsions and had seven in rapid succession. After this succession of attacks she was again completely unconscious. The temperature and pulse did not rise with those attacks. Later in the day she became semi-conscious and had no return of the fits.

Urine. Quantity 15 ozs; dark coloured; acid; Specific Gravity 1032; Albumen abundant; tube casts; no blood; Urea 5 grains per oz.

Treatment. After the series of convulsions 1 pint

Saline fluid was transfused into the sub-mammary tissue. Chloral Hydrate and Potassium Bromide a a 25 grains were given per rectum four hourly. Nutrient enemata were also given.

26th April 1908: The patient is now quiet and there have been no further convulsions. She is semi-conscious and can swallow fluids.

Quantity of urine measured. 30 ozs. were collected per catheter, and a quantity was lost, being passed involuntarily in bed and at stool. Albumen 3 per thousand Esbach. Urea 6 grs. per oz.

Treatment. Milk diet.

27th April 1908: Consciousness has now almost fully returned, only a dazed condition remaining. The quantity of urine passed is 102 ozs., and contained no Albumen. Urea 8 grs. per oz.

28th April 1908: The patient is now fully conscious. No Albumen is present in the urine. Urea 9 grs. per oz.

5th May 1908: The patient was delivered naturally today of a macerated female child weighing 3 lbs. and measuring 17" in length.

No Albumen is present in the urine.

The Puerperium was quite normal and she was dismissed on the 14th April 1908, well.

C A S E XXII

Chart I

DISEASE.

Eclampsia

Notes of Case.

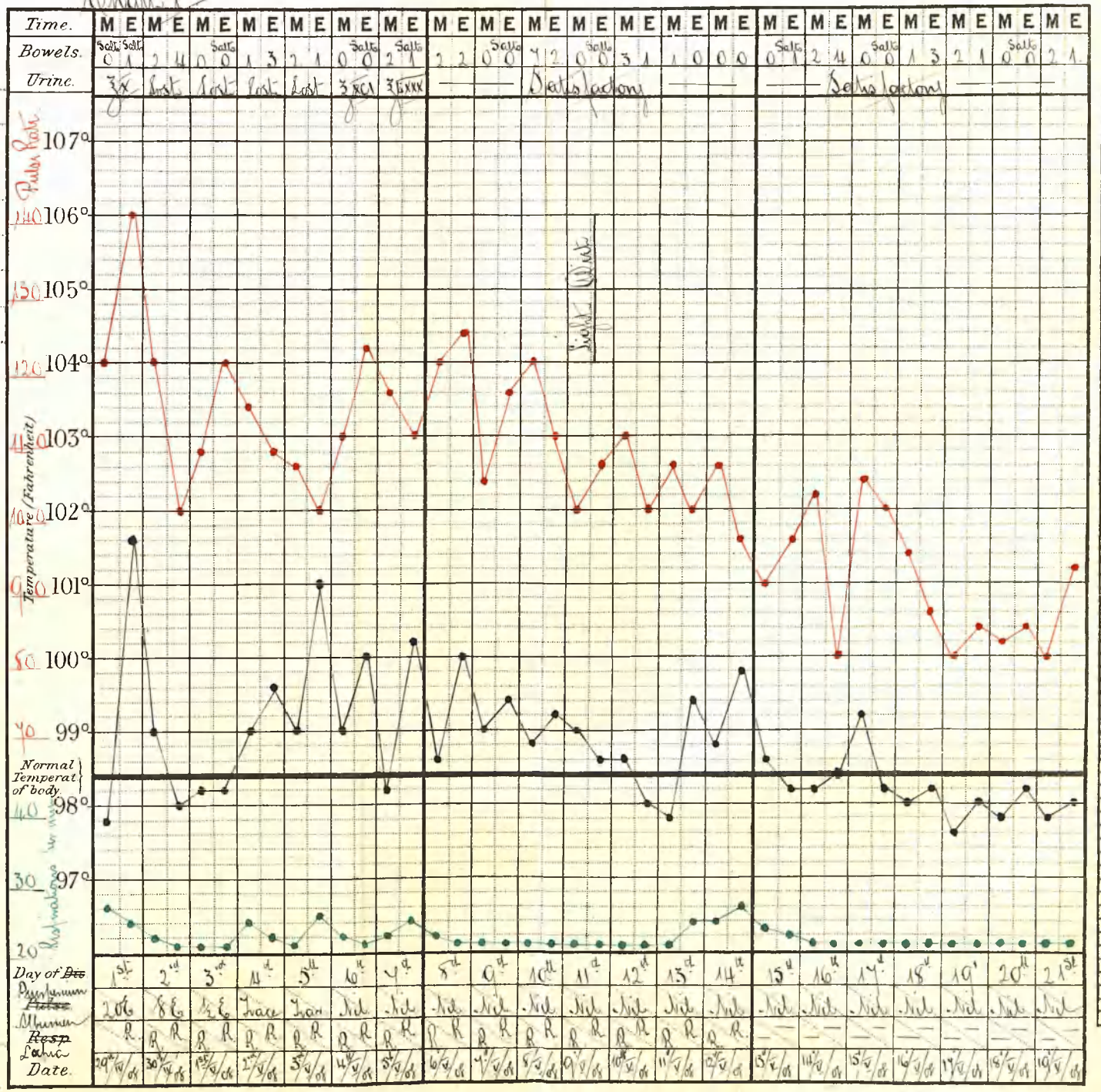
Name: Margaret Barr

Age: 19 years

Occupation: Milk - Lasht

Book No. XXII

1 Para



Date of admission. 20th April 1908.

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DISEASE.

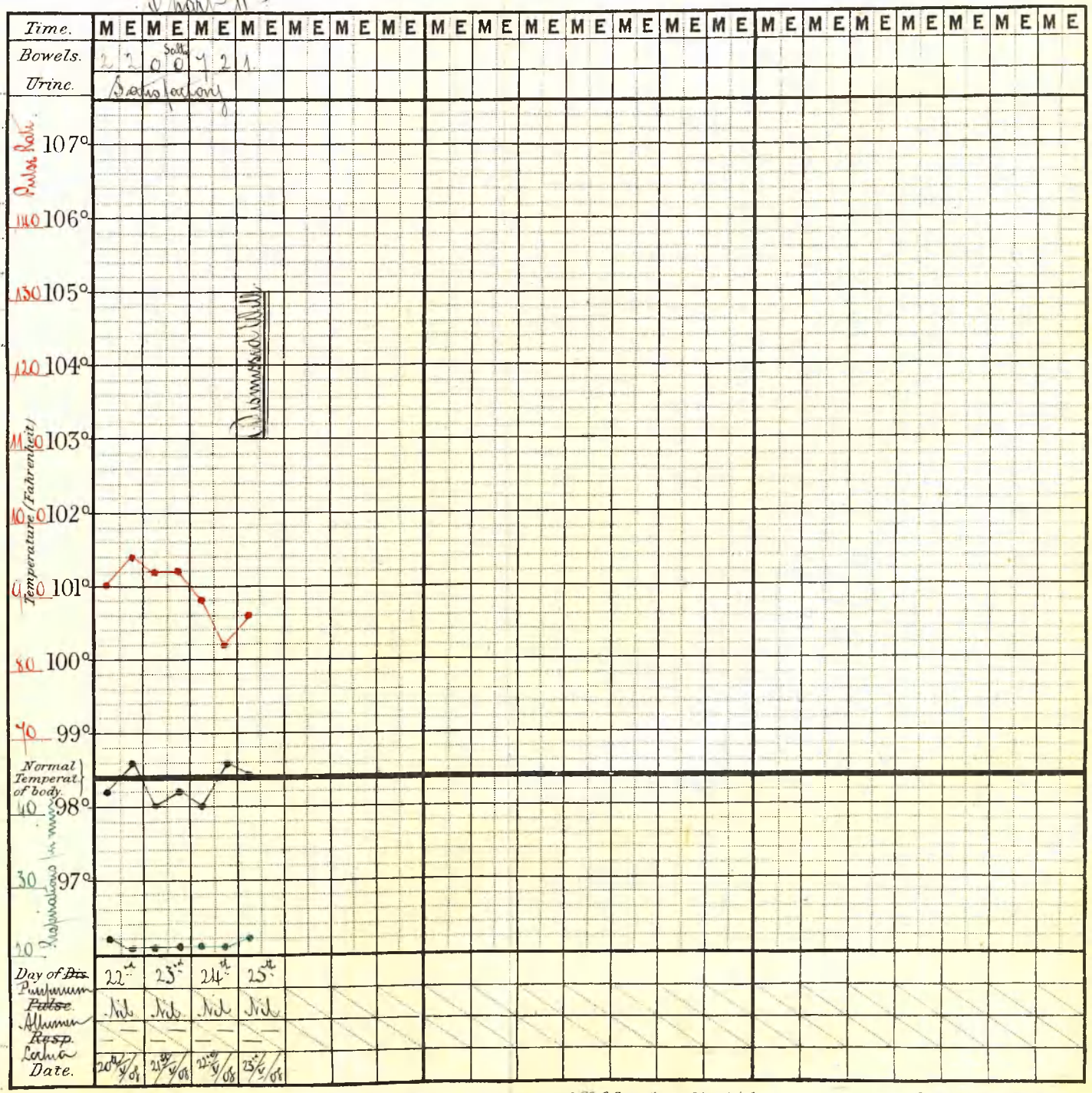
Malaria

Notes of Case.

Margaret Barr
19 Years
Mrs. Light
Book No. XXII

Para

Date of admission.
20th April 1908
Result Recovery



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CASE XXII MARGARET BARR, aet 19 years, primipara. Full time. Recovery.

Admitted on 29th April 1908 at 5.15 a.m.

Delivered on 29th April 1908 at 9.50 p.m.

(chloroform forceps)

Child Female dead, 7½ lbs. weight.

HISTORY: There was no history of previous illness that can be discovered. The patient did not notice any swelling of the face or extremities. Last evening (28th April 1908) she was seized with a severe epigastric pain which she thought was due to eating a large quantity of cheese to supper. The pain gradually became worse and at an early hour on the day of admission she took a severe convulsion. This was followed by three others.

PRESENT CONDITION: The patient is cyanosed and quite unconscious. The pupils are dilated and do not react to stimuli. There is no oedema on any part of the body. The uterus is equal in size to that of a full time pregnancy. Auscultation of the abdomen fails to reveal any foetal heart's sounds. No foetal movements can be felt. Slight uterine contractions are in progress. Per vaginam the os uteri admits the tip of a finger. The cervix is fully taken up.

Urine. Quantity 10 ozs. per catheter; muddy; Specific Gravity 1035; acid; Albumen 20 per thousand Esbach; granular casts; blood distinct.

Shortly after admission the patient began taking eclamptic seizures and had three in the first hour before

transfusion, and five in the succeeding seven hours. During the clonic stage in each of the fits the movements were largely confined to the left side, the right side of the body only moving slightly.

The os uteri was fully dilated and the membranes were ruptured artificially prior to the application of the instruments. Labour progressed and she was delivered by forceps under an anaesthetic at 9.50 p.m. The placenta and membranes were adherent and were removed manually. An intrauterine douche of sterile water at 118° F. was then given. After delivery the temperature was 101.8° F. Pulse 140, feeble and a low tension. Respirations numbered 35 per minute.

Treatment. Magnes. Sulph. 2 ozs. per nasal tube. Bled from right median basilic vein 1 pint; transfused Saline 2 pints. After six convulsions Chloral and Bromide a a 30 grs. were given per rectum. A hot wet pack was given in the forenoon.

Just before delivery the pulse became very rapid (150 beats per minute) and running, and an additional Saline 2 pints were transfused into the left median basilic vein. Strychnine $\frac{1}{60}$ grain was given, $\frac{1}{30}$ grain was injected hypodermically. Brandy 2 ozs. was given four times per rectum at two hourly intervals.

30th April 1908: Early this morning the patient rallied. There were no further convulsions. She is still unconscious and nutrient fluids are being given by a tube. The pulse is slower, stronger, and more regular.

Urine. Quantity abundant; milky; acid; Specific Gravity 1022; Albumen 8 per thousand Esbach.

Treatment. Strychnine $\frac{1}{60}$ gr. hypodermically four hourly. Brandy 1 oz. four hourly per rectum.

1st May 1908: The patient has been extremely restless. Consciousness has been returning gradually. She can now swallow fluids.

Urine contains Albumen $\frac{1}{2}$ per thousand Esbach. Strychnine was stopped today.

2nd May 1908: She is still semi-conscious and restless although she slept well during the night. Stimulant was stopped. Albumen - trace.

3rd May 1908: The patient is now quite conscious. The temperature is slightly elevated. A slight cystitis is present. Urine contains a trace of Albumen. Pus cells and mucus are also present.

Treatment: Urotropine 10 grs. four hourly.

The cystitis continued until May 7th 1908, after which date the urine gradually became normal, and she was dismissed on 24th May 1908, well.

C A S E XXIII

CASE XXIII MRS MacDOUGAL, aet 38 years, VI para. Full time.
Death.
Admitted on 29th April 1908 at 4 a.m.
Died undelivered on 29th April 1908 at 5.45 a.m.

HISTORY: There was no history of antecedent disease. The five previous children were born naturally, and without any symptoms of toxaemia. During the present pregnancy she had had slight swelling of the feet and ankles of unknown duration. No other symptoms seem to have been felt. About 10 p.m. on the 28th April 1908 she was seized with a convulsion which was reported to have lasted for 15 minutes. She had five succeeding seizures.

PRESENT CONDITION: The patient is cyanosed and completely unconscious. The tongue is lacerated. The pupils are dilated and the reflexes gone. No oedema is noticed on any part of the body. The temperature is 97.8° F. Pulse numbers 145 beats per minute, and is feeble, irregular, and of poor tension. Respirations are 25 per minute, and are short and gasping. The size of the uterus is equal to that containing a full time child. No signs of the foetal life can be detected. No uterine contractions are in progress.

Urine (Catheter specimen). Quantity 2 ozs; colour is almost black; consistence is muddy; Specific Gravity 1040; reaction neutral; Albumen solid; casts (granular blood) plentiful; blood abundant.

Shortly after admission she took another exceedingly severe convulsion or rather series of convulsions which lasted ¾ hour. These consisted mainly of a continuation of clonic movements. These movements were very slight

and feeble. The pulse could not be felt during or after this attack. After the subsidence of this continued seizure the patient was practically moribund. Respirations were gasping, spasmodic, and were accompanied by facial contortions. She gradually sank and died undelivered within two hours of admission.

Treatment. Saline 2 pints transfused. Stimulants given freely. Hot applications were placed over the cardiac region. Chloral Hydrate and Potassium Bromide were given per rectum. Chloroform was considered during the continuous seizure, but was not given as the cardiac condition was too feeble and the pulse gone.

C A S E XXIV

DISEASE.

Pneumonia

Broncho-pneumonia

Notes of Case.

Mrs Evans

23 Years

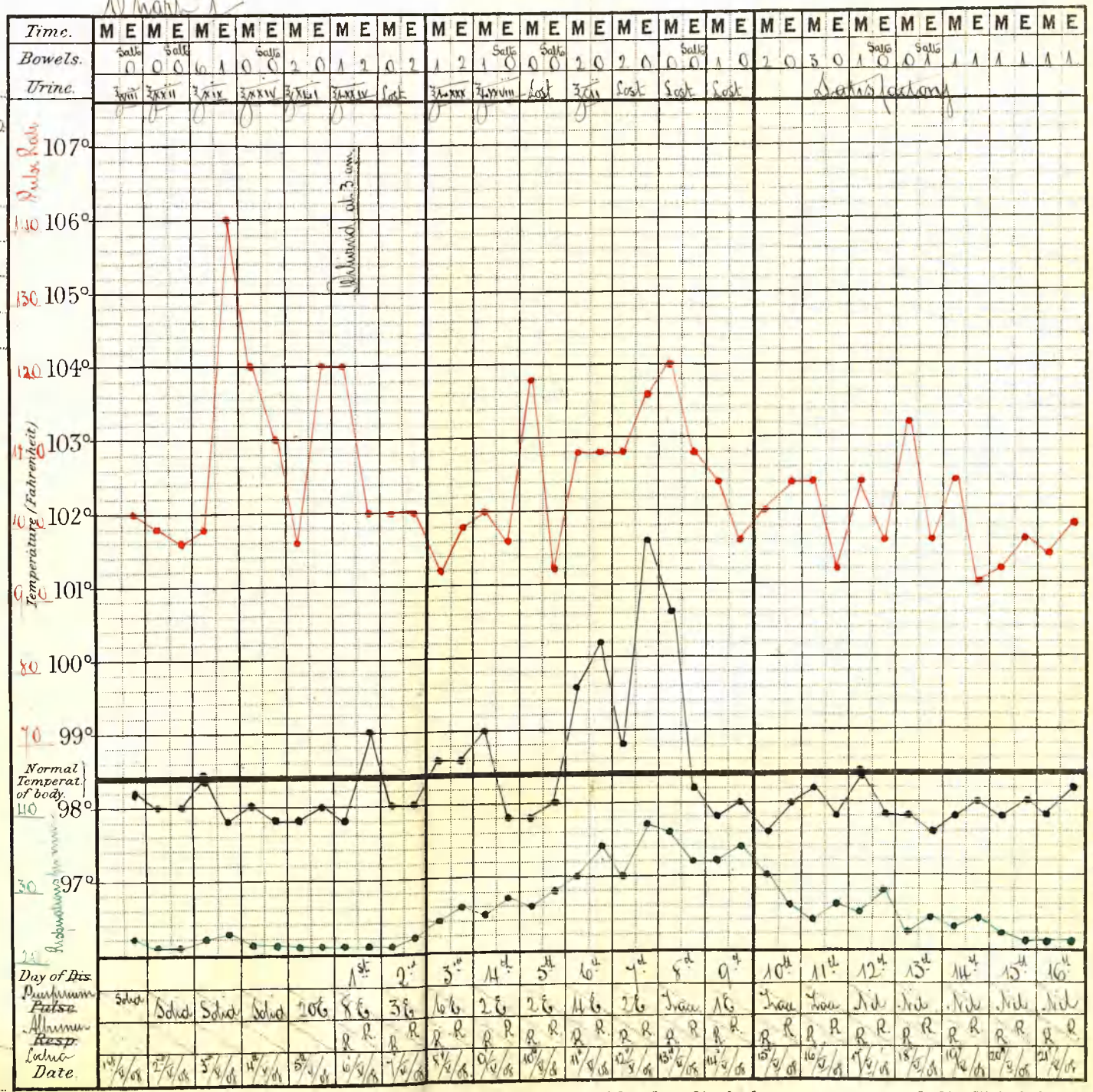
Nurse - Light

Book No XXIV

Date of admission.

1st May 1908

Result Recovery



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Gould's Clinical Chart.

Chart II

DISEASE.

Melancholia

Bronchitis

Notes of Case.

Mrs. Young

23 Years

Night Nurse

Book No. XXIV

1 Para

Date of admission.

1st May 1908

Result Recovery

Time	M	E	M	E	M	E	M	E	M	E	M	E
Bowels.	0	0	0	2	0	0	1	3	1	3	1	2	0
Urine.			Satisfactory									

Pulse Rate

Temperature (Fahrenheit)

Normal Temperat of body.

Day of Dis Puerperium

Pulse. Albumen

Resp. Lactic

Date.

Day of Dis Puerperium	14th	18th	19th	20th	21st	22nd	23rd
Pulse. Albumen	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Resp. Lactic	—	—	—	—	—	—	—
Date.	22/4/08	23/4/08	24/4/08	25/4/08	26/4/08	27/4/08	28/4/08

Commenced Milk

Temperature (Centigrade)

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CASE XXIV MRS CRAIG, aet 23 years, primipara. 27 weeks.

Recovery.

Admitted on 1st May 1908 at 6.25 p.m.

Delivered on 6th May 1908 at 3 a.m.

Presentation vertex.

Child Male dead, 3½ lbs. weight.

HISTORY: There is no history of any disease previous to the pregnancy. From about the twentieth week of pregnancy there had been a slight swelling of the feet and ankles. A neighbour noticed the scantiness of the urine passed, but the patient herself did not think that the quantity was abnormally small. She is in the 27th week of pregnancy. Seven days ago she began to take convulsions, and had had thirty in all. The fits were distributed fairly evenly over those days. Between the attacks she had lain in a semi-comatose condition. Restlessness had been very marked at intervals. She was brought to Hospital from Whiting Bay, Arran, where she had been for a holiday. Morphia is said to have been given for the controlling of the convulsions.

PRESENT CONDITION: Patient is in a semi-comatose condition and appears to be under the influence of morphia. The pupils are 'pin point' and do not react to stimuli. The temperature and respirations are normal. The pulse numbers 100 per minute, is regular and is of poor tension. There is no oedema on any part of the body. The fundus of the uterus reaches to the level of the umbilicus. There is no evidence of foetal life. Per vaginam the os

is closed and the cervix uteri is not wholly taken up.

Urine, per Catheter, 8 ozs; dark amber, acid; Specific Gravity 1032, granular casts; no blood; Albumen solid; Urea, 4 grs. per oz.

Treatment. Magnes. Sulph. 2 ozs. per nasal tube. Saline 2 pints transfused into right median cephalic vein. Milk per tube.

2nd May 1908: The patient is slightly more conscious today. She can be aroused and made to swallow milk and Imperial drink. There have been no convulsions. Pulse very quick at times.

The urine is practically similar to that of yesterday. The quantity for the 24 hours is 22 ozs.

Treatment. Strychnine $\frac{1}{60}$ gr., Digit. $\frac{1}{100}$ gr. Brandy $\frac{1}{2}$ oz. four hourly.

3rd May 1908: She is rather collapsed and unconscious today. The pulse is very feeble, fast, and irregular.

Urine. Quantity per Catheter 19 ozs; Albumen abundant; Urea, 4 grs. per oz.

Treatment. Strychnine $\frac{1}{60}$ grain given hypodermically four hourly. Saline 2 pints transfused under the right breast with beneficial results. Milk per tube.

4th May 1908: The patient is completely unconscious today. At times the pulse is barely perceptible at the wrist. The subconjunctival tissue is very oedematous and the cornea appears as if it were sunk in the surrounding tissue. About 6 p.m., the pulse being imperceptible, Saline 2 pints was again transfused under the left

breast. Strychnine $\frac{1}{60}$ to $\frac{1}{30}$ gr. was given four hourly. Nutrient and stimulating enemata were given frequently.

Urine. Quantity 24 ozs.; milky; Albumen solid; Urea, 6 grs. per oz.

5th May 1908: Improvement has taken place. The patient is semi-conscious and restless. The pulse is still weak, but slower and fairly regular.

Later. Uterine contractions have commenced and the os uteri is dilating.

Urine. Quantity 13 ozs.; Albumen 20 per thousand Esbach.

Treatment. Strychnine discontinued. Nutrient and stimulating enemata.

6th May 1908: Delivery took place at 3 a.m. The child is a dead male child which weighed 3½ lbs. Since delivery the patient has markedly improved. She is less comatose. The subconjunctival oedema has almost disappeared. The swallowing of fluids can now be performed without difficulty. The strength and regularity of the pulse have improved.

Urine. Quantity 74 ozs.; Albumen 8 per thousand Esbach; Urea; no casts or blood.

Treatment. Milk, Pot. Imp.

7th May 1908: Improvement continued. Albumen 3 per thousand Esbach; Urea, 7 grs. per oz.

8th May 1908: There is a slight cough and muco-purulent expectoration present today. Examination of the lungs re-

veals the presence of moist rale at the left base. There is also a quantity of blood in the urine. Albumen 6 per thousand Esbach. Urea. The patient's mental condition improves.

9th May 1908: The cough continues and in addition to the other signs at the left base there is a slight dulness to percussion. Temperature has risen slightly.

Urine. Quantity 80 ozs.; no blood; Albumen 2 per thousand Esbach; Urea, 8 grs. per oz.

10th, 11th May 1908: The general condition of the patient remains much as above. The dulness at the left base of the lung, however, has deepened.

Urine (10th May 1908). Quantity 78 ozs.; Albumen 2 per thousand Esbach; Urea, 8 grs. per oz.

Treatment. Expectorant mixture. Poultice to left side of chest.

12th May 1908: The temperature is raised. The face is flushed and there is a distinct twitching of its left side. The mind is again obfuscated. The lung condition remains much as before.

Urine contains clumps of cocci, pus cells, and granular casts; Albumen 4 per thousand Esbach; Urea.

Treatment. In addition to the above treatment Urotropine 10 grains t.i.d. were given.

13th May 1908: The general condition of the patient has undergone improvement. The temperature has fallen somewhat, and the mental condition is clearer. There only remains a slight and occasional facial twitching of the

left side. The dulness of the left base of the lung still persists, and there is more rale and muco-purulent expectoration.

Urine. Quantity 52 ozs.; bacteria and casts scanty; Albumen 2 per thousand Esbach; Urea, 8 grs. per oz.

Treatment as above.

By the 16th May 1908 the lung and bladder condition had cleared up, and convalescence was established and continued. No Albumen was present in the urine, and the Urea had risen to normal

She was dismissed on 28th May, 1908, well. (To south of England)

C A S E XXV

Chart I

DISEASE.

Eclampsia

Parametritis

Notes of Case.

Mrs.

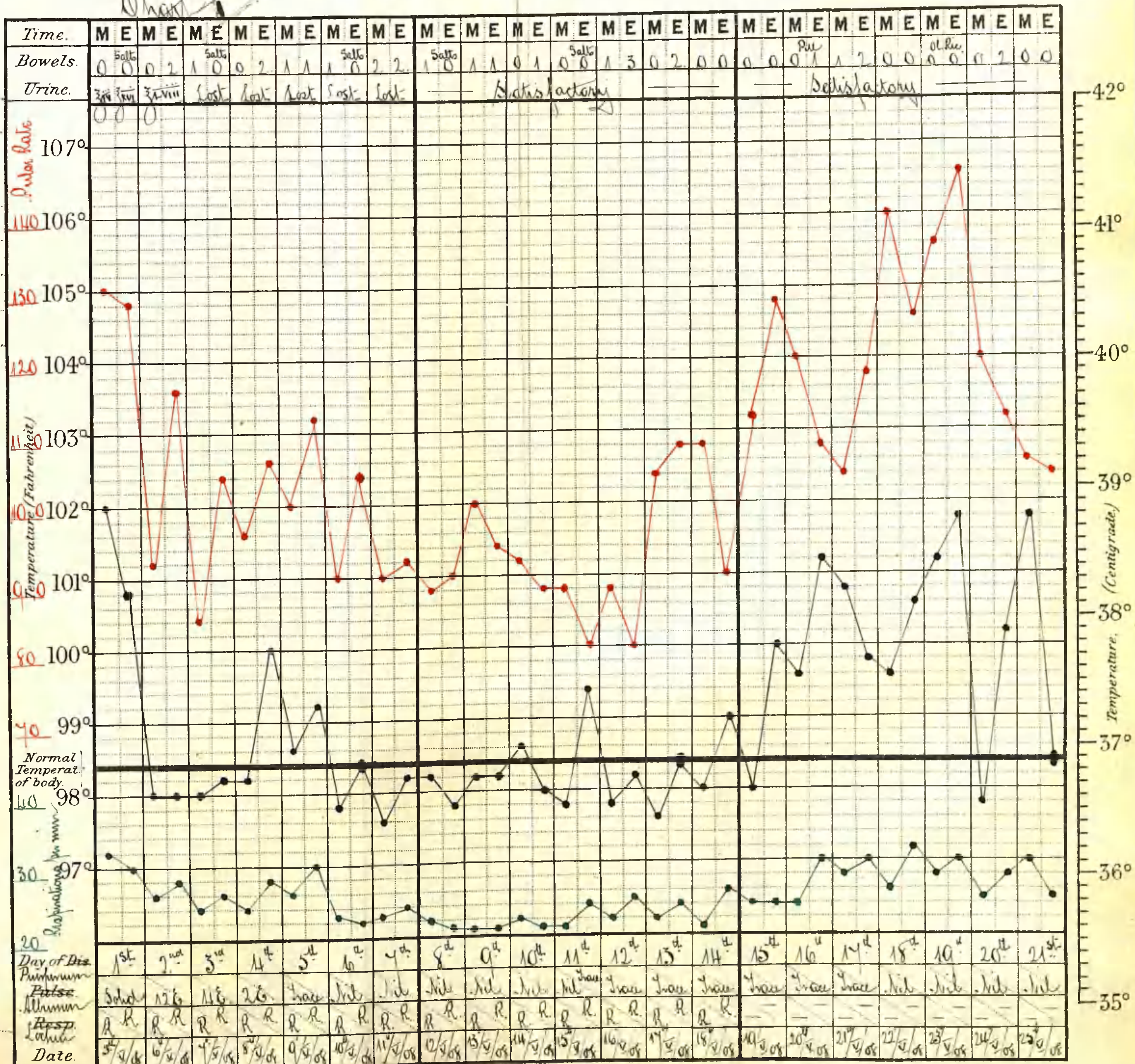
Name Lloyd

Age 25 Years

Diet Milk - Light

Case Book No. XXV

I Para



Date of admission.

4th May 1908

Result Recovery

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Chart 11

DISEASE.

Pneumonia

Parametritis

Notes of Case.

Mrs

Lloyd

25 Years

Milk Light

Book N° XXV

L Para

Time. M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E

Bowels. D 1 1 2 0 1 0 2 1 0 2 1 0 1 0 2 1 0 1 1 0 0 1 1 0 1 0 1 0 1 0 0 1 1 0 2 0

Urine. — Satisfactory — — — Satisfactory — — — Satisfactory — —

Pulse Rate

Temperature (Fahrenheit)

Normal Temperat. of body

Respirations

107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

140 130 120 110 100 90 80 30

Discharged well

42° 41° 40° 39° 38° 37° 36° 35°

Temperature (Centigrade)

Day of Dis

	22nd	23rd	24th	25th	26th	27th	28th	29th	30th	31st	32nd	33rd	34th	35th	36th	37th	38th	39th	40th											
Pulse. Minimum	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil											
Resp. Minimum	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—											
Date.	26/9/08	27/9/08	28/9/08	29/9/08	30/9/08	31/9/08	1/10/08	2/10/08	3/10/08	4/10/08	5/10/08	6/10/08	7/10/08	8/10/08	9/10/08	10/10/08	11/10/08	12/10/08	13/10/08												

Date of admission 24th May 1908.

Result Recovery

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Gould's Clinical Chart.

CASE XXV MRS LLOYD, aet 25 years, primipara. Full time.
Recovery.
Admitted on 4th May 1908 at 7.30 p.m.
Delivered on 4th May 1908 at 9 p.m. under chloroform.
Presentation Breech.
Child Male alive, 7½ lbs. weight.

HISTORY: No history can be obtained about any symptoms which the patient felt before or during the pregnancy. There does not seem to have been any headache, oedema, or epigastric pain. She had had seven convulsions before admission.

PRESENT CONDITION: The patient is quite unconscious. The breathing is stertorous. She is markedly cyanosed and has bitten the tongue and cheek. The pupils are moderately contracted and react sluggishly to stimuli. Palpation of the abdomen shows that the head of the child is at the fundus. The uterus is contracting strongly. On vaginal examination the os was found to be almost fully dilated. When the os had fully dilated she was anaesthetised, the membranes were ruptured, and the child delivered. Auscultation of the lungs shows that there is crepitant rale at the bases behind. Between 10 p.m. and 12 midnight she had eight eclamptic convulsions, each lasting from two to three minutes.

Urine (per Catheter) 4 ozs.; dark; neutral; Specific Gravity 1035; Albumen solid; numerous finely and coarsely granular casts; blood.

Treatment. Magnes. Sulph. 3 ozs. per nasal tube. Bled one pint; transfused two pints Saline solution.

Later, hot wet pack. Chloral Hydrate and Potass. Bromide 20 grs. per rectum twice.

5th May 1908: Between 12 midnight and 8 a.m. she had four more convulsions which were however of less severity than the previous ones. After 8 a.m. the patient remained unconscious for a few hours, then consciousness slowly returned, and with it restlessness set in. The swallowing of fluids can now be accomplished.

Urine. Quantity 16 ozs.; dark; neutral; Albumen solid; casts; blood.

Treatment. To promote urinary secretion two pints Saline solution were again transfused, and hot applications were made over the kidneys. Chloral and Bromide a a 15 grs. once.

6th May 1908: The patient is more conscious today. She has perspired freely and is now resting quietly.

Urine. Quantity 57 ozs. some lost at stool; milky; acid; Specific Gravity 1025; Albumen 12 per thousand Esbach; casts; trace of blood.

7th May 1908: She is now conscious. On opthalmoscopic examination of the eyes, one or two small haemorrhages are present on both retinal discs.

Urine, abundant; Albumen 4 per thousand Esbach; few casts; no blood.

8th May 1908: The patient is in a condition of mild dementia and there are present choreiform movements of the head. These movements are principally in the antero-posterior direction.

Albumen is 2 per thousand Esbach; no casts or blood.

Treatment. Milk diet. Imperial drink. Chloral and Bromide a a 10 grs. four hourly.

9th May 1908: The mental condition has improved somewhat. The involuntary movements have almost gone.

Urine contains only a trace of Albumen.

Treatment. The Chloral and Bromide were stopped.

From 10th May to the 15th May 1908: Improvement continued.

Albumen nil.

15th May 1908: Dementia and involuntary movements ceased entirely some days ago. Temperature raised slightly this evening.

16th May 1908: The patient began to complain of pain in the right iliac region. Tenderness and resistance could be made out in the region of the vermiform appendix. No dulness could be discovered in this area. Per vaginam nothing abnormal could be detected.

The lochia had practically ceased, and it had been normal throughout.

Treatment. Hot applications. Vaginal douches.

17th May 1908: The condition of the patient is much the same today. There is practically no change in the physical signs.

18th May 1908: A small area of dulness could be detected over the appendix region today, otherwise the patient's condition is unchanged.

Albumen, trace.

19th May 1908: The area of abdominal dulness has increased and is now about the size of an orange. The temperature is considerably raised.

Albumen, trace.

Examination of the retinae shows that the above mentioned haemorrhages have almost disappeared.

20th May 1908: The area of pain and dulness rapidly spread, and extended from the iliac crest to about one and a quarter inches below the area of liver dulness. Per vaginam there was a sense of fulness in the right fornix and the uterus was pushed slightly towards the left.

Albumen, trace.

21st - 23rd May 1908: The above symptoms persisted, and in addition the fulness of the right fornix became a definite swelling, although at no time did it give the least indication of containing pus.

Albumen, trace.

24th May 1908: The temperature fell suddenly today, and the pain was much less. The physical signs are much the same.

Albumen gone.

31st May 1908: The physical signs had gradually subsided and beyond a slight degree of thickening to the right of the cervix, nothing abnormal can be detected.

Convalescence now continued, and the patient was dismissed well, on 13th June 1908.

C A S E XXVI

DISEASE.

Edwardsia

Notes of Case.

No.

Surname

22 Years

Male - Irish

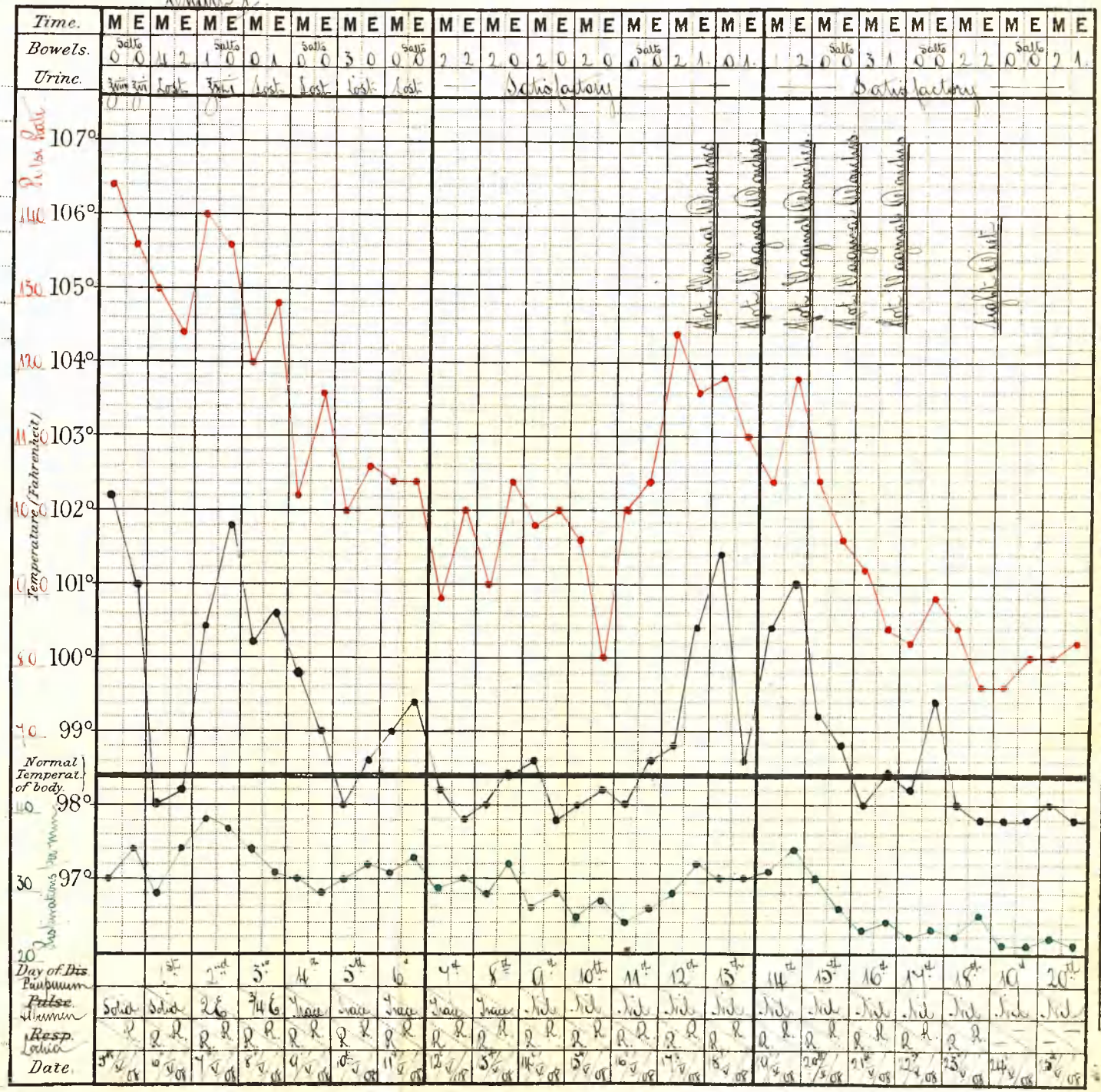
Book No. XXVI

Para

Date of admission.

5th May 1908

Result Recovered



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DISEASE.

Adamsson

Notes of Case.

Mrs. Turnbull

22 Years.

Milk Light.

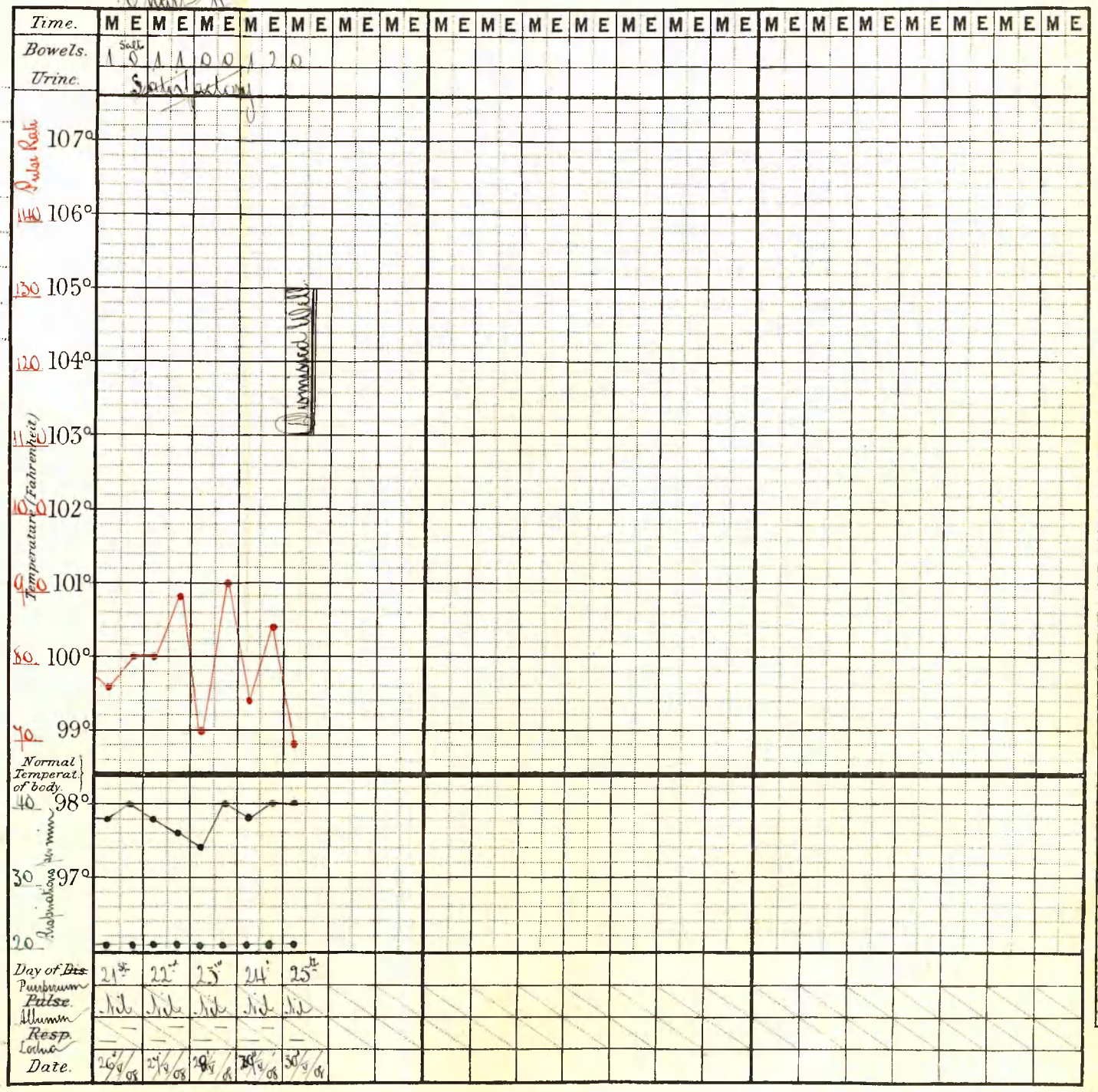
Chart N° XXVI

Para

Date of admission.

2 May 1908

Date of Recovery



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CASE XXVI MRS TURNBULL, aet 22 years, primipara. 27 weeks.
Recovery.
Admitted on 5th May 1908 at 8 a.m.
Delivered on 5th May 1908 at 9 p.m.
Child Female dead, 3½ lbs. weight.

HISTORY: As far as can be discovered, the course of pregnancy had been normal up to the morning of admission, when severe frontal headache was complained of. No oedema or other symptom beyond the one mentioned above had been felt. In the few hours before admission she had had six fits.

PRESENT CONDITION: The patient is unconscious, and reflexes gone. She took several convulsions when being prepared for the Labour Ward. The tongue is severely lacerated. Temperature is 102.2° F. Pulse is 144 per minute, regular and of fairly high tension. Respirations number 30 per minute.

On abdominal palpation the fundus uteri reaches to the umbilicus. It is contracting very slightly. No signs of foetal life can be found. Per vaginam the cervix uteri is found to be still elongated, and the os admits one finger. The patient had ten convulsions before transfusion one hour after admission. and seven after it. These seizures were typically eclamptic in character, and lasted on an average two minutes each. The total number occurring before delivery, which took place naturally at 9 p.m., was twenty-three. After delivery she returned to a semi-conscious condition, and was extremely restless.

Urine. 8 ozs. per Catheter; dark; slightly acid; Specific Gravity 1030; Albumen solid; granular casts; no blood.

Treatment. One hour after admission she was bled 20 ozs. and transfused 2 pints Saline. Magnes. Sulph. 3 ozs. per nasal tube. Chloral Hydrate and Potass. Bromide a a 30 grains were given per rectum, two hours after admission. Milk and Imperial drink were given as soon as the patient could be got to swallow.

6th May 1908: There have been no further convulsions. The breathing is short and distressed. There is also a slight irritative cough. No sputum has been expelled. Dulness over the pulmonary area is absent. On auscultation moist crepitant rale can be heard over the lungs, especially at the bases behind.

Urine. The exact quantity was lost owing to the large number of the stools, but it is estimated to be scanty; milky; acid; Specific Gravity 1032; Albumen abundant; granular casts; no blood.

Treatment. Linseed jacket poultice over lung area, afterwards gauze jacket. Stimulants; brandy half an ounce four hourly, and stimulating cough mixture four hourly. The position of the patient in bed was changed from time to time.

7th May 1908: The patient is fairly conscious. There is herpes on the nose and the upper lip. She has an irritating cough and a viscid sputum. Temperature and pulse are raised today. There is pain felt below the right scapula and on percussion commencing dulness is elicited

in this region. In addition to the widespread rale there is a slight tubularity of breath sounds at the right base.

Urine 41 ozs.; clear amber; Specific Gravity 1030; Albumen 2 per thousand Esbach; no casts; no blood.

Treatment. In addition to the treatment of yesterday Strychnine $\frac{1}{60}$ gr. hypodermically was given three times daily.

8th May 1908: The patient's condition is practically unchanged. The temperature has fallen slightly.

The urine contains Albumen to the extent of $\frac{3}{4}$ per thousand Esbach.

10th May 1908: The condition in the right lung shows signs of resolving.

Albumen, trace.

12th May 1908: The base of the right lung has cleared considerably. The dulness and tubularity have gone, and only a moist rale persists.

Albumen, trace.

14th May 1908: The lungs have practically cleared.

Albumen, nil.

16th May 1908: The patient complained of pain in the left ovarian region. Tenderness is present both on palpating abdominally and on examination per vaginam.

Urine. Albumen, nil.

Treatment. Hot poultices were applied to the abdomen. Hot vaginal douches were given three times daily. Morphia $\frac{1}{4}$ grain hypodermically at night.

17th May 1908: The pain and tenderness are still severe. No dulness or localised swelling can be detected.

18th May 1908: The physical condition of the patient remains as on the 17th May.

20th May 1908: The pain has considerably abated. The temperature and pulse have come down.

22nd May 1908: The abdominal pain has practically subsided.

The patient rapidly recovered strength, and was dismissed, on 30th May 1908, well.

C A S E XXVII

CASE XXVII MRS WYLLIE, aet 36 years, II para. 36 weeks.

Death.

Admitted on 5th May 1908 at 10.15 a.m.

Delivered on 5th May 1908 at 5.30 p.m. Chloroform and Forceps.

Presentation vertex. Left occipito anterior position.

Child Female dead.

Died 6th May 1908 at 4.20 p.m.

HISTORY: The patient's first pregnancy and confinement were, as far as can be ascertained, normal. From the fifth month of this pregnancy there had been swelling of the feet and ankles, slight at first, and increasing as the pregnancy advanced. From the 6th month she had had intermittent headache. There had been no oedema of the face. Early on the morning of admission she had severe epigastric pain. Shortly afterwards she began to take convulsions, and had fifteen before admission. A Doctor was called who transfused her one and a half pints of Saline solution.

PRESENT CONDITION: The patient is quite unconscious. The pupils are moderately contracted and do not respond to stimuli. She is slightly cyanosed. The size of the uterus is equal to that of a thirty-sixth week pregnancy. Faint uterine contractions can be felt. No foetal heart's sounds and movements can be heard or felt. Per vaginam the cervix is not quite fully taken up, and the os admits the tip of the finger. Temperature is 101.8° F. Pulse is 120 per minute, regular and of high ten-

sion. Respirations number 32 per minute.

Urine. 4 ozs. per catheter; dark muddy; Albumen; blood and tube casts (finely and coarsely granular)

She had one convulsion shortly after admission, and after this seizure had passed over, the left median basilic vein was opened and one pint blood withdrawn. Afterwards two pints Saline solution was transfused. Per nasal tube Magnes. Sulph. two ounces was given. During the day the uterine contractions became stronger. At 4 p.m. there were other nine seizures, and as the pulse was becoming very weak and irregular, the os, which had dilated to the size of a four shilling bit was fully dilated manually under chloroform. The membranes were then ruptured, forceps applied, and the child delivered. After three of the above mentioned fits Chloral Hydrate and Potassium Bromide a a 25 grs. were given per rectum. The patient was also enveloped in a hot wet pack for 20 minutes. She was taken out of this as the pulse became more rapid. The perspiration was profuse. As the fits became more frequent, the Chloral and Bromide were repeated. After the effects of the chloroform had passed off she took another convulsion, and she was again transfused two pints of Saline solution under the right breast. She had three further convulsive seizures. After these last she seemed very exhausted, and the pulse was scarcely perceptible. Strychnine $\frac{1}{30}$ gr., Digitalin $\frac{1}{100}$ gr. were given four hourly, also Brandy was given per rectum.

She had in all thirty convulsions.

6th May 1908: The patient is quite unconscious. The heart's action is weak and irregular. It numbers between 140 and 160 per minute. The temperature and respirations are normal. The amount of urine for the twenty-four hours obtained per Catheter is 13 ozs. The Albumen is solid on boiling. Tube casts and blood are also present. Urea 3 grains per ounce. Stimulants were given freely.

The patient collapsed, and died at 4.20 p.m.

C A S E XXVIII

CASE XXVIII MRS WILSON, aet 30 years, primipara. Full time. Death.

Admitted on 14th May 1908 at 6.15 p.m.

Undelivered.

Died, 14th May 1908 at 7.30 p.m.

HISTORY: During the last three weeks the patient is said to have had oedema of the face, feet, and ankles. At times she had complained of "sore head". At 8 a.m. on the morning of the day of admission she complained of severe epigastric pain. This was accompanied by a "dull feeling" in the head, and a "mist" before the eyes. These symptoms persisted, and became of greater intensity until 3 p.m. when she took a convulsion. Two further seizures followed, and about 5.30 p.m. she took a fourth, which lasted more or less until admission.

PRESENT CONDITION: The patient is completely unconscious, and deeply cyanosed. The pupils are widely dilated, and the reflexes are gone. The tongue is bitten, and there is a quantity of blood-stained frothy mucus about the mouth. The temperature is sub-normal. Respirations number 25 per minute, and are almost stertorous in character. The pulse, when it can be felt, numbers over 160 beats per minute. It is very weak and irregular. There is a persistent twitching of all the muscles of the body, as if the clonic action of the spasm still persists. although of much less violence. There is no sign of foetal life, nor is there any evidence of the onset of labour.

Urine. Six ounces were drawn off per catheter;

bloody; neutral; Specific Gravity 1040; Albumen solid; tube casts (blood and granular) abundant; blood abundant; Urea 4 grains per ounce.

Treatment. The patient was kept warm on a bed heated by hot water. Brandy was given twice per rectum. Saline, two pints, was transfused into the median basilic vein.

She never rallied, and died undelivered at 7.30 p.m., one and a quarter hours after admission.

CASE XXIX

Chart 1

DISEASE.

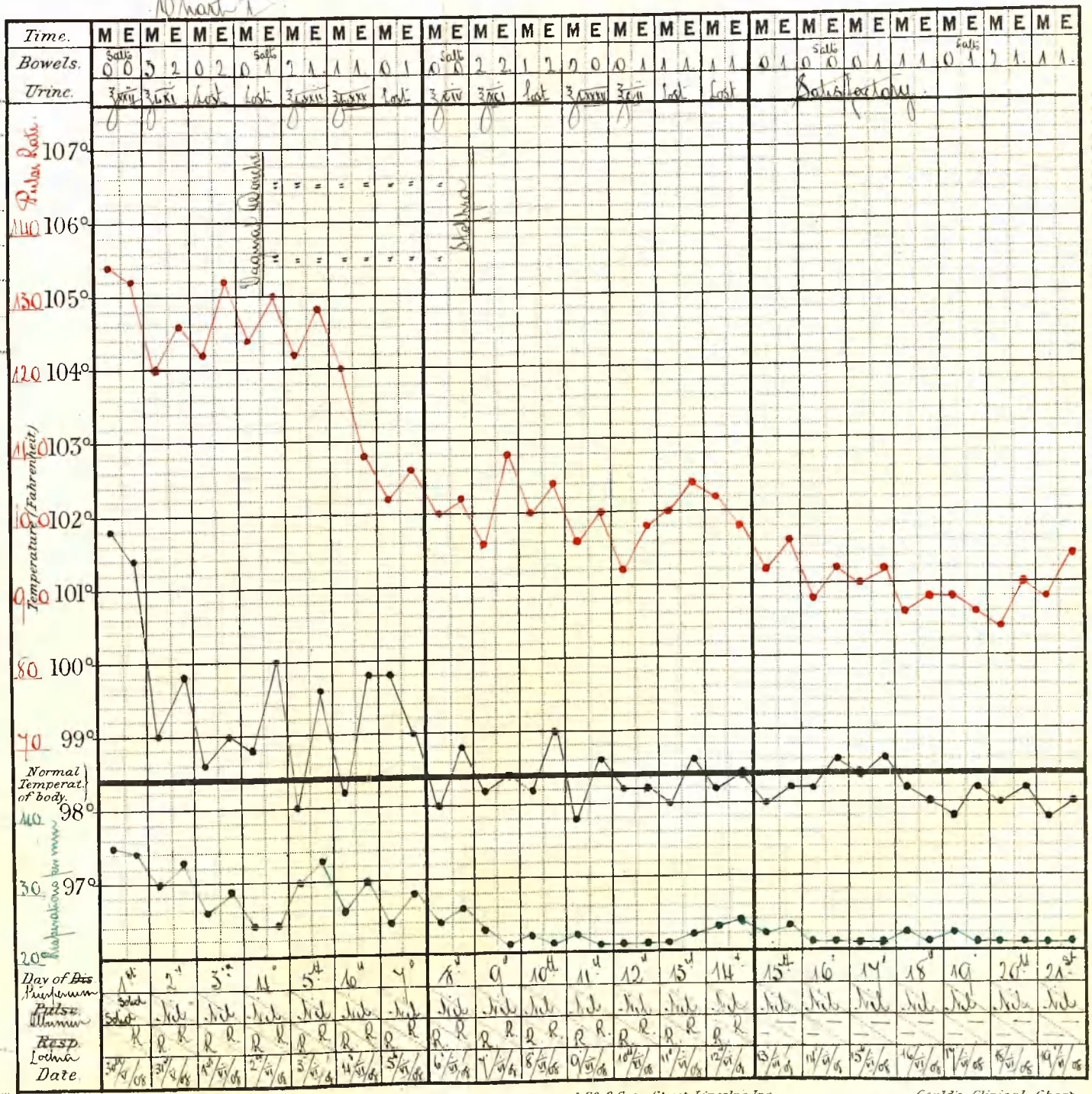
Pneumonia

Notes of Case.

Mrs.
[illegible] years.
22 years.
Milk - Light
Book N° XXIX

1 Para

Date of admission.
30th May 1908.
Result Recovery



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Gould's Clinical Chart.

Chart II

DISEASE.

Eclampsia

Notes of Case.

[illegible]

[illegible]

22 [illegible]

Milk + Light

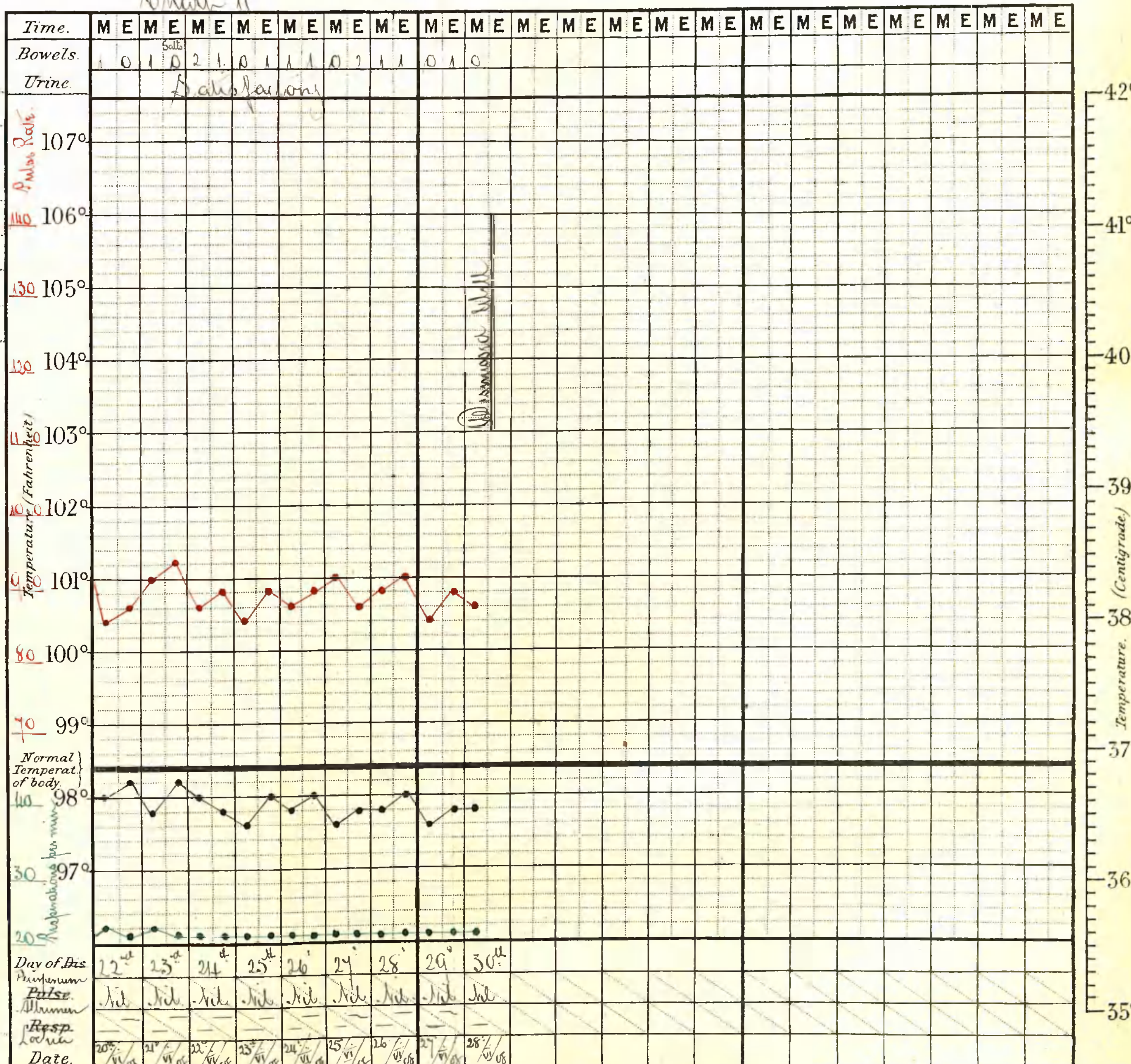
Book No. XXIX

I Para

Date of admission.

30th May 1908

Result Recovery



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CASE XXIX MRS JARVIE. aet 22 years, primipara. Full time.
Recovery.
Admitted on the 30th May 1908 at 8 a.m.
Delivered on the 30th May 1908 at 12 noon.
Presentation vertex.
Child Male dead, 7 lbs. weight.

HISTORY: Previous to her pregnancy there is no history of any previous disease. During the last two months of pregnancy she had suffered from an increasing swelling of the lower extremities. For the last five weeks she had noticed that there had been swelling of the lower eyelids, which had been worse in the mornings. Headache of an ever increasing, and intermittent variety had been felt for some weeks previous to onset of the convulsions. There is no history of epigastric pain. On the evening before admission the patient had felt "dazed", and had "difficulty in seeing". Convulsions came on about midnight, and she had twenty before admission at 8 a.m.

PRESENT CONDITION: The patient is semi-conscious, and very restless. The pupils are normal, and react to stimuli. Temperature is 101.8^{0} F. Pulse is 135 per minute, and is of high tension. Respirations number 35 per minute. The uterus is contracting strongly. No signs of foetal life can be discovered. The os uteri is nearly fully dilated, and the membranes are ruptured. The child's head presents, and is well down into the cavity of the pelvis. While being prepared for admission to the Labour Ward she took two convulsions, the second was very severe, lasting fully five minutes. After transfusion

she had a third and last, which was very slight. Later, she was placed under chloroform, and the child delivered by forceps. The vagina was much bruised in delivery. Opthalmoscopic examination of the retinal discs shows the presence of haemorrhages, and there is also blurring of the discs.

Urine, 22 ozs. per Catheter; dark amber; acid; Specific Gravity 1025; Albumen solid; tube casts; no blood.

Treatment. After the two convulsions on admission she was bled one pint, and transfused Saline solution two pints into the median cephalic vein. Magnes. Sulph. 3 ozs. was given per nasal tube. Milk, Potus. Imperialis

31st May 1908: The patient slept well during the night, and today is almost quite conscious. She states that she only feels "dazed", and expresses surprise at being in Hospital, as she has not any recollection of being brought in. There have been no return of the convulsions.

Urine abundant; milky; Specific Gravity 1018; acid; no Albumen, blood or tube casts.

Treatment. Milk diet. Potus. Imperialis.

1st June 1908: On account of the bruised condition of the cervix and vagina she was douched vaginally twice daily. The top of the bed was also raised, to allow of free drainage.

No Albumen was present in urine.

Treatment. As pulse continued somewhat rapid, Strychnine $\frac{1}{60}$ grain given four hourly.

2nd June 1908: As above.

5th June 1908: Pulse regaining normal state. Stimulant stopped.

6th June 1908: Vagina healed. Vaginal douching stopped. Convalescence established.

25th June 1908: The retinae are more distinct, and the haemorrhages have quite disappeared.

27th June 1908: Dismissed, well.

C A S E XXX

DISEASE.

Eclampsia

Notes of Case.

Name Mrs Mac Murrough

Age 21 Years

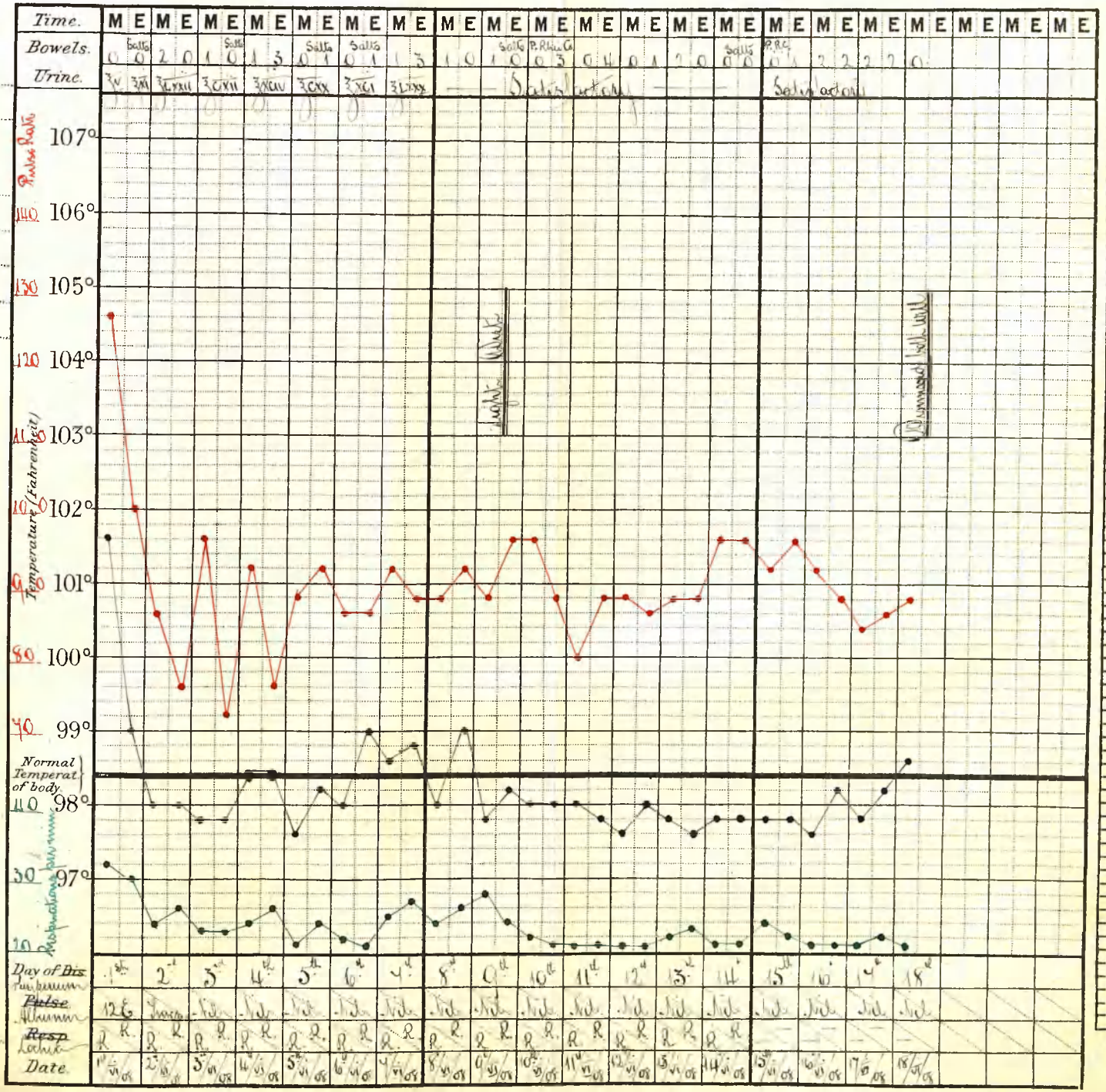
Ward Kirk Ward

Case Book No. XXX

J. Dunn

Date of admission. 1st June 1908

Result Recovery



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CASE XXX MRS MacILRAITH, aet 21 years, primipara. 36 weeks pregnant. Recovery.

Admitted on 1st June 1908 at 3.10 a.m.

Delivered on 1st June 1908 at 7 a.m.

Presentation vertex.

Child Female alive, 5 lbs. weight.

HISTORY: There is no history of any illness previous to pregnancy. For the last month patient had noticed that her feet and ankles had been swollen, and also that during the last two weeks her face had been puffy in the mornings. There had been no headache, epigastric pain, or eye symptoms. Yesterday afternoon, however, the patient had been seized with a severe headache, and about midnight had taken a convulsion. This fit had been followed by three others.

PRESENT CONDITION: The patient is semi-conscious, and slightly cyanosed. The tongue is lacerated. Temperature is 101.6° F. Pulse numbers 120 per minute, is regular and of moderate tension. Respirations number 32 per minute. There is slight oedema of the lower extremities. The pupils are equal and moderately contracted; they react to light. The uterus reaches almost to the ensiform cartilage, and is contracting strongly. Foetal heart's sounds and foetal movements can be heard and felt. Per vaginam the os is equal in size to a five shilling piece and is dilatable. The cervix is fully taken up. The head is found engaging in the first obstetrical position. The membranes are unruptured. Immediately after admission

she had one typical eclamptic convulsion. She was anaesthetised with chloroform, the os dilated manually, and the membranes ruptured. The child was then delivered with forceps.

Urine. 5 ozs. per Catheter; dark, muddy; faintly acid; Specific Gravity 1040; tube casts (granular); blood; Urea 5 grains per ounce; Albumen 12 per thousand Esbach.

Treatment. After delivery she was transfused Saline solution two pints. (Patient was not bled, as a fair amount of blood was lost post-partum.) Purge. Milk diet. Imperial drink.

2nd June 1908: The patient is quite conscious today. No further convulsions have occurred. The oedema has gone.

Urine. 72 ozs; milky; acid; Specific Gravity 1025; Albumen, trace; Urea 8 grains per ounce.

The patient made an uninterrupted recovery, and was dismissed well, on June 18th 1908. The child was also well.

C A S E XXXI

DISEASE.

Glamorgan

Pneumonia

Notes of Case.

Inger Hunter

23 Years

N.W. – Light

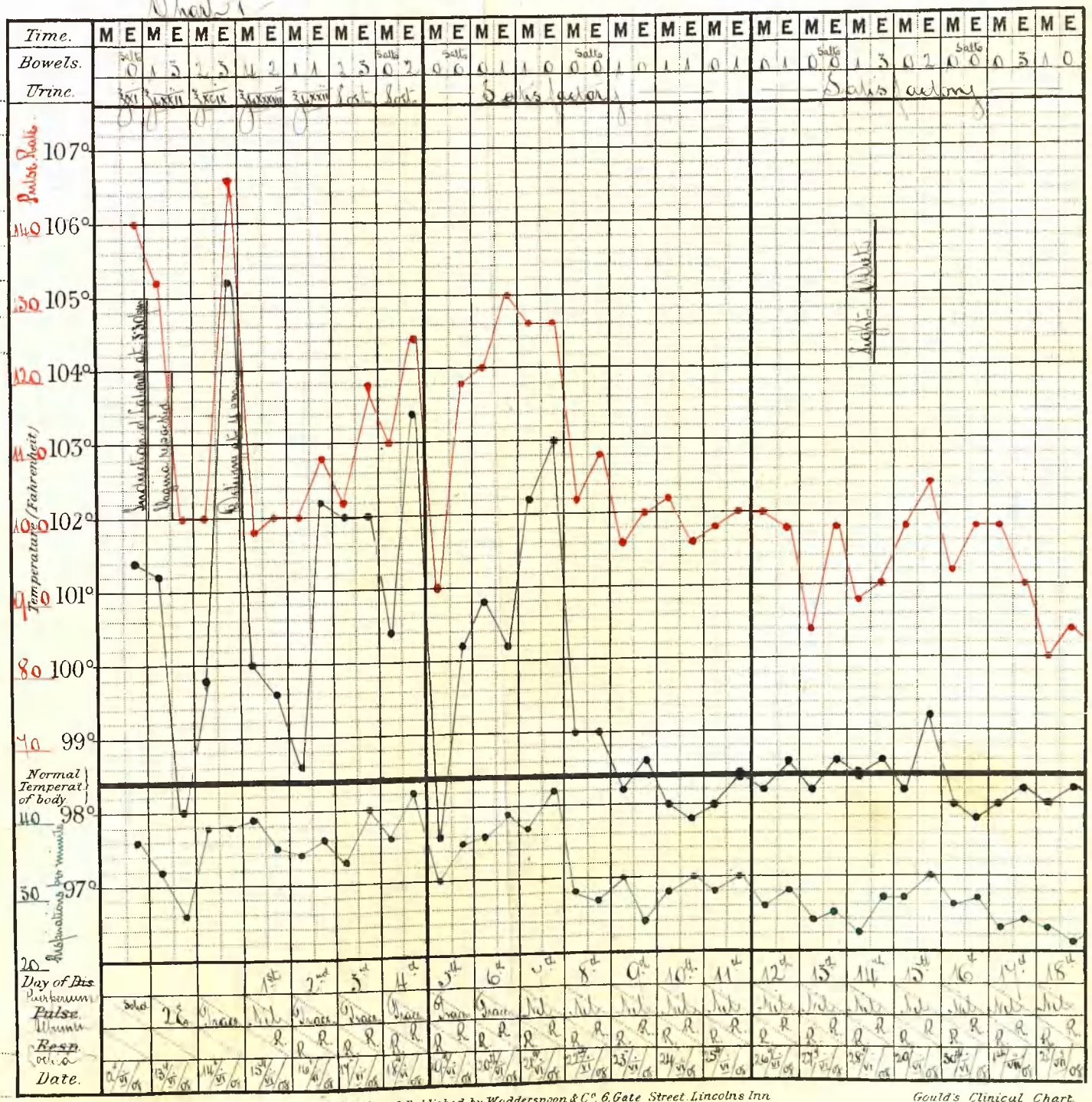
Book No. XXXI

Para

Date of admission.

12th June 1908

Result Recovery



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Gould's Clinical Chart

Chart II.

DISEASE.

Diabetic Pneumonia

Notes of Case.

Age 25 Years

Light

Book No XXXI

1 Para

Date of admission. 12th June 1908

Result Recovery.

Time.	M	E	M	E	M	E	M	E	M	E	M	E	M	E	M	E	M	E	M	E	M	E	M	E	M	E	M	E	M	E
Bowels.	Sallo						Sallo																							
	0	0	1	2	1	0	0	0	2	2	0	2	0	2	0	1	1													
Urine.					Satis factory																									
Pulse Rate																														
140																														
130																														
120																														
110																														
100																														
90																														
80																														
70																														
Normal Temperat. of body.																														
40																														
30																														
20																														
Day of Dis	10th		20th		21st		22nd		23rd		24th		25th		26th		27th													
Puerperium																														
Pulse	Nil		Nil		Nil		Nil		Nil		Nil		Nil		Nil		Nil													
Albumin																														
Resp.																														
Lochia																														
Date	5/VII/08		6/VII/08		7/VII/08		8/VII/08		9/VII/08		10/VII/08		11/VII/08		12/VII/08		13/VII/08													

Pneumonia well

Temperature (Fahrenheit)

Temperature. (Centigrade.)

42° 41° 40° 39° 38° 37° 36° 35°

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CASE XXXI **LIZZIE HUNTER**, aet 23 years, primipara. Full time.
Recovery.
Admitted on 12th June 1908 at 12 noon.
Delivered on 14th June 1908 at 4 p.m.
Child Female dead, 7½ lbs. weight.

HISTORY: The patient had always been healthy until about four weeks ago, when she had to discontinue her work (mill-worker) on account of "not feeling well". About one week later she had noticed that her feet and ankles were swollen. Swelling of the eyelids, epigastric pain, disturbances of vision, or alteration in the quantity of urine passed were not observed. Headache had been present from time to time, but had never been severe. At 5.30 a.m. on the day of admission, and without any warning, the patient began to take convulsions, and had twenty before admission. Her mother stated that her daughter had not "felt life" for two weeks before admission.

PRESENT CONDITION: The patient is unconscious, and looks very ill. The pupils are contracted, and do not react to stimuli. The face is puffy and cyanosed. She has bitten the tongue severely, the laceration on it extending from the tip three quarters of an inch down the centre. Abdominal palpation shows that the uterus is that of a full time pregnancy, and is not contracting. The head presents, and is above the pelvic brim. No sign of foetal life can be discovered. Per vaginam the os uteri admits the tip of a finger, and the cervix is fully taken up. There is a flattening of the pelvis. At the brim the true conjugate being three and three quarter inches, and the

obliques four and a half inches. Intercristal measurement is ten and three quarter inches, and Interspinous is ten and a quarter inches. Temperature 101.4° F. Pulse 140, irregular. and of poor tension. Respirations number 36 per minute. In the lungs, especially at the bases behind, numerous moist rales can be heard. She had severe eclamptic seizures at 1.30 p.m., 2.15 p.m., 3.30 p.m., 3.40 p.m., 5 p.m., and 5.15 p.m., and 5.35 p.m., being seven in all. Their duration averaged from two and a half to four minutes.

Urine. 11 ozs. per Catheter; very dark; faintly acid; Specific Gravity 1035; Albumen solid; Urea 6 grains to the ounce; quantities of finely and coarsely granular tube casts, and blood.

Treatment. Magnes. Sulph. 4 ozs. per nasal tube. Bled 15 ozs. Transfused 2 pints Saline solution. (The seven convulsions occurred after transfusion.) Chloral Hydrate 10 grains and Bromide 15 grains at 2.5 p.m. and 5 p.m.. A hot wet pack was given at 2.30 p.m., and sinapisms applied over the Kidney region. At 3.30 p.m. induction of labour was decided on, and this was performed by the slow (Krauses) method. The vagina was thoroughly cleansed and douched. Three No. 10 sterile gum elastic bougies were introduced into the uterus, and the vagina packed. At this time one and a half pints Saline was transfused under the right mamma. (Three convulsions occurred before this Saline, and four after it.)

13th June 1908: There have been no further fits. The patient, however, is still in a grave condition. Tempera-

ture is still raised, and the pulse rapid and irregular. The bowels have moved four times. She has vomited several times. The lung condition is much the same as it was yesterday.

Later, consciousness seems to be returning, and between short periods of sleep the patient is very restless. The packing in the vagina was renewed. There are no symptoms of commencing labour. She can swallow fluids when roused.

Urine. 72 ozs; milky; acid; Specific Gravity 1028; Albumen 2 per thousand Esbach; Urea 8 grains per ounce; tube casts scanty; blood, faint trace.

Treatment. Milk; Brandy four hourly; Mouth swabbed out with Hydrogen peroxide.

14th June 1908: Today the patient is semi-conscious, and very restless. She has taken fluid nourishment in fair quantity, and without much difficulty. Both lungs are oedematous, and there is a commencing consolidation of the bases behind. There have been uterine contractions going on during the morning. The membranes ruptured at 12 noon, and at 4 p.m. a macerated female child was delivered. The child's head was slightly hydrocephalic and very soft. During the delivery the passage of the head was aided manually. An intrauterine douche was given after the delivery. At 5.30 p.m. the temperature rose considerably, and she complains of pain in the left side of the chest. There is dulness to percussion, and friction on auscultation, over the lower lateral part of the lung area.

Urine, 99 ozs.; clear amber; acid; Specific Gravity 1020; Albumen, trace; no blood; no tube casts; Urea 9 grains per ounce.

Treatment. Milk diet. Poultice to affected side. Strychnine $\frac{1}{60}$ gr. four hourly. Brandy half ounce four hourly.

15th June 1908: The patient is very weak, but fairly conscious. The pain and friction have gone from the left chest. The dulness persists, and on auscultation the respiratory murmur and vocal fremitus are diminished. Crepitant rales are still to be heard over the entire pulmonary area, although the base of the right lung seems to be mostly involved.

Urine, 88 ozs.; clear amber; acid; Specific Gravity 1022; no Albumen; no tube casts; no blood.

Treatment. As above.

16th June 1908: There is dulness over the lower lobe of the right lung. The vocal fremitus is increased. The breathing is becoming tubular. The vocal resonance is increased. The left chest condition remains much as before. There does not seem to be any increase in the amount of fluid present. The patient is cyanosed, and semi-conscious. There is a slight irritating cough, but little expectoration.

Urine, 72 ozs.; dark amber; acid; Specific Gravity 1025; Albumen, trace; few casts; no blood; Chlorides diminished.

Treatment. As before.

17th June 1908: The patient still remains cyanosed and weak. A large slough is now present on the front portion of the tongue. She has a rusty viscid sputum and a troublesome cough. The lower and middle lobes of the right lung now show distinct signs of consolidation. The fluid in the left pleural sac is now diminishing.

Urine. Albumen, trace; Urea 8 grs. per oz; Chlorides diminished.

Treatment, As before.

From 18th June 1908 to 20th June 1908: The patient remained in much the same critical condition; the middle and lower lobes of the right lung remaining solid. The left chest cleared gradually, and remained normal but for the presence of rale. The patient was kept well stimulated.

21st June 1908: The slough on the tongue separated and left the tongue in a curious bifid condition. The temperature has come down, but the right lung still shows signs of consolidation. No Albumen in urine.

23rd June 1908: The right lung condition is now resolving. The patient, although extremely weak, is improving. No Albumen.

By the end of June the lungs were clear, and the patient was dismissed on 11th July 1908, well.

C A S E XXXII

DISEASE.

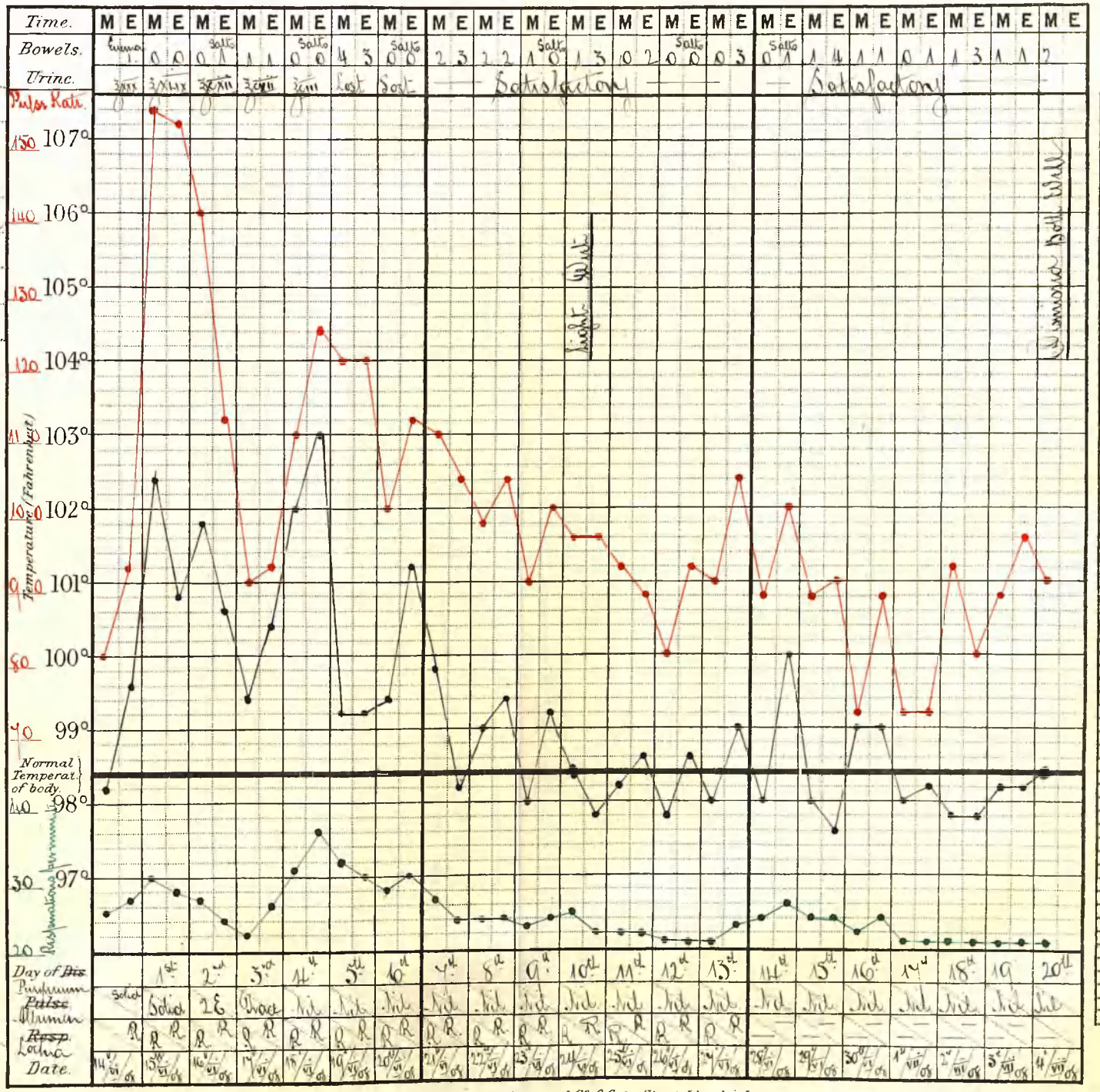
Adamassa

Notes of Case.

Sarah
Mrs. Dermot
32 Years
Milk - Light
Ward No. XXXII

II Para

Date of admission.
11th June 1908
Result Recovery



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Gould's Clinical Chart.

CASE XXXII SARAH MacDERMOT, aet 32, II para. Full time.
Recovery.
Admitted on 14th June 1908 at 11.15 a.m.
Delivered on 14th June 1908 at 5.50 p.m.
Presentation vertex. Left occipito-anterior position.
Child Male alive, 6¼ lbs.

HISTORY: The patient can give no history as to her previous health. Eight years ago she had been delivered normally in the Glasgow Maternity Hospital.

PRESENT CONDITION: The patient is grey-haired, and looks much older than her stated age. There is a well marked Arcus Senilis. The arteries seem degenerate. There is no swelling visible on any part of the body. She is at full time. The cervix is taken up, the os admits two fingers, and the membranes are unruptured. The child is alive. There are slight uterine contractions in progress. The patient was very noisy and restless in the first and second stages of labour. At 3.30 p.m. the membranes ruptured, and the os at this time was almost fully dilated. At 5.30 p.m., when the foetal head was on the perineum, she took a typical eclamptic seizure. The patient was anaesthetised, and the child delivered with forceps. Between 5.50 p.m. and 11.50 p.m. she had nine convulsions. These convulsions were of about two to three minutes duration, and the subsequent coma was deep and lasting. In this case also the spasmodic actions were more violent on the left side than on the right.

Urine, (6 o'clock specimen) quantity 30 ozs.; dark

amber; Specific Gravity 1035; Albumen solid; tube casts; no blood.

Treatment. Purge. After delivery, Chloral and Bromide a a 25 grs. per rectum. Bled one pint. Transfused Saline solution two pints (after nine fits had occurred). 11.30 p.m.; Chloral and Bromide a a 20 grains were again given. Shortly before midnight a hot wet pack was given.

15th June 1908: Between midnight and 3 a.m. she had three convulsions. Chloral and Bromide 20 grains again given. At 6 a.m. the pulse began to flag, and stimulants, Brandy 2 ozs., were given per rectum. At 7 a.m., as her condition did not improve, 2 pints Saline solution was transfused intravenously. At this time the patient was quite unconscious, and there was a generalised oedema over the entire body surface. The sclerotics showed signs of oedema. There were abundant moist rale in the lungs. At 7.45 a.m. Strychnine $\frac{1}{60}$ gr. was given hypodermically. At 8.45 a.m. she had a slight fit, which was the last, the total number being 14. At 9 a.m. the pulse was exceedingly rapid and irregular, and Strychnine $\frac{1}{60}$ gr. and Digitalin $\frac{1}{100}$ gr. were given. The lips were also moistened frequently with Brandy and water. In the evening, breathing was difficult, and the lungs contained numerous moist rales. Feeding has been carried out by tube. The temperature and pulse, however, are down slightly.

Urine, 49 ozs.; milky; acid; Albumen solid; tube casts; no blood.

16th June 1908: The patient is slightly more conscious today. Swallowing small quantities of fluid can be carried out without much difficulty. There is no dulness in the chest, but abundant rale is to be heard over the whole pulmonary area. The eyes were examined with the opthalmoscope today, and recent haemorrhages are to be seen in addition to slight retinitis. Strychnine and Digitalin have been given four hourly by hypodermic.

Urine, 96 ozs.; clear amber; acid; Specific Gravity 1020; Albumen 2 per thousand Esbach; no tube casts; no blood.

17th June 1908: The patient is still semi-conscious. The lung condition remains much the same as yesterday. The pulse is stronger. The cough has become troublesome.

Urine 112 ozs.; amber; acid; Specific Gravity 1020; Albumen, trace; no tube casts; no blood.

Treatment as before.

18th June 1908: She is more conscious, and complains of severe headache; otherwise her condition is the same as yesterday.

Urine, 103 ozs.; straw; acid; Specific Gravity 1018; Albumen, nil; tube casts and blood, nil.

19th June 1908: Consciousness returned today, and the patient seems much improved. Cough is less. The rales in the lung are clearing up. The hypodermic injections of Strychnine and Digitalin were stopped today. No Albumen is present in the urine.

Convalescence now was uninterrupted, and the patient and the child were dismissed well, on July 4th 1908.

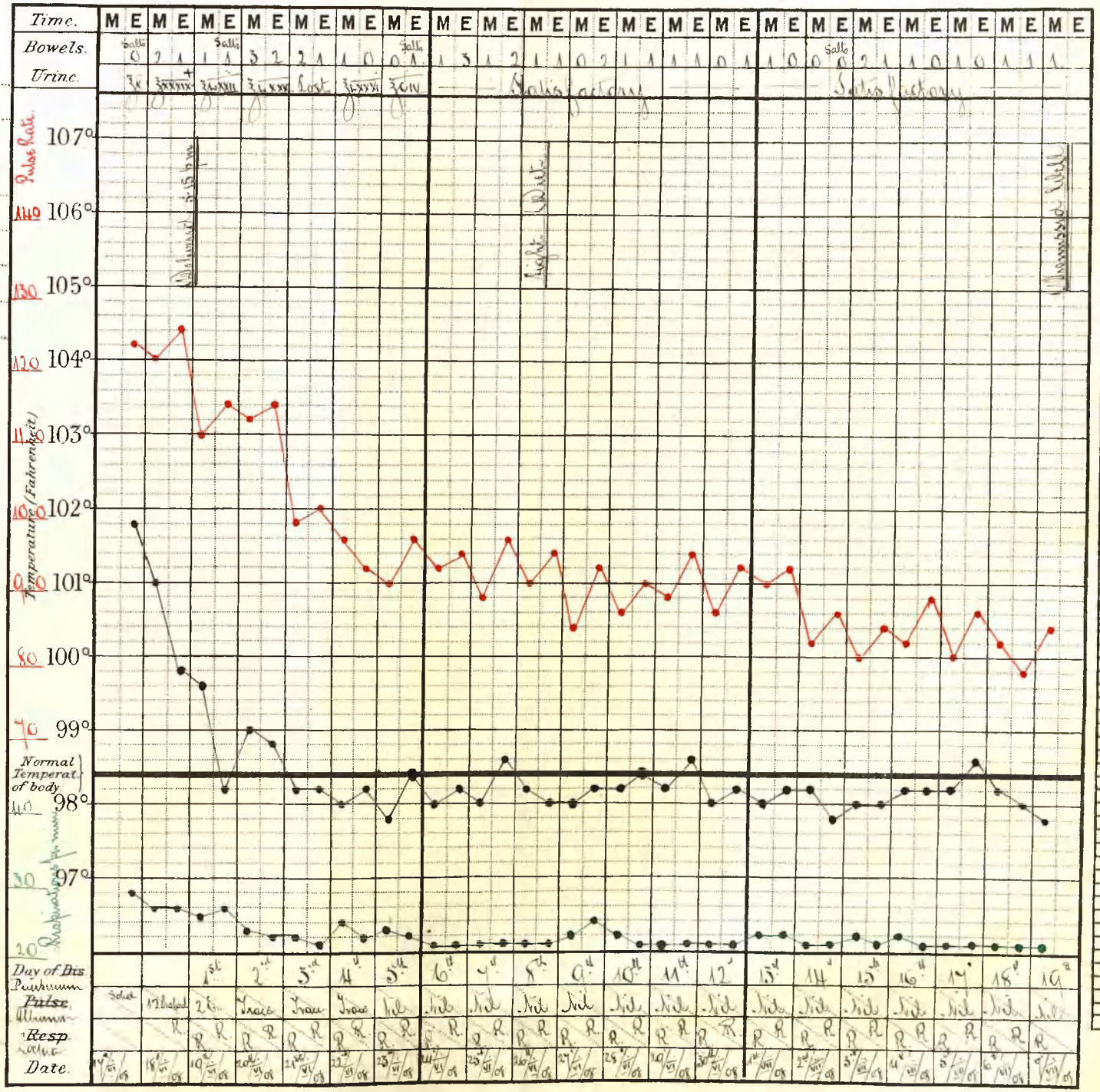
C A S E XXXIII

DISEASE.

Notes of Case.

Book N° XXXIII

Date of admission.



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CASE XXXIII MRS TURNBULL, aet 25 years, primipara. Full time. Recovery.

Admitted on 17th June 1908 at 7.45 p.m.

Delivered on 18th June 1908 at 5.15 p.m.

Presentation vertex. Left occipito-anterior position.

Child Male dead, 6½ lbs. weight.

HISTORY: The patient seems to have had no symptoms of any kind before or during her pregnancy. When her husband went to work at 6 a.m. on the 17th June she was quite well, but on his return at 6.30 p.m. he could not get into the house. The door being forced, he found his wife "in convulsions". The exact number of fits taken could not be ascertained, but there must have been several, as the skin is bruised over the elbows, scapulae and buttocks.

PRESENT CONDITION: The patient is quite unconscious, and cyanosed. The pupils are dilated, and insensitive to stimuli. Immediately after admission she took three convulsions in quick succession. Each convulsion was of about one and a half to three minutes duration. Temperature 101.8° F. Pulse 122 beats per minute, regular, and of high tension. Respirations number 28 per minute. No sign of foetal life can be discovered. The head presents in the first position. The abdomen is equal in size to that of a full time pregnancy. Per vaginam the pelvis shows signs of flattening at the brim, the true conjugate measuring three and five eighth inches, obliques four and a half inches. Inter-

cristal measurement was ten and three quarter inches, and the Interspinous ten and a quarter inches. The os is closed, and the cervix is fully taken up. There is no sign of commencing labour. She had further convulsions at 10.5 and 10.25 p.m.

Urine, 10 ozs.; acid; dark; Specific Gravity 1025; Albumen solid; granular tube casts; blood; Urea 4 grains per ounce.

Treatment. After the first three fits, Magnes. Sulph. 3 ozs. were given per nasal tube. She was bled 1 pint, and transfused 2 pints Saline solution. At 10.25 p.m. she was put in a hot wet pack. Milk was given per tube.

18th June 1908: The patient passed a fairly good night, having slept for several hours. She had a fit at 12.15 a.m., and another at 2.35 a.m. She then slept until 7.30 a.m. At this time it was noticed that uterine contractions had commenced. Up till 1 p.m. she had seven more convulsions, but these were of the slightest description, lasting only from one quarter to one half minute. She was semi-conscious in the intervals between the seizures, and could swallow fairly. At 1 p.m. the membranes ruptured, but the os was not fully dilated until 4 p.m. The patient was anaesthetised, forceps applied, and the child delivered. While the head was passing the brim of the pelvis the patient was placed in the Walcher position. The perineum was ruptured half way backwards. This was stitched immediately after delivery of the pla-

centa.

Urine 39 ozs. a quantity lost at stool; milky; acid; Specific Gravity 1028; Albumen 12 per thousand Esbach; tube casts; trace, blood; Urea 7 grains per ounce.

Treatment. Milk diet. Chloral and Bromide a a 20 grains at four hourly intervals.

19th June 1908: The patient is semi-conscious, and very restless. There has been no return of the convulsions.

Urine 72 ozs.; clear; Specific Gravity 1022; Albumen 2 per thousand Esbach; no tube casts or blood; Urea 8½ grains per ounce.

20th June 1908: She is quite conscious and quiet today. The Albumen in the urine is only a trace. Urea measures 9 grains per ounce.

Treatment. The Chloral and Bromide was discontinued.

Improvement continued, the urine being free from Albumen on 23rd June, and light diet given on the 26th June. The perineum healed without difficulty. She was dismissed, well, on July 7th 1908.

C A S E XXXIV

DISEASE.

Lumbago

Post-Partum

Notes of Case.

No

Livesay

40 Years

Milk - Light

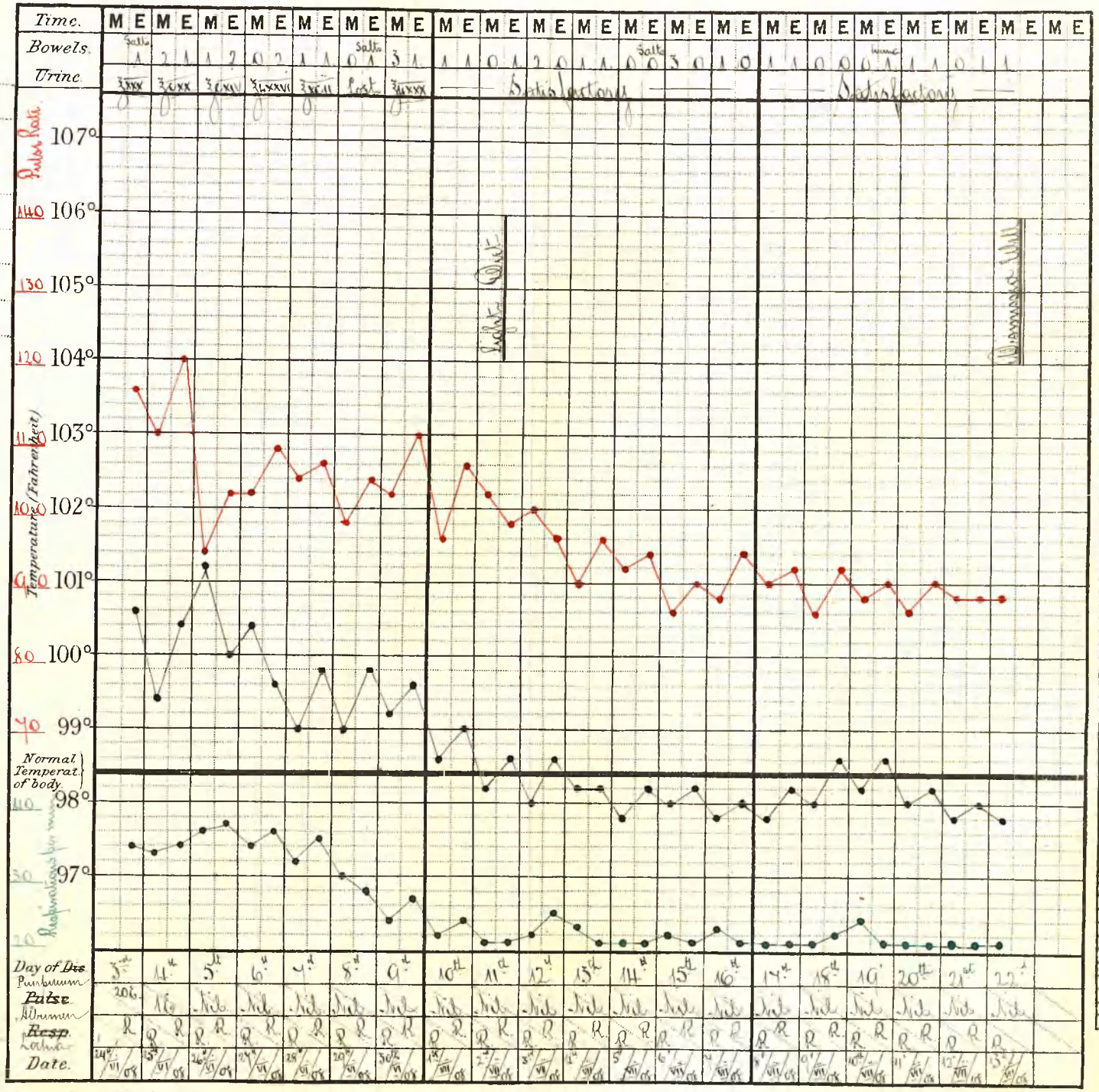
~~Sick~~ No XXXIV

Para

Date of admission.

14th June 1908

Recovery



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Gould's Clinical Chart.

CASE XXXIV MRS LINDSAY, aet 40 years, VI para. Delivered before admission. Recovery.

Post-Partum Eclampsia.

Admitted on 24th June 1908 at 8 p.m.

Delivered on 22nd June 1908 at 3 a.m.

HISTORY: Previous to her pregnancies the patient had always been healthy. From two to three days after every pregnancy she had had attacks of violent and uncontrollable vomiting, which had lasted from twenty four hours to three days. Her Doctor had ascribed these attacks to a toxaemia. During the present pregnancy (the sixth) when about twelve weeks pregnant she had been in the Royal Infirmary, Glasgow, for six weeks, with pernicious vomiting. She had been dismissed well. She had remained in fair health until after the confinement. She was delivered of a live female child at 3 a.m. on June 22nd. At 9 a.m. on June 24th the convulsions commenced, and she had nine before admission.

PRESENT CONDITION: The patient is unconscious, but can be roused to a semi-conscious state, and can swallow fluids. There is no cyanosis. The tongue is lacerated. The temperature is 100.6° F. Pulse numbers 116 beats per minute; it is regular and of poor tension. Respirations are 34 per minute. The uterus reaches to four inches above the umbilicus, and seems well retracted and contracted. She took an eclamptic convulsion at 9.35 p.m., before transfusion, which lasted three minutes.

Urine. Quantity 30 ozs., per Catheter; dark amber; acid; Specific Gravity 1025; Albumen 20 per thousand Esbach; few tube casts; no blood.

Treatment. Magnes Sulph. 2 ozs. Saline solution 2 pints transfused. Chloral and Bromide a a 25 grains were given twice at a four hourly interval.

25th June 1908: The patient spent a restless night. She slept only for short periods. Convulsions occurred at 1.40 a.m., 2.30 a.m., 6.45 a.m., and 10.45 a.m. The length of these fits averaged two minutes. She regained semi-consciousness between them, and was able to swallow small quantities of milk.

Urine, 120 ozs.; milky; acid; Specific Gravity 1018; Albumen one per thousand Esbach; no casts or blood.

Treatment. Chloral and Bromide a a 20 grains were given at 2.30 a.m. and at 7 a.m.

26th June 1908: The patient had slept at intervals, between which she was restless. She is fairly conscious, but in a state of mild dementia. At 10.30 a.m. she vomited violently, the vomit consisting only of curdled milk and a watery material. There has been no return of the convulsions.

Urine, 114 ozs.; clear pale straw; acid; Specific Gravity 1015; no Albumen, tube casts or blood.

Treatment. Milk and Imperial drink. Chloral and Bromide a a 10 grains four hourly.

She remained in this condition of mild dementia

until the 2nd July 1908, when her mental condition became normal. It was not until this day that she could be prevailed on to acknowledge the child as her own. She knew and recognised her other children.

She was dismissed well on July 13th, 1908.

C A S E XXXV

DISEASE.

Thombson

Notes of Case.

Mrs
Thombson
22 Years
Male - Single
Book N° XXXV

II Para

Date of admission.
30th June 1908
Mrs Thombson

Time. M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E

Bowels. Manna Salts 1 0 | 3 1 | Salts 1 0 | 2 0 | 1 1 | Salts 1 0 | 2 1 | Salts 1 1 | 3 0 | 2 1 | 1 0

Urine. 3 xii | 3 xx | 3 xiv | 3 xxviii | 3 xxxvi | Lost | Lost | 3 xxxvi | 3 xxxvii | 3 xiv | 3 xii | Lost

Pulse Rate

Temperature (Fahrenheit)

Normal Temperat of body.

Respirations

Temperature. (Centigrade)

Fahrenheit	Centigrade
107°	42°
106°	41°
105°	40°
104°	39°
103°	38°
102°	37°
101°	36°
100°	35°
99°	
98°	
97°	

Day of Puerperium: 1st 2nd 3rd 4th 5th 6th 7th 8th 9th 10th

Pulse: Solid Solid 1/6 Trace Trace Trace Trace Trace 1/6 1/26 Trace 1/6

Albumen: X R R R R R R R R R R

Date: 30th/VI/08 1st/VII/08 2nd/VII/08 3rd/VII/08 4th/VII/08 5th/VII/08 6th/VII/08 7th/VII/08 8th/VII/08 9th/VII/08 10th/VII/08

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CASE XXXV MRS THOMPSON, aet 22 years, II para. Full time. Recovery.

Admitted on 30th June 1908 at 11.10 a.m.

Delivered on 1st July 1908 at 12.o'cnoon.

Presentation vertex. Left occipito-anterior position.

Child Male dead, 8¼ lbs. weight.

HISTORY: When a schoolgirl, after an attack of measles, the patient had suffered from "an acute inflammation of the Kidneys". Five years ago she had had another attack of Kidney trouble. In her first pregnancy, eighteen months ago, she had had headache, and swelling of the face and lower extremities. These symptoms had disappeared after delivery. During this pregnancy she had had swelling of the face and lower extremities for eight weeks before admission. She had also been troubled with intermittent frontal headache. For three of four days before the eclampsia commenced the headache had become constant and very severe.

She was found unconscious on the floor, and was brought to the Glasgow Maternity and Women's Hospital.

PRESENT CONDITION: The patient is unconscious, and cyanosed. The tongue is lacerated. There is a general anasarca present, being most marked on the face and ankles. Temperature is 100° F. Pulse is 100 beats per minute, regular and of high tension. The uterus is that of a full time pregnancy. No signs of foetal life can be discovered. Per vaginam the os admits one finger, and the cervix is fully taken up. No uterine contractions are going on.

The vertex presents in the first obstetrical position. She had eclamptic seizures at 1.30 p.m., 4.10 p.m., 6.5 p.m., and 8.15 p.m. (all after transfusion) The average length of these attacks was two and a quarter minutes. In the intervals between them she lay quite quiet and comatose. Towards evening uterine contractions commenced. After 8.30 p.m. she became very restless.

Urine, 12 ozs., per Catheter, on admission; dark colour; Specific Gravity 1020; Albumen solid; granular and hyaline tube casts; no blood.

Treatment. On admission Magnes. Sulph. 2 ozs. was given per nasal tube. She was bled 1 pint, and transfused Saline solution 2 pints. At 6.5 p.m. she was placed in a hot wet pack. Chloral and Bromide ā ā 25 grains were administered per rectum at 2 p.m., 4.30 p.m., and 8.30 p.m. At midnight, the os being fully dilated, the patient was anaesthetised, membranes ruptured, forceps applied, and child delivered.

1st July 1908: The patient slept well, and awakened semi-conscious. There have been no further convulsions. She is now able to swallow fluids.

Urine 54 ozs.; milky, acid; Specific Gravity 1025; Albumen 4 per thousand Esbach; tube casts; no blood.

Treatment. Milk and Imperial Drink.

2nd July 1908: The patient is now quite conscious. She has a cough, and a muco-purulent expectoration. At the bases of the lungs moist rales can be heard. The temperature, pulse and respirations are rising. The fundus

of each eye shows traces of old and recent retinal haemorrhages, and there is distinct blurring of the discs.

Urine 72 ozs.; clear; acid; Specific Gravity 1022; Albumen, trace.

3rd July 1908: The patient remains in the same condition as on the 2nd July.

Albumen is still a trace in the urine.

6th July 1908: The cough and expectoration are less, and the condition in the lungs has improved.

Albumen, faint trace.

7th July 1908: The patient complains of headache this morning.

The Albumen in the urine has risen to 2 per thousand Esbach.

Treatment. Purge. Hot wet pack.

8th July 1908: The headache is less today, and the urinary output has risen.

Albumen is ½ per thousand Esbach.

9th July 1908: No headache is present today, and a faint trace of Albumen is present.

10th July 1908: The patient insisted on leaving the Hospital, taking the responsibility of so doing.

The Albumen in the urine = 1 per thousand Esbach.

C A S E XXXVI

DISEASE.

Lambeth

Notes of Case.

No.

Brighton

32 Years.

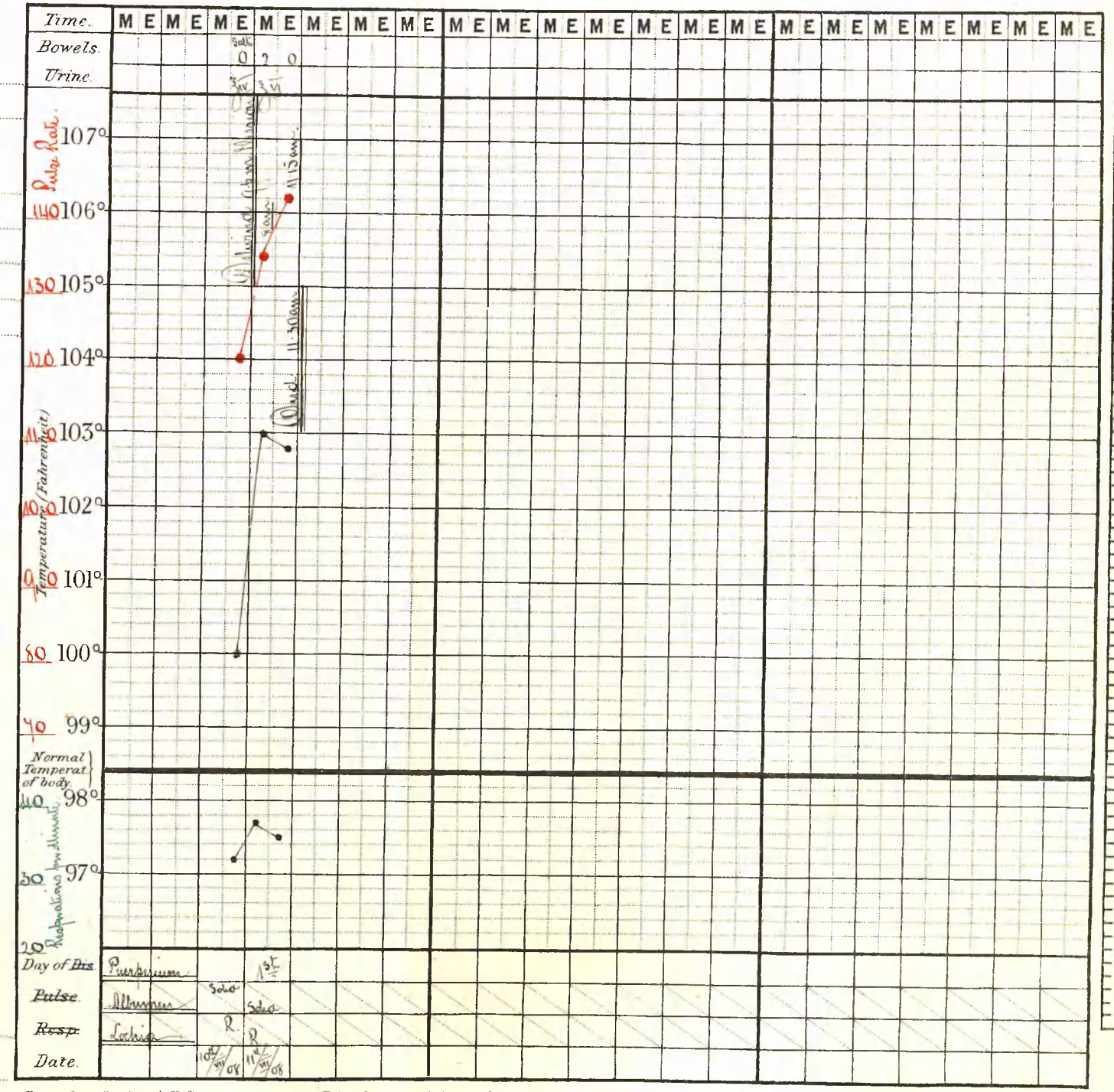
Book No. XXXVI

Para

Date of admission.

July 1908.

Date of death.



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CASE XXXVI MRS DEIGHTON, aet 32 years, primipara. Full time. Death.

Admitted on 10th July 1908 at 3.50 p.m.

Delivered on 10th July 1908 at 9 p.m.

Presentation vertex (version)

Child Male dead, 7¼ lbs.

HISTORY: Several years ago the patient had been treated in the Victoria Infirmary, Glasgow, "for Gastric Ulcer". This had been her only illness previous to the onset of pregnancy. For three weeks previous to admission she had been troubled with swelling of the feet and ankles, and a week later, intermittent swelling of the face had been also noticed.

During the night 9th - 10th July 1908 she had complained of severe headache, and of a great pain in the epigastrium, but in the morning had said that she felt better. Her husband states that at 6 a.m., when he left her, she was "fairly well". Shortly after he left the house his wife suddenly "lost her sight", but managed to grope her way to the door and call a neighbour. The blindness continued for two hours, and was accompanied by "a numb feeling in the head". The patient then had a very severe convulsion. This was followed by a second fit, which also seems to have been severe. A third seizure took place in the ambulance.

PRESENT CONDITION: The patient is greatly cyanosed, and completely unconscious. The tongue is lacerated, and there is a blood-stained froth around the mouth. The pupils

are "pin-point", and do not react to stimuli. There is no history of the administration of any drug. Temperature 100° F. Pulse 120, regular and of high tension. Respirations 32 per minute. The lungs are comparatively clear. The uterus is equal in size to that of a full time pregnancy. The head presents in the first obstetrical position. No signs of foetal life can be discovered. Per vaginam the os admits the tip of a finger. The cervix is fully taken up. There are slight uterine contractions in progress. At 4 p.m. she had a series of convulsions which lasted twenty minutes. This series was of exceptional severity, and it seemed as if she would not survive it. Chloroform was given at this stage, in order to control the fits and perform phlebotomy and transfusion. The pulse became fast, weak, and irregular during the narcosis. Between 6.15 p.m. and 8.30 p.m. she had six convulsions, the average duration of which was four minutes. Between these attacks she was very restless, and great difficulty was experienced in controlling her.

At 9 p.m. the os uteri was equal to a florin in size. Under an anaesthetic a Champetier de Ribes bag of medium size was inserted through the os, and distended with sterile water. When the os was sufficiently dilated podalic version was performed, and the child delivered without difficulty. Between 10.15 p.m. and 12 midnight she had five more fits of great severity.

The pulse and temperature had now risen considerably.

Urine. Quantity 4 ozs.; muddy; slightly acid; Specific Gravity 1035; Albumen solid; granular tube casts; blood; Urea 3 grains per ounce.

Treatment. On admission to Labour Ward, Magnes. Sulph. 3 ozs. per nasal tube. Bled 1 pint, transfused Saline 2 pints. Chloral and Bromide a a 20 grains per rectum, two, three or four hourly as required. Later in the evening a hot wet pack was given, also hot sinapisms were applied over the Kidney region. After the attacks at midnight, as the pulse was almost imperceptible, Strychnine $\frac{1}{30}$ gr. was given hypodermically, also Brandy 2 ozs. per rectum.

11th July 1908: At 1 a.m. there was another seizure, and at 5 a.m. the last. The patient at this time was very exhausted, and another Saline (2 pints) was transfused under the breast. Strychnine $\frac{1}{30}$ gr. and Digitalin $\frac{1}{100}$ gr. were given four hourly, also Brandy 2 ozs. per rectum two hourly.

The patient, however, gradually sank and died during the forenoon.

C A S E XXXVII

DISEASE.

Diarrhoea

Notes of Case.

Harriet

Burns.

17 Years

Milk.

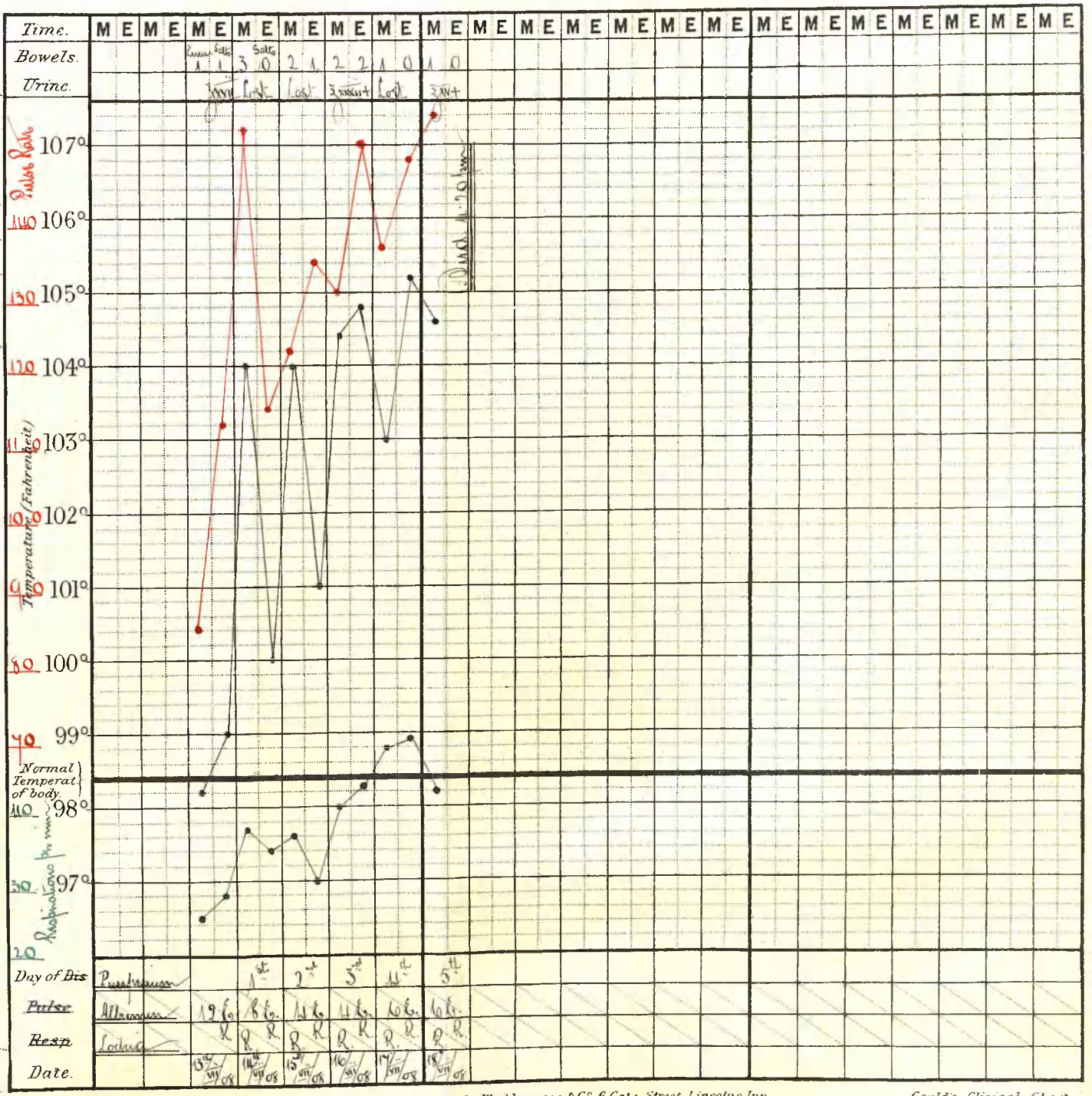
~~Book~~ N° XXXVII

1 Para

Date of admission.

15th July 1908

Result Death.



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CASE XXXVII HARRIET BURNS, aet 17 years, primipara. Full time. Death.

Admitted on 13th July 1908 at 2.5 a.m.

Delivered on 13th July 1908 at 6.45 p.m.

Presentation vertex.

Child Male alive, 6½ lbs. weight.

HISTORY: It was found out after the patient's death that there had been some oedema of the lower extremities of three weeks' duration. No advice was sought regarding this swelling, as the condition of the patient was being concealed. There had been no other symptom complained of.

PRESENT CONDITION: No oedema is noticeable on admission. Labour has commenced. The os admits one finger. The vertex presents in the fourth position, right occipito-anterior. Foetal heart's sounds can be distinctly heard. Temperature is normal. Pulse rate is 84 beats per minute, regular and of rather high tension. Respirations number 25 per minute.

At 6.30 p.m. when the head was protruding from the vulva, she took the first convulsion. From this time until 7.40 a.m. on the 14th July 1908 twenty-seven fits were taken. The labour had not been severe.

Urine 22 ozs., per Catheter; clear amber coloured; acid; Specific Gravity 1020; Albumen 12 per thousand Esbach; few granular tube casts; trace, blood.

Treatment. After the first convulsion the patient was anaesthetised, and the child was delivered with for-

ceps. At this time also 1 pint blood was withdrawn from the median basilic vein, and 2 pints Saline transfused. Magnes. Sulph. 2 ozs. were given. Chloral and Bromide a a 25 grains were given twice per rectum when the convulsions were severe and followed each other closely. At 10 p.m. a hot wet pack was given.

14th July 1908: The patient is still unconscious. Temperature, pulse and respirations are considerably raised. Over the whole pulmonary area moist and crepitant rales are to be heard. These are, however, most numerous at the bases of the lungs. Later in the day the percussion note at the bases of the lungs behind showed impairment.

Urine. Exact quantity of urine unknown; milky; acid; Specific Gravity 1022; Albumen 8 per thousand Esbach; tube casts; no blood.

Treatment. At 12.20 a.m. (when the fits were in progress) 2 pints Saline solution were transfused under the right breast. Later, when pulmonary oedema was commencing, Strychnine $\frac{1}{30}$ grain was given four hourly. The position of the patient was changed frequently. Brandy 2 ozs. was given four hourly per rectum. Poultices were applied to the bases of the lungs when the position of the patient permitted of it

15th and 16th July 1908: In spite of the treatment, the oedema of the lungs is slowly increasing. The patient is semi-conscious, and swallows liquid nourishment freely.

The urine is fairly abundant, and the Albumen is decreasing.

The treatment is similar to that carried out on the 14th July.

17th July 1908: The condition of the patient is much worse today. She is quite unconscious, and feeding is carried out by means of a nasal tube. The bases of the lungs are quite solid.

Urine contains Albumen to the extent of 6 per thousand Esbach.

The treatment is much the same as before.

18th July 1908: She is still unconscious, and the pulse is beginning to flag.

Later. She gradually sank and died at 4.20 p.m.

C A S E XXXVIII

DISEASE.

Pneumonia

Notes of Case.

Mrs

Skelton

34 Years

Milk Nurse

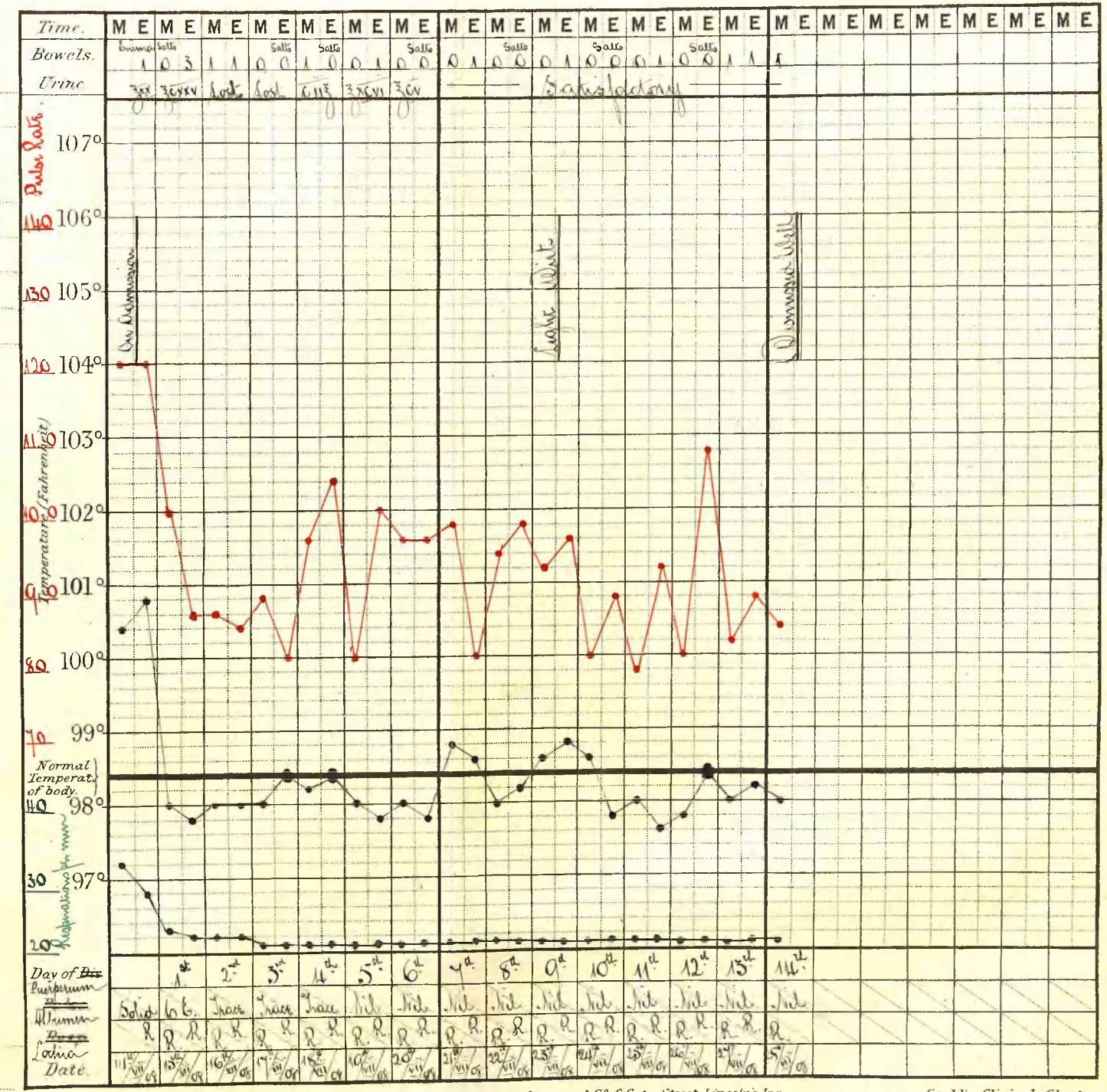
Ward No XXXVIII

Para

Date of admission.

14th July 1908

Result Recovery



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Gould's Clinical Chart.

CASE XXXVIII MRS SKELTON, aet 34 years, II para. 36 weeks.
Recovery.
Admitted on 14th July 1908 at 12.15 p.m.
Delivered on 14th July 1908 at 11.30 p.m.
Presentation vertex. Left occipito-anterior position.
Child Male dead, 5 lbs. weight.

HISTORY: The patient had never known a day's illness apart from the pregnancies. The first pregnancy had been, and had ended normally. About one month ago she had noticed that the feet and ankles were swollen, especially after much standing. A Doctor had been consulted, who found a considerable quantity of Albumen in the urine. She had been put on treatment, and although the Albumen in the urine had diminished, it had not entirely disappeared. She had had frequent frontal headache. At midnight (13th - 14th July 1908) she was suddenly seized with an agonising pain in the "pit of the stomach". Her Doctor was summoned, and he administered an opiate, which relieved the pain. At 9 p.m. she took a severe convulsion which lasted, according to her friends' statement, ten minutes. Two other convulsions occurred before her admission to Hospital.

PRESENT CONDITION: The patient is conscious, but rather dazed. The pupils are normal in size, and react to stimuli. The temperature is 100.4° F. Pulse is 120 per minute, regular and of high tension. Respirations number 32 per minute. The uterus reaches almost to the

ensiform cartilage of the sternum. No signs of foetal life can be discovered. There are no uterine contractions going on. Per vaginam the os uteri admits one finger, and the cervix is not fully taken up. The head presents in the left occipito-anterior position. A few minutes after admission to Hospital she took a severe eclamptic fit which lasted three quarter minutes. Half an hour later she took another convulsion, also of great severity. After this attack she was very restless, and remained semi-conscious. About 4 p.m. uterine action had definitely set in. At 6.40 she had a third and last seizure. It was also severe, and was followed by vomiting. Labour proceeded quite naturally, the membranes ruptured at 8.15 p.m., and she was delivered of a dead male child at 11.30 p.m.

Urine. 20 ozs., per Catheter; almost black; alkaline; Specific Gravity 1038; Albumen solid; tube casts of all sorts very abundant; blood in great quantity. There was an enormous sediment which consisted almost entirely of tube casts and blood cells.

Treatment. Magnes Sulph. 2 ozs., aided later by a large soap and water enema. After the first fit she was bled 1 pint, and 2 pints Saline solution was transfused. After the second attack, Chloral and Bromide a a 30 grains were given per rectum. Milk diet and Imperial drink.

15th July 1908: The patient is quite conscious today. Temperature, pulse and respirations are normal. She is

drinking great quantities of fluid.

Urine, 125 ozs.; milky; acid; Specific Gravity 1025; Albumen 6 per thousand Esbach; tube casts scanty; trace, blood.

Treatment. Milk diet and Potass. Imperialis.

From the 16th July 1908 - 28th July 1908, the date of dismissal, convalescence was uninterrupted.

The Albumen disappeared from the urine on the 19th July 1908.

Light diet was given on the 23rd July 1908.

C A S E XXXIX

DISEASE.

Lambsia

Post Partum

Notes of Case.

Jessie

Leggat.

18 Years

Milk - Light.

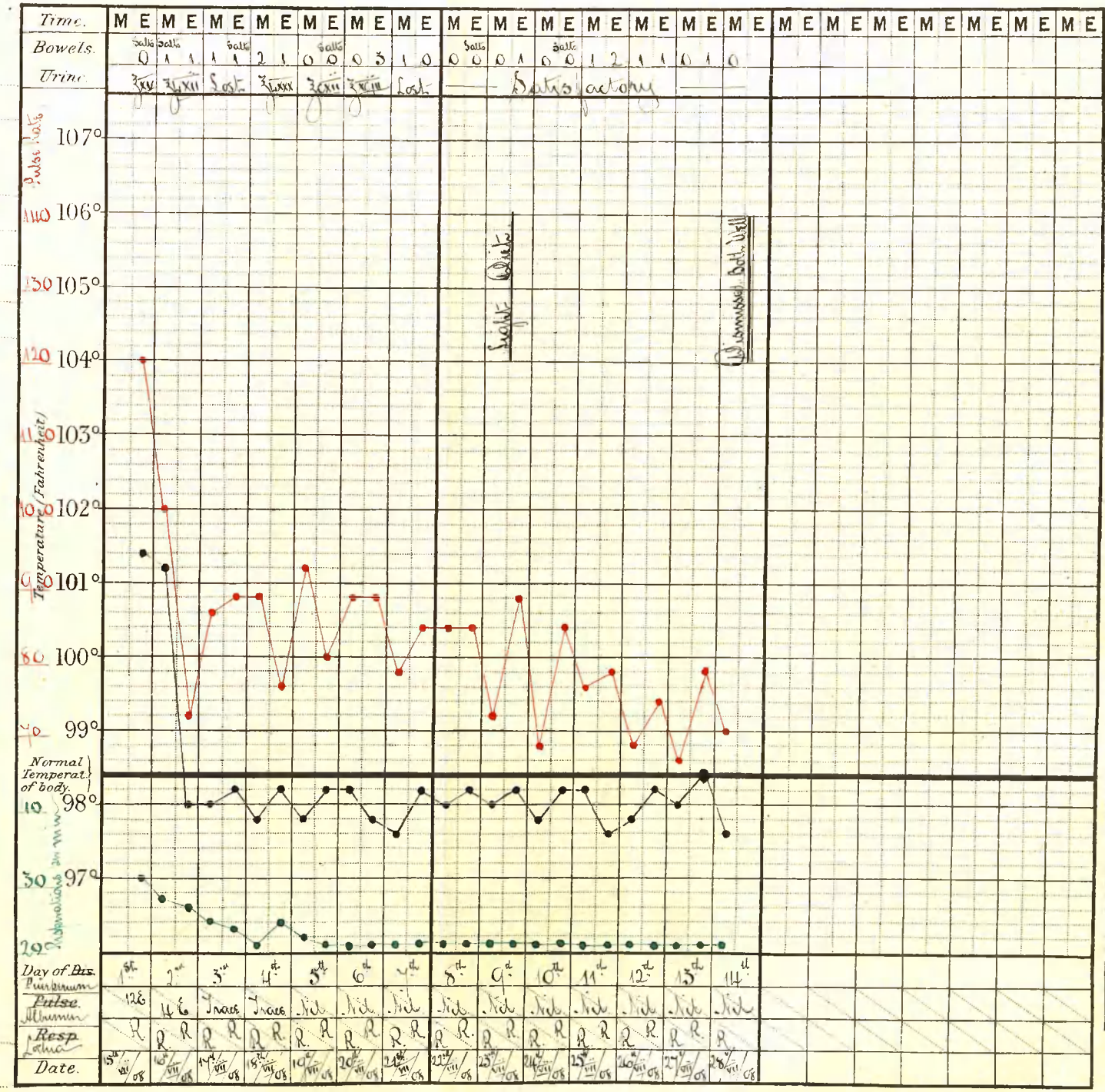
No XXXIX

Para

Date of admission.

July 1908

Recovery



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CASE XXXIX LIZZIE LEGGAT, aet 18 years, primipara. Full time. Recovery.

Post Partum Eclampsia.

Admitted on 15th July 1908 at 6.45 p.m.

Delivered on 15th July 1908 about 12 noon before admission.

Child Female alive.

HISTORY: There is no history of antecedent disease. During the pregnancy there was no oedema, headache, or disturbance of vision. A nurse from the Hospital was summoned at 7 a.m. on the day of admission, and found the patient at the commencement of labour. The nurse did not notice oedema on any part of the body. The labour ended naturally at 12 noon.

At 3.30 p.m., without any warning, the patient was seized with a convulsion. Two more seizures occurred, and she was sent to Hospital.

PRESENT CONDITION: The patient is slightly cyanosed, and semi-conscious. She can be roused, and is able to swallow fluids in small quantities without difficulty. The pupils are normal. The tongue is slightly lacerated. The uterus extends to the umbilicus, and is well retracted and contracted. Temperature 101.4° F. Pulse 120 beats per minute, regular and of high tension. Respirations number 30 per minute. Shortly after admission she had two eclamptic seizures lasting three and two minutes respectively. After the period of coma had passed she remained much in the same condition as she was in on admission.

Urine. Dark coloured; acid; Specific Gravity 1030; Albumen 12 per thousand Esbach; tube casts; no blood.

Treatment. Bled 1 pint, and transfused 2 pints Saline solution (four fits before, and one after transfusion). Magnes. Sulph. 3 ozs. Chloral and Bromide a a 20 grains (once after admission and once three hours later).

16th July 1908: The patient is quite conscious.
Albumen is 4 per thousand Esbach.

17th July 1908: Improvement continues.
Albumen, trace.

18th July 1908: Improvement continues.
Albumen, trace.

19th July 1908: Improvement continues.
Albumen, nil.

23rd July 1908: Improvement continues.
Albumen, nil.
Light diet.

28th July 1908: Mother and child dismissed, well.

CASE XL

DISEASE.

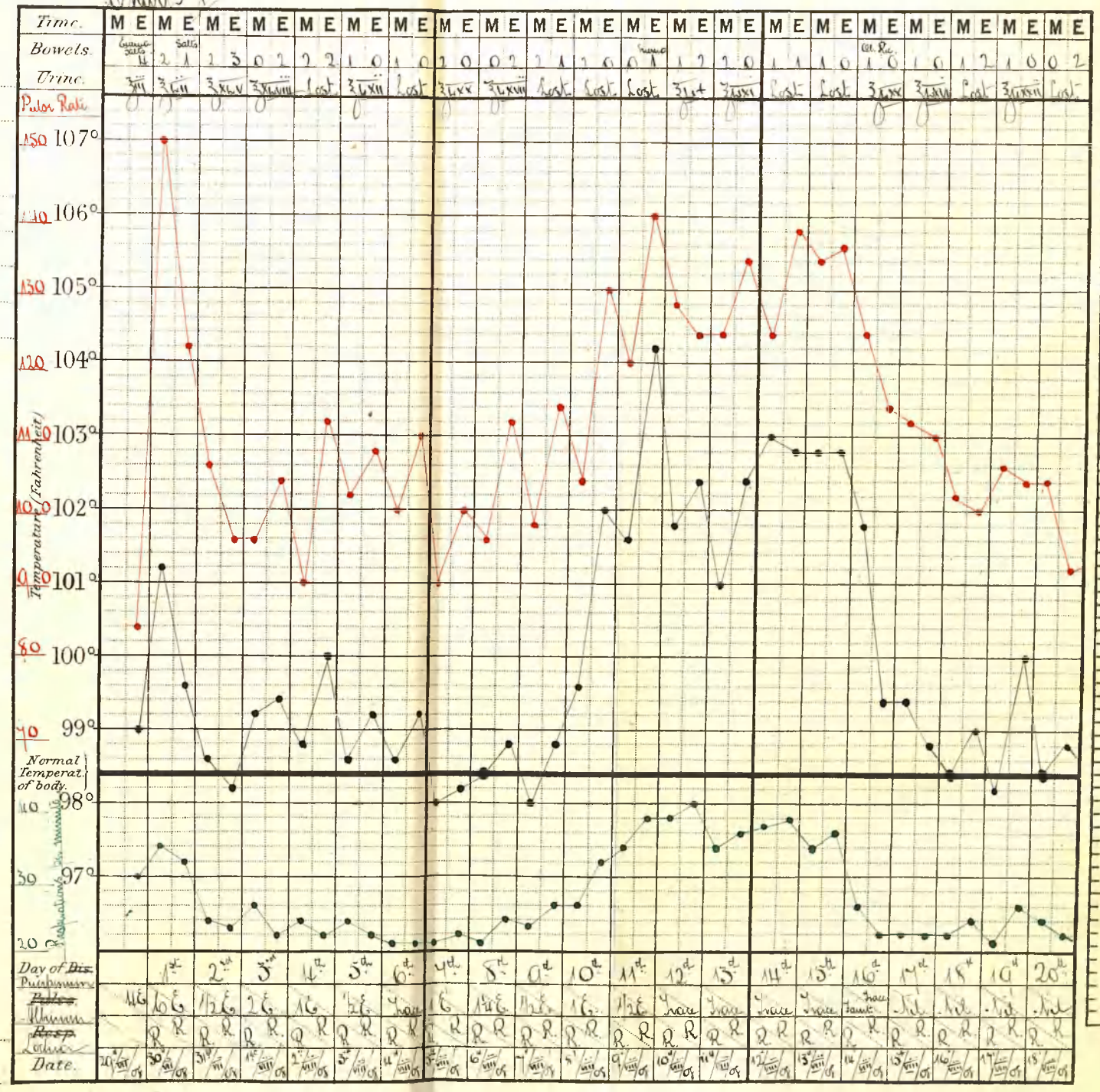
Adamsson

Notes of Case.

Mrs
O'Donnell
10 Years
M.R. - Light
Book N° XL

Para

Date of admission.
10th July 1908
Recovery



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Gould's Clinical Chart

DISEASE.

Diarrhoea

Notes of Case.

Mr. O'Donnell

10 Years

Milk - Light

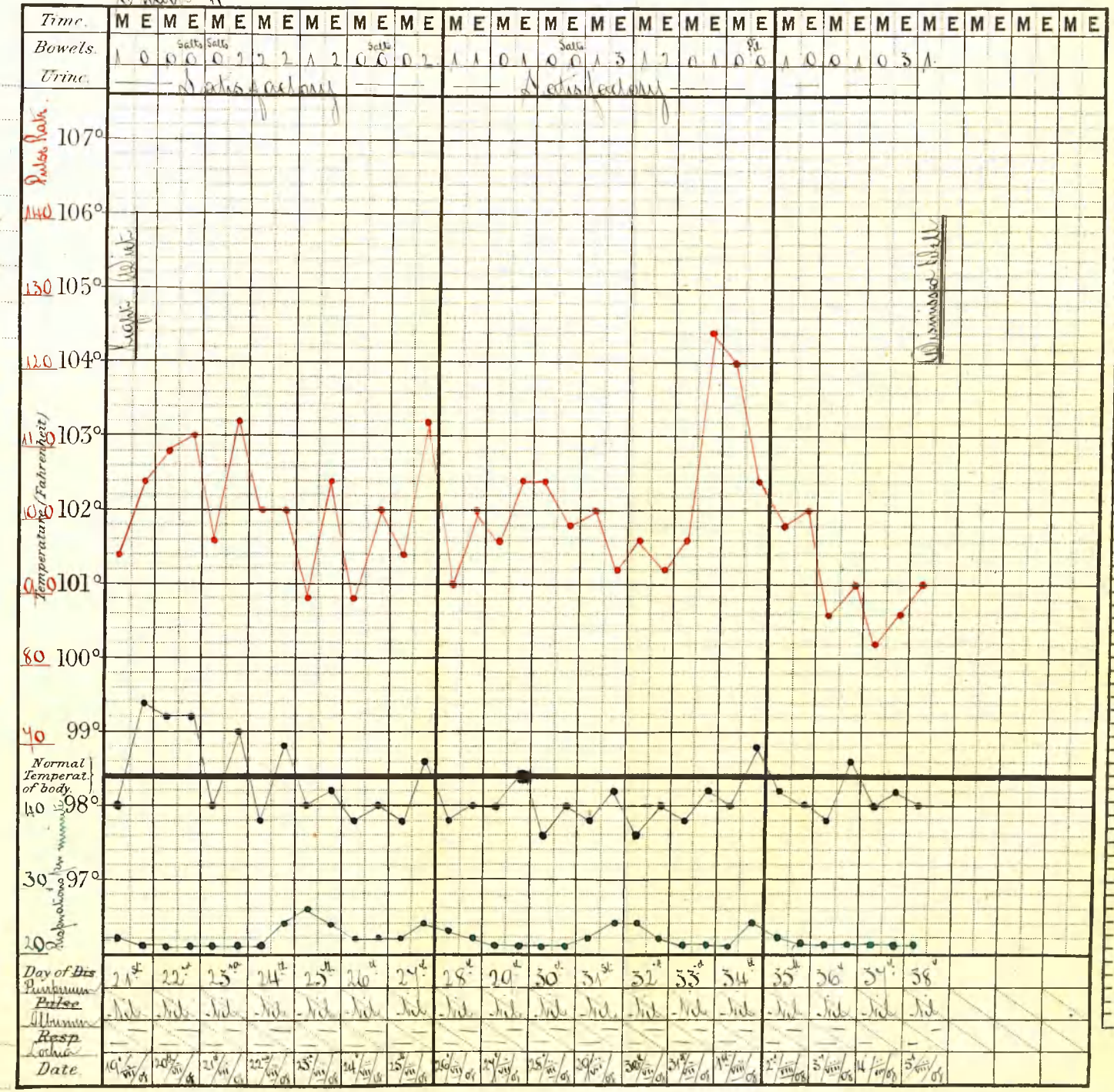
~~Cot~~ No XI.

Para

Date of admission.

July 1908

Recovery



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Gould's Clinical Chart.

CASE XL MRS O'DONNELL, aet 19 years, primipara. Full time. Recovery.

Admitted on 29th July 1908 at 5.15 p.m.

Delivered on 30th July 1908 at 6.25 a.m.

Presentation vertex.

Child Male dead, 6¼ lbs. weight.

HISTORY: There is no history of antecedent disease. From the third or fourth week of the pregnancy she has been troubled with intermittent vomiting and frontal headache. On the evening of the 28th July she had severe epigastric pain, accompanied from time to time by vomiting; the vomited matter consisting of bile-stained mucus. No oedema on any part of the body had been noticed. There had been no alteration in the power of vision. A neighbour states that the patient took a fit lasting "one and a half hours". A Doctor was then called, who sent the patient to Hospital.

PRESENT CONDITION: The patient is fairly conscious, and can answer questions intelligently. She is very pale and anaemic. No oedema is present on any part of the body. The tongue is lacerated. The pupils are normal in size and react normally. Temperature is 99° F. Pulse is 84 beats per minute, regular and of fair tension. Respirations number 30 per minute. The uterus is that of a full time pregnancy. The vertex presents, and is well into the cavity of the pelvis. No signs of foetal life can be discovered. Per vaginam the os admits one finger, and the cervix is fully taken up. The position of

the head is the left occipito-anterior one. There are slight uterine contractions going on. She had no fits from admission until 8.10p.m., when a typical eclamptic fit was taken. This was followed by ten others, at 8.20 p.m., 9.25 p.m.; 30th July 1908 - 1.45 a.m., 3.40 a.m., 3.48 a.m., 3.50 a.m., 3.55 a.m., 3.58 a.m., 4.10 a.m., and 5.5 a.m. These attacks varied in intensity and duration. The average length of the fits was 55 seconds. In each successive interval between the convulsions the patient became more deeply comatose.

Urine, on admission, Catheter Specimen, 3 ozs.; dark coloured; acid; Specific Gravity 1025; Albumen 4 per thousand Esbach. Granular tube casts (scanty); blood, trace; Urea 5 grains per ounce.

Treatment. On admission Magnes. Sulph. 2 ozs. Hot wet pack. Milk. Pot. Imperialis. After the third fit at 9.25 p.m., bled 1 pint, transfused Saline 2 pints. At 9.30 p.m. Chloral Hydrate and Potass. Bromide a a 25 grains were given per rectum. (In Hospital, therefore, she had three convulsions before the transfusion, and seven afterwards; although it seemed as if the convulsions were of less severity after transfusion.)

30th July 1908: The patient had eight fits in the early morning (see above). After the last one she was deeply comatose, and remained so for three hours. The temperature at this time rose to 102.8° F., but quickly dropped to 101° F. About 6 a.m. the os uteri had dilated to the size of a five shilling piece. The

patient was anaesthetised, very little chloroform being required. The os was then dilated manually, membranes ruptured, and the child delivered with forceps. The perineum was torn half way back to the rectum. This was immediately repaired. After delivery the pulse became very fast, fluttering and irregular, but after stimulants had been given it rallied considerably.

Later:- The patient is now semi-conscious, and sleeps at intervals. The temperature has fallen to 99.4° F., and the pulse to 122 beats per minute. The cyanosis and other signs of the convulsions have completely passed off.

Urine. Quantity 52 ozs.; milky; acid; Specific Gravity 1028; Albumen 6 per thousand Esbach; Urea 4 grains per ounce; granular tube casts; trace, blood.

Treatment. Milk. Potas. Imperialis. At 2 a.m. and 4 a.m., when the convulsions were in progress, Chloral Hydrate and Potass. Bromide a a 25 grs. were given per rectum. When the pulse became so rapid and irregular after delivery, Strychnine $\frac{1}{30}$ grain was given, and Strychnine $\frac{1}{60}$ grain was continued afterwards four hourly. At the same time Brandy 3 ozs. was given per rectum.

31st July 1908: The patient is greatly improved today. She is quite conscious. The temperature is normal, and the pulse numbers 106 beats per minute, is regular, and of good tension.

Urine, 45 ozs.; clear amber; acid; Specific Gravity

1022; Albumen ½ per thousand Esbach; Urea 7 grains per ounce; no tube casts; no blood.

Treatment as before.

1st August 1908: This morning the improvement continues. The patient passed a good night, having slept for a number of hours. Urine was passed naturally.

Urine, 48 ozs.; amber; acid; Specific Gravity 1025; Albumen 2 per thousand Esbach; Urea 6 grains per ounce; no tube casts; no blood;

Treatment. Brandy discontinued.

2nd August 1908: The patient continues to improve.

Albumen 1 per thousand Esbach. Urea 6 grains per ounce.

3rd August 1908: Improvement continues. The perineum shows signs of breaking down. It was swabbed carefully with Hydrogen peroxide.

(From this date until the 9th August 1908 I was away from the Hospital, and no more observations except those found on Chart were made.)

9th August 1908: I transferred the patient to the Isolation Block on my return today. The pulse and temperature are high. She complains of headache and of feeling unwell. The perineum is dirty and sloughing, and there is a degree of local redness and tenderness. Careful examination of the body elsewhere does not reveal any further lesion than might account for the temperature. The lungs are normal. The uterus and appendages are

free from any sign of inflammation. The rate of Involution of the uterus is normal. The lochia remains fresh and sweet.

Urine, 71 ozs.; pale straw; acid; Specific Gravity 1018; Albumen ½ per thousand Esbach; Urea 9½ grains per ounce; no casts or blood.

Treatment. The perineum was thoroughly cleansed with Perchloride solution, and the sloughs and remnants of ligatures removed. It was then swabbed with H_2O_2 Strychnine continued.

The temperature and pulse remained up until the perineum had become thoroughly cleansed, and the sloughs separated. The urine contains on an average Albumen ½ per thousand Esbach.

14th August 1908: The temperature fell to 99.4° F. today. The perineum is now clean, and has commenced to granulate. The patient looks very pale, and is very weak. after the days of high temperature.

Urine, 70 ozs.; straw coloured; acid; Specific Gravity 1020; Albumen, trace; Urea 9¼ grains per ounce.

Treatment. Swabbing perineum with hydrogen peroxide. Strychnine. Milk diet. Ferri et Ammon. Citrate 15 grains were given three times daily.

15th August 1908: The temperature is normal today, and the patient seems better and stronger.

The urine contains no Albumen, and the Urea is 8¾ grains per ounce.

Convalescence was now uninterrupted. Albumen was

never again present in the urine. The execretion of Urea remained normal. Light diet was given on the 19th August. The raw surface on the perineum healed, but the loss of tissue still persisted.

She was dismissed, well, on the 4th September 1908.

C A S E XLI

DISEASE.

Malaria

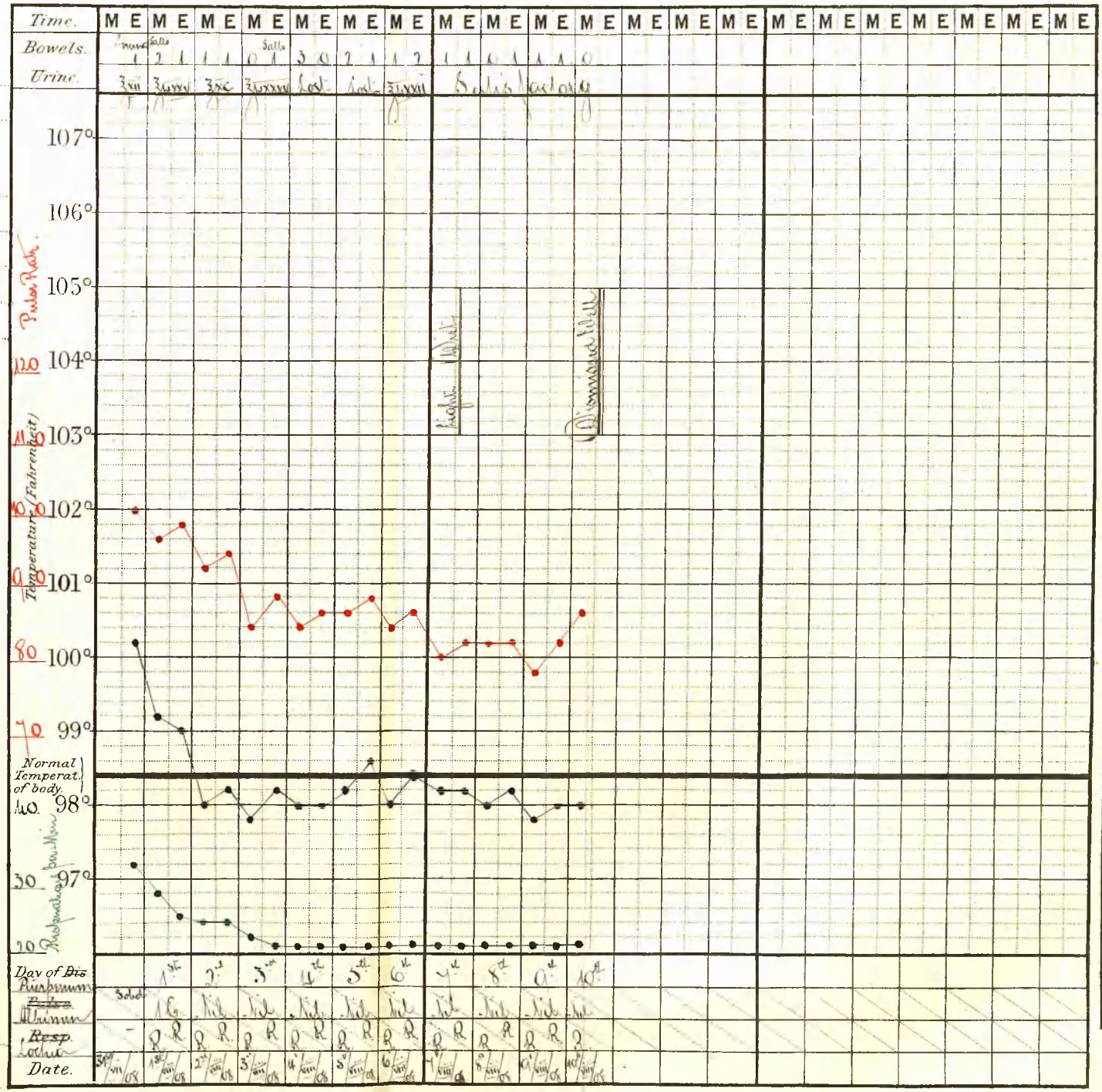
Notes of Case.

Mrs
Palmer
40 Years
Milk Trade
~~Book~~ N°

Tertian Para

Date of admission.
3rd July 1908

Recovery



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CASE XLI MRS PALMER, aet 40 years, XIX para. Full time. Recovery.

Admitted on 31st July 1908 at 9.15 p.m.

Delivered on 31st July 1908 at 11 p.m.

Child Female alive, 6¼ lbs. weight.

HISTORY: The patient has had sixteen living children, and two abortions. In all her pregnancies she had had swelling of the feet and ankles. Headache, epigastric pain, and defects of vision had never been complained of. During the present pregnancy she has had swelling of the lower extremities to a greater extent than formerly. Headache had been complained of on the day of admission, when it is said to have been very severe. There had been no abdominal pain, or disturbance of vision. About 7 p.m. she took a convulsion, and half an hour later a second.

PRESENT CONDITION: The patient is an exceedingly stout and powerfully built woman. She is maniacal, and it is with the greatest difficulty that she can be controlled. There is oedema of the lower extremities, but it is difficult to detect any swelling elsewhere. The size of the abdomen is that of a full time pregnancy. Strong uterine contractions are in progress. The foetal heart's sounds can be distinctly heard. Per vaginam, the os uteri is found to be fully dilated, and the head well down in the cavity of the pelvis. The membranes are ruptured. Temperature is 100.2° F. Pulse is 100 beats per minute, regular and of fair tension. Re-

spirations number 32 per minute, and are rather irregular, as she holds her breath while struggling. Shortly after admission to the Labour Ward she was delivered naturally of a live female child. There was a fair amount of post partum haemorrhage, the uterus being soft and failing to retract and contract after delivery of the placenta. Immediately after delivery she took a very severe eclamptic seizure, lasting eight and a half minutes. After the coma of this fit had passed over the patient was again restless.

Urine, 12 ozs.; amber; acid; Specific Gravity 1028; Albumen solid; tube casts; trace, blood.

Treatment. Chloral and Bromide a a 30 grains were given twice per rectum. These injections were returned. Magnes. Sulph. 2 ozs. was given. Milk Pot. Imp. After the eclamptic seizure 2 pints Saline transfused under right breast.

1st August 1908: The patient was very restless until 4 a.m. when she became quiet and slept for some hours. She awakened quiet, and, beyond a dazed feeling, sensible.

Urine, 75 ozs.; milky; acid; Specific Gravity 1015; Albumen one per thousand Esbach; no casts or blood.

Treatment. Milk and Pot. Imp. Chloral and Bromide a a 15 grains four hourly.

2nd August 1908: The patient is very well today. She is quite sensible. Examination of the retina was made, and no changes were found in them.

Urine, 90 ozs.; pale straw; Specific Gravity 1010; acid; no Albumen; blood and casts.

Treatment. Milk and Pot. Imp. Chloral and Bromide stopped.

Convalescence was uninterrupted, and she was dismissed, well, on August 10th 1908. The child was also well.

C A S E XLII

DISEASE. Eclampsia

Notes of Case.

Mrs Norland

28 Years

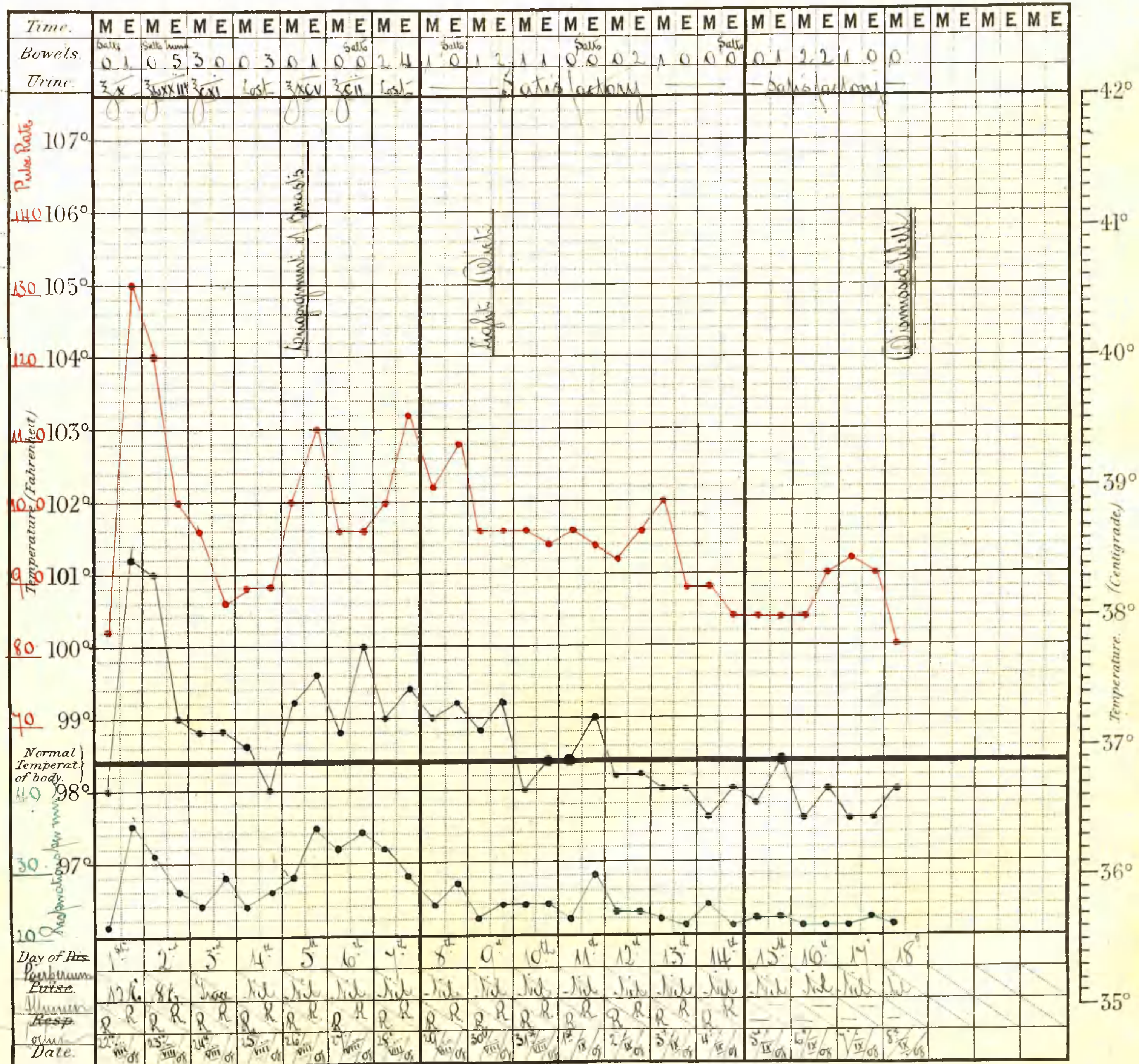
Milk + light

Book No XLVII

I Para

Date of admission. 22nd August 1908

Result Recovery



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CASE XLII MRS BORLAND. aet 28 years, primipara, twins. Full time. Recovery.

Admitted on 22nd August 1908 at 6.15 a.m.

Delivered on 22nd August 1908 at 7.15 a.m. and 7.25 a.m.

Presentation vertex; vertex (version).

Children Female/Female, dead/dead, 5½lbs./5½lbs. weight.

HISTORY: The pregnancy had been normal until three months ago, when the patient was six months pregnant, when she had noticed oedema of the feet and ankles. This swelling was at first slight, and on resting entirely disappeared. During the three weeks before admission the face and hands became swollen, and the swelling of the lower extremities became marked and permanent. There never had been any definite headache. She had had severe epigastric pain two days ago. This pain had gradually passed off. "Blurring of the eyes" while reading had been complained of, for a few days before admission. At 5 a.m. on the day of admission the patient had awakened in a dazed condition, and had taken a fit. This convulsion was followed by other two, and she was brought to Hospital.

PRESENT CONDITION: Immediately on admission she took a severe eclamptic seizure, lasting three and a half minutes. On recovering from this fit she was quite conscious, and able to converse intelligently. Temperature is normal. The pulse numbers 120 beats per minute, regular, and of moderate tension. Respirations are 28 per

per minute. Uterine contractions are taking place. The foetal heart's sounds are feeble, and can be heard with equal distinctness over the whole uterine area. This fact, combined with the very large size of the abdomen, raises the suspicion of it being a twin pregnancy. Two sets of foetal parts cannot be palpated. Per vaginam, the os uteri is found to be fully dilated, and the membranes unruptured. A foetal head presents in the first position. Under chloroform, the membranes were ruptured, and the child delivered with forceps. A second foetal head was discovered, podalic version was performed, and the child delivered. The perineum was slightly torn, and was immediately repaired.

After transfusion and delivery, at 11.5 a.m., 1.15 p.m., 2.30 p.m., 3 p.m., 3.10 p.m., 5.15 p.m., 6.25 p.m., 9 p.m., and 11.15 p.m. convulsions occurred. They were typically eclamptic in character, and the average duration was one and three quarter minutes. After each convulsion the coma became deeper, and more lasting, and after the last seizure the patient was completely unconscious. She was able to swallow liquids early in the day, but on becoming unconscious, this did not continue.

Urine. Quantity 10 ozs.; acid; dark muddy; Specific Gravity 1028; Albumen 12 per thousand Esbach; few Granular tube casts; trace of blood.

Treatment. Magnes. Sulph. 2 ozs. After the seizure on admission bled 1 pint, 2 pints Saline solution transfused into the median basilic vein. At 2.30 p.m. a hot wet pack was given. Chloral Hydrate and Potass.

Bromide a a 25 grains were given per rectum at 11.30 a.m., 3.20 p.m., 6.30 p.m., and 11.30 p.m. Milk. Potus Imperialis.

23rd August 1908: At 1.30 a.m. the patient had a very slight convulsion. This was the last to occur. After the fit, patient slept for a considerable time. Later in the day, consciousness returned, but the mental condition was not quite normal. She perspired freely during the day.

Urine, 73 ozs.; milky, acid; Specific Gravity 1022; Albumen 8 per thousand Esbach; granular tube casts; no blood.

Treatment. Milk and Potus. Imperialis. Chloral and Bromide discontinued.

24th August 1908: The patient passed a good night, having slept for seven hours. She is quite conscious today. The mental condition has also returned to normal.

Urine, 111 ozs.; clear pale straw; acid; Specific Gravity 1015; Albumen ½ per thousand Esbach; no casts or blood.

25th August 1908: Improvement continues.

Urine contains trace of Albumen.

26th August 1908: Albumen has disappeared from the urine, the quantity of which is large. Engorgement of Breasts occurred.

Improvement continued, and she was dismissed on September 8th, 1908, well.

C A S E XLIII

CASE XLIII MRS GALLACHER, aet 27 years, VI para. Full time. Death.

Admitted on 27th August 1908 at 2.15 p.m.

Delivered on 27th August 1908 at 3.15 p.m.

Presentation vertex.

Child male dead, 6¼ lbs. weight.

Died 27th August 1908, 5 p.m.

HISTORY: The first child was born alive at full time. The four succeeding children were born dead prematurely. No reason can be given for the premature deliveries, as no Doctor was in attendance. As far as can be ascertained, she had had no previous illness, nor had she suffered from any symptoms of Kidney involvement or toxaemia during the previous pregnancies. On August 21st 1908, after a day's washing, the patient had noticed that the feet were swollen, and that the face was puffy. She had also felt a severe pain in "the small of the back". She complained of these symptoms to a neighbour. These symptoms had continued, and had become progressively worse, until at 12 o'clock noon on the day of admission, when on awakening, she had taken a severe convulsion. This fit had been quickly followed by a second, from which she never recovered.

PRESENT CONDITION: The patient is completely unconscious. There is complete dilation and insensitiveness of the pupils. The face is cyanosed, and the tongue is lacerated. There is slight oedema of the lower extremities. The temperature is 99.4° F. Pulse is very rapid, un-

certain, and scarcely perceptible. Breathing is slow and sighing. There is much blood-stained froth at the mouth. Without, an anaesthetic Saline solution 2 pints were transfused into the right mammary region. Strychnine $\frac{1}{30}$ grain was given hypodermically. The patient did not react to this stimulation. As the membranes appeared at the vulva they were ruptured. The foetal head was found to be well down in the pelvic cavity, forceps were applied, and the child delivered. No anaesthetic was used, and the patient gave no indication of having felt the above manipulations, as she lay perfectly quiet and unconscious through them. A second hypodermic injection of Strychnine $\frac{1}{30}$ grain was now given, also Brandy 2 ozs. was given per rectum. The reaction to these stimulants was practically nil, and the patient died at 5 p.m., having been one and three quarter hours in Hospital.

Urine, 3ozs per catheter; neutral; Albumen very abundant; trace of blood. Tube casts were present.

C A S E XLIV

DISEASE.

Lumbago
Pneumonia

Notes of Case.

Mary
Stirker
20 Years

~~Book~~ N° XLIV

L Para

Date of admission.
31st August 1908

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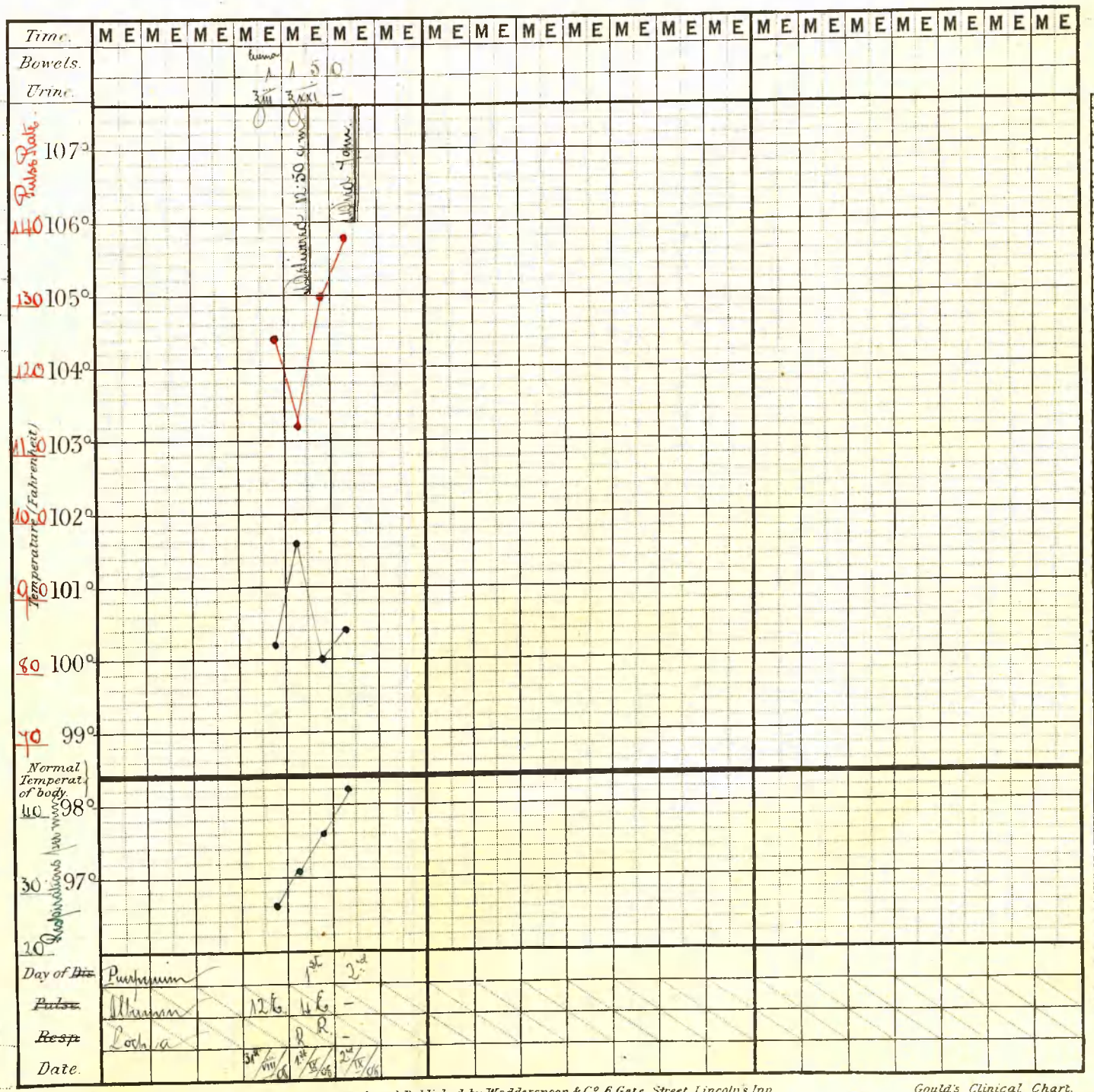

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Gould's Clinical Chart.

CASE XLIV MARY STRIKE, aet 20 years, primipara. Full time. Death.

Admitted on 31st August 1908 at 2.20 p.m.

Delivered on 1st September 1908 at 12.50 a.m.

Presentation vertex (Right occipito-anterior position).

Child Male dead, 6¼ lbs. weight.

Died 2nd September 1908, at 7 a.m.

HISTORY: During the fortnight immediately prior to admission the patient had been treated for Albuminuria of about one month's duration. When seen for the first time by her Doctor she had complained of headache, and had great oedema of the face and lower extremities. At this time also there had been a great Albuminuria. Under treatment these symptoms had subsided somewhat, and as labour was impending, she was sent to Hospital to be confined. There had been no previous illnesses, as far as can be made out.

PRESENT CONDITION: Almost immediately after admission the patient took a convulsion, or rather a series of convulsions lasting thirty minutes. After these fits she was very ill indeed, the pulse being 160 beats per minute, small, irregular, and almost imperceptible. At the bases of the lungs behind there were a few fine crepitant rales. No other physical sign could be discovered. Abdominal examination showed that the uterus was equal in size to that of a full time pregnancy. There are slight uterine contractions in progress. No signs of foetal life can be discovered. The head of the child

presents. Per vaginam, the os uteri admits one finger. The cervix is fully taken up. The foetal head presents in the Right occipito-anterior position.

Later. The uterine contractions did not increase in force until the evening. The os was almost fully dilated, and the membranes ruptured at 9 p.m.

Urine (Catheter specimen) 3 ozs.; acid; dark; Albumen 12 per thousand Esbach; Urea 4 grains per oz.; trace, blood; granular tube casts.

Treatment. When the seizure commenced Chloral Hydrate and Potass. Bromide a a 20 grains were given per rectum. This was repeated in three quarters of an hour. Saline solution 2 pints were transfused into the median basilic vein. Chloroform was administered, and was successful in checking the convulsions, although the patient became very collapsed during the inhalation of the drug. At the time of collapse, Brandy 3 ozs. was given per rectum. Strychnine $\frac{1}{30}$ grain was given subcutaneously, and $\frac{1}{60}$ grain was given one hour later.

Later. As the cardiac condition of the patient began to improve she was placed in a hot wet pack for a short period, with an excellent result. Two ozs. Magnes Sulph were given per nasal tube.

1st September 1908: At 12.50 a.m. delivery occurred easily and naturally. The pulse or the general condition of the patient was not much disturbed by this occurrence. The patient is very restless, and semi-conscious. Percussion at the bases of the lungs behind gives a slightly

duller note, and there is more resistance. Auscultation shows that the rales are more abundant over this area. Respiration is unduly prolonged, and there is a tendency towards tubularity. Over the other areas of the lung, moist rales are to be heard. Cough is present, and expectoration is scanty and of a muco-purulent character.

Later. The pulse is rapid and feeble. The right lung gives a dull note to percussion to the level of the fourth dorsal spine behind. This dulness is carried round into the axillary region. Tubularity is well marked over this region. The left lung still has the impaired percussion note at the base. There are signs of pulmonary oedema elsewhere. Diarrhoea set in, and the motions were passed involuntarily.

Urine (Catheter specimen) 31 ozs.; milky; acid; Specific Gravity 1028; Albumen 4 per thousand Esbach; Urea 6½ grains per ounce; granular tube casts; no blood.

Treatment. Stimulants;- Brandy 2 ozs. per rectum four hourly; Strychnine $\frac{1}{60}$ grain hypodermically four hourly. Jacket poultices to lungs.

2nd September 1908: The patient had a bad night, being restless and semi-conscious. The whole right lung solidified. She died at 7 a.m.

C A S E XLV

DISEASE.

Eclampsia

Notes of Case.

Ellen Sutton

24 Years.

Milk Light.

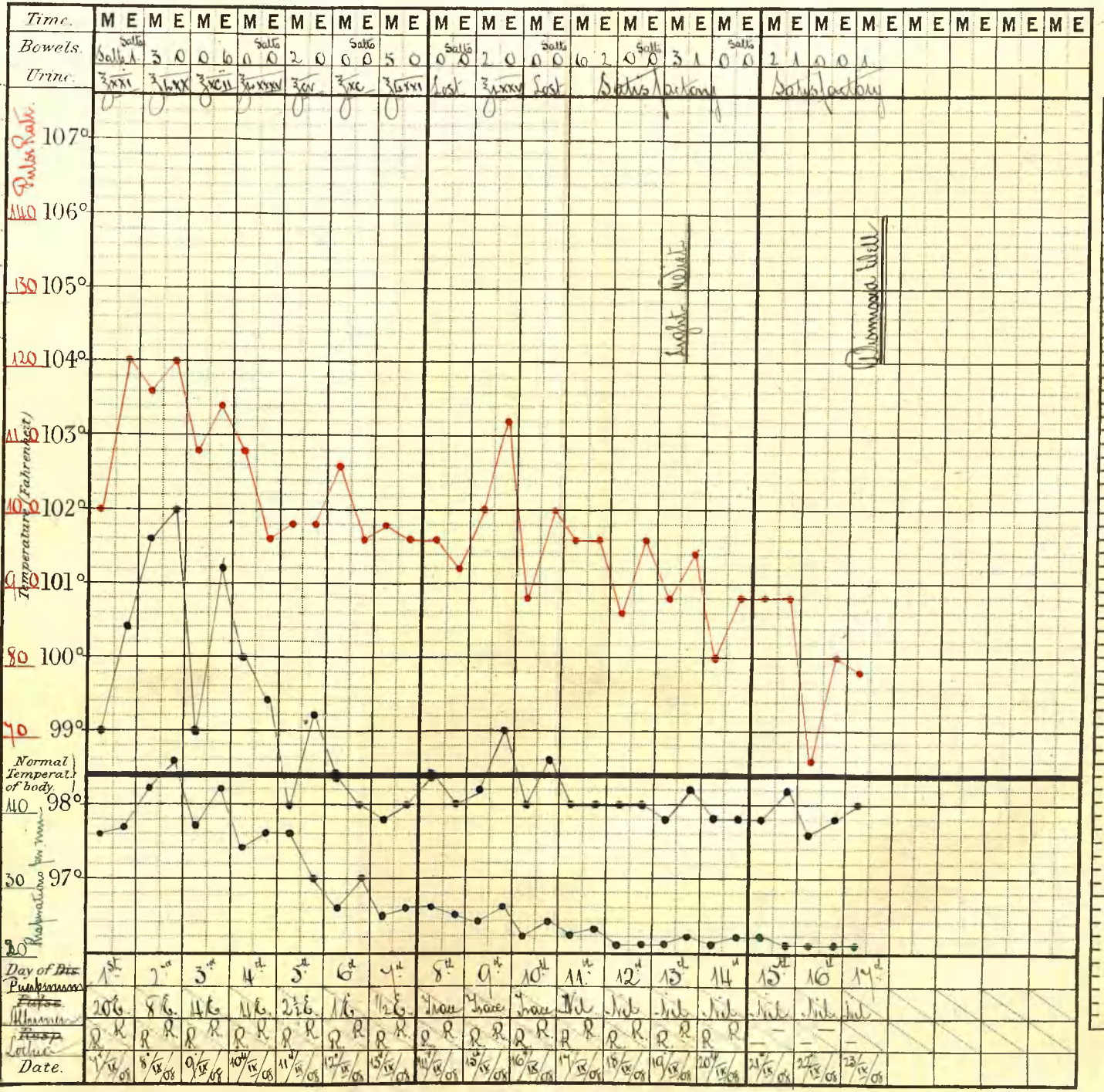
Book N° XLV

II Para

Date of admission

December 1908

Recovery



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Gould's Clinical Chart.

CASE XLV HELEN FULTON, aet 24 years, II para. 36 weeks.
Recovery.
Admitted on 7th September 1908 at 1.45 a.m.
Delivered, before admission, on 7th September. 1908.
Child Male dead.

HISTORY: On June 29th 1905 the patient had been admitted to the Glasgow Maternity Hospital with puerperal eclampsia. At that time she had had eight fits in all. There had been no symptoms of nephritis previous to that pregnancy, although she had headache and oedema during the later months of pregnancy. She was dismissed after the last attack without a trace of Albumin in the urine. Between the pregnancies, as far as can be ascertained, there had been no symptoms of Kidney disease. When seven months gone in the present pregnancy she began to suffer from oedema, and headache, which increased as the pregnancy advanced. On 6th September labour commenced. About 6 p.m. she was attended by two students who found the os partially dilated, and the foetal head well down in the pelvic cavity. About midnight she took an eclamptic seizure. Six further fits were taken in rapid succession. Labour had made but little progress. One of the outdoor physicians saw the case, and delivered her with forceps, under an anaesthetic. There seems to have been considerable post partum haemorrhage. The patient was then sent to Hospital.

PRESENT CONDITION: On admission, the patient is pale, and slightly cyanosed, and there is considerable oedema over

the whole body. It is most marked, however, under the lower eyelids, and in the lower extremities. She is comatose, and completely unconscious. The pupils are moderately contracted, and react slowly to stimuli. The tongue is lacerated. The uterus is well contracted and retracted. No dulness is made out on percussion of the lungs, but on auscultation over the lower lobes crepitant moist rales are to be heard. There is a slight cough, but no expectoration. Temperature is 99° F. Pulse is 100 beats per minute, regular, and of poor tension. Respirations number 36 per minute. While being bathed, the patient took a typical eclamptic seizure of two minutes duration, which was the last. She slept at intervals during the day, and during the periods of sleeplessness she was restless and semi-conscious. Fluids were swallowed in small quantities.

Urine (per Catheter) 21 ozs. in 14 hours; amber; acid; Specific Gravity 1030; Albumen 20 per thousand Esbach; Urea 5 grains per ounce; granular tube casts; trace, blood.

Treatment. Magnes. Sulph. 2 ozs. per tube. Transfused 2 pints Saline. Milk. Pot. Imp.

8th September 1908: The patient is semi-conscious today. She passed a good night, having slept for several hours. There have been no more convulsions. Oedema has disappeared. Cough is frequent, and there is a muco-purulent expectoration. The percussion of the bases of the lungs behind is slightly impaired, and moist crepitant rales

are heard all over the pulmonary area, especially at the bases. The temperature, pulse, and respirations are considerably raised.

Urine, 70 ozs.; milky; acid; Specific Gravity 1025; Albumen 8 per thousand Esbach; Urea 7 grains per ounce; tube casts; faint trace blood.

Treatment. Milk. Potus Imperialis. Jacket poultices. Strychnine $\frac{1}{60}$ grain four hourly.

9th September 1908: The patient is more conscious. The lung condition is much the same as yesterday, although perhaps the rale is not so abundant. Temperature, pulse and respirations are reduced.

Urine, 92 ozs.; pale straw coloured; acid; Specific Gravity 1022; Albumen 4 per thousand Esbach; Urea $8\frac{1}{2}$ grains per ounce; no casts; no blood.

10th September 1908: The patient is quite conscious today. The cough is less, and what there is of it is loose. The expectoration is free. The lungs are almost quite clear to percussion. A few rales are still present on auscultation.

Urine, 85 ozs.; amber; acid; Specific Gravity 1018; Albumen 4 per thousand Esbach; Urea 8 grains per ounce; no casts or blood.

Treatment as above.

11th September 1908: Improvement continues. Rale almost gone. Urine 105 ozs.; Albumen 2 per thousand Esbach; Urea $8\frac{3}{4}$ grains per ounce.

12th September 1908: Improvement continues. No rale can be detected. Urine 90 ozs.; Albumen 1 per thousand Esbach; Urea 8 grains per ounce.

14th September 1908: Improvement continues. Strychnine stopped. Urine: Albumen ½ per thousand Esbach; Urea 9 grains per ounce.

16th September 1908: Improvement continues. Urine: Albumen a trace; Urea 10½ grains per ounce.

18th September 1908: Improvement continues. Urine: Albumen none; Urea 10 grains per ounce.

The patient made an uninterrupted recovery, and was dismissed on the 23rd September 1908, well.

C A S E XLVI

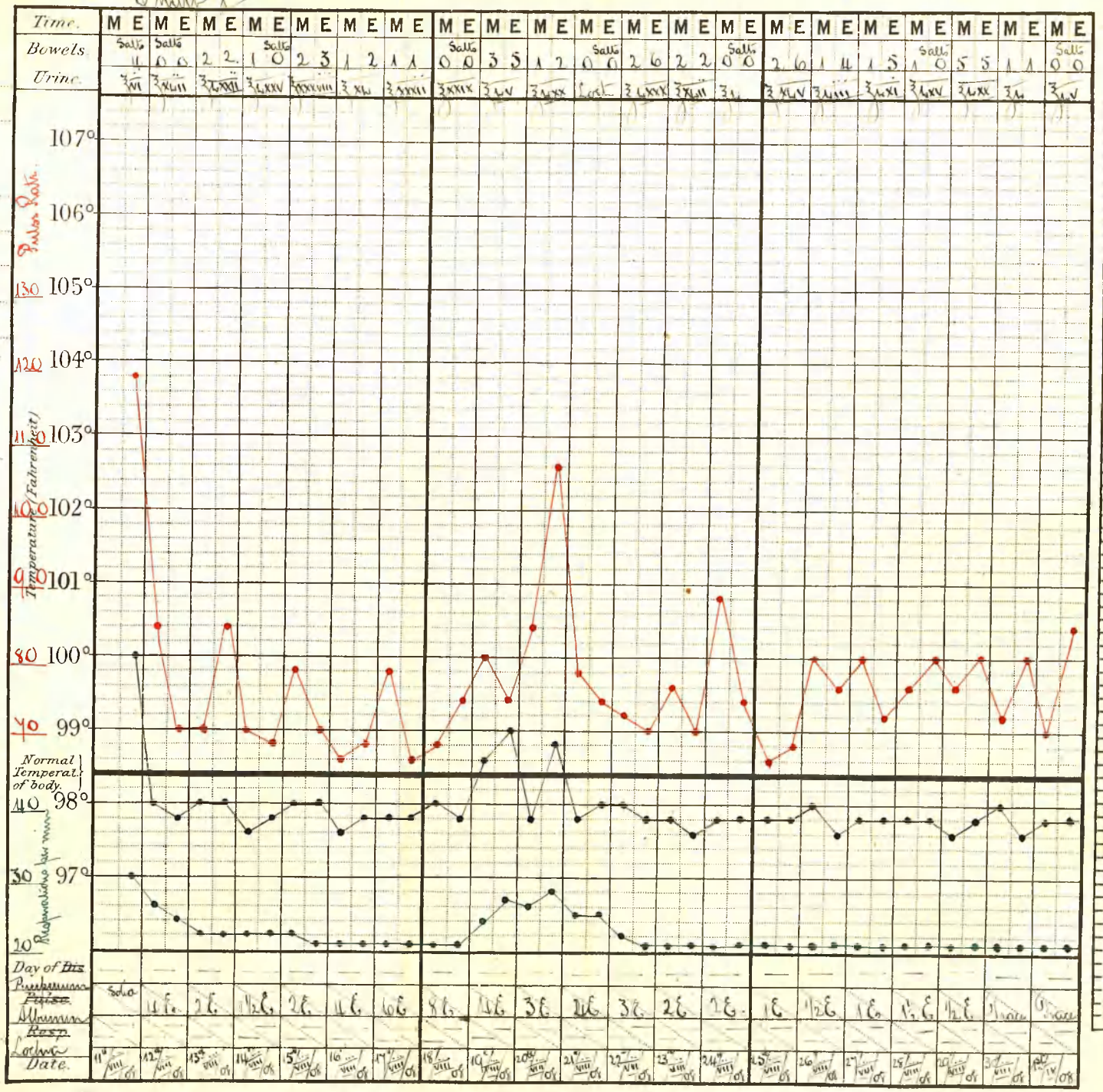
Chart I.

DISEASE.

Notes of Case.

Book No XLVI

Date of admission.



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Gould's Clinical Chart.

DISEASE.

Diarrhoea

Notes of Case.

Jessie Rodgers

22 Years.

Light - Milk

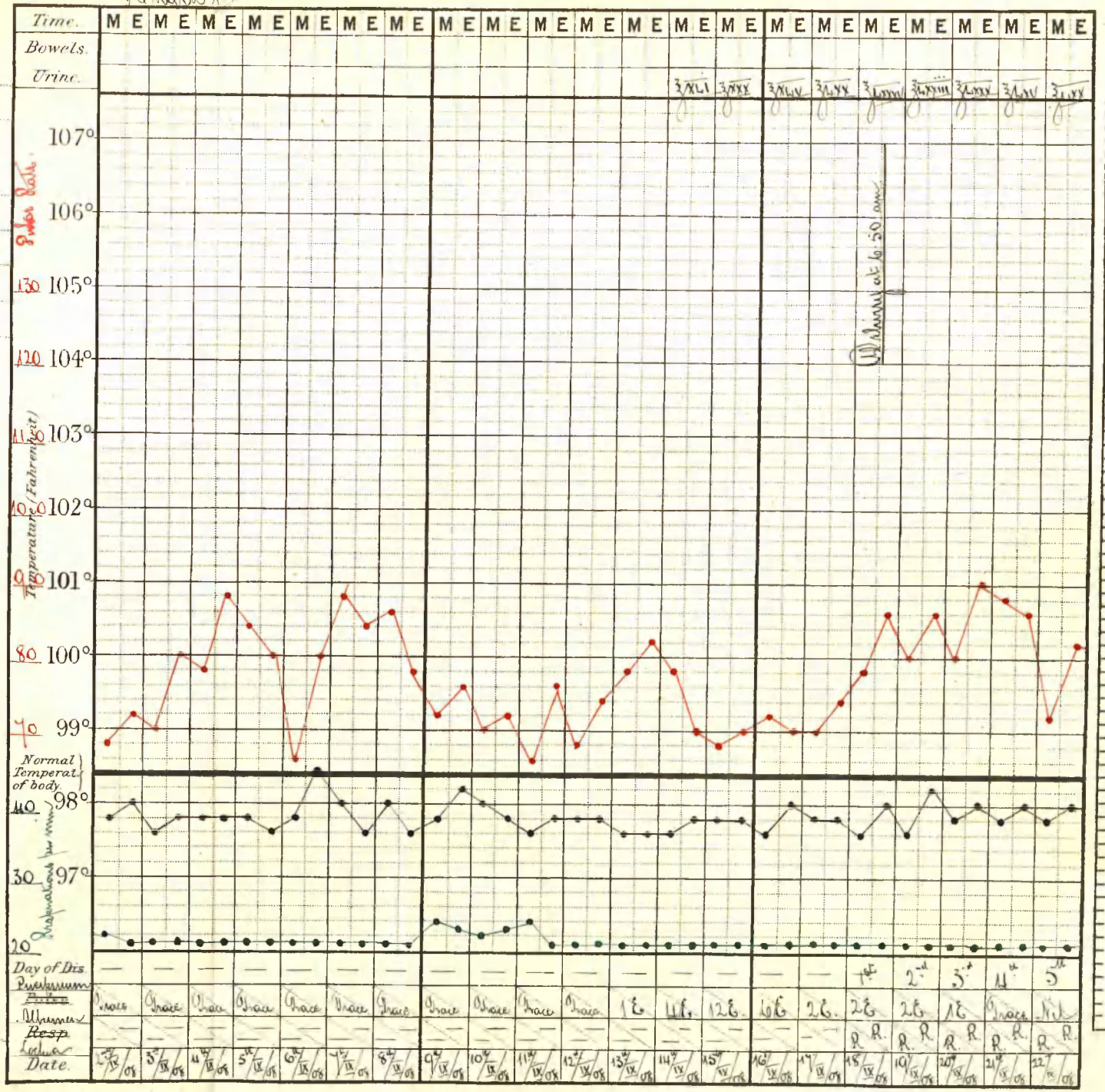
Book No XLVI

1 Para

of admission.

September 1908

Recovery.



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Gould's Clinical Chart.

Chart III.

DISEASE.

Pneumonia

Notes of Case.

Jessie Rodgers.

22 Years.

Milk - light

~~Chart~~ No. XLVII

L. Para

Time. M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E

Bowels. Salts 0 0 1 3 3 0 1 1 0

Urine. — Satisfactory —

Pulse Rate. 130 120 110 100 90 80 70 60 50 40 30 20

Temperature (Fahrenheit) 107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

Light Diet

Ordinary Diet

Normal Temperat. of body.

Respirations per Minute

Temperature. (Centigrade) 42° 41° 40° 39° 38° 37° 36° 35°

Day of Dis. 6th 7th 8th 9th 10th

Puerperium

~~Pulse~~ Nil Nil Nil Nil Nil

~~Resp.~~ R R R R R

Lochia

Date. 23/9/08 24/9/08 25/9/08 26/9/08 27/9/08

Date of admission. September 19 08

Pneumonia

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CASE XLVI JESSIE RODGERS, aet 22 years, primipara. 26 weeks. Recovery.

Admitted on the 11th September 1908 at 11.15 a.m.

Delivered on the 18th October 1908 at 6.50 a.m.

Child Male, premature, dead, 3 lbs. weight.

HISTORY: As the patient had been living alone, no history can be obtained as to the previous condition, or the number of convulsions taken. The Glasgow Maternity and Women's Hospital nurses had been sent for by neighbours. They had found the patient unconscious. She was sent to Hospital. When the patient recovered from the attack she could give no account of her previous health.

PRESENT CONDITION: The patient is very restless, and semi-conscious. The face is pale, and there are lacerations on the tongue. There is no oedema on any part of the body. The pupils are normal in size, and react to stimuli. The temperature is 100° F. Pulse is 118 beats per minute, regular, and of high tension. Respirations number 30 per minute. The fundus uteri appears one inch above the level of the umbilicus. Foetal heart's sounds and movements can be distinctly made out. Per vaginam, the os uteri is found to be closed, and the cervix is still elongated. Shortly after admission she took two very severe convulsions, of eight and six minutes duration respectively. Those were the last fits to be taken. For the remainder of the day she remained

in a state of stupor.

Urine (per Catheter) 6 ozs.; amber coloured; acid; Specific Gravity 1030; Albumen solid; Urea 3 grains to the ounce; granulated tube casts; trace, blood.

Treatment. Magnes. Sulph. 2 ozs. After the fits Chloral and Bromide a a 25 grains per rectum. Bled 20 ozs. Saline transfusion 2 pints. The patient was placed in a continuous hot bath for half an hour, and perspired freely after being taken out. (The pulse rose while in the bath, and the patient was removed.)

12th September 1908: Patient has recovered consciousness this morning, and beyond being dazed is intelligent.

Urine, 42 ozs.; milky; acid; Specific Gravity 1028; Albumen 4 per thousand Esbach; Urea 5 grains per ounce; tube casts scanty; faint trace blood.

Treatment. Milk. Potus Imperialis.

13th September 1908: Patient has now fully regained consciousness.

Urine, 72 ozs.; straw coloured; acid; Specific Gravity 1022; Albumen 2 per thousand Esbach; Urea 7 grains per ounce; no casts; no blood.

14th September 1908: Urine, 75 ozs.; straw coloured; acid; Specific Gravity 1022; Albumen 1½ per thousand Esbach; Urea 8 grains per ounce; no casts; no blood.

15th September 1908: Urine, 38 ozs.; pale amber; acid; Specific Gravity 1025; Albumen 2 per thousand Esbach; Urea 6 grains per ounce; no casts; blood.

16th September 1908: The patient complained of headache today. No oedema is to be found on any part of the body.

Urine, 40 ozs.; pale amber; acid; Specific Gravity 1028; Albumen 4 per thousand Esbach; Urea 5 grains per ounce; tube casts very scanty; no blood.

Treatment as above.

17th September 1908: The headache is still present, but not so severe as yesterday.

Urine, 32 ozs.; amber; acid; Specific Gravity 1028; Albumen 6 per thousand Esbach; Urea 4 grains per ounce; tube casts; no blood.

18th September 1908: The headache is very severe today. There is also considerable swelling of the lower eyelids. The pulse is of higher tension than it had been since the cessation of the convulsions.

Urine, 29 ozs.; dark; acid; Specific Gravity 1032; Albumen 8 per thousand Esbach; Urea 3 grains per ounce; tube casts; no blood.

Treatment. Brisk purge administered. A hot wet pack was also given. Milk diet. Abundant Imperial drink.

19th September 1908: The patient's symptoms are relieved. The headache and oedema are quite gone. The arterial tension is also reduced.

Urine, 55 ozs.; amber; acid; Specific Gravity 1022; Albumen 4 per thousand Esbach; Urea 6 grains per ounce; tube casts scanty; no blood.

20th September 1908: Improvement continues. Signs of foetal life still made out.

Urine, 70 ozs.; pale amber; acid; Specific Gravity 1018; Albumen 3 per thousand Esbach; Urea 7½ grains per ounce; no tube casts; no blood.

21st September 1908: Urine lost; amber; acid; Specific Gravity 1020; Albumen 4 per thousand Esbach; Urea 7 grains per ounce; no tube casts or blood.

22nd September 1908: Urine 80 ozs.; straw; acid; Specific Gravity 1015; Albumen 3 per thousand Esbach; Urea 7 grains per ounce; no tube casts or blood.

23rd September 1908: Urine 42 ozs.; pale straw; acid; Specific Gravity 1010; Albumen 2 per thousand Esbach; Urea 8 grains per ounce; no tube casts or blood.

24th September 1908: Urine, as yesterday.

On the 30th September the urine only contained a trace of Albumen, and the Urea excreted was normal, 9 grains per ounce. This condition remained present until the 13th October 1908, when the Albumen in the urine = 1 per thousand Esbach, and the Urea output was 8 grains to the ounce.

14th October 1908: The patient complains of slight headache today. No oedema can be discovered. The tension of the arteries tends to be high.

Urine, 41 ozs.; amber; acid; Specific Gravity 1025; Albumen 4 per thousand Esbach; Urea 7 grains per

ounce; no casts or blood.

15th October 1908: The headache has increased considerably. The oedema under the eyes has re-appeared. The patient complains of a pain in the epigastrium. The arterial tension is now high. The signs of foetal life are present, although they indicate some enfeeblement of the foetus.

Urine, 30 ozs.; amber; acid; Specific Gravity 1028; Albumen 12 per thousand Esbach; Urea 4 grains per ounce; tube casts present; no blood.

Treatment. Hot water pack. Later, 2 pints Saline transfusion under right breast. Milk. Imp. drinks.

16th October 1908: The patient's condition is much relieved. Under the treatment of yesterday the skin, kidneys and bowels acted freely. The high tension in the vessels is reduced. She remarked that she felt "no life" today, and on examination no foetal heart could be heard.

Urine, 45 ozs.; milky; acid; Specific Gravity 1025; Albumen 6 per thousand Esbach; Urea 6 grains per ounce; tube casts scanty; no blood.

17th October 1908: No symptoms of toxamia are present today. During the night labour commenced.

Urine, 70 ozs.; straw; acid; Specific Gravity 1022; Albumen 2 per thousand Esbach; Urea 8 grains per ounce; no casts or blood.

Treatment. Milk. Pot. Imp.

18th October 1908: The membranes ruptured at 4.30 a.m. and

the patient was delivered at 6.50 a.m.

Urine, 85 ozs.; pale straw; acid; Specific Gravity 1010; Albumen 2 per thousand Esbach; Urea 8 grains per ounce; no casts or blood.

19th October 1908: Urine, 73 ozs.; pale straw; acid; Specific Gravity 1015; Albumen 2 per thousand Esbach; Urea 7½ grains per ounce; no casts; no blood.

20th October 1908: Urine, 80 ozs.; Albumen 1 per thousand Esbach; Urea 8 grains per ounce.

21st October 1908: Urine, 65 ozs.; Albumen, trace; Urea 9 grains per ounce.

22nd October 1908: Urine, 70 ozs.; Albumen, nil; Urea 9 grains per ounce.

Convalescence was now uninterrupted, and the patient was dismissed on October 27th 1908, well.

The diet during the patient's whole stay in Hospital was entirely milk.

C A S E XLVII

CASE XLVII POLLY MEARNS, aet 23 years, primipara. Full time. Death.

Admitted on 14th September 1908 at 4.45 a.m.

Delivered on 14th September 1908 at 8.45 a.m.

Presentation vertex (left occipito-anterior position).

Child Male alive, 7 lbs. 10 ozs. weight.

HISTORY: This patient was admitted at the commencement of labour. There is no history of headache, oedema, or eye trouble. She remained well until the onset of the first convulsion at 6 a.m. Labour had been up to that point easy. She had six convulsions before delivery. At 8.30 a.m., as the os uteri was fully dilated, the membranes were ruptured. Under chloroform, forceps were were applied to the child's head, and delivery effected. She had fits at 11.15 a.m. (3 minutes); 11.30 a.m. (2 minutes); 12.10 p.m. (2 minutes); 1.10 p.m. (1½ minutes); 1.20 p.m. (2 minutes); 1.50 p.m. (5½ minutes). After this convulsion the temperature was 104° F., pulse 128 beats per minute, respirations 28 per minute. She was completely comatose, and remained so until the end. At 3.40 p.m. three convulsions occurred within ten minutes. At 5 p.m., temperature 105° F., pulse 136 beats per minute, respirations 32 per minute. Auscultation over the lung area, especially at the bases, reveals signs of oedema. The temperature gradually ran up to 106.2° F. During the night, in spite of active stimulation, the patient gradually became weaker, and died at 7.50 a.m., 15th September 1908.

Urine, 10 ozs.; amber; acid; Specific Gravity 1025: Albumen solid; Urea 3 grains per ounce; tube casts; trace, blood.

Treatment. Enema before onset of convulsions. After onset of fits, Magnes. Sulph. 2 ozs. Bled half a pint. Transfused Saline two pints. Chloral and Bromide a a 20 grains at 7 a.m., 11.30 a.m., 1 p.m., and 4 p.m. Cold sponged frequently when the temperature rose over 102° F. At 1.45 p.m., 3 p.m., 10 p.m. Strychnine $\frac{1}{30}$ grain. At 2.15 p.m. Saline 2 pints into right breast. At 4 p.m., 8 p.m. Brandy 2 ozs. per rectum. The first dose was returned. During the night Strychnine and Brandy were given alternately every two hours.

C A S E XLVIII

DISEASE.

dampsia
Post-Partum
Pneumonia

Notes of Case.

Mrs Lawler

æt. 22 Years

Male - Light

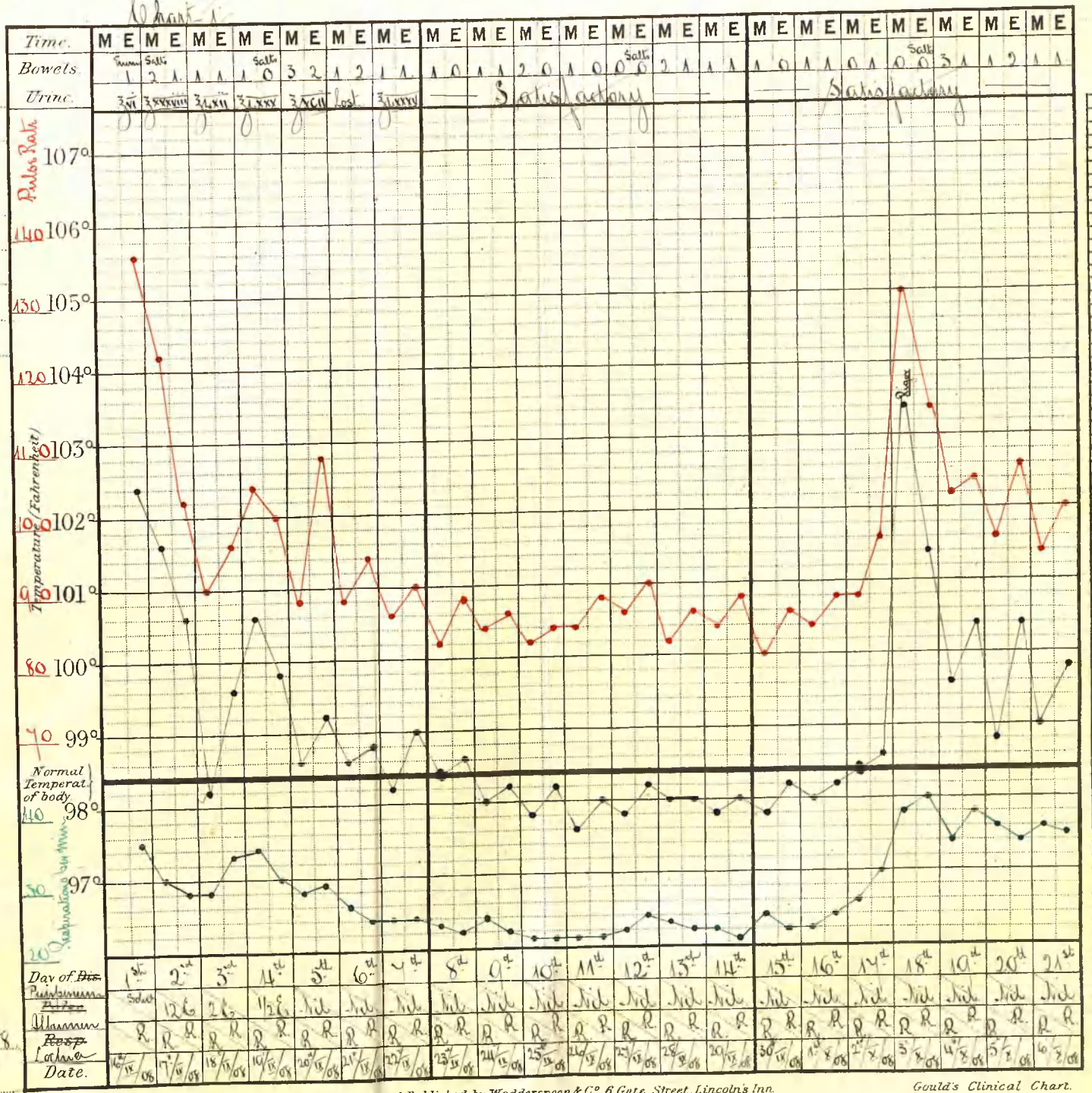
~~Book~~ No XLVIII

1 Para

Date of admission.

16th November 1908

ult Recovery



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Gould's Clinical Chart.

Chart II

DISEASE.

Glandular Post Partum Pyæmia

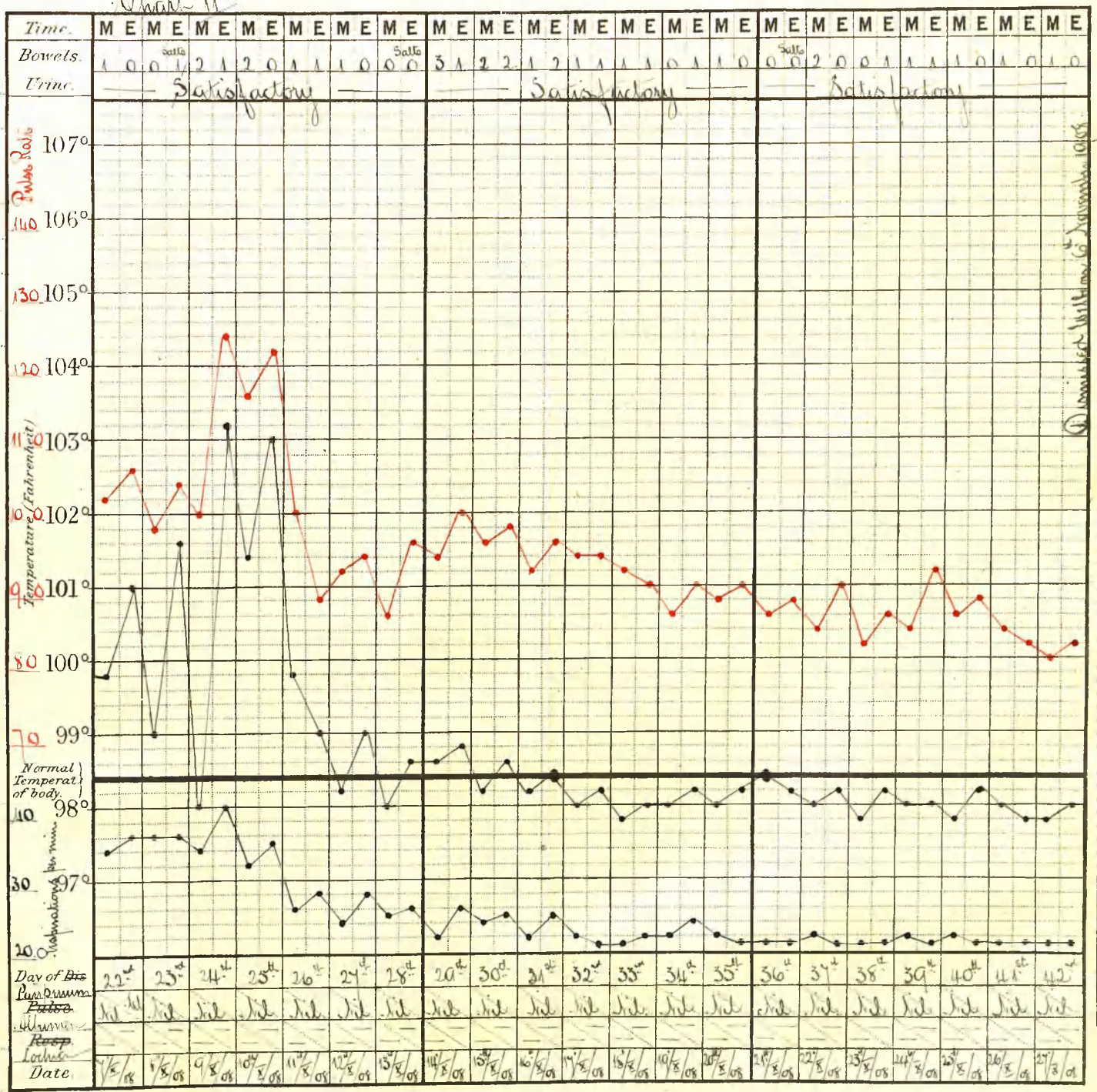
Notes of Case.

Mrs [illegible] Lawlor
Aet 22 Years
Milk - Light

Book No XLVIII

1 Para

Date of admission.
September 1908
Recovery



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CASE XLVIII MRS LAWLOR, aet 22 years, primipara. Full time. Recovery.

Post Partum Eclampsia.

Admitted on 16th September 1908 at 4 p.m.

Delivered, before admission, on 16th September 1908 at 9 a.m.

Child Female alive.

HISTORY: For four weeks previous to the delivery the patient had suffered from oedema of the lower extremities. The amount of swelling had been growing progressively greater. Headache had been present for three weeks before the onset of the convulsions. There had been no epigastric pain or eye symptoms. She was delivered of a live Female child at 9 a.m. on the day of admission. The delivery had been effected by forceps, the patient being under chloroform. The perineum had been lacerated during the operation. About one hour after delivery she had begun to take convulsions, and had taken eleven before admission.

PRESENT CONDITION: The patient is very restless, and is in a semi-conscious condition. There is slight cyanosis. The tongue is lacerated. The temperature is 104° F. The pulse numbers 136 beats per minute, and is regular, and of high tension. Respirations are 35 per minute. The perineum is ruptured almost to the rectum. The patient is very anaemic, the blood count being as follows:-

Red Blood Corpuscles	2,400,000
White Blood Corpuscles	5,000
Haemoglobin	55%

The uterus is at the level of the umbilicus, and is well retracted and contracted. At 4.15 p.m. she took a severe eclamptic fit, of eight minutes duration. At 6 p.m. and 6.35 p.m. she took convulsions lasting 2 and 3 minutes respectively. At 8 p.m., 9.15 p.m., 10.20 p.m., 11.5 p.m., and midnight, convulsions occurred, and their average duration was 2½ minutes. After the earlier seizures she was extremely restless, and she could be made to swallow small quantities of milk. As the fits became more numerous, however, the intra paroxysmal period was spent in a practically comatose condition. Later in the evening, at the bases of the lungs behind, fine crepitant rales could be heard.

Urine (Catheter specimen) 6 ozs.; dark coloured; acid; Specific Gravity 1035; Albumen solid; Urea 5½ grains per ounce; trace, blood; epithelial tube casts abundant.

Treatment. Immediately on admission the perineum was thoroughly cleansed, trimmed, and repaired. At 5 p.m. the patient was bled from the median basilic vein to the extent of one pint. After phlebotomy she was transfused with two pints of Saline solution. At 6 p.m. Chloral Hydrate and Potassium Bromide a a 20 grains was given per rectum. At 6.30 p.m. a hot pack was given with good results. At 9.30 p.m. a second dose of Chloral and Bromide was given. At 11.30 p.m. a second Saline transfusion was given into the submammary tissue.

17th September 1908: There were convulsions at 12.50 a.m., 1.5 a.m. and 2.10 a.m. Their duration was 2½ minutes, 2 minutes, and 3 minutes respectively. After the last seizure the patient passed from the comatose state into a deep sleep, from which she awakened at 9 a.m., fairly conscious, but in a dazed condition. The temperature, pulse, and respirations are coming down. Towards evening the patient fully recovered consciousness, and could swallow, and talk sensibly.

Urine, 38 ozs.; milky; acid; Specific Gravity 1020; Albumen 12 per thousand Esbach; Urea 6 grains per ounce; blood, faint trace; tube casts scanty.

Treatment. At 1 a.m. a dose of Chloral and Bromide was given per rectum. Milk diet and Imperial drink.

18th September 1908: The patient is fully conscious today. The maximum temperature was 99.6° F. The bases of the lungs have quite cleared up.

Urine, 62 ozs.; amber coloured; acid; Specific Gravity 1018; Albumen 2 per thousand Esbach; Urea 8 grains per ounce; no blood; no tube casts.

Treatment. Milk diet. Imperial drink.

19th September 1908: The patient's condition is satisfactory. She passed a quiet night, and slept well. The perineum shows signs of breaking down.

Urine, 80 ozs.; pale straw coloured; acid; Specific Gravity 1012; Albumen ½ per thousand Esbach; Urea 9½ grains per ounce.

Treatment. Milk diet.

20th September 1908: The general condition of the patient shows continued improvement. The perineum has commenced to slough, and there is consequently a slight rise of temperature.

Urine, 92 ozs.; straw coloured; acid; Specific Gravity 1012; Albumen, nil; Urea 9 grains per ounce; no blood or tube casts.

Treatment, as above.

For the ensuing thirteen days the patient made good progress. The stitches in the perineum had given way, but granulation tissue had formed, and the wound appeared healthy. On an iron tonic, the red blood corpuscles had risen in number to 3,500,000, and the haemoglobin to 65%.

3rd October 1908: Today, at 6 a.m., the patient took a rigor, and the temperature rose to 103.4° F. She complained of a severe pain on the right side. Pain is aggravated on deep inspiration. On auscultation over the right lung in the mid axillary, at the level of the 9th rib pleural friction can be detected. The percussion note is materially altered. There is a short cough, but no expectoration. The urine contains no Albumen.

For the following three days this condition remained much the same.

7th October 1908: In addition to the pleural friction at the right base, the percussion note is impaired, and

on auscultation, respiration is prolonged. There are also to be heard numerous fine rales.

A definite broncho-pneumonic patch developed in this situation, and remained so until the 20th October 1908.

The friction, tubular breathing, and other signs of consolidation gradually disappearing, she was dismissed on 6th November 1908, well.

C A S E XLIX

DISEASE.

Dambsie

Notes of Case.

No.

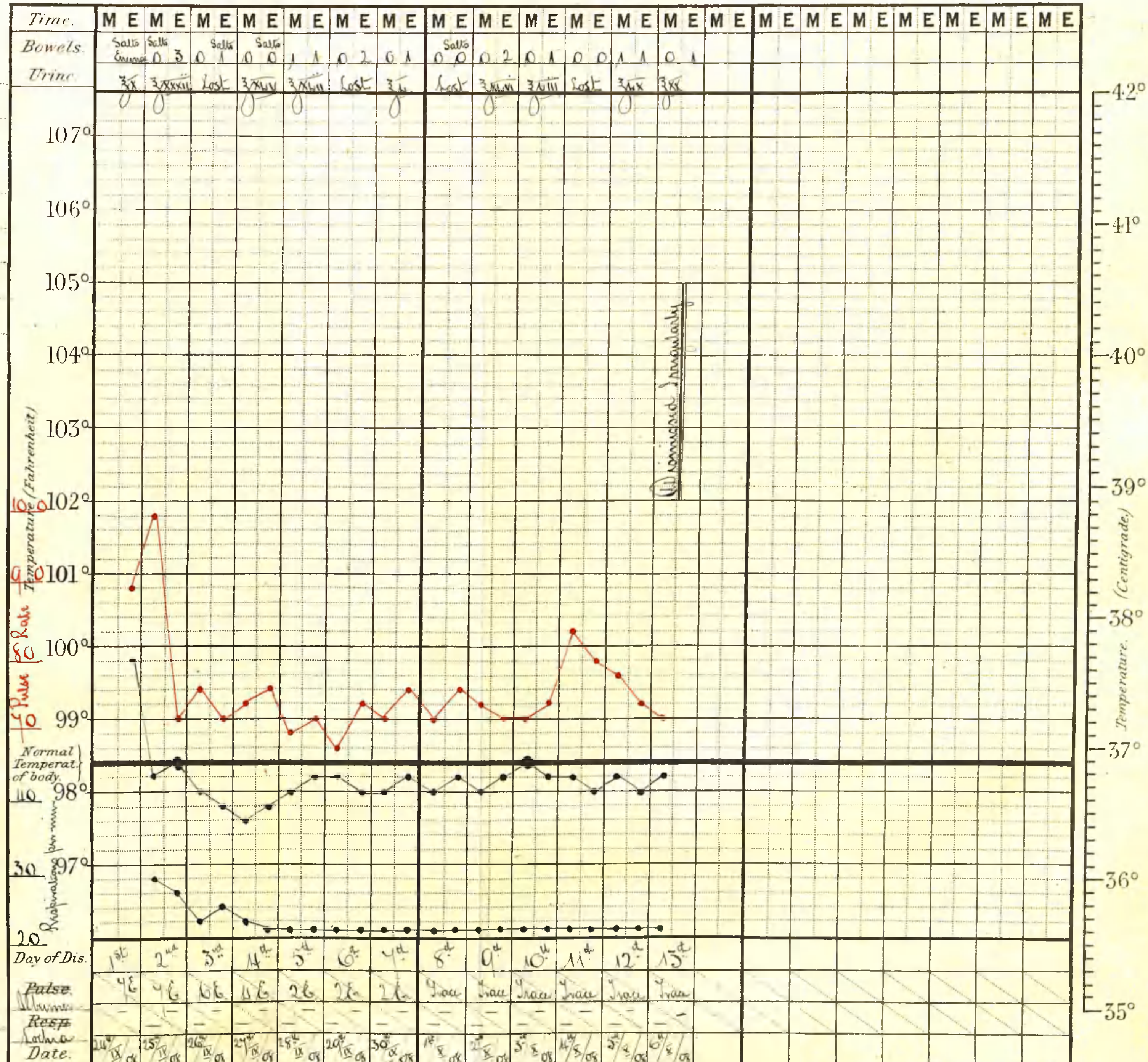
... Mills

Aet. 20 Years.

Wife

~~Book~~ No. XLIX

1 Para



Date of admission.

24th September 1908

Result Improved

CASE XLIX MRS MILLS, aet 20 years, primipara. Improved.
Admitted on 24th September 1908 at 3 p.m.
Dismissed irregularly, undelivered, on 6th October 1908.

HISTORY: There is a history of a fall from a step-ladder eight weeks ago. Since then she had complained of pain in the left iliac region. Epigastric pain had been present, but it is uncertain whether this was a premonitory symptom of Eclampsia, as it always had become worse after food. Vomiting, and on one occasion haematemesis had occurred. There seems, however, to be no doubt that the pain had been much more severe on the day before admission. Frontal headache had been present for three days previous to admission. Oedema of the lower extremities had been noticed for two days before the onset of the convulsions. On the morning of the day of admission the patient had taken two convulsions.

PRESENT CONDITION: The patient is quite conscious, although her memory is defective. The temperature is 99° F. Pulse numbers 88 beats per minute. It is hard, and of high tension. Respirations are 28 per minute. There is considerable oedema of the lower extremities. A few crepitant rales can be heard at the bases of the lungs. She is 34 weeks pregnant, and the uterus is of a corresponding size. The os is closed, and the cervix is not taken up. Uterine contractions cannot be detected. The foetal heart's sounds and movements can be readily detected. At 4 p.m. and 6 p.m. she vomited,

and the vomited matter consisting of dark "hare soup" material, and a quantity of red blood. Two hours after admission she had a slight eclamptic seizure of 40 (forty) minutes duration.

Urine, 15 ozs.; amber; acid; Specific Gravity 1025; Albumen 7 per thousand Esbach; Urea 7 grains per ounce; trace, blood; granular tube casts.

Treatment. Magnes. Sulph. 2 ozs. A hot pack was given at 5.30 p.m., with good results. Milk. Pot. Imp.

25th September 1908: The patient is conscious. There have been no further convulsions. The skin, kidneys and bowels are acting satisfactorily.

Urine, 32 ozs.; amber; acid; Specific Gravity 1028; Albumen 7 per thousand Esbach; Urea 6½ grains per ounce.

Treatment, As above.

26th September 1908: The patient's condition is much about the same.

Urine, 35 ozs.; straw; Specific Gravity 1024; Albumen 6 per thousand Esbach; Urea 7 grains per ounce.

Treatment. As above.

27th September 1908: The patient continues to improve.

Urine, 50 ozs.; pale straw; Specific Gravity 1022; Albumen 4 per thousand Esbach; Urea 8 grains per ounce.

Treatment. As above.

28th September 1908: Urine lost. Albumen 2 per thousand Esbach; Urea 7½ grains per ounce.

29th September 1908: Urine (lost); Albumen 1 per thousand Esbach; Urea 8 grains per ounce.

30th September 1908: Urine (lost); Albumen 2 per thousand Esbach; Urea 7 grains per ounce.

1st October 1908: Urine 40 ozs.; Albumen 2 per thousand Esbach; Urea 7 grains per ounce.

2nd October 1908: Urine 31 ozs.; Albumen 1 per thousand Esbach; Urea 8 grains per ounce.

3rd October 1908: Urine 42 ozs.; Albumen - trace; Urea 8½ grains per ounce.

4th October 1908: Urine 51 ozs.; Albumen - trace; Urea 9 grains per ounce.

5th October 1908: Urine 55 ozs.; Albumen - trace; Urea 8 grains per ounce.

6th October 1908: Patient left irregularly, undelivered.

CASE L

DISEASE.

Eclampsia

Notes of Case.

Name { Mrs. Richmond

30 Years

Milk Sight

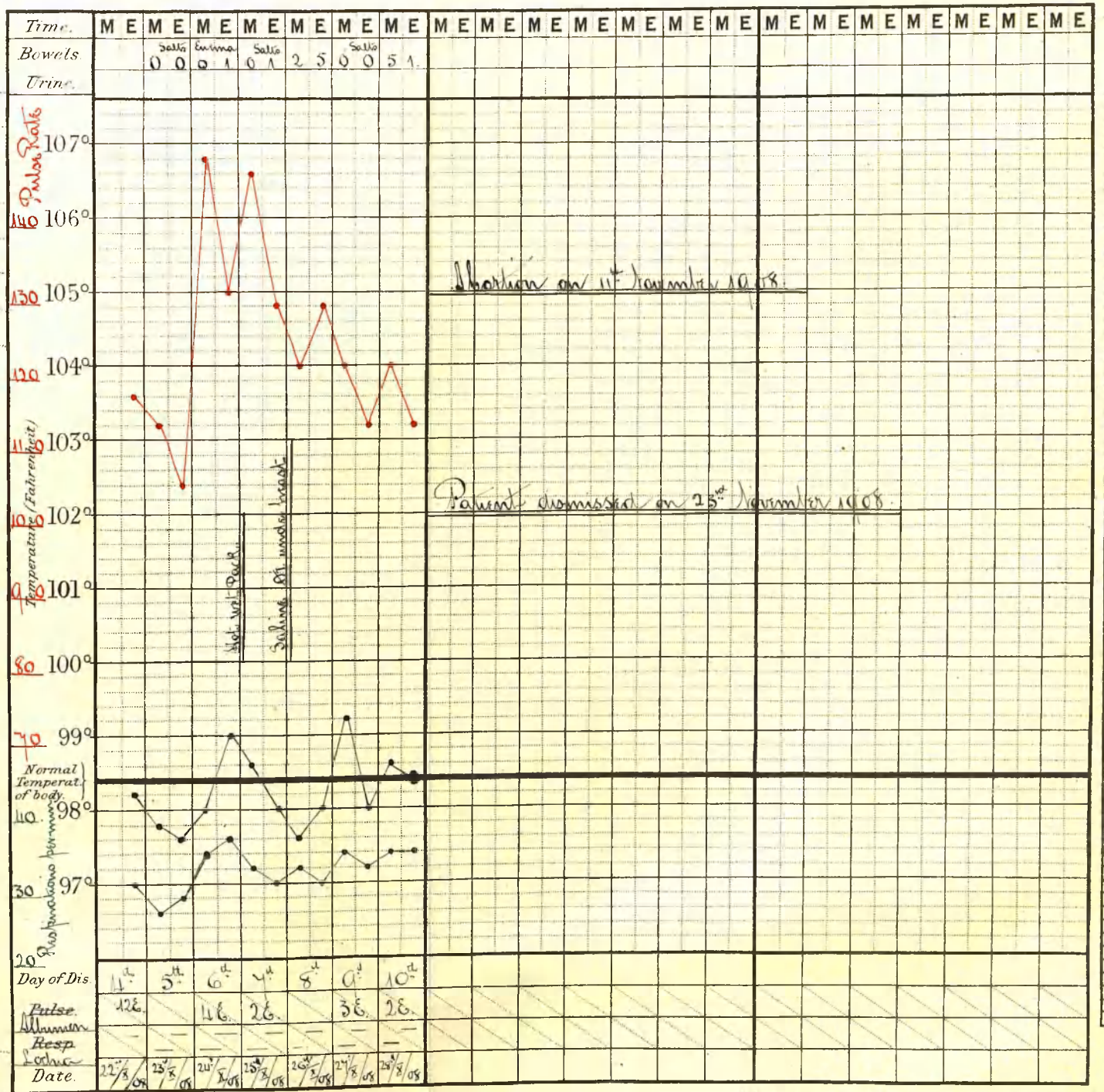
~~Book~~ N° 1.

III Para

Date of admission.

22nd October 1908

Result Recovery



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Gould's Clinical Chart.

CASE L MRS RICHMOND, aet 30 years, III para. 13 weeks pregnant. Recovery.

Admitted on 22nd October 1908.

Delivered prematurely on 11th November 1908.

HISTORY: After the two previous confinements the patient suffered from melancholia, for which she was treated on both occasions in Asylums. The mental condition of the patient at other times seems to have been erratic. Her husband states that his wife had been quite well until the 20th October 1908, when she complained of violent headache, and a shivering feeling. Later in the day a severe pain in the epigastrium was experienced, and the same night she had four eclamptic convulsions. No oedema had been noticed. Early on the 21st October 1908 she was removed to the Western Infirmary, Glasgow.

The notes received from that Institution are:-

"She was admitted semi-comatose, and could not answer "any questions. Any attempt at examination was vio- "lently resisted. Pulse 140 beats per minute. Res- "pirations 25 per minute. Temperature sub-normal. "The urine was withdrawn and found to contain Albumen "in large amount. Epithelial hyaline and granular "tube casts were present. The Bowels were moved by "an enema. One pint Saline solution was injected per "rectum. The patient was put in a hot pack.

"22nd October 1908: The patient is much better. She is "quite conscious, but somewhat maniacal, and strongly "resists any attempt at examination. She refused to

"take anything by the mouth until tonight, when she had "some milk."

She was then sent to the Maternity Hospital.

PRESENT CONDITION: The patient is semi-comatose, and when touched is very violent. The temperature is 98° F. Pulse is 120 beats per minute, irregular, and of poor tension. Respirations number 30 per minute.

Examination of the abdomen shows that the uterus extends about one and a half inches above the symphysis pubis. No internal examination could be made. There were no further convulsions.

Urine. The quantity is abundant, and could not be measured, as it was sometimes passed in bed; acid; Specific Gravity 1028; Albumen 12 parts per thousand per Esbach's tube; tube casts; no blood.

Treatment. Hot wet packs. Strychnine $\frac{1}{60}$ grain four hourly.

23rd October 1908: The patient is much quieter, and fairly sensible. There have been no further convulsions.

Urine. Quantity 20 ounces; acid; Specific Gravity 1022; Albumen 6 per thousand Esbach; tube casts; no blood.

The following notes are taken from the Hospital Journal:-

24th October 1908: Oedema of lungs. Albumen 4 per thousand Esbach.

25th October 1908: Oedema of lungs. Albumen 2 per thousand

Esbach. No casts.

26th October 1908: Patient shows symptoms of insanity.

27th October 1908: Insane. Albumen 3 per thousand Esbach.

28th October 1908: Insane. Albumen 2 per thousand Esbach.

30th October 1908: Insane. Albumen 1 per thousand Esbach.

2nd November 1908: Mental condition improving. Albumen ½ per thousand Esbach.

4th November 1908: Patient is sometimes quite rational; at other times insane. Albumen - trace.

6th November 1908: Patient generally more rational. Trional 15 grains at night.

9th November 1908: Improvement of mental condition. Albumen - trace.

11th November 1908: Patient aborted early this morning. Four months' foetus expelled. Albumen - trace.

14th November 1908: Since the 11th November the patient's condition has greatly improved. She is very quiet, and her mental condition seems quite good.

23rd November 1908: The patient has shown signs of steady improvement. She remains quite quiet, and is quite rational. Although there is still a faint trace of Albumen in the urine, she was dismissed today.