AN ANALYSIS OF

50 CASES OF ECLAMPSIA.

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AN ANALYSIS OF

50 CASES OF ECLAMPSIA.

In the field of obstetrics there is at present no subject occupying more attention than the treatment of Eclampsia, and there is no question calling more loudly for answer than the precise nature of its causation.

The differences of opinion amongst authorities on almost every point in connection with the disease demonstrate our want of exact knowledge more clearly than anything else. There are few diseases which more completely forbid generalisations of classes, and the laying down of definite rules in aetiology, prognosis and treatment.

While I was house surgeon in the Glasgow Maternity Hospital I had an opportunity of seeing eight cases of Eclampsia and of

helping to treat them. I have tried to report these cases as fully and carefully as possible, and especially any points which bear on the more recent ideas of treatment.

There is also tabulated a series of forty-two cases occurring in the Maternity Hospital during the three years 1900-1902.

I thought that the best way to bring out these points of interest most clearly would be to write a description of the disease and refer to these cases when they seemed to have a definite bearing on any of the questions raised. I had also three cases during the same time who were admitted apparently in the pre-eclamptic condition, but as I have accurate notes of only one, it is the only case included here. I think it is also interesting as showing the benefit that may be hoped for when this condition is early recognised and appropriate treatment instituted.

I am indebted for permission to embody these reports in my thesis to Dr. Robert Jardine and Dr. Munro Kerr, physicians to Glasgow Maternity Hospital.

C A S E I.

Nellie Swanson,

primipara, aet. 28.

HOUR CHART.

DISEASE.

Name { Nellie Swainson -

28 years -

Book No

Notes of Case

1 Para -

1 Fits previous to Admission at 11.30 p.m.

Date of admission 7th December 1902

Result Dismissed Well -

	AM			PM			AM			PM			AM			PM			AM			PM			AM			PM			AM			PM		
Time	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10
Bowels													2	0	1	1	1	1	2	1	1	1	0	0	1	3	3	1	1	2	0	1	1	3	1	1	0
Urine	Oz						40						60?																								
Albumen Esbach	Solid						Solid						7		3		5		3		3		3		3		1		Trace		Trace		Trace		Faint		
Day of Dis.	Puerperium												1		2		3		4		5		6		7		8		9		10		11		12		13
Pulse.			110				98		88				94	78	96	90	80	90	90	100	99	92	90	96	80	80	80	86	80	82	84	84	84	84	76	72	64
Resp.													R		R		R		R		R		R		R		R		R		R		R		R		R
Date.	Lochia		7th				8th						9th		10th		11th		12th		13th		14th		15th		16th		17th		18th		19th		20th		21st

Temperature (Fahrenheit): 107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

Normal Temperat of body

Temperature (Centigrade): 42° 41° 40° 39° 38° 37° 36° 35°

Intolerance of Uterus

Mag. Sulph. 3iii

Delivery at 1.15 p.m.

1 Fit at 3 p.m. & 1 Fit at 4.30 p.m.

Milk only & Potus Imperialis ad lib

Milk Diet

Light Diet

S. & C. Enema

Dismissed -

1 Fit on Admission at 11.30 p.m.

11.30 p.m.

1.30 p.m.

Entered at Stationers Hall

Printed and Published by Wodderspoon & Co 6, Gate Street, Lincolns Inn

Goulds Clinical Chart

Nellie Swanson, aet. 28, primipara, single, was admitted at 11.30 p.m. on 7th. Decr., 1902,to Glasgow Maternity Hospital.

She was seven months pregnant.

For the last fortnight she had had slight swelling of the face and legs but this was not constant, being sometimes quite away. During the four days before admission she had rather severe frontal headaches which were becoming more constant and painful. She occasionally had a feeling of nausea during this time but there was no vomiting or epigastric pain. She had not noticed that her urine was diminished in amount, but she had been very constipated for some weeks back.

In the early morning and during the afternoon of 7th. Decr., headache was very severe, and she felt so ill generally that she had to go to bed. She had no eye symptoms.

About 6 o'clock in the evening she took a convulsion, and before she recovered consciousness another followed in about half an hour. Between this and 8 o'clock the patient had two more fits, which, from her friends' statements, appear to have been general epileptiform seizures. She never recovered consciousness after the first, though on stimulation she could be partially roused since about 9 o'clock.

There was no history of antecedent renal disease or of epilepsy.

On admission the patient was unconscious, but on handling her she made apparently voluntary movements of the head and arms. The pupils were of medium size and responded to light naturally. There was slight oedema of the legs and more marked

swelling of the face and eyelids. The pulse was regular, about 110, and of moderately high tension. Auscultation of the heart and lungs revealed no abnormality save accentuation of the second sound at the aortic cartilage.

Her temperature was 100°. The tendon reflexes were normal. The abdomen was distended to about the size expected at the 7th. month of pregnancy; the uterus reached about three inches above the umbilicus. Uterine contractions were present and the head of the child was palpated at the fundus.

No foetal heart could be heard. Per vaginam the os admitted two fingers and the membranes were unruptured.

3oz. urine was withdrawn by catheter and turned almost solid on boiling. There was no blood present, and no tube casts could be found.

Diluted with an equal quantity of water the urine yielded albumin above the U mark on Esbach's albuminometer. Under the microscope large numbers of rod-shaped motile bacteria were seen, and the urine had the characteristic "watered silk" appearance generally present with bacteruria.

Shortly after admission she had a typical eclamptic seizure which lasted about two minutes from the commencement of the movements of the eyeballs till the finish of the clonic spasm. The temperature taken ten minutes afterwards had not risen, and the pulse was quite regular at 110 per minute.

Ophthalmoscopic examination at this time revealed nothing abnormal in the fundi.

At 12.40 a.m. (Decr. 8th.)dilatation with Bossi's dilator was commenced and was continued gradually until the index 9 on the instrument was reached; this occupied about half an

hour. The os was now about two-thirds dilated, and the legs were brought down, the aftercoming head perforated and extraction completed.

A sample of the child's urine collected as the breech was just born, contained a distinct quantity of albumen, but the amount of urine obtained was too small to allow of an accurate estimation of the percentage.

Post partum haemorrhage was rather encouraged, but the uterus contracted firmly, and very little bleeding occurred. There was a slight tear in the left anterior fornix which was not stitched. An intra-uterine douche of sterilised water was given.

About 2.30 a.m. (an hour and a quarter after delivery) the patient was still unconscious but was able to swallow, and 3oz. Epsom salts was given.

Her pulse was now about 98 per minute and less hard; the temperature was 99°. At 3 a.m. and again at 4.30 a.m. there were very slight fits, limited to some twitching of the face and arms, and neither lasting as long as a minute. As she was able to swallow she was given milk and imperial drink freely and she took a great deal of fluid by the mouth.

About mid-day (8th. Decr.) the patient began to recover consciousness and in the afternoon complained of headache. She could see quite well and answer questions intelligently by the evening. During the first twenty-four hours of her residence she passed two pints of urine, containing about 1.5 per cent of albumin; the bowels moved loosely twice.

9th. Decr.

Patient has been quite conscious since 6 p.m. yesterday

but has been dozing most of the time. Her headache is quite gone, and she feels very comfortable. The face is still somewhat oedematous but there is only a suspicion of swelling in the legs. The uterus is well retracted and the lochia are healthy. Pulse 80, quite soft and regular; temperature 98°.

She has taken a great deal of liquid (six and a half pints) and the bowels have been moving freely.

The urine could not be measured but was probably about three pints. A catheter specimen contained .7 per cent of albumin; motile bacteria were numerous.

10th. Decr.

Except for the discomfort of engorged breasts patient is feeling quite well. She maintains a copious diuresis, the albumin in a catheter specimen being .3 per cent. Bacteruria is still present. Pulse 90; temperature normal. The uterus and lochia are satisfactory. Face still a little puffy. She is taking milk and imperial drink well.

15th. Decr.

Progress has been perfectly satisfactory. All oedema is gone, and the patient is quite bright and intelligent. The uterus has involuted well, and the lochia are serous. A catheter specimen of the urine contains .3 per cent albumin and bacilli are present in undiminished quantities. She is having light diet and is to-day allowed to sit up.

Temperature normal; pulse 72.

22nd. Decr.

Convalescence has been most satisfactory. The patient

has now no symptom of her illness except a faint trace of albumin in the urine.

The lungs and heart are healthy and the uterus is well involuted. P.V. a slight tear is felt in the cervix.

During the puerperium the patient had 1oz. mag. sulph. every second day and for a week took copious quantities of imperial drink. The bacteruria persisted till dismissal. Plate cultures were made from the urine but nothing grew on incubation. Fundi are healthy.

The patient was dismissed to-day.

CASE I.

Urine. There was no history of previous renal disease, but a trace of albumin still persisted three weeks after delivery. The bacteruria may have been associated with this. There was a copious diuresis (two pints) in the first twenty-four hours although no saline was infused.

Prodromals. Premonitory symptoms were well marked although some of the most constant were absent in this case.

Convulsions. There were five fits ante partum, none being of the severest type; the coma always tended to disappear after the convulsions, and was never (except just after the seizures) very profound. From 8 p.m. till 11.45 p.m. there were no fits and the coma was lessening. The fit occurring at 11.45 p.m. succeeded the washing and examination of the patient which may have determined its onset.

There were two slight fits post partum, both greatly mitigated compared with the ante partum convulsions, and occurring two hours and three and a half hours respectively post partum.

This was rather a mild case as evidenced by the slightness of and tendency to recover from the coma, the number of the fits (7), and their comparatively slight intensity.

CASE 2.

Mrs. Greinert,

primipara, aet. 22.

Owing to the patient's inability to speak English, an accurate history could not be obtained in this case.

HOUR CHART.

DISEASE.

Name { Mrs Greiner

Age 22 years

Diet

Case Book No

Notes of Case

- Para -

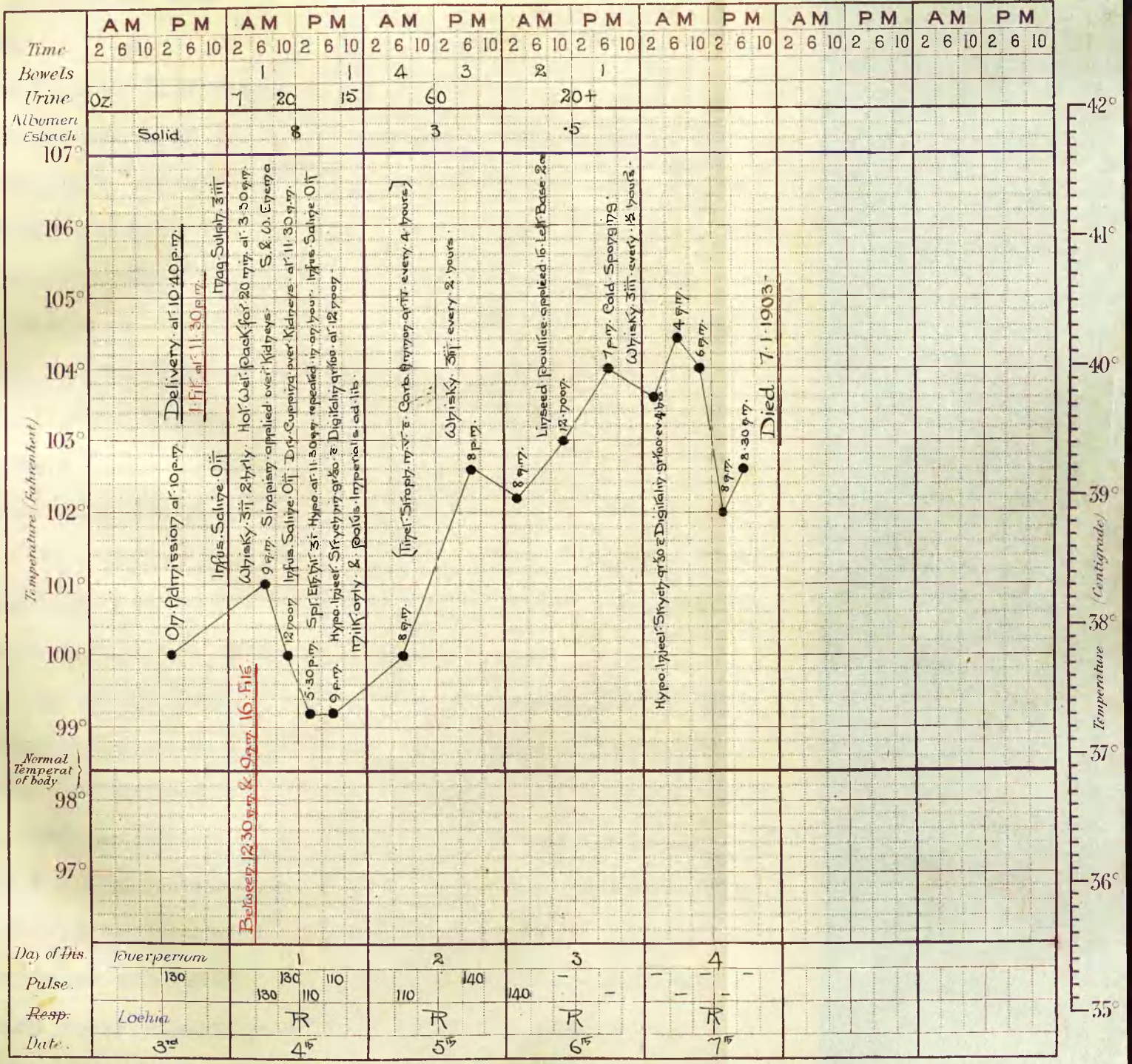
4 Fits previous 16.
Admission at 10 p.m.

Pneumonia

Date of admission

3rd January 1903

Result Died



Entered at Stationers Hall. *Printed and Published by Wodderspoon & Co 6, Gate Street Lincolns Inn.* *Gould's Clinical Chart.*

Mrs. Greinert, aet. 22, primipara, a Polish woman, was admitted to Glasgow Maternity Hospital at 10 p.m. on Jan'y 3rd., 1903.

She was about full time.
During the afternoon of 3rd. Jan'y, she was out walking with her husband, when she began to feel ill, went home and took to bed. She had two fits between 6 and 7 p.m., and was then seen by a doctor, who found her semi-conscious, rather cyanosed, and with foam at the mouth. At 8 p.m., while he was present, the patient had a severe general convulsion, eclamptic in nature; during her transit in the ambulance waggon she had a fourth seizure.

On admission the patient was semi-conscious.
The pupils were widely dilated and reacted sluggishly to light; she had had some herb treatment at home before the doctor saw her and this may have affected the pupils.

There was no oedema of the face or legs. The pulse was 130 per minute, regular, and not of high tension. The heart sounds were rather weak, but free from murmur. The temperature was 100°. Strong uterine contractions were present, the uterus being well retracted, and the head in the cavity of the pelvis.

The foetal heart sounds were distinct, of good quality, and best heart below and to the left of the umbilicus. P.V. the os was found fully dilated and the child presenting by the vertex. The catheter was passed and 4oz. of bloody urine withdrawn; it turned solid on boiling.

Microscopically blood cells and granular and blood casts were present in abundance.

Chloroform was administered and the child delivered easily by forceps at 10.40 p.m. 3oz. Epsom salts was given.

At 11.30 p.m. the patient had a severe eclamptic seizure lasting for about three minutes, and soon after she had a painful attack of retching and vomiting; the vomited matter was dark green in colour and measured 10 oz.

Two pints of saline solution was then infused into the lax abdominal wall. The temperature was now 101° having risen about a degree since delivery. At 3 a.m. whisky 2 dr. every two hours was commenced, and a soap and water enema was given with good result. A hot wet pack was then employed for twenty minutes, the skin acting well.

During the night the patient had in all sixteen fits of varying intensity (some of them lasting five or six minutes), was very restless, and had several attacks of painful retching and vomiting. The vomit was never of great quantity and never contained blood.

The last fit occurred at 9 a.m. 4th. Jan'y, the temperature having remained at 101° all night. The catheter was passed at 10 o'clock, and, as only 7 oz. urine had been secreted in about 12 hours, sinapisms were applied to the renal regions, and two pints of saline solution again infused. At 11.30 a.m. the patient was dry-cupped and poulticed over the loins. 1 dr. spirit.aether.nit. was given hypodermically and repeated in an hour.

The pulse was now (1 p.m.) very feeble, beating about 130 to the minute; the patient was somewhat cyanosed, and the temperature was 100°. One-thirtieth grain strychnine and one-fiftieth grain digitalin were injected hypodermically.

At 3 p.m. the catheter was again employed and nearly a pint of urine was withdrawn, this amount having been secreted in less than four hours.

At 5.30 p.m. she was again given two pints of saline solution into the abdominal wall.

By 12 midnight the patient had passed since admission about two pints of urine, more than thirty ounces of this amount having been secreted in the last twelve hours. The urine contained .8 per cent albumin. Microscopically a few granular casts and a comparatively small number of red blood corpuscles and leucocytes were found. The bowels had moved loosely twice.

She had had in all twenty convulsions, four previous to delivery and sixteen since, the last occurring at 9 a.m. - ten hours after the birth of the child.

She can now (midnight) be easily roused and seems to have a clear conception of where she is, though she is still very drowsy. The temperature is 99.2°, the pulse 110 and very small. The patient seems to have some epigastric pain. The tendon and eye reflexes are normal.

5th. Jan'y.

The patient passed a good night, sleeping quietly most of the time. This morning she was quite conscious and apparently free of pain. She is taking plenty of milk and imperial drink. The pulse is still very weak and compressible, about 110 per minute; the temperature is 100°. Her bowels have moved loosely four times and she is passing abundance of urine.

During the afternoon she carried on an animated conversa-

tion with her husband for about twenty minutes. He informed us that she was feeling well except for a slight headache.

About 9 p.m. the patient began to complain of pain in the left side of the chest. The temperature rose to 102.6°, and the pulse became irregular and more feeble, about 140 to the minute. On examination of the chest there was found moderate dulness and fine râle at the left base; the breathing was tubular.

During the second twenty-four hours in hospital (till 10 p.m. 5th. Jan'y), she passed probably about 3 pints of urine, though a great deal was lost. A catheter specimen yielded .3 per cent albumin; no blood or casts were to be found. The bowels moved in all seven times in the twenty-four hours.

The patient this evening was put on tincture of strophanthus m 5, and carbonate of ammonium gr. 4 every four hours, and the whisky increased to 3 dr. every two hours.

6th. Jan'y.

The patient passed a restless night and appeared to suffer a good deal from pain in her side. A hot poultice was twice applied to the left base.

This morning there is increase of dulness both in quality and extent on the left side, and the right base is also dull. There is marked tubularity at both bases. The pulse is steadier, 140 per minute, but is very feeble and small. Temperature 102.2°.

The patient appears more comfortable to-day, although she is distinctly more cyanosed and the face wears an anxious expression.

In the evening the temperature rose to 104° and only fell after repeated cold sponging. The pulse became uncountable. Strychnine and digitalin hypodermically were commenced and the whisky was increased to 3 dr. every hour and a half.

Diuresis is still good, the urine containing about half a part albumin per 1000 parts. The bowels moved thrice. Ophthalmoscopic examination to-day revealed no change in the retinae.

The patient is drinking peptonised milk and meat juice in good quantity.

7th. Jan'y.

The patient is in a very low state to-day. Cough and expectoration are painful and frequent. The pulse is running and cannot be steadied. The respirations are shallow and hurried, 44 per minute.

The temperature ranges from 102° to 104°, and cold sponging is frequently necessary to keep it at the lower limit. The patient is still quite conscious but evidently realises she is dying, as she talked with her husband this afternoon about the care of her child when she was gone.

The lungs were not examined to-day but the heart sounds have become very weak and irregular.

As far as the eclamptic condition is concerned the patient may be considered cured, but she is evidently moribund with pneumonia.

8th. Jan'y.

The patient died at 8.30 this morning.

REPORT OF POST MORTEM EXAMINATION.

9th. January, 1903.

The patient is a well-developed, healthy-looking woman. Rigor mortis is well marked; the abdomen is distended.

Thorax. Pericardial sac contained a considerable amount of straw-coloured fluid. The heart was small, the right ventricle very thin, dilated, and fatty; the left small and hypertrophied. Auricles normal. Tricuspid valve ring much dilated and the valve incompetent.

The right lung was partly adherent from an old pleurisy and showed marked consolidation of the middle and upper lobes. The left was also adherent and showed distinct engorgement with actual consolidation.

Abdomen. Liver, spleen, and pancreas were healthy. The intestinal canal throughout appeared normal. Left kidney congested and hyperaemic, almost haemorrhagic. The right kidney was little affected save for some diminution in the cortex. Uterus well contracted and quite healthy. Death was due to pneumonia and cardiac failure, subsequent to eclampsia.

The post mortem examination was made by Dr. Carstairs Douglas - pathologist to the Hospital.

C A S E 2.

No oedema was present in this case - a very severe one. Cyanosis appeared early and continued throughout.

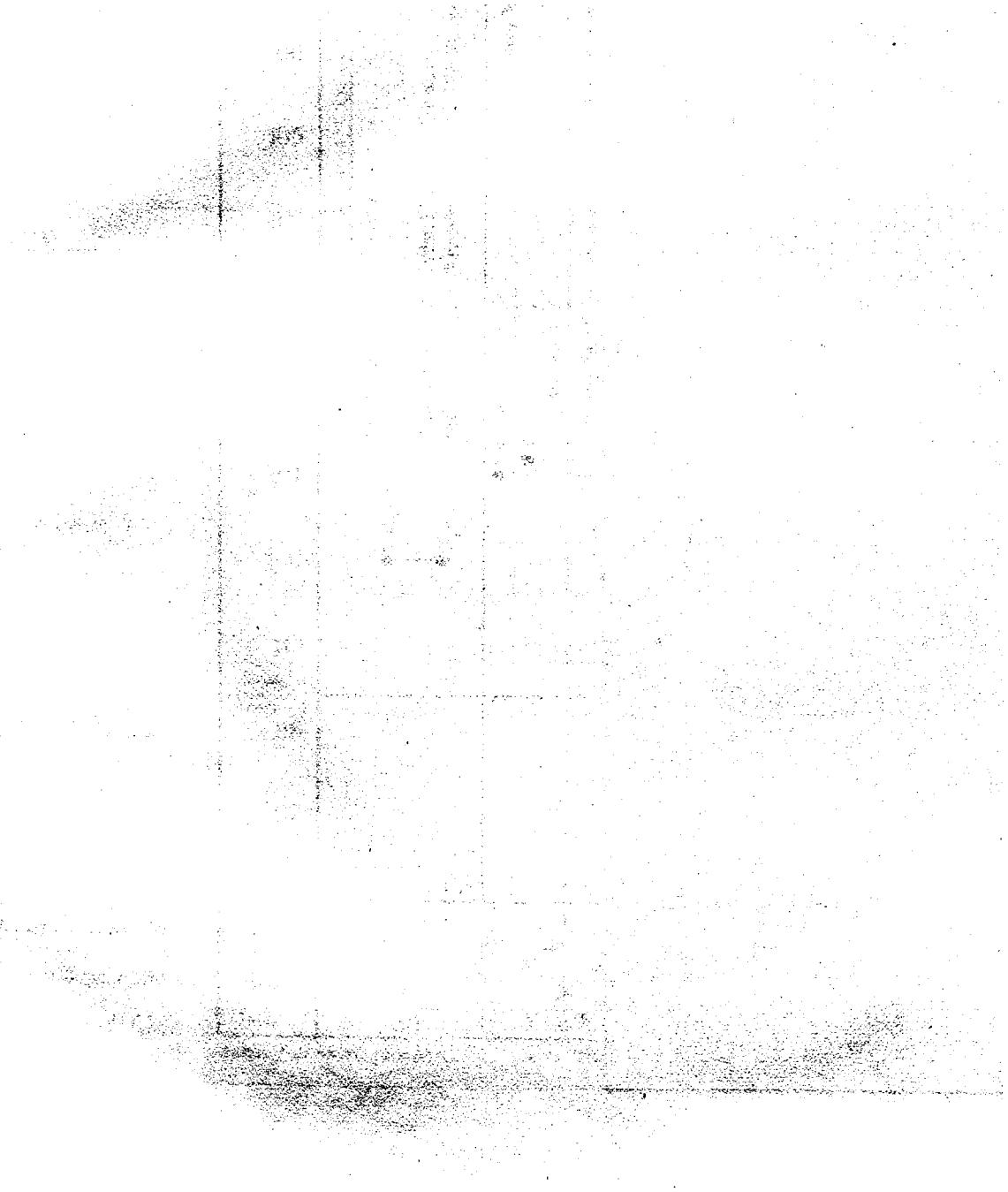
Urine. Rapid decrease in percentage of albumin after delivery. In the first twelve hours in hospital only 3 oz. urine was secreted, notwithstanding saline infusion on admission. In the second twelve hours, she passed about thirty-three ounces, nearly a pint of this in the five hours subsequent to the stimulation of the kidneys by poulticing and dry cupping.

Pneumonia. Forty-eight hours after delivery, thirty-six hours after the last fit, and after twenty-four hours of consciousness and intelligence, pneumonia supervened, this grave sequel commencing when the heart was already flagging from the eclampsia. The prognosis was of the gravest immediately this was diagnosed.

Retinae. Ophthalmoscopic examination was negative.

This was a severe case as evidenced by the number of the fits (20), their severity (an intense seizure), the depth of coma, the effect of the convulsions on the heart. The convulsions were not influenced by delivery in any favourable way, and it is doubtful if our treatment had any more beneficial in-

fluence than to promote a somewhat tardy diuresis, and to keep her heart beating a little longer by stimulation after the pneumonia supervened.



OUR CHART.

DISEASE.

Mrs Boyd.

42 years.

Book No.

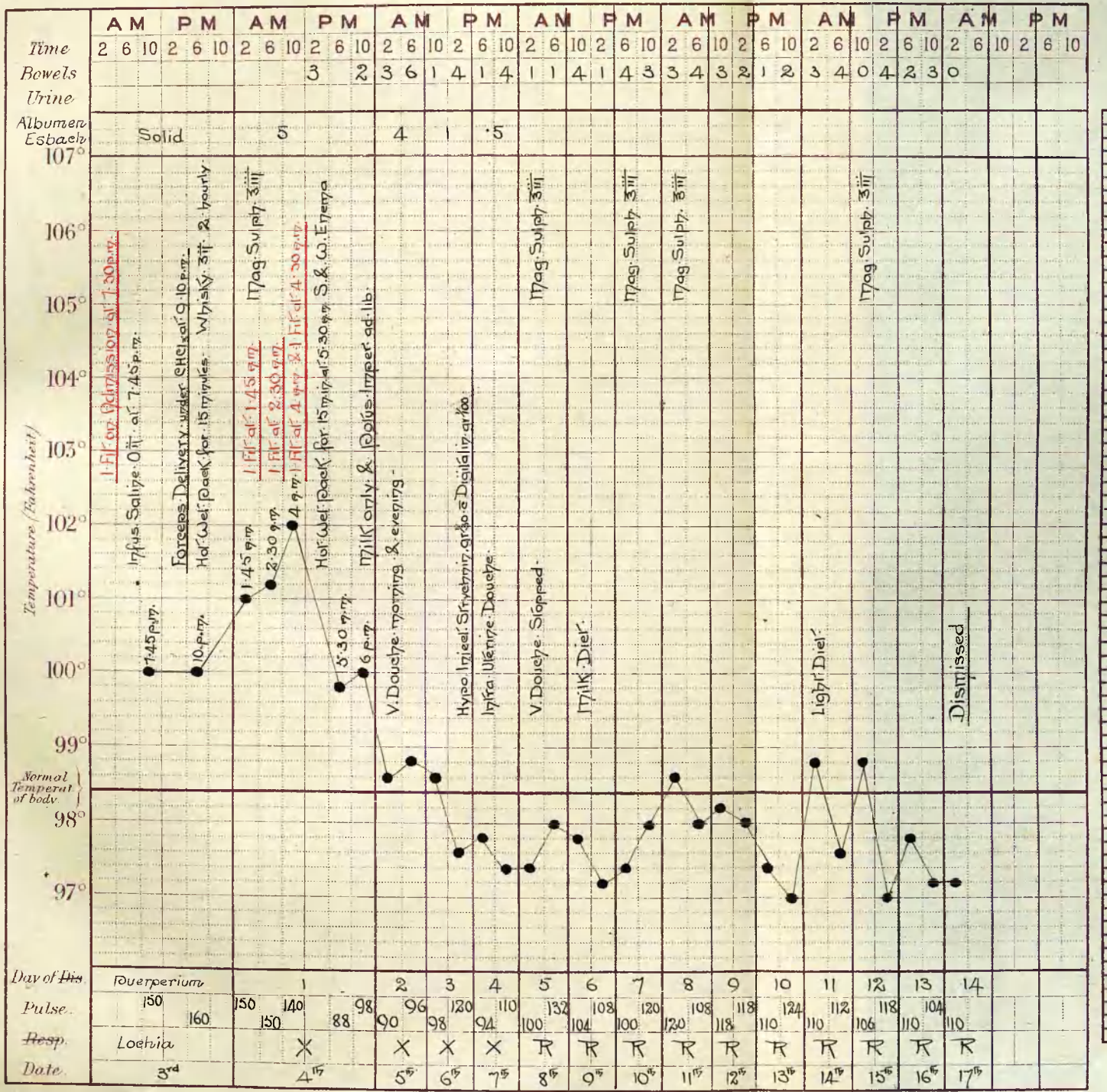
Notes of Case

VIII Para.

4 Fils previous to Admission at 7 30 p.m.

Date of admission 3rd January 1903

ult Dismissed Well.



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Gould's Clinical Chart.

C A S E 3.

Mrs. Boyd,

VIIIpara, aet. 42.

Mrs. Boyd, aet. 42, VIIIpara, was admitted to Glasgow Matemity Hospital at 7.30 p.m. on 3rd. Jan'y, 1903.

She was within a fortnight of full time.
All her previous labours had been natural, and her first two pregnancies uneventful, but twelve years ago, while five months pregnant with her third child, she began to have pain in the small of the back, and her legs, but not her face,became swollen.

There was no cough, headache, or vomiting and the swelling disappeared with the termination of her pregnancy. During her succeeding pregnancies the same symptoms always manifested themselves (oedema and lumbar pain) but always disappeared after delivery. She never had any headache, vomiting (except "morning sickness"), or eye symptoms. All the children were born alive at full time and were all large, healthy babies.

Her last pregnancy terminated two years ago; she had a healthy puerperium and suckled her child for sixteen months - until she missed a period. She had menstruated four times before conception occurred nine months ago.

Until midterm the present pregnancy was uneventful, except that the usual morning sickness was absent. When about four and a half months pregnant she did not feel so well as when carrying her former children. She was weaker, less able for her duties, and especially on certain days had feelings of great lassitude. Her legs and feet became much more swollen than formerly and she suffered from severe pain in the back and an excessive feeling of weight in the abdomen. For the last three months she has had an almost constant dry cough which interfered with her sleep. About two months ago, for the

first time in her life her eyesight began to fail and soon she was unable to sew or to read small type.

During the last month all her symptoms became greatly aggravated, especially the cough which became so constant and painful that she was "afraid to go to bed." She began to have attacks of giddiness and, to a great extent, lost her appetite. Her abdomen was "bigger" than it had ever been before. The swelling of the feet became so great that she could not get on her boots and had to wear "overshoes." Latterly she became very weak and had to stay in bed most of the day. For three days previous to admission she had epigastric pain, gradually growing more constant and severe.

On 1st. Jan'y she had intense frontal headache, became very sick and had a prolonged attack of vomiting, after which the headache was mitigated. She does not remember that her eyesight was worse then than it had been during the previous month.

During the night she had several attacks of vomiting, severe epigastric pain and frontal headache; the last thing she remembers was telling her husband early in the morning of 2nd. Jan'y that she "felt very ill."

She seems to have had constant abdominal pain, severe headache and increasing dimness of vision during the 2nd. and 3rd. Jan'y, becoming at the same time very drowsy. Vomiting too was frequent. The urine was not diminished in quantity and the bowels had been moving freely for some days. She was apparently in a state of complete stupor for some hours before the first fit occurred. Between 4 p.m. and 7 p.m. 3rd. Jan'y

the patient had four general convulsions. She did not regain consciousness after the first and the breathing all along was stertorous.

She was admitted in this condition at 7.30 p.m. The pulse was 150 per minute and of moderately high tension; the cardiac sounds were healthy. There was well-marked oedema of the face and lower extremities. The pupils were contracted, the right slightly larger than the left. Immediately after admission she had an epileptiform seizure. Dilatation of the pupils and a twitching of the eyelids were first noticed and double internal squint appeared; this condition lasted about twenty seconds. The jaws then became clenched and the head was thrown rapidly from side to side.

Synchronously with the commencement of the head movements the hands and arms flexed, and in a moment clonic spasm began in the arms, the hands remaining clenched with the thumb in the palm. The body and legs did not seem to be affected. The movements ceased in about two minutes and the breathing became stertorous, the cheeks being blown out and in with each respiration. The pulse was now 160 and very small; the temperature was 100°.

The abdomen was much distended and no uterine contractions could be felt. The foetal heart was distinct in a normal situation. The os uteri admitted two fingers and, for a multipara, was rather rigid; the head was presenting. 6 oz. urine was withdrawn by catheter. It turned solid on boiling and contained blood, and granular and blood casts in quantity. Chloroform was now administered and with the aid of a hot vaginal douche

the os was dilated manually sufficiently to allow the application of forceps. An unusually large quantity of liquor amnii escaped, though it could not be said that there was a great degree of hydramnios. In the meantime two pints of saline solution was infused under the right breast. Traction was gradually exercised on the forceps, and delivery, which was difficult owing to the large size of the child, was accomplished in about an hour. The child was a very large one and was still-born; it weighed ten and a half pounds. The cervix was torn and there was some bleeding.

The pulse post partum was of much the same quality and ran about 150 to the minute. The temperature was 100°. The pupils continued contracted.

About a quarter of an hour after delivery a hot wet pack was given, the skin reacting well. The patient remained unconscious and restless during the early part of the night. At 1.45 a.m., 4th. Jan'y, 4 oz. magnesium sulphate was given and immediately after a severe fit ensued, the temperature rising to 101°. At 2.30 another convulsion occurred, not lasting so long as its predecessor, the temperature rising to 101.2°. The pulse remained fairly good but rapid (150). 2dr. whisky every two hours was commenced. Two more fits occurred at 4 and 4.30 a.m., each preceded for about half an hour by great restlessness. The temperature was now 102°, but the pulse was steadier at 140 per minute.

A large soap and water enema was given to start purgation, and a second hot wet pack, both with very good result.

By 11 a.m. the patient was somewhat quieter; she was still unconscious, but could be roused to answer "yes" or "no" and to

take a drink. The last of the four fits post partum occurred at 4.30 a.m.

Throughout the day the patient lay in a state of stupor, fre-:quently very restless but at times sleeping quietly for some minutes.

There was no excessive lochial discharge, and the uterus remained well retracted. Her temperature in the evening was 100°, and the pulse which was regular and of good tension numbered 98 per minute. The patient by midnight had taken about a pint of fluid (milk and imperial drink). The bowels had moved loosely five times, and a large quantity of urine was passed, which however could not be measured. A catheter specimen contained .5 per cent albumin, no blood and no bile. It had no influence on Fehling's solution. Microscopically granular casts and debris were abundant.

5th. Jan'y.

The patient slept well and quietly during the night and this morning can be easily roused to consciousness, though she is still very drowsy. The pulse is 90 and the temperature normal. She is drinking plenty of milk and passing urine copiously. The oedema is a good deal less in the legs and feet, but the face is still rather swollen. Both lung bases are moderately dull, and fine mucus râle is abundantly present. She is not coughing much and there is but little expectoration.

The lochial discharge is a little heavy and a vaginal douche has been given. Throughout the day the day the patient drank about three pints of fluid and her bowels were freely

opened. In the evening she was less somnolent and spoke occasionally. She said she felt very well except for a little frontal headache, but could not remember anything. She complains of a "haze before her eyes" when she looks at anything. The pupils are equal and respond naturally to light.

6th. Jan'y.

The patient had a quiet night. The temperature continues normal, and there has been little coughing, but the pulse has been rapid and sometimes irregular. Bowels and kidneys are acting well; a catheter specimen of urine gives .1 per cent albumin. As the lochial discharges have still a heavy odour vaginal douches were given night and morning. Although quite conscious to-day the patient is very quiet and complains of great weakness and slight constant headache. She says she can see clearly to-day.

There were no changes found on ophthalmoscopic examination, but the visual field, roughly tested, seems a good deal narrowed in each eye.

In the evening the pulse became more rapid (120) and very irregular and small. No alteration of the cardiac or respiratory sounds could be detected. The pupillary, cutaneous, and tendon reflexes did not seem altered. A hypodermic injection of strychnine and digitalin was administered.

7th. Jan'y.

The patient has had a quiet night. The oedema of the face and lower extremities is gone. Albuminuria is present only to a small extent. The kidneys are acting well. The pulse is small and at times irregular; temperature subnormal.

The patient is suffering from mental depression and complains of constant headache. The lochial discharge is rather foetid and an intra-uterine douche was given. The interior of the uterus seemed healthy.

8th. Jan'y.

There is a faint trace of albumin in the urine, and a few granular casts are present. The patient feels very weak, and the melancholia is greater.

Except for her anaemic condition nothing can be found to account for her great weakness and mental depression. The lochial discharge is improved to-day; the uterus is well retracted and there is no tenderness in the abdomen. In the evening there was a discharge of blood from the vagina, amounting to about 10 oz.

9th. Jan'y.

During the night a purpuric rash appeared over the left scapula about 8" by 4" in area. There has been no more bleeding from the uterus and the lochial discharge is healthy. There is a trace of albuminuria. The condition of mental depression is unchanged.

This evening a purpuric rash appeared over the left forearm, the centre of it corresponding to the point where a hypodermic injection had been given. She is taking food fairly well.

10th. Jan'y.

The patient is somewhat brighter to-day. Both rashes are fading and no more have appeared. The pulse is stronger and the headache is gone. Albumin a faint trace.

13th. Jan'y.

Progress since last night has been very satisfactory. The

patient seems much stronger and is quite bright and interested in her surroundings. She is sitting up in bed and taking her food well. The pulse is stronger, the cough gone, and improvement general. There is still a faint trace of albumin; no casts are present, in the urine.

17th. Jan'y.

The patient has been out of bed and going about for three days. She is looking and feeling very well, and is in excellent spirits. For the last two days there has been no albuminuria but to-day there is a faint trace. She says she has not felt so well for a long time. The first thing she remembers after her delivery was being carried from one ward to another about 11 a.m. on Jan'y 4th.

At that time she seemed to be still in a condition of stupor and could only be roused with some difficulty to say "yes" and "no."

She was dismissed to-day.

C A S E 3.

Patient was an VIIIpara, aged 42, with a history of renal trouble becoming more and more severe with each successive pregnancy and culminating in the present case with a long drawn out preeclamptic condition.

Prodromals. The premonitory symptoms of the near approach of eclampsia were the headache, vomiting, and epigastric pain, together with a very profound feeling of general malaise for three days prior to the appearance of convulsions. There was also a condition of stupor for some hours before the first paroxysm. These symptoms probably denoted the onset of the acute renal disease of which Eclampsia is one of the manifestations.

Predisposition. Everything predisposed this patient to a severe illness. She was pregnant with a large child (the 8th) at the age of forty-two. Kidney mischief was already present to an alarming extent. Cough and want of sleep had told on her strength and the consequent loss of strength and appetite had reduced her still further. She had suckled her child until conception (16 months) and had menstruated four times before conception.

Urine. The urine was not diminished prior to the attack.

Temperature. Temperature rose with each fit post partum.

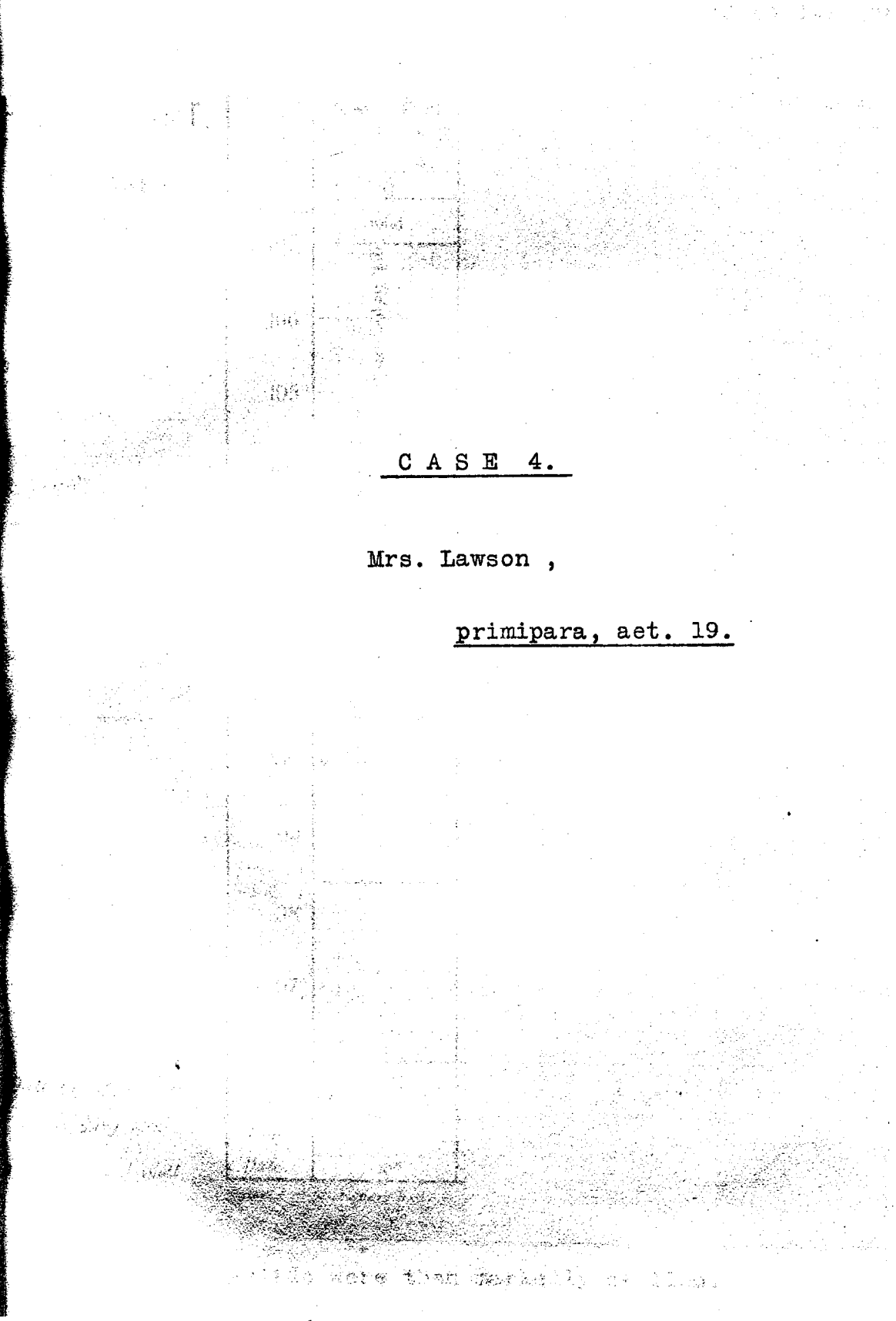
Salts. The large dose of Epsom salts (4 oz.) seemed to determine a convulsion immediately after.

Packs. The hot wet packs had a quietening effect.

Purpuric Eruptions. These, being coincident with a marked anaemia and a discharge of blood from the uterus, suggested at first the possibility of an idiopathic anaemia, but they were in all probability only due to the changes in the vascular tension which had occurred so rapidly during the week, and the anaemia was probably the result of the general condition of malnutrition of the patient consequent upon the long period of lactation, nephritis, and pregnancy.

The evidences of a profound toxaemia were well marked in this case, although there were only nine convulsions.

The chronic changes which must have been present in the kidneys accounted for the persistence of these symptoms for an unusually long time after the cessation of the fits.



C A S E 4.

Mrs. Lawson ,

primipara, aet. 19.

HOUR CHART.

DISEASE.

me { Mrs Lawson

e 19 years.

t

se Book N°

Notes of Case

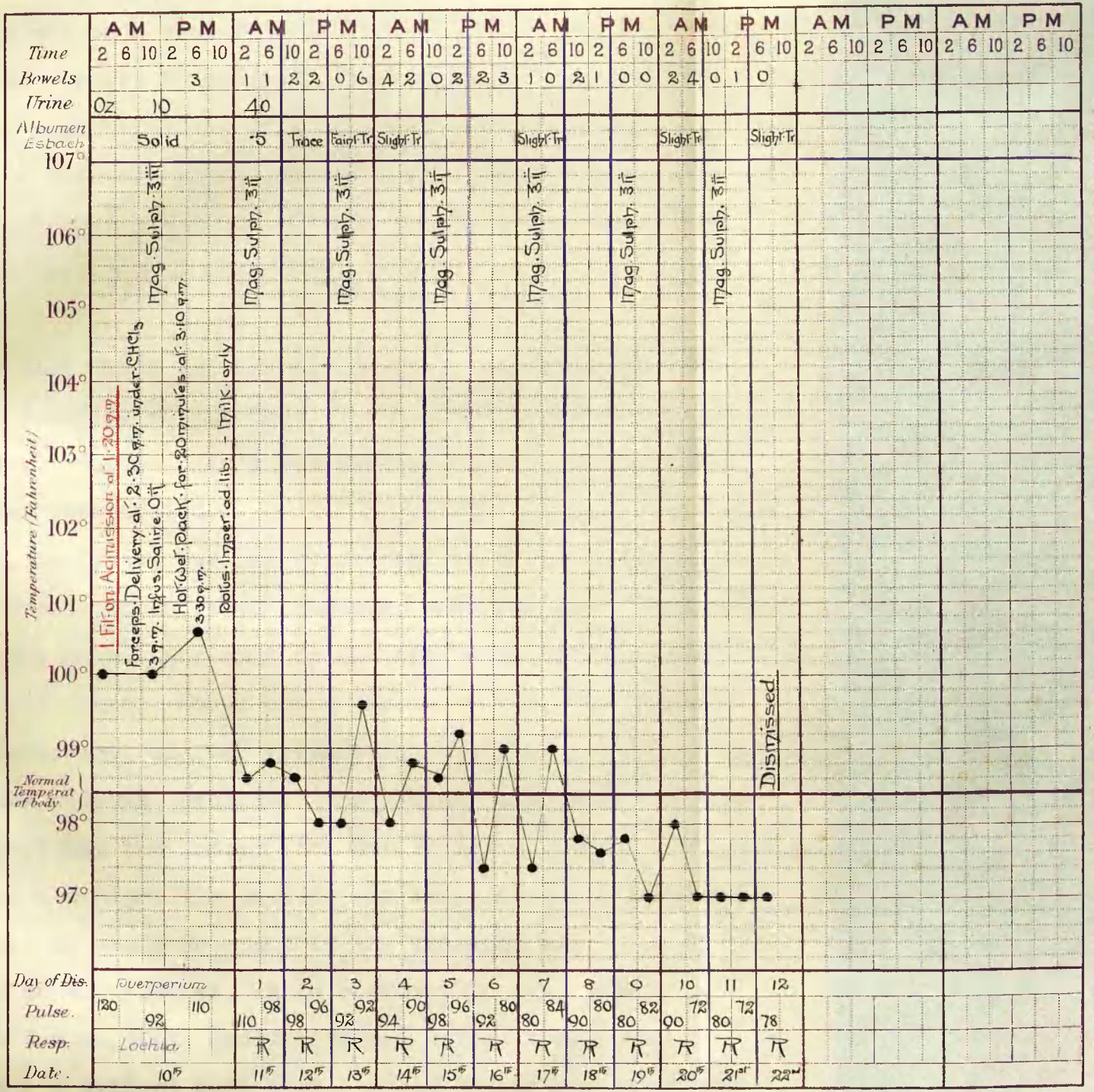
Para.

8 Fits previous to Admission at 1·20 p.m.

Date of admission

10th January 1903.

sult Dismissed Well



Entered, at Stationers Hall. *Printed and Published by Woddersposn & Co 6, Gate Street, Lincolns Inn.* *Goulds Clinical Chart.*

Mrs. Lawson, aet. 19, primipara, was admitted to Glasgow Maternity Hospital at 1.30 p.m. 10th. Jan'y, 1903.

She was within a week of full time.

There was no history of previous renal trouble, and she has always been a particularly healthy woman. Menstruation began at theage of thirteen years and has always been normal. She had quite a normal pregnancy until a few days ago, and seems to have suffered little even from the discomforts usually present.

On the 6th. Jan'y she had an attack of sickness and vomiting with flushing of the face; these symptoms disappeared the same day. On 7th. Jan'y she had polyuria. For some days she has been constipated.

On 8th. Jan'y she had slight frontal headache during the day; this became worse in the evening, when she also complained of epigastric pain. She went to bed and seems to have passed a fairly good night. About 6 p.m. on Jan'y 9th. the headache became more severe, and her friends noticed that her face and eyelids were somewhat swollen. The patient said that she "did not feel anything the matter with her eyes."

During the night the patient was very restless, complaining bitterly of headache, pain in the stomach, and pains in the lower part of the abdomen, which recurred at intervals.

About 4 a.m. 10th. Jan'y, frontal headache was very severe and the patient felt sick. A cup of tea was given her and was immediately followed by an attack of vomiting. Her face and eyelids were then markedly swollen.

She said she felt very ill but could see quite well. This condition lasted until 7 a.m., when the patient had a convulsion which lasted "about a minute;" and in half an hour, before she quite regained consciousness, was succeeded by another.

There were seven more fits before admission at intervals of from thirty to forty-five minutes; after the second she never was otherwise than completely unconscious.

Immediately on admission at 1.30 p.m., the patient had a fit in the reception-room. It lasted about two minutes, and after it the breathing was stertorous and the face pale.

The pulse was 120 and of high tension; the cardiac sounds were healthy with marked accentuation of the second aortic sound.

The temperature was 100°. The pupils were of medium size and reacted sluggishly and imperfectly to light. She was very restless and quite unconscious.

There was a moderate amount of oedema in the cheeks and eyelids, but the legs were not at all swollen. The abdomen was not larger than usual at full time. Strong uterine contractions were occurring every few minutes; the foetal heart was distinct in a normal situation.

The bag of waters was protruding in a pouch at the ostium vaginae; the os was fully dilated. The specific gravity of the blood was 1062 and the percentage of haemoglobin 94.

Chloroform was administered, the membranes ruptured, and forceps applied. The child was easily delivered; it was alive, a male, weighing five and a half pounds. The third stage occupied five minutes and there was no post partum bleeding, the

uterus contracting firmly without artificial stimulation.

While the patient was still under chloroform two pints of saline solution was infused under the right breast, and 3 oz. Epsom salts given by the stomach tube. The catheter was passed and 2 oz. of muddy, bloody urine withdrawn. It turned completely solid on boiling and contained a large quantity of blood. Microscopically numerous red blood corpuscles, leucocytes, and epithelial cells were found; epithelial, granular and blood casts were present in quantity. The urea estimated by Doremus' ureometer, was 5 grains per 1 oz. The pulse, post partum was 92, and of much the same quality - very high tension. The temperature was 100°.

Half an hour after delivery a hot wet pack for twenty minutes was given, the skin not answering well. The temperature rose after it to 100.6°, and the pulse to 110. Immediately on being taken out of the pack she vomited about ten ounces of dark greenish fluid, the filtrate of this containing about 1 per cent urea.

At 3.15 p.m. - about an hour after delivery - the patient was very restless and asked to be left alone. Half an hour later she said she was "very dry" and drank about 4 oz. milk . At this time she could not answer questions but was not deeply unconscious. She was given milk and imperial drink ad libitum and soon became much quieter, sleeping most of the time and occasionally groaning.

The catheter was passed at 5.30 p.m., and 3 oz. urine withdrawn, and again at 7.30 p.m., when 2 oz. was obtained. The specific gravity was 1042, the reaction alkaline; it became solid with albumin. Fehling's solution was not influenced

and there was no bile. The urine was clearer, but had the same microscopical characters. Her bowels moved loosely at 7.45; no urine was lost. As the patient had only secreted 5 oz. urine in the first five hours since delivery, it was thought advisable to attempt to stimulate the kidneys, and a hot mustard and linseed poultice was applied to the loins. She began to grow more restless about 8 o'clock, and by 10 p.m. was very much so, trying to get out of bed and to bite and scratch the nurse. By 10.30 her bowels had moved loosely thrice; the catheter was passed then, but only 3 oz. urine was withdrawn.

At 11 p.m. two pints of saline solution was again infused under the right breast, and the patient was dry cupped over the kidneys. A second poultice was then applied.

By midnight the patient was much quieter, and during the night dozed almost continuously. She drank copiously and twice complained of headache. She had two further slight attacks of vomiting, each specimen being dark green and the filtrate containing urea.

The specific gravity of the blood at midnight was 1060. At this time the tendon reflexes were apparently normal.

11th. Jan'y.

The patient this morning is still very drowsy, but can answer questions slowly.

Her temperature is normal, and the pulse, which is not of such high tension is 110, regular and strong.

She has had no fits since delivery.

Throughout the day she remained quiet and dozing most of the time. By 10 p.m. she had passed since delivery about three

pints of urine besides a good deal which was lost. The bowels had moved in all eight times. She had taken about eight pints of milk and imperial drink. She has talked a little, but that not very intelligently. She says she has no headache or pain anywhere. The uterus is well retracted and the lochia healthy.

A catheter specimen of the urine contained a copious suspension and deposit of urates. Reaction acid: specific gravity 1030: percentage of albumin .05: urea 8 grains per 1 oz. Microscopically a few red blood corpuscles and epithelial cells were to be seen but no casts. A specimen in the afternoon was clear of urates. A sample of the child's urine obtained to-day contained a considerable quantity of albumin.

On ophthalmoscopic examination a comet-shaped haemorrhage of recent date was observed in the macular region of the left eye. No other changes in either eye were discoverable. The visual acuity and field of vision seem normal on applying a rough test. The specific gravity of the blood to-day is 1060. She is quite conscious and intelligent this evening.

12th. Jan'y.

The patient has had a quiet restful night and appears much better this morning. The temperature continues normal, and the pulse is 94 , and of ordinary tension. The oedema is quite gone.

She is passing urine freely; it is clear and straw-coloured with a specific gravity of 1016. There is no blood and no deposit, but a trace of albumin persists. The urea is 8 grains per 1 oz.

She has no pain anywhere and no discomfort. The lochial

discharge is healthy.

A specimen of the child's urine to-day contains considerable albumin, but owing to the small quantity obtained it was impossible to estimate the percentage exactly.

13th. Jan'y.

Improvement in every respect continues. The patient is much brighter, though she still sleeps for the greater part of the day. Diuresis is copious, the urine being pale and clear and containing only a faint trace of albumin.

14th. Jan'y.

Progress is quite satisfactory. The patient is having milk diet and is quite bright, looking and feeling very well. The albuminuria is still present but is very slight.

17th. Jan'y.

Progress has been satisfactory in every way. There has been no rise of temperature since delivery, and the only symptom remaining is a very slight albuminuria.

Ophthalmoscopic examination to-day shows no fresh changes in either fundus. The haemorrhage in the left macular region is still visible but is much paler in colour.

20th. Jan'y.

The patient feels well in every way. She is allowed up to-day. There is a slight trace of albumin persisting in the urine.

22nd. Jan'y.

The patient was up most of the day yesterday and felt very well. The albumin has not disappeared from the urine, a faint haze being still present on boiling.

She says that the last thing she remembers before coming into hospital was sitting in a chair feeling very ill at 7.30 p.m. on 9th. Jan'y., and she has a confused recollection of getting a cup of tea and vomiting in the early morning of the 10th. This was only about three hours before the first convulsion occurred.

She was dismissed to-day.

C A S E 4.

Prodromals. Premonitory symptoms well marked.

Effect of delivery. There were nine fits before delivery at fairly regular intervals. She never became conscious after the first, but on admission she was not deeply comatose, so that even before delivery occurred she had begun to improve. This is an example of the kind of case which is fallaciously made to show the benefit of delivery in immediately stopping or mitigating the fits.

Urine. This patient, like Case 3, did not respond as quickly as usual to saline injections, there being only 5 oz. urine secreted in the first five hours. Even with hot poulticing of the kidney regions only 3 oz. urine was passed. With the second saline infusion at 11 p.m. dry cupping and poulticing were resorted to and copious diuresis followed.

The albuminuria persisted till dismissal, although no history of symptoms pointing to previous renal disease was obtainable.

When convalesence began there was a rise in the output of urea, after separation of the albumin, to 8 grains per ounce even with a diuresis of three pints in twelve hours and the patient taking only milk and

imperial drink.

Memory. Her memory of events prior to the onset of convulsions was retained in this case much later than usual.

This was not a very severe case. There were nine fits; the coma was never very profound; the patient spoke and swallowed within an hour and a half of delivery. The only fit that occurred in hospital was not very intense. Cyanosis was never present; even immediately after a fit the face was pale.

The tension of the pulse did not decrease with the delivery, nor did it become lower during the period when the greatest improvement took place (first twelve hours).

C A S E 5.

Mrs. Faichnay,

XIIIpara, aet. 40.

...UR CHART.

DISEASE.

Mrs
Faichney
...0 years

...ook No

Notes of Case

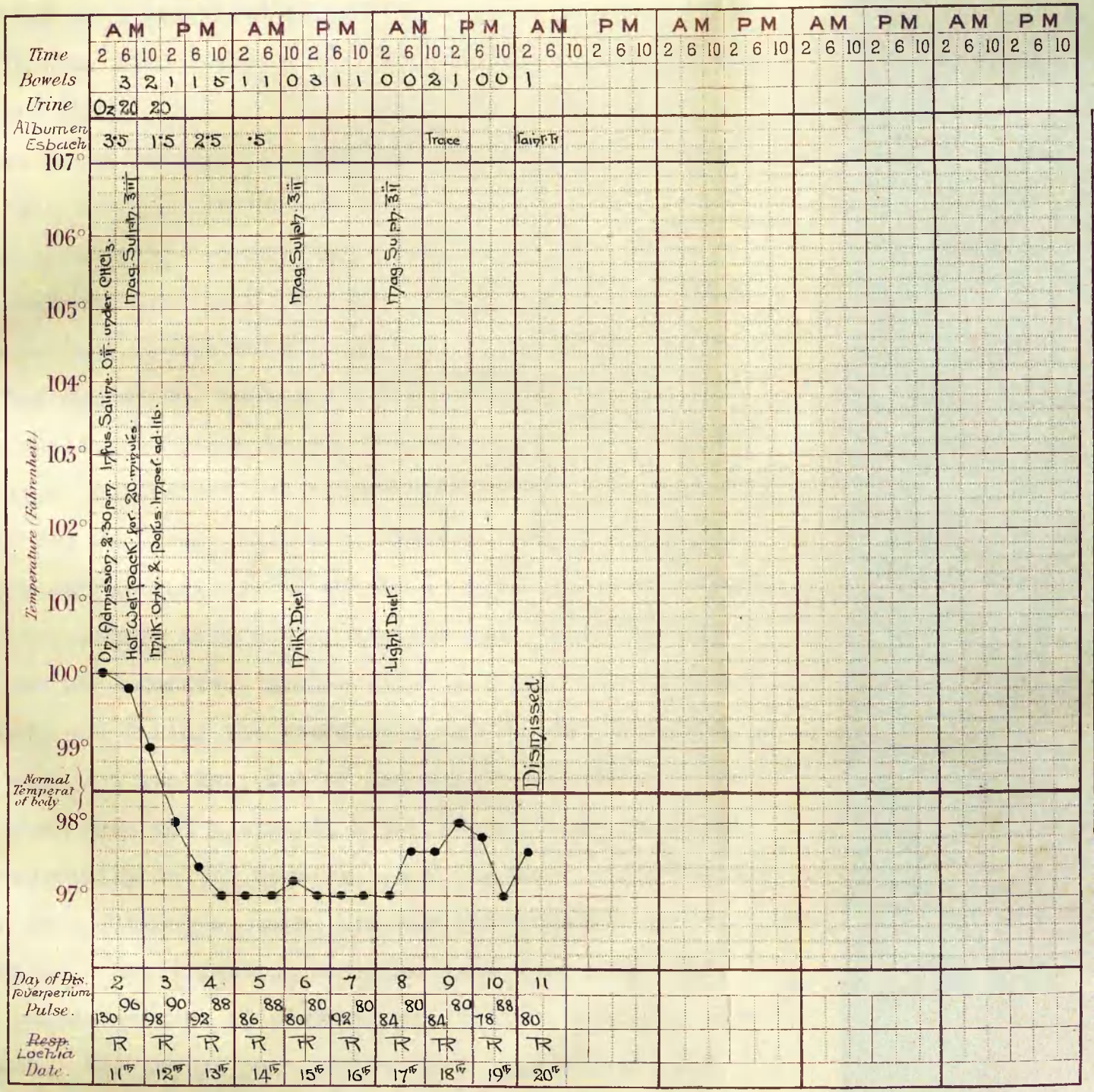
...- xiii para -

...elivered at home -
...p.m. 9th January 1903

...fits previous to
...mission at 2·30 p.m.
...e last fit occurring
...11·15 p.m. 10th January

...te of admission
...January 1903

...t Dismissed Well



Entered at Stationers Hall. *Printed and Published by Wodderspoon & Co 6. Gate Street, Lincolns Inn.* *Goulds Clinical Chart*

Mrs. Faichnay, aet. 40, XIIIpara, was admitted to Glasgow Maternity Hospital, at 2.30 p.m., 11th. Jan'y, 1903.

She had been delivered at home of a large stillborn child at 11 p.m. 9th. Jan'y. The cord had been prolapsed and forceps were used.

There is no history of renal disease previous to childbearing. She has had thirteen pregnancies, all, with the exception of this last, terminating naturally at full time. Thirteen children have been born in nineteen years, two years being the longest period between any two consecutive births.

At the age of 21, when about eight months pregnant with her first child, she began to get oedematous, at first in the face and legs, but before the termination of her pregnancy the whole body was swollen. Delivery, however, was quite natural, and all the oedema disappeared within a week. In each succeeding pregnancy the oedema made its appearance earlier and before delivery was more marked. Always after delivery the oedema disappeared, and during the times when she was not pregnant her legs were not swollen; but of late years she frequently noticed, even when not pregnant, a puffiness of the face and eyelids especially in the morning. For the last ten years she has never been without a cough, and has suffered a great deal from headache. Her digestion has also been very poor, and, for some years, attacks of sickness and bilious vomiting have been common. Her bowels have always been constipated, and gastralgia has been frequent.

The twelfth child was born on Decr. 2nd., 1901; she did not nurse it.

During the first four months of this last pregnancy she was in her usual state of poor health, but about a fortnight before quickening the swelling of the feet and legs began, and soon became more marked than it had ever been before.

During the latter half of pregnancy she has been in very poor health. The oedema has been excessive; headaches were frequent and often very severe; she became very weak, suffering much from cough and "indigestion." She has been almost constantly confined to bed for the last month, all her symptoms being greatly aggravated. She often had "needles and pins" sensations in her arms and legs, sometimes lasting the whole day, and generally rather painful. Attacks of abdominal pain and bilious vomiting were common, and her bowels never moved without large doses of medicine. She is very certain that during this month her eyesight did not fail, although she says it has been getting gradually weaker for some years. She was always giddy if she sat up, but never had any singing in her ears.

For a week before labour and especially for the last few days she passed very little urine; her bowels had not moved for four days.

Severe frontal headache, sickness, vomiting, and epigastric pain were almost constant for three days before the onset of labour. She had almost no sleep or food during that time, she says. Her eyesight seems to have been as usual. She had a sense of fear very present for two days before labour and was greatly pleased that the child had been born"without anything going wrong."

During the first night of her puerperium she felt very much better, sleeping for some hours, but at 10 a.m., 10th. Jan'y, the frontal headache and epigastric pain returned. Two hours later she became suddenly blind, so that she could not see her friends.

About 2 p.m. she had a convulsion of short duration - "the first in her life" - and this was very soon followed by a similar seizure. At 2.45 she was seen by the outdoor house-surgeon who found her quite conscious and able to answer questions intelligently.

She complained then of severe frontal headache and loss of vision. She could not distinguish any object, but could just discern a light which was used to test her pupil reflex.

The pupils were dilated and equal and responded readily to light; there appeared to be slight internal squint of the left eye. The eyes followed the light laterally, but not vertically.

Hearing and feeling were evidently normal, but intelligence was somewhat dulled.

The face was very puffy especially under the eyes, and there was moderate oedema of the legs and feet. The face was sallow and the mucus membranes dusky. The tongue was moist and tremulous with a white fur in the centre.

The pulse was 96, regular, and of high tension. The heart sounds were free from murmur, but there was accentuation and inconstant reduplication of the second sound. The respirations were regular, easy and free from stertor.

She was passing urine in fair quantity since delivery. She refused to come into Hospital.

At 5.30 the condition of the patient was unchanged; she had had no more fits. 1 dr. pulv. jalapae co. with ½ a dr. pot. bromidi and 15 gr. chloral hydrate were given.

At 11 p.m. the doctor was told that she had been passing urine freely (probably about two pints). Her bowels had not moved and she had vomited at 9 p.m. The condition seemed unaltered.

At 11.15 p.m. she had an eclamptic fit - the third. It began with marked twitching of the left side of the face and eyelids and almost immediately after the right side of the face followed, but the movements were not so marked.

The mouth began to make chewing movements, the lips , through which frothy saliva began to appear, remaining closed. The head then began to jerk towards the left very rapidly, not rolling from side to side. The pupils were contracted and the eyes upturned to the left.

The arms were then raised from the body, and the hands and arms became flexed, the thumbs not being in the palms. The abdomen seemed also to be affected but not the legs. No urine or faeces escaped.

The movements ceased very abruptly, about four minutes from the time when the twitching of the eyelids was first noticed. The breathing after the fit was not stertorous. The saliva was running from her mouth, but apart from this she appeared simply to be asleep. Immediately after the fit, the pulse was 136, regular, and hard.

The patient had no more fits and seems to have remained in a state of stupor until her admission at 2.30 p.m. the following day, (11th. Jan'y, 1903).

On admission she was quite unconscious, but not deeply comatose. There was slight general oedema, most marked in the legs and feet. The pupils were moderately contracted and hardly responded to light; there was no squint. The pulse was 130, strong, regular and of high tension.

The cardiac sounds seemed pure, and there was accentuation of the second aortic. The temperature was 100°. The respiratory murmur was deficient, especially over the front of the chest, and at both bases there were dulness and numerous moist râles.

The catheter was passed and 6 oz. urine withdrawn. It was of a muddy appearance containing abundant blood, and .35 per cent albumin. There was no bile and the specimen had no effect on Fehling's solution. Reaction was neutral; specific gravity 1020. Microscopically white and red blood corpuscles, casts of all kinds, and epithelial and granular debris were present in quantity. The abdomen was distended but the uterus was well retracted.

Chloroform was administered and two pints of saline solution run in under the right breast. 3 oz. Epsom salts was given by the stomach tube, and the patient was then put into a hot wet pack for twenty minutes which caused a profuse diaphoresis.

She continued in a state of stupor, occasionally very restless, for the next twelve hours, when she began to appear more intelligent. By 10 a.m. 12th. Jan'y she was easily roused to answer questions. She then complained of headache, dimness of vision and slight giddiness. She was only able to distinguish that there were people in the room and to make out

a white object before her eyes. The pupils were of medium size and reacted well to light; the eyes followed a light in all directions.

Her bowels had moved loosely thrice, and she had passed about three pints of urine. She slept most of the day but took her milk and imperial drink well, giving little trouble in the nursing. Her pulse which is softer is about 100 this evening. On examination with the ophthalmoscope no changes whatever were found in the retinae. Her temperature since admission has been a little above normal.

13th. Jan'y.

The patient has slept well during the night and taken her nourishment satisfactorily. A copious diuresis is maintained and the bowels are moving freely. A catheter specimen of the urine is clear and amber-coloured; no blood is present, and the percentage of albumin is .25. The patient is fairly intelligent to-day, and except for a slight continuous headache, feels well. To-day she can distinguish objects in the room fairly well. The temperature and pulse rate have settled to normal. The uterine condition is entirely satisfactory. The oedema of the face is still present, but elsewhere there is the merest trace. Dulness and crepitations are still present at both bases, but the patient is not coughing much, and there is very little expectoration.

14th. Jan'y.

The patient is very much improved to-day. Her headache is gone, and she can see quite well. The bowels are moving freely and diuresis is copious. The albumin in the urine has fallen to .05 per cent. Except for a little puffiness under

the eyes all trace of oedema is gone.

16th. Jan'y.

Improvement in every respect continues. She says she has not been so well for a long time. She has no memory whatever of her labour or of the events immediately preceding it.

The cough is almost gone and there is no expectoration. The lung bases are clearer but moist râles are still present.

18th. Jan'y.

Progress is quite satisfactory. The patient is having light diet and is allowed up. A trace of albuminuria is present; a little granular debris, but no casts, can be found microscopically.

20th. Jan'y.

Convalesence has been rapid and satisfactory. Except for a faint trace of albumin in the urine all symptoms of the illness have disappeared.

She was dismissed to-day.

C A S E 5.

A somewhat similar history to that of Case 3.

XIIIpara, aged 40.

There were the same predisposing conditions.

This patient also had a well-marked sense of fear that something was going to happen to her.

Onset of Fits. Fits began fourteen hours post partum, during which time she had been feeling much better, and passing more urine than she had done for several days.

Coma. Within 30 minutes of the first convulsion she regained consciousness, but with her memory for recent events quite gone. Coma was never deep.

Retinae. Even after many years of renal insufficiency no changes could be found in the fundi.

This was a mild case.

C A S E 6.

Mrs. Chalmers ,

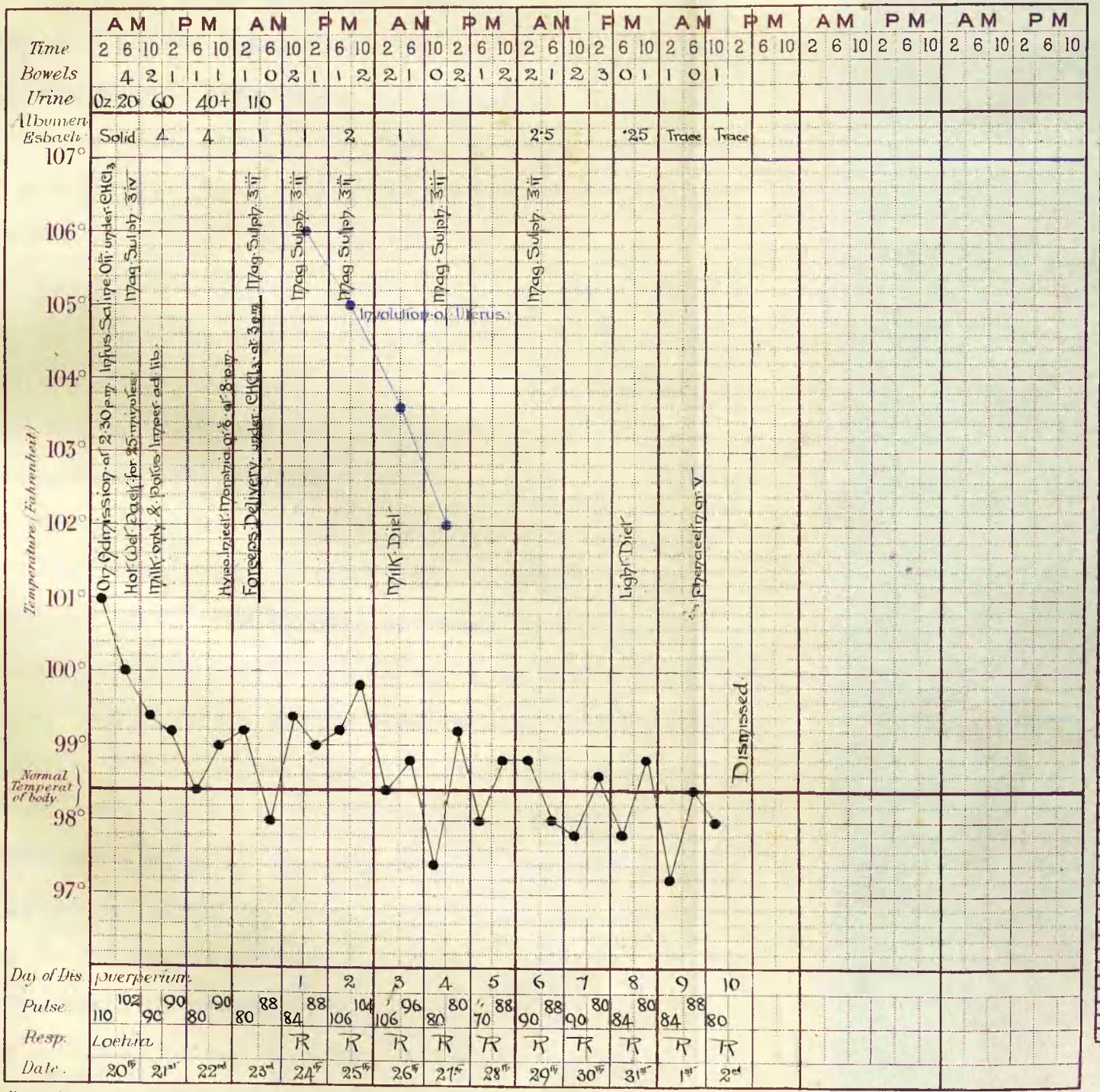
primipara, aet. 22.

IR CHART.

DISEASE.

Mrs
Chalmers
Years
No
tes of Case
Para
previous to
ssion at 2.30 p.m.

of admission
January 1903
Dismissed Well



Entered at Stationers Hall.

Printed and Published by Wedderspoon & Co 6, Gate Street, Lincolns Inn

Mrs. Chalmers, aet. 22, primipara, was admitted to Glasgow Maternity Hospital at 2.30 p.m., 20th. Jan'y, 1903.

She was about seven and a half months pregnant. She had had a severe attack of scarlatina when a young girl which had been complicated with nephritis. At the age of 16 she had swelling of the legs, and for the last two years has had occasional pain in the loins, though there has been no return of the oedema in the legs or face.

For some years before marriage she often had severe headaches, so bad as to make her stay at home from her work (sewing). There is no history of cough nor of progressive dimness of vision. Menstruation began at the age of thirteen and was always easy and regular.

She was married at the age of 21, and altered thrice since, the last time being in the first fortnight of May.

There was but little morning sickness in the early months of her pregnancy, but even as early as the third month the legs and feet became a little swollen; this oedema during the rest of the pregnancy was sometimes well-marked and sometimes almost gone, but it never altogether disappeared. She noticed that her headaches were always worst when there was most swelling - a voluntary statement. She first felt quickening in the second week of December. About the end of December the swelling became more constant and more marked; it was always confined to her legs and feet; she "had to get new boots."

Headache at this time became more frequent and severe. Ever since the oedema first made its appearance about the end of July there has been "a kind of mist" over her eyes, and for

the last two months she was never able to read or sew on this account until she had tried for some time; her eyes then would "water" profusely and after about ten minutes of this she began to be able to see more distinctly. Since the New Year her eyesight got steadily worse so that she noticed a difference almost daily.

During these last three weeks she has had attacks of epigastric pain, quite distinct from the movements of the child, and frequently nausea and vomiting ensued. The vomiting always relieved the pain. The vomiting seemed to bear no relation to the ingestion of food, and she never noticed any blood in the vomited matter.

Of late she has often had as many as three attacks of vomiting in the day.

The movements of the child have been very vigorous during this time. The oedema during the last three weeks became general, the eyelids being much swollen. She thinks she passed less urine of late though she had to make water oftener; the urine was high-coloured. She has also been very constipated, the bowels only moving about twice a week and only with large doses of Castor oil.

She frequently felt very weak, and often had "giddy" turns, having to sit or lie down.

During the week before admission all her symptoms became more urgent. Her legs were much swollen and painful; headache was severe; her eyesight was very bad; and she had much epigastric pain and vomiting. She was often so weak and giddy that she felt as if she were about to faint though she never lost consciousness. She does not remember anything after

16th. Jan'y - three days before the first convulsion. She has never had any tinnitus aurium.

On the 17th. and 18th. Jan'y she remained in much the same condition, complaining most of headache, blindness, and pain in the upper part of the abdomen. She became more swollen generally, and was restless though inclined to be drowsy all day on the 18th.

When her husband returned from work at 7 a.m. on Jan'y 19th., he found her lying on the floor unconscious. She was put into bed and lay quite quietly until 11 a.m., when she took a convulsion. An hour later she began to regain consciouness and soon appeared to be very much clearer, although inclined to lie still and doze.

She continued in this condition till about 3 p.m., when she had a general convulsive seizure, soon followed by another. She never regained consciousness after this, lying quite comatose until the evening when she had a series of "seven fits with :in an hour."

During the day she had passed urine in bed, but her bowels did not move.

About 11 p.m. a doctor saw her and gave her chloroform; she had not had any more fits. She remained very quiet and apparently sleeping for most of the night, and by early morning was able to take sips of milk and hot water, although she could not speak at all.

On admission at 2.30 p.m., there had been no return of the convulsions. She was unconscious, but, on handling her, she moved and cried "stop it!"

Her face was of a good colour, but was very much swollen;

there was moderate oedema over the rest of the body. The pupils were of medium size and responded well to light. The temperature was 101°, and the pulse 110, of high tension, regular, and strong. Cardiac and respiratory sounds were healthy.

The uterus reached midway between the umbilicus and xiphisternum, and contractions were occurring though not very frequent or strong. The position was a "first vertex;" no foetal heart sounds or movements could be detected. Per vaginam, the membranes were unruptured and the head was presenting through a somewhat rigid os, capable of admitting two fingers.

The catheter withdrew about 1 oz. of muddy urine, acid in reaction, with a specific gravity of 1030, and a copious deposit of urates on standing. It turned solid on boiling; it contained no bile, and had no effect on Fehling's solution.

Microscopically red blood cells, and granular and epithelial casts were to be seen in large quantity. It contained about 6 grains per 1 oz. of urea.

The blood was very dark in colour, the specific gravity was 1064, and the precentage of haemoglobin 98.

Chloroform was administered and two pints of saline solution infused under the right breast. 4 oz. Epsom salts was given by the stomach tube, and the patient was then put into a hot wet pack for 25 minutes; she sweated very freely.

The patient remained unconscious and restless during the rest of the day, sleeping quietly in all about two hours.

By midnight she had passed about a pint of urine, the bowels had moved loosely four times, and there had been a gentle perspiration most of the time. The temperature at midnight was 100°; the pulse was 102 and somewhat softer. The

uterine contractions had ceased.

st. Jan'y.

The patient was restless for a good part of the night, but slept quietly for about six hours. By 7.30 this morning she was able to answer questions intelligently. She is still in a certain degree of stupor, however, but she can be easily awakened and says she has no pain. She has passed in the last twelve hours (the second twelve hours since the infusion) probably nearly three pints of urine, the skin and bowels also acting satisfactorily.

The os is not further dilated and uterine contractions have not returned. Foetal heart sounds cannot be heard.

Throughout the day the patient continued very somnolent, although becoming more restless in the afternoon. During the evening she became quieter and more sensible and said that she was feeling better but had some pain in the abdomen when her bowels moved. Diuresis to-day has continued abundant. A catheter specimen of the urine is clear and amber-coloured; there is a slight mucus deposit; reaction acid; specific gravity 1018. Esbach's instrument showed .4 per cent of albumin. Urea 8 grs. per 1 oz.

Microscopically blood cells and casts are to be found, but in smaller numbers.

She is drinking a great deal of milk and potus imperialis. The oedema is somewhat less marked to-day, especially in the legs.

2nd. Jan'y.

The patient passed a quiet, comfortable night, and this

morning her condition is greatly improved. She is much brighter and is looking better; she can talk sensibly although her memory is still very confused.

She says she can see more clearly than she has been able to for some weeks, and she has no headache or abdominal pain. The oedema is decreased both in the legs and face. Renal and intestinal activity continue; she is drinking a great deal of fluid.

The temperature is normal, the pulse 80, of moderate tension. On ophthalmoscopic examination the right fundus was found healthy, but in the left, just outside and above the macula, which is well seen, there is a thin-looking circular haemorrhage about the apparent size of a split pea. There are no other changes evident in the fundus. The percentage of albumin still stands at .4; blood cells and casts are present in small numbers. The urea is at 7 grains per 1 oz.

The patient passed a good day, but in the evening about 7 p.m. began to complain of abdominal pain. By 8 o'clock it was evident that this was due to the return of uterine contractions. The os was no further dilated. One-eighth grain morphia was given hypodermically and a tight binder applied to the abdomen. She suffered but little pain from the labour and slept between the pains.

3rd. Jan'y.

The patient was delivered easily and naturally without chloroform at three o'clock this morning. The child was male, weighing three and three-quarter pounds, badly macerated, having probably been dead since the evening of the 20th. The

third stage lasted ten minutes. The uterine surface of the placenta was half covered with blood clot, probably three days old. There was evidence of fatty change in small areae. The temperature post partum was 99.2°, the pulse 80, and of moderate tension.

The patient slept for eight hours after delivery. By twelve noon (nine hours) she had passed five and a half pints of urine. The percentage of albumin was one-tenth.

24th. Jan'y.

The patient has been very well since last note. Her eyesight is quite clear, and she can read newspaper type. She complains this morning of slight frontal headache. The breasts are engorged and rather painful, and have been rubbed with olive oil and tightly bound. The uterus is well retracted. A catheter specimen of the urine is pale and clear; specific gravity 1008, albumin 1 per 1000; no blood and a few casts to be seen microscopically.

25th. Jan'y.

Progress is eminently satisfactory. The uterus is involuting very rapidly, being only three inches above the symphysis; the lochial discharge is small in amount, but quite healthy. The breasts are still painful and were again rubbed and bound up. Little fluid has been given during the last twenty-four hours on this account, and 1 oz. magnesium sulphate was administered yesterday evening. The urinary output is nevertheless two pints and the percentage of albumin is .2.

26th. Jan'y.

The breasts are much more comfortable to-day. The invol-

ution of the uterus has been rapid, the fundus being now only two inches above the pubes. Except for slight puffiness of the eyelids all oedema has disappeared. The patient remembers everything clearly until 16th. Jan'y, but from then until the time of her labour, which she remembers only in a confused way, everything is a blank. The albumin is still .1 per cent in a catheter specimen.

She is allowed light diet to-day.

29th. Jan'y.

Progress is very satisfactory. All the oedema has now disappeared, even from the eyelids. She is feeling much stronger, and has no discomfort whatever. Albumin $\frac{1}{4}$ per 1000; no blood cells or casts are now to be found by the microscope.

31st. Jan'y.

Except for slight albuminuria ($\frac{1}{4}$ per 1000), all symptoms of her illness have gone. On ophthalmoscopic examination it was found that the haemorrhage was almost entirely absorbed and that all that remains is a small brownish red spot.

She was allowed out of bed this evening.

3rd. Feb'y.

The patient is feeling well in every way. There is still a slight trace of albuminuria.

She was dismissed to-day.

C A S E 6.

Prodromals. There is a history of previous renal disease. Oedema of the legs appeared early in pregnancy (3rd. month). Headache and a "mist over the eyes" appeared early also; the latter was always relieved when she looked steadily for about ten minutes at her reading or sewing. "Watering" of the eyes occurred at the same time. Was the improvement due to a relief of ocular tension which this afforded, or was the accommodation of the eyes slower?

The earlier and later prodromals were both well-marked.

Urine. In this case the increase in the output of urea after the saline infusion was well marked. Although diuresis commenced soon after the cessation of the fits, there was not the usual fall in the percentage of albumin until after delivery. There was a very large diuresis (five and a half pints) in the first nine hours post partum.

Retinae. This is one of the only two cases in which retinal haemorrhage was present; it absorbed very quickly.

Labour. The labour passed off with the convulsions, and

did not occur till fully three days later. It was quite natural in every way, and the pains seemed to cause less disturbance than usual. She had had one-eighth grain of morphia subcutaneously.

A case which illustrates a typical attack of Eclampsia thoroughly. Had accouchement forcé been here resorted to when fits occurred, the result would probably have been very different, and in any case could not have been more successful than was the expectant treatment resolved upon.

The involution, as in all these cases, was rapid; in three days the fundus was only two inches above the pubes, the position that is usually found on the 6th. to the 8th. day in a case of a normal labour.

C A S E 7.

Mrs. Howie ,

primigravida, aet 22.

This was a case in which the patient, about seven months pregnant, appeared, on admission, to be threatened with eclamptic convulsions. The report is inserted as showing the benefit of prophylactic treatment, and as an example of the good result that may be hoped for in such cases.

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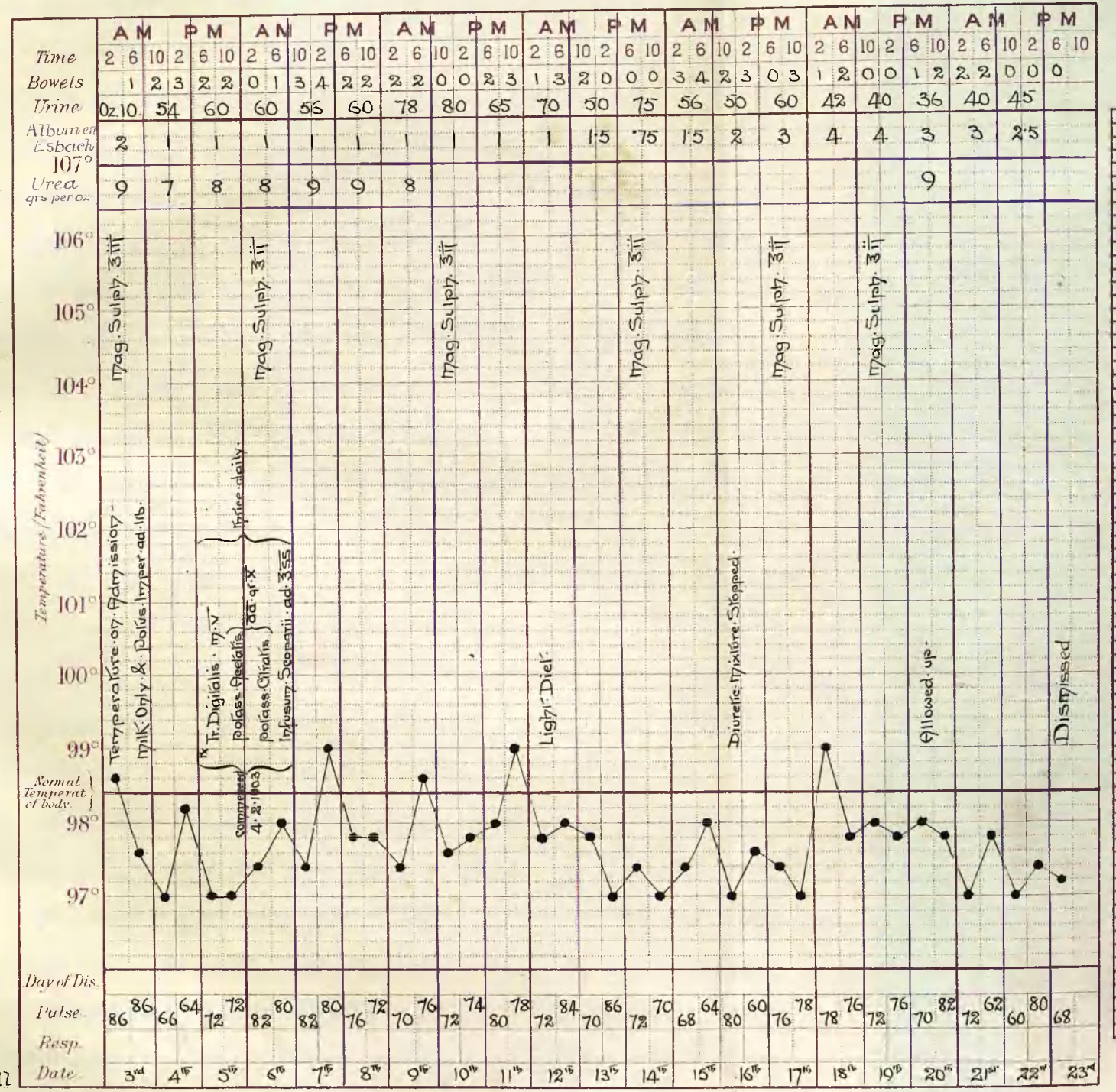

Entered at Stationers Hall. Printed and Published by Wodderspoon & Co 7. Serle Street, Lincolns Inn. Goulds Clinical Chart

Mrs. Howie, aet. 22, primigravida, was admitted to Glasgow Maternity Hospital at 12 noon, 3rd. Feb'y, 1903, complaining of frontal headache and epigastric pain of three days' duration, and of swelling of the legs of a week's duration.

The patient is about seven months pregnant. She has never had scarlatina nor is there any history of renal disease previous to her pregnancy, but she has always been rather a delicate young woman, suffering from "weak digestion," epigastric discomfort, and habitual constipation. At the age of 17 she had acute rheumatism, but she seems to have made a good recovery. During the illness she never had any precordial pain nor shortness of breath, and at no time was there any oedema of the face or legs. She has never been afflicted with any rheumatic pain since.

Menstruation began at the age of 15, and after an omission of two months became regular; from the beginning it was accomplished with sacral and lumbar pain. She had a seven days habit; the pain was always during the molimen and the early part of the flow.

She has never had more than an occasional headache previous to her pregnancy, and her eye-sight has always been good.

She was married at the end of May, 1902, and menstruated twice afterwards, the last time in the early part of July. Morning sickness was rather severe and lasted about two months; she then remained in fairly good health as regards her strength, digestion, and freedom from headache until two weeks ago, i.e., shortly after midterm. At this time she began to have attacks of sickness and vomiting soon after going to bed.

This at first came only occasionally, but soon became regular, appearing every night within a few minutes of her lying down. The sickness always continued until she vomited; this gave her immediate relief. There was never any blood or bile in the vomited matter; at no time after food during the day had she these symptoms.

She generally had supper about 8 o'clock and went to bed about 10 p.m., but the sickness and discomfort in the stomach never ensued till she lay down.

It was quite independent of coitus, or of any particular kind of food she was taking.

About the same time (the beginning of December) she began to have headaches at intervals of a few days. During the month of January the headaches became more frequent and severe, and during the last few days have been almost constantly present; they were always frontal in situation.

Eight days prior to admission she noticed that on taking off her boots at night her legs were a little swollen, and next morning she found that her face was oedematous, especially under the eyes. The oedema became more marked within the next few days, and the attacks of vomiting became no longer confined to the patient's lying down, but occurred also throughout the day.

During the last week also epigastric pain unrelated to food-taking became more constant. She became very weak but was never drowsy or giddy, nor has she ever felt as if she were about to faint. Her eyesight remained perfectly clear and she never had any singing in her ears.

For a fortnight before admission she was passing less urine than usual, and at the time the oedema began the output became still less.

During the last three days she has been passing about eight to ten ounces of highly-coloured urine daily, her bowels have not moved at all, epigastric pain has been almost constant, vomiting frequent, and headache severe.

In the same time the oedema of her legs and face became still more distinct, and she had a continual feeling of weakness and general malaise. Her memory was not at all affected.

On admission the patient was markedly oedematous. In the legs the pitting on pressure was deep; over the trunk there was moderate swelling; the face was very puffy. She was of a good colour, and lay easily in bed in any position.

She had a well marked goitre about the size of a large, flattened orange extending most to the right side of the neck. It has gradually grown larger since she first noticed it about twelve years ago; she thinks it has increased in size more rapidly since she became pregnant.

It has never given her any trouble, and there is no exophthalmos or tachycardia.

The cardiac dulness is not enlarged; the apex beat normal in character and situation. A loud blowing systolic murmur is heard over the whole cardiac area, least marked at the apex, and best heard in the pulmonic region; it is well heard in the vessels of the neck, but is not conducted into the axilla. A bruit is distinct in the thyroid tumour. There is no visible

pulsation in the carotids; the pulse is 66, regular, and of moderate tension; the temperature is 97.8°.

There is no abnormality in the percussion of the chest, and the breath sounds are clear and healthy except for some crepitations at the bases.

The uterus reaches midway between umbilicus and xiphoid, and the abdomen is of the usual size at the seventh month. Foetal movements are present and the foetal heart is distinct. There is no tenderness in the epigastrium or other region of the abdomen. Nothing abnormal is to be made out on vaginal examination.

There is no subconjunctival oedema; the pupils are of medium size and respond naturally to light and on accommodation. Visual acuity and the field of vision, roughly tested, appear normal. Ophthalmoscopic examination reveals nothing abnormal. Her tongue is coated with whitish-yellow fur and she has very good teeth.

A catheter specimen of the urine is clear and straw-coloured, of neutral reaction and specific gravity 1010. It contains no blood or bile and does not affect Fehling's solution. There are two parts per 1000 of albumin, and the urea is 2 per cent. Microscopically only a little granular debris is to be found.

The specific gravity of the blood is 1058. The percentage of haemoglobin is 88.

The patient was kept in bed for twenty-four hours without further treatment. During this time she had a good deal of epigastric pain which had no relation to foodtaking; it was

constant through the night as well as the day.

Frontal headache was almost constant, and at times seemed to be very bad. She had an attack of vomiting in the evening; this was almost two hours after a bowl of gruel; the vomit was simply mucus.

During the first twenty-four hours of her residence in hospital, she passed about sixteen ounces of urine.

th. Feb'y.

As the patient does not seem any better this morning and, in a catheter specimen of urine, the urea has fallen to 7 grains per ounce, it was thought expedient to begin some eliminant treatment.

She was put on milk and imperial drink only, 1 oz. of Epsom salts given, and the following diuretic mixture begun:-

℞

Tincturae Digitalis	5 m	
Potassii Citratis	āa 10 gr.	To be taken in water 3ce daily.
Potassii Acetatis		
Infusum Scoparii ad ½ oz.		

ɜth. Feb'y.

From noon yesterday till noon to-day the patient has passed more than four pints of urine. Percentage of albumin .1; urea 8 grains per ounce.

There has been no more vomiting and the epigastric pain is quite gone. The bowels have moved loosely several times. The oedema is somewhat less. She still complains of frontal headache but it is comparatively slight.

th. Feb'y.

The headache has been quite gone since yesterday

morning, and there has been no more sickness or epigastric pain. The oedema is almost gone from the legs and feet, though the eyelids are still a trifle swollen. The bowels have been moving freely, and she is passing about three pints of urine in the twenty-four hours. Albumin 1 per 1000; urea 9 grains per ounce.

th. Feb'y.

There is no trace of oedema to-day except a slight puffiness under the eyes. A profuse diuresis is still kept up and encouraged, the percentage of albumin being .1 and of urea 1.8. All subjective symptoms are quite gone, and there has been no recurrence. The cardiac condition remains as it was on admission; no râle can now be heard at the bases of the lungs.

2th. Feb'y.

The improved condition of the patient continues. The oedema is now completely gone, and the patient is allowed to take a little exercise and to have light diet.

6th. Feb'y.

The diuretic mixture was stopped to-day. Progress is satisfactory in every respect. The patient is looking well and feeling much stronger.

0th. Feb'y.

The patient continues well in every way. She is still taking imperial drink and passes about two pints of urine daily. The percentage of albumin since she has been out of bed and on light diet remains between .3 and .4; the urea continues about 8 grains per ounce.

She is going about the ward for a good part of each day.

3rd. Feb'y.

The patient has been in very fair condition for the past

fortnight. She has none of the symptoms from which she suffered on admission.

As all risk of an immediate appearance of Eclampsia seems gone, and as the patient is only about seven and a half months pregnant, it was considered safe to dismiss her to-day, with instructions how to take care of herself and to communicate with her doctor at once should any of the former symptoms return.

She was dismissed to-day.

C A S E 7.

"Evening sickness."

The "evening sickness" after midterm is a rather unusual condition, and was probably due to the adoption of the horizontal position, being, like "morning sickness," of reflex origin.

Prodromals.

The prodromal symptoms were well marked on admission, even epigastric pain and vomiting being present; these latter are symptoms which herald the near approach of convulsions usually.

In this case, however, there was neither drowsiness nor restlessness, pulse and temperature were normal, the eyesight was quite good, and the intelligence perfectly clear. It was therefore considered quite safe to allow some time for observation before starting treatment.

It is, of course, very difficult to say what was the exact condition of affairs causing her symptoms-whether the chronic nephritis of pregnancy was becoming very severe, or whether the commencement of the acute renal disease of pregnancy had supervened.

Some of my cases who fell victims to the acute renal disease and eclampsia had not more severe premonitory symptoms than this patient, and I think it very likely that this case was on the high road to eclamptic convulsions when our treatment was instituted. There is no difficulty in supposing that the/

renal disease of pregnancy may stop short of fits, since even when they have occurred, there are such differences in the severity of its manifestations in individual subjects.

Goitre.

The goitre was thought to have increased rapidly during pregnancy. The fact that she had a goitre has very little if any bearing on the views of Nicholson that eclampsia is due to "thyroid inadequacy." I suspect it was really an adenoma, and that the new tissue had shared with the thyroid gland the usual hypertrophy of pregnancy.

Urine.

There was a history of scanty diuresis for some days before admission, but the first catheter specimen obtained was pale and clear. In the first twenty-four hours before diuresis was encouraged the urea fell from 9 grains per ounce to 7 grains per ounce. It was a very noteworthy fact that when the patient in the next twenty-four hours was passing a great deal of urine (four pints) the urea rose to 8 grains per ounce. The albumin also fell after treatment was commenced, but rose to more than double when she was allowed exercise and light diet.

This brings out the point so strongly insisted upon by most writers that, to control metabolism and nitrogenous waste, rest complete and continuous is essential.

C A S E 8.

Mrs. Hall ,

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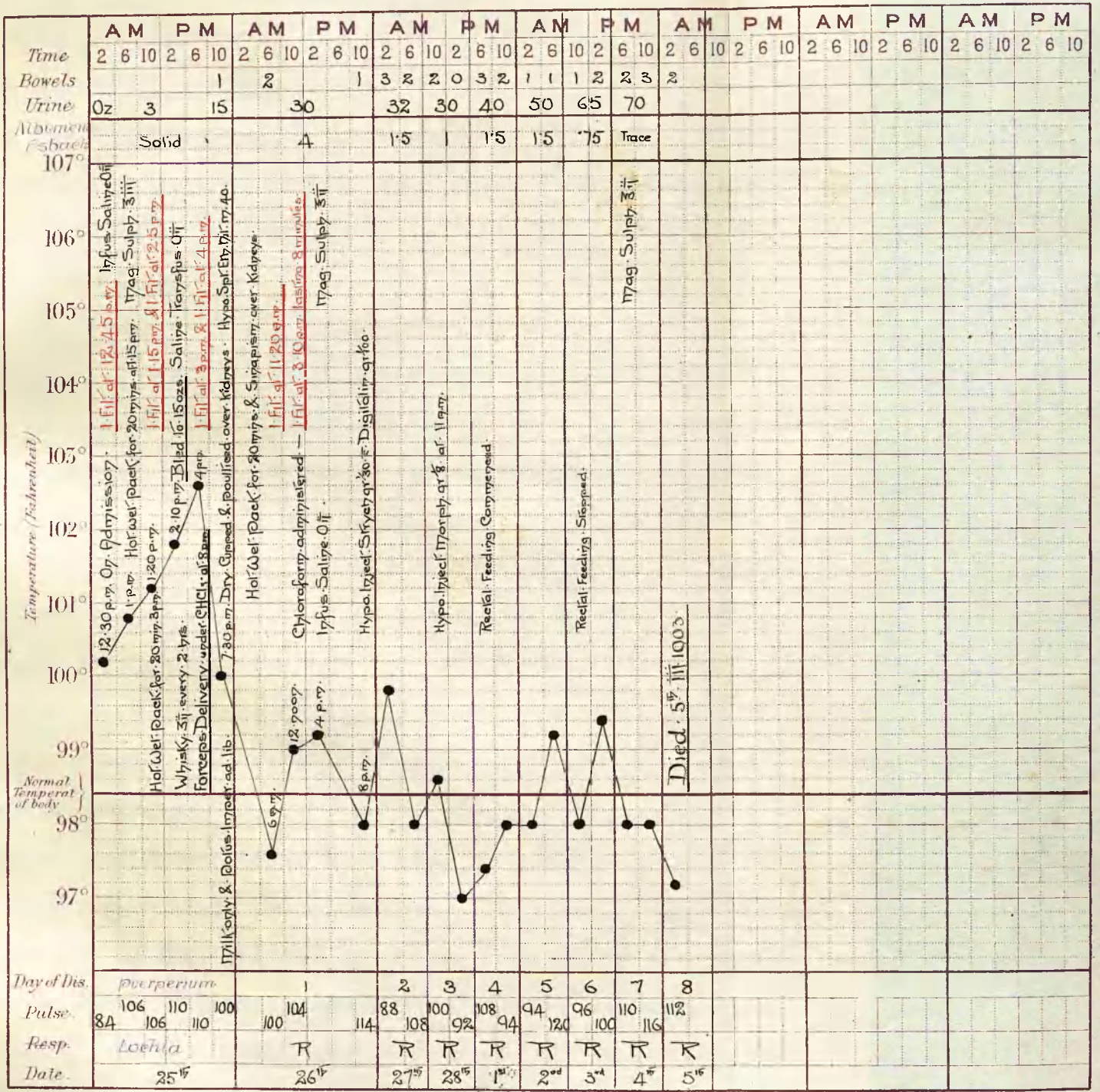

Entered at Stationers Hall. Printed and Published by Wodderspoon & Co 7 Serle Street Lincolns Inn. Goulds Clinical Chart.

Mrs. Hall, IIpara, aet. 24, was admitted to Glasgow Maternity Hospital at 12.30 p.m. 25th. Feb'y, 1903.

She was six and a half months pregnant.

She had had a miscarriage at the 4th. month in May, 1902 - eight and a half months ago.

For the last eight days there has been a continually increasing oedema of the legs and feet. Until this oedema made its appearance the pregnancy was quite normal and the patient in good health.

On the morning of the 24th. oedema of the eyelids was first noticed by the patient's mother, and she then complained to her of headache. The headache seems to have lasted the whole day, but there was no complaint of epigastric pain, dizziness, amaurosis, or tinnitus aurium, and no vomiting occurred.

On the morning of the 25th. at 3 o'clock, after a very restless night, the patient complained to her husband of intense frontal headache.

She raised her hands to her brow and kept them there for a few minutes, when a convulsion occurred, which, he says, affected her whole body.

In the next three hours four similar fits occurred, after each of which she became semiconscious, but at 6 a.m. a much longer and more severe seizure occurred and this was succeeded by others of equal intensity about every twenty minutes until her admission. After 6 p.m. she never regained consciousness.

On admission at 12.30 p.m. the patient was in deep coma, and could not be roused by stimulation. She had had about twenty-four convulsions. Her face was pale and the lips and tongue livid, the latter somewhat lacerated at the tip; bloody

frothy mucus was issuing from her mouth. The pupils were moderately contracted and equal but did not respond to light; there was external squint of the left eye. No conjunctival oedema was present, but the sclerotics were bloodshot.

There was slight but general oedema, most marked in the face and legs. The respirations were stertorous and laboured and numbered 36 per minute.

The pulse was 84, regular, and of very high tension; there was strong pulsation visible in the carotids. The apex beat was in a normal situation and thrilling in character. A presystolic murmur was present and there was marked accentuation of the second sound in the aortic area. There was no dulness at either of the bases and no râles could be distinguished. The temperature was 100.2°.

The uterus reached about two inches above the umbilicus and contractions were present. The position of the child was a "first vertex," and the foetal heart could be heard in a corresponding situation. Per vaginam, the membranes were unruptured, and the vertex was presenting through an os capable of admitting three fingers, which had thin, regular and rigid margins. The catheter was passed but there was no urine in the bladder.

About fifteen minutes after admission the pupils were observed to dilate to a medium size, and about ten seconds later the head and eyes were turned stiffly to the left, the mouth became wide open, and the elbows and wrists flexed and rigid, the hands clenched, the thumbs not being in the palms.

This condition of tonic spasm lasted about five seconds. The eyelids then began to twitch very rapidly and immediately

after the head began a to-and-fro movement to the left, never passing the middle line. The mouth became firmly closed, and the eyes were upturned to the left. The contractions now affected the arms, and with each there was flexion of the wrist and elbow, and abduction of the arm at the shoulder. The left arm made more extended movements than the right; it was estimated that the contractions would occur at the rate of about 200 per minute at their height.

About five seconds after the movements of the arms had begun, clonic contractions of the muscles of the back and legs ensued of such a nature that the pelvis was lifted clear of the bed at each spasm.

The latter movements continued for about fifteen seconds and were the first to subside, the movements of the arms at the same time becoming slower and soon afterwards stopping.

The movements of the head and neck which were the first to begin were the last to cease, continuing intermittently for about five to ten seconds after all other motion had stopped.

During this time the patient was becoming very livid; after respiration was again established it was some minutes before she regained any good colour at all. The pupils at the close of the seizure became contracted again. The carotid arteries were pulsating very strongly; the pulse which was hardly altered in quality was 106; the temperature had risen .6 of a degree, and was now 100.8°.

At 1 p.m. two pints of saline solution was infused under the right breast and 3 oz. Epsom salts given by the stomach

tube. Almost immediately after this another severe seizure occurred lasting about three and a half minutes. The pulse after it was still 106; the temperature had risen to 101.2°. At 1.30 p.m. the patient was put into a hot wet pack for twenty minutes; there was no reaction to this. At 2.5 the patient took another fit equal in intensity to the last, the temperature rising to 101.2°.

As the fits were continuing with unabated severity, and as the last three had occurred within an hour and a quarter, it was determined to bleed her. At 2.15 the right median basilic vein was opened and 15 oz. of blood withdrawn. The great tension in the vascular system was evident here, for on opening the vein the blood rose in a steady stream to about eighteen inches; it was very dark in colour. With the object of diluting the toxaemic condition of the blood two pints of saline solution was at the same time run into the vein.

At 3 p.m. the fourth fit since admission occurred; it was not so severe as the others. She then had another hot wet pack for half an hour, the skin reacting much better this time. At 3.45 the catheter was passed and 3 oz. of very dark brown, opaque, bloody urine was withdrawn It turned solid on boiling; no bile was present and it produced no change on Fehling's solution. Microscopically numerous red blood corpuscles, leucocytes and epithelial cells were present. A great many casts were also found, mostly granular or of the epithelial type undergoing granular degeneration.

At 4 p.m. the fifth fit since admission occurred; it lasted about a minute and a half, and was not nearly so severe as the last. This was the second convulsion since she had been

bled; it was much mitigated in force and duration, and occurred at an hour's interval from the last.

The patient lay in a state of complete stupor until 8 p.m. She had had no more fits, but did not appear to be any less deeply comatose. It was then found that the os was well dilated and the head in the cavity. The membranes were ruptured, forceps applied, and a small female child delivered.

There was a short third stage and there was no tendency for the uterus to relax, even when uncontrolled by the hand. The temperature post partum was 100°, and the pulse regular, of high tension and 100.

The patient was then dry cupped over the kidneys and 40 m spirit. aether. nit. given hypodermically. A hot mustard and linseed poultice was applied to the loins, and the patient was put on 2 oz. whisky two hourly. As much milk and imperial drink as she could be got to swallow was given.

th. Feb'y.

The patient has remained deeply comatose since her delivery. The bowels have thrice moved loosely, and about 10 oz. urine was collected, probably as much more being lost with the motions. A catheter specimen contained .4 per cent albumin; blood was distinct and casts numerous. In the early morning another hot wet pack was given and mustard leaves applied for ten minutes to the loins. She has been taking fluids much better. Her temperature this morning is 97.6°, and the pulse remains of high tension, regular, and 112.

At 11.20 a.m. the patient had another convulsion, the duration being five minutes, but the movements not very violent. She remained completely unconscious during the rest of the day.

At 3.10 p.m. she had another seizure which differed from any other eclamptic convulsion I have seen. In the order of the parts involved it exactly resembled the fit she had on admission, but the progression of the spasmodic wave from one group of muscles to another was very slow, the movements being confined for a longer time to each portion of the body attacked. The movements of the arms and head could not have been more frequent than twenty per minute. After eight minutes by the clock, while head and neck, arms, body and legs were still convulsed and there was no sign of subsidence of the movements chloroform was given and in less than a minute the patient became quite still.

In the evening 2 oz. Epsom salts was given by the stomach tube and two pints saline solution infused. The temperature this evening is 98°. The pulse is 114, not so hard, and at times irregular.

During the twenty-four hours she has taken about eight pints of fluid, and has passed nearly two pints of urine. Her bowels have continued to move loosely. A hypodermic injection of strychnine (one-thirtieth grain) and digitalin (one-hundreth grain) was given.

7th. Feb'y.

The patient lay very still all the night, and showed no sign of returning consciousness even on stimulation. Her bowels have moved loosely eight times, and, besides a great deal lost, 12 oz. urine was collected. A catheter specimen was opaque and straw-coloured. There was a slight trace of blood and also of bile; albumin .15 per cent. She is taking fluids well and the skin is keeping gently moist. There have

been no more convulsions.

This morning the patient remains deeply comatose. The pupils are of medium size and react to but do not follow a light. She is somewhat jaundiced. The uterus is well retracted and the lochia healthy. The pulse is 88 and steadier, the temperature 99.8°.

28th. Feb'y.

About 12.30 a.m. the patient became very restless and continued so during the rest of the night. This morning she is still in deep stupor and cannot be roused to intelligence. Slight jaundice continues. She is very restless, tossing herself about the bed almost continuously. One-eighth grain morphia was given hypodermically about 11 a.m. The bowels have twice moved loosely and she has passed about a pint and a half of urine in the twenty-four hours. The percentage of albumin is .1. A faint trace of blood is present; no casts can be found; the presence of bile is still evident.

She continues to drink freely.

This afternoon there was a sharp epistaxis, about a cupful of blood being lost. Nothing could be found to account for it, and it ceased without treatment.

1st. March.

Last night the front of the patient's neck became swollen, and this morning it is more so. Her tonsils are a good deal swollen but it is impossible to make a thorough examination of her throat. She has no difficulty in breathing but swallowing seems troublesome and painful, and she is not taking nourishment well. She is not conscious but is not so

deeply comatose; her eyes follow the nurse sometimes and it was thought that she tried to speak about 7 a.m. She is very restless again, but it cannot be made out that she is suffering any pain. She is passing abundance of urine, the albumin being one and a half per thousand; no blood or casts are present. The uterine condition is satisfactory. Rectal feeding was started this evening as supplementary to the small amount of nourishment that she can be got to swallow.

The temperature is normal, and the pulse strong and regular, about 100 per minute.

1d. March.

The patient was very restless all night, but is a little quieter and apparently clearer to-day. No fresh condition has manifested itself. Her neck is much swollen; the general oedema has hardly improved since admission notwithstanding renal and intestinal activity.

This evening when asked if she had a headache the patient said "No."

rd. March.

To-day the patient seemed better and recognised her husband, although she was unable to speak to him. She is very restless and complains of headache. The jaundice has disappeared to-day and the oedema is distinctly less. Her throat seems better and she is willing to take plenty of fluid again. Rectal feeding has been stopped to-day.

th. March.

The patient was very restless all night, and to-day does not look well. The temperature is normal, but the pulse is

very much weaker than it has yet been.

p.m.

The patient is not so well. She is very restless and inclined to delirium. She has been sick and vomiting. The temperature is normal; the pulse 116, very weak, and irregular. The oedema is much less; she has passed three and a half pints of urine to-day, the amount of albumin being small. The lungs are quite clear.

h. March.

The patient died to-day. She had been very restless and delirious all night, and often sick. A post mortem examination was not obtained.

C A S E 8.

nvulsions. There were five fits in the first three hours after each of which she tended to regain consciousness; then a more severe seizure occurred, followed by others every twenty minutes till admission, after which she never was otherwise than deeply unconscious.

nild. The child was alive after twenty-four seizures, though it was stillborn eight hours later during which time there were only five fits and these less severe than the others.

Temperature. The temperature rose a fraction of a degree with each convulsion after admission.

Pulse. The pulse in this case was of exceedingly high tension, and remained so after improvement began.

Bleeding. There were three severe fits within the hour previous to bleeding. Immediately after the bleeding and transfusion of saline solution, she had a fit but not so severe; the next did not occur till one hour later although they had been coming every half hour before, and this seizure was much mitigated in intensity.

The bleeding seemed to benefit this patient very much.

Coma. It was four complete days after delivery before she appeared at all improved as regards the coma.

Jaundice. There was slight jaundice and bile in the urine two days after delivery.

This was a very severe case, as evidenced by the number and severity of the convulsions, the profound coma, and the long duration of the coma. The oedema was slight but very intractable.

C A S E 9.

Mrs. Hendry,

primipara, aet 23.

Mrs. Hendry, aet. 23, primipara, was admitted to Glasgow Maternity Hospital at 3 a.m., 3rd. March, 1903.

She was within a month of full time.

She has never had symptoms of any kind pointing to renal disease and has never had a fit of any kind. Except for occasional fainting turns she has always been a healthy woman.

Her pregnancy seems to have been quite a normal one until three weeks ago when she noticed in the evening that her feet and legs were a little swollen; this had disappeared by next morning but returned later in the day.

The oedema soon became more constant and its disappearance in the morning less complete. For the last week there has always been a good deal of swelling of the lower extremities, and in the last few days this has been increasing quickly. She never noticed any swelling of the eyes, but her friends had noticed that her face was somewhat swollen on the evening of the 2nd. March.

During the last week she has had vague abdominal pains, which for the last three days have been more definitely localised to the epigastrium. She had no headache, disturbance of vision, singing in the ears, or pain in the back.

On the 2nd. March the epigastric pain became more intense, and she felt very ill generally. In the evening both these symptoms became very severe; her face was then oedematous. She was quite conscious and complained of giddiness and pain in the stomach. Her doctor saw her about 11.30 p.m., and gave her some pills, soon after taking which she had a sharp attack of vomiting. She then lay drowsy and restless until about 1.30

a.m. 3rd. March, when she took a convulsion. Four others followed within an hour, during which time she never became conscious. She had a sixth in the ambulance waggon while being conveyed to the Hospital, and on admission at 3 a.m. was quite unconscious.

There was slight oedema of the face, legs and feet. The face was rather cyanosed, the pupils contracted, equal, and responsive to light.

The pulse was 100, regular, and of moderately high tension; the temperature was 98.6°.
Auscultation of the heart and lungs revealed nothing abnormal. The abdomen was about the size expected at the eighth month. No foetal heart sounds or movements could be distinguished. On vaginal examination the os admitted the tip of the finger; the cervix was still recognisable and the child was presenting by the vertex.

Immediately after admission the patient had a typical eclamptic convulsion. She was then washed and put to bed, and almost at once had another seizure.

Under chloroform two pints of saline solution was infused under the right breast and 4 oz. Epsom salts given by the stomach tube.

4 oz. of urine was withdrawn by the catheter. It turned almost solid on boiling and contained a large quantity of blood and of granular, epithelial, and blood casts.

She had a very severe convulsion at 4 a.m., and became very cyanotic, the commencement of respiration being delayed for almost a minute after the cessation of clonic spasm. She

then slept quietly until 5.15, when she had another, even more severe seizure - the 10th. in all and the 5th. since admission.

She was then bled to 18 oz. and a pint of saline solution transfused into the vein. The catheter was passed and 3 oz. bloody urine withdrawn. She had no further convulsions for three hours after the bleeding and transfusion, and slept quietly until 9 a.m., when she had a slight seizure.

By 10 a.m. she had taken about 8 oz. of milk and imperial drink but had not passed urine; nor had her bowels moved. The catheter was passed and 6 oz. urine obtained. Two pints of saline solution was then infused and a large soap and water enema given with good result. The os was now dilated to admit two fingers. The temperature has never been higher than 99°.

During the early afternoon, the patient showed signs of returning consciouness, but slept quietly most of the time. She drank freely and had another loose motion.

About 3 p.m. labour pains set in definitely, and as the patient seemed to suffer a good deal at each contraction, and as it was feared that a prolonged or painful labour might influence a recurrence of convulsions one-fourth grain morphia was given hypodermically, with good effect, i.e. dilatation went on rapidly and comparatively painlessly and at 5 p.m. the os was fully dilated.

The membranes were ruptured, and, under chloroform, forceps applied; a small female child was easily delivered stillborn. It was then found that another child remained in utero. It also presented by the vertex and was extracted stillborn by

forceps. The placenta was moderately adherent and removed manually; there were one placenta, two amnions, and one chorion. The uterus contracted firmly.

A catheter specimen of urine obtained just before delivery contained albumin up to the U mark on Esbach's tube, a good deal of blood, and casts. After her labour the patient slept quietly until about midnight. When she awoke she was very dazed, but could answer questions quite intelligently and complained of slight headache.

4th. March.

The patient had a good night, sleeping quietly most of the time, and taking fluids well. About 3 a.m. she was sick and vomited a small amount of bile-stained mucus. This morning she is conscious though still somewhat dazed; she complains of severe occipital headache. She has passed about two pints of urine, containing a few casts, a trace of blood and .05 per cent albumin.

The patient passed a quiet day, drowsy but conscious and answering satisfactorily when spoken to. There has been no more vomiting and her headache this evening is easier.

5th. March.

The patient has had a good night and this morning feels very well. The oedema is much less, but she still has slight headache; there has been no return of the sickness. A copious diuresis is maintained and the bowels are being kept open with Epsom salts. There is only a trace of albumin in the urine, a few casts and no blood. As the skin was somewhat dry and harsh a hot wet pack was given in the afternoon. She has had a satisfactory day. The uterine condition is healthy.

6th. March.

The patient complains to-day of pain in the right breast, and there is some oedema of the right forearm and hand probably due to the ligature of the medianbasilic vein.

She has passed nine and a half pints of urine in the twenty-four hours, the albumin a faint haze and casts absent. The oedema is almost gone.

7th. March.

The patient still feels her right arm rather stiff, but, apart from this and slight occasional headache, is very well. A trace of albuminuria is still present; all oedema has now disappeared and she is having light diet.

9th. March.

Progress is quite satisfactory. The fundus of the uterus to-day (6th. day of puerperium) is on a level with the symphysis; the lochia are healthy. A trace of albuminuria persists.

The last incident the patient remembers, previous to her convulsions, occurred about 8 p.m., 2nd. March; she states that her husband says she gave him his supper after that, but she has no recollection of it. She thinks that for about three weeks prior to the onset of convulsions she was passing rather more urine than usual.

17th. March.

Since last note the patient has been very well, and has been kept in bed and on light diet only on account of a faint haze of albumin still present in the urine.

24th. March.

For the last week the urine has been quite clear of al-

bumin. The patient is feeling very well and for some days has been going about the ward.

She was dismissed to-day.

C A S E 9.

Prodromals. No history of previous renal disease, but well marked premonitory symptoms.

Fits. The accession of fits was rapid - 6 in first 90 minutes.

Bleeding. There were 6 fits from 1.30 till 3 a.m.
There were 4 fits from 3 till 5 a.m.

She was then bled and there was only one more fit, and that a slight one three hours later. The bleeding seemed to have a very beneficial influence.

Urine. Diuresis began at once after treatment, but was not abundant till after delivery.

Temperature. The temperature did not rise with the fits

Coma. The coma was at first profound, but an improvement was manifested soon after the bleeding and cessation of fits.

Headache, etc. The headache persisted for two days post partum.

The oedema disppeared in three days.

There was the usual loss of memory for

events preceding the convulsions.

Involution. There was the customary rapid involution.

There were in all eleven fits, and moderate coma, but from the first the patient reacted well to treatment.

SYNOPSIS OF CASES.

No	Age	Birth	Period	Onset of fits	Prodromals	Previous health	Urine	No. of fits, ante partum	No. of fits, post partum	TIME BETWEEN last fit and delivery	TIME BETWEEN delivery and last fit	TREATMENT MEDICAL	TREATMENT OBSTETRICAL	RESULT Mother	RESULT Child	REMARKS.
1	28	1	7 mths	Pregnancy	Oedema, Headache, Nausea, Malaise.	Good	Solid. Casts & blood absent. Bacter-:uria.	5	2	-	3½ hrs.	3 oz.mag.sulph., Milk, Potus Imperialis.	Rapid dilatation a la Bossi, Perforat'n of aftercoming head.	Well	Dead	Coma always tended to lessen after the fits: was never profound except just after a fit. The 2 post partum seizures were slight. No retinitis.
2	22	1	Full time.	Labour (1st)	Not well marked. Malaise for 4 hrs.	Not ob-tain :able.	Solid. blood casts.	4	16	-	10½ hrs.	3 oz.mag.sulph., Sal.infus:Enema, Hot wet pack, Dry :cupping, poultic-ing kidneys. Free stimulation.	$CHCl_3$ & forceps on admission, os being fully dilated.	Died	Well	Pneumonia ensued on the second day of puerpery, when fits and coma were long past. Died on fifth day.
3	42	8	8½ months	Pregnancy	Headache, Vomiting, Epigastric pain, Pro-found mal-aise, Stupor	Renal disease for some years.	Solid. blood, casts.	5	4	-	7 hrs.	4 oz.mag.sulph., Sal.infus., Enema, Hot wet pack.	$CHCl_3$, Manual dilata-:tion. Forceps.	Well	Dead	C.V. 4". Large child (10½ lbs.). Slight hydramnios. Melancholic for first four days of puerpery. Discharge of blood p.v. and purpuric rashes.
4	19	1	8½ months	Labour (1st)	Headache, Epigastric pain, Vomit-:ing, Oedema restless.	Good	Solid. blood, casts. Urea, 5 gr.pr. ounce.	9	-	1 hr.	-	3 oz.mag.sulph., Sal.infus., Hot wet pack, Dry-cupping of Renal region, Poultic'g.	$CHCl_3$ & forceps on admission, os being fully dilated.	Well	Died on 5th. day.	Never conscious after first fit though coma never profound. Patient could speak and swallow 1½ hr. post partum. Left retinal haemorrhage.
5	40	13	about full time.	Puerpery (1st.day)	Headache, Oe-:dema, Vomit-:ing, Blind-ness, Pain in epigast".	Nephrit-:is, Bad health during some years.	Albumin .35% blood, casts.	-	3	-	15 hrs.	3 oz.mag.sulph., Saline infusion, Hot wet pack.	-	Well	Dead	13 children in 19 years, longest interval between any two consecutive births being two years. 90 minutes after first fit was quite conscious but memory for recent events gone. 3rd. fit nine hrs. later; then stupor for 15 hours. No retinal changes notwithstanding long history of renal disease.
6	22	1	7½ months	Pregnancy	Headache, Oe-:dema, Vomit-:ing, Amaur-osis, epigas-:tric pain, drowsiness.	Scarlet fever as a girl. Nephrit-:is. Oedema.	Solid. blood, casts. Urea 6 gr.per ounce.	10	-	3½ days	-	$CHCl_3$ (outside). 4 oz.mag.sulph., Saline infusion, Hot wet pack.	Expectant, (natural delivery).	Well	Dead	"7 fits in one hour." Retinal haemorrhage in left macular region. Retro-placental bloodclot.

No	Age	Birth	Period	Onset of fits	Prodromals	Previous health	Urine	No. of fits ante Partum	No. of fits post partum	TIME BETWEEN last fit and delivery	TIME BETWEEN delivery and last fit	TREATMENT MEDICAL	TREATMENT OBSTETRICAL	RESULT Mother	RESULT Child	REMARKS.
8	24	2	6½ months	Pregnancy	Oedema, Headache (frontal), Restlessness.	Good	Solid. Blood, casts.	29	2	-	18 hrs.	3 oz.mag.sulph.,Sal. infus.,Hot wet pack, Bled to 18 oz., Sal. transfus.,Drycupping and poulticing,Spt. aether.nit.hypodermically,Stimulation with strychnine,digitalin and whisky.	Expectant. $CHCl_3$ and forceps when os fully dilated.	Died	Dead	2 prolonged fits 14 and 18 hours after delivery. Jaundice from third to 6th. day of puerpery. Epistaxis on third day. Prolonged coma after delivery and cessation of fits. Subconjunctival ecchymosis on admission.
9	23	1	8 mths	Pregnancy	Oedema, Epigastric pain,Vomiting,Drowsiness, Restlessness.	Good	Solid. blood & epithelial casts, Blood.	11	-	9 hrs.	-	3 oz.mag.sulph.,Sal. infus., Bled to 18 oz., Sal.transfus., Morphia ¼ gr.	Expectant. $CHCl_3$ and forceps when os fully dilated.	Well	Both dead.	Twin pregnancy. Bleeding seemed to exert a decided and immediate influence in checking the fits.

No	Age	Birth	Period	Onset of fits	Prodromals	Previous health	Urine	No. of fits ante Partum	No. of fits post partum	TIME BETWEEN last fit and delivery	TIME BETWEEN delivery and last fit	TREATMENT MEDICAL	TREATMENT OBSTETRICAL	RESULT Mother	RESULT Child	REMARKS.
1	20	1	Full time.	Labour (1st)	Headache, Oedema, Sickness, Vomiting.	Renal dis:ease for some yrs.	Albumin.	1	-	3 days	-	2 oz.mag.sulph.	Expectant (natural delivery)	Well	Well	Twenty-hour hours after commencement of labour os the size of half a crown.
2	18	1	Full time.	Puerperium (1 hr.post partum)	Headache, Sickness, Vomiting.	Nephritis	Albumin.	-	17+ "Status epilepticus" for 1 hour.	-	13 hrs.	$CHCl_3$ during "status epilepticus."	-	Well	Well	Normal delivery. Jaundice, clay motions and biliuria for twelve days.
3	23	-	Full time.	Labour (2nd)	Headache, Oedema, No eye symptoms.	-	Slight albumin. Casts.	1	4	-	?	KBr. Mag.sulph. Saline infusion.	Forceps delivery 15 minutes after 1st. fit.	Well	Well	Post partum convulsions lasted about three minutes.
4	38	1	?	Pregnancy	Headache, Oedema.	-	Albumin	4	-	2½ days	-	Chloral, mag.sulph. Saline infusion.	Expectant (forceps delivery)	Well	?	4 fits at long intervals: not comatose between fits. Mental symptoms on second day of puerpery. Retinae normal.
5	22	1	Full time.	?	-	-	-	6	-	?	-	4 oz.mag.sulph. Saline infusion.	Hysterectomy (took 75 minutes)	Died	Well	C.V. 3" - "Small, anaemic, badly developed." Pulse always rapid. Died of oedema of lungs and cardiac failure, 7th.day
6	23	1	Full time.	Pregnancy	Headache, Oedema.	-	-	19	-	Died undelivered		4 oz.mag.sulph. Enema. Hot wet pack. Saline infusion.	-	Died	Died	"Delay in getting case into G.M.H." "Fits lasted from 5 to 15 mins." 19 in 3 hrs. Os undilated on admission. No pains.
7	19	1	Full time.	Puerperium	Oedema.	Enteric fever, & "bad with kidneys" 2 yrs.ago	Albumin. Casts.	-	2	-	6 hrs.	Mag.sulph. Saline infusion.	-	Well	Well	Normal labour. 1st. fit 2 hours, 2nd. fit 4 hours post partum. No retinitis.
8	32	4	Near Full time.	Labour (1st)	Headache, Oedema.	-	-	4	-	?	-	Mag.sulph. Enema. Saline infusion.	$CHCl_3$ Rapid manual dilatation. 1st. Version: 2nd. Forceps.	Died	Both dead.	During 8 hours the os had not at all dilated, and as no progress was made, artificial dilation was adopted.
9	-	1	Near full time.	?	Headache, Amaurosis, Vomiting, Coma for 3 hours.	Good	Albumin.	7	-	1 hour	-	Mag.sulph. KBr. Chloral. Hot wet pack. Sal.infusion.	Symphysiotomy.	Died	Well	C.D. 3⅞". Os fully dilated when operation performed. No fits post partum, but wildly excited. Temperature rose on 2nd. day; died on 5th. day.
10	21	1	Full time.	End of first stage.	-	-	Albumin.	4	3	-	9 hrs.	Chloroform.	Expectant. (forceps delivery)	Well	Well	Labour began 10.30 p.m. 27th: 4 fits from 7 a.m. till 9.30 a.m. 28th: Os fully dilated at 10.30 a.m.-12 hours from 1st. fit. Almost conscious at 4 p.m. Improvement rapid.
11	-	1	8½ mths.	Labour (1st)	Slight oedema only.	Has had a fit at her menstrual period for 5 years.	Marked albumin.	9	-	10 minutes	-	-	Forceps delivery	Well	?	Hemiparesis of right side when she became conscious, ultimately disappearing.

No	Age	Birth	Period	Onset of fits	Prodromals	Previous health	Urine	No. of fits ante partum	No. of fits post partum	TIME BETWEEN last fit and delivery	TIME BETWEEN delivery and last fit	TREATMENT MEDICAL	TREATMENT OBSTETRICAL	RESULT Mother	RESULT Child	REMARKS.
12	34	-	6½ months	Pregnancy	-	-	Albumin	6	-	1 month	-	3 oz.mag.sulph. Potus Imperialis.	-	Well	?	6 fits in 7 hours: diuresis established: oedema disappeared. Dismissed undelivered. Confined at home 1 month later.
13	20	1	Full time.	Labour (2nd)	Headache, Epigastric pain. No oedema.	-	Albumin	1	-	10 minutes	-	-	Forceps delivery. $CHCl_3$	Well	Well	1 fit when head was on perinaeum. Immediate delivery.
14	21	1	8 months	?	Epigastric pain & dis:comfort, Headache.	Good	Albumin	13	-	10 hours	-	Mag.sulph.Hot wet pack.Sal.infusion. Morphia.	Expectant. $CHCl_3$ & forceps when os fully dilated.	Well	Dead	"It was observed that any stimulation of the uterus by hand caused extreme restlessness which subsided on leaving it alone."
15	21	1	Full time.	Pregnancy	Oedema, Scanty urine for 4 days.	"Enuresis for many years."	Albumin	20	-	41 hours	-	Mag.sulph. Saline infusion.	Expectant.	Well	Dead.	20 fits in 30 hours. Oedema lessened and consciouness returned. Labour began 6 hours later and was completed in twenty-four hours.
16	25	1	7 months	Pregnancy	Headache, Vomiting, Oedema.	Oedema of legs at times	Albumin	2	-	4¼ hours	-	Saline infusion.	Rapid dilatation, Version.	Well	?	Albuminuria for three weeks before admission, improving under treatment. 2 fits at 6 and 8.30 a.m. Delivery at 12.45 p.m.
17	30	6	6 months	Pregnancy	Headache, Vomiting. No Oedema	Good	Albumin	7	-	23 hours	-	Mag.sulph. Saline infusion.	Expectant.	Died	Dead	7 severe fits in five hours. Child born 23 hours later. Passed very little urine for 2 days, restless and vomiting. Died on third day. P.M. kidneys cystic.
18	30	1	8½ months	Pregnancy	Amaurosis, Headache, Vomiting for 24 hr.	-	Albumin lasted till dis:missal.	8	-	2 days	-	Saline infusion. Morphia ¼ gr.	Expectant.	Well	Dead	8 fits in 16 hours, the last ½ hr. after infusion. Gradual improvement for two days. Delivery of macerated foetus. "Improvement was not more rapid after than before delivery."
19	26	5	?	?	Headache, Vomiting, No eye symptoms.	Aborted at 4th. mth. last 3 times	Albumin	3	8	-	12 hours	-	Forceps delivery. $CHCl_3$	Well	Well	The 3 fits ante partum were "slight;" the 8 fits post partum were "graver and more severe," the 1st. occurring shortly after delivery.
20	20	2	6 months.	Pregnancy	Headache, Nausea, Vomiting, Oedema.	-	Albumin (Solid)	9	19	-	16 hours	Chloral,KBr.,Tr. Verat.Virid. Mag. sulph.Sal.infus.re-:peated post partum	Accouchement forcé	Well	Dead	After 9 fits in 8 hours accouchement forcé. 19 fits followed in the next 16 hours. Tr. Verat.Virid. 10 m had little effect on the pulse.
21	17	1	Full time.	Pregnancy	Occipital headache.	-	Albumin, gone in 3 days.	10	4	-	17 hours	Saline infusion. Tr.Verat.Virid.m 10. Morphia ½ gr.	Accouchement forcé	Well	Dead	10 fits in 11 hrs. The first 7 at 20 min.intervals, the last two"perceptibly delayed by morphia." Accouchement forcé: 4 fits in next 17 hours.
22	16	1	Full time.	Labour (2nd)	No Oedema.	-	Albumin	2	6	-	14 hours	Mag.sulph.Sal.infus. Tr.Verat.Virid.m 10.	Forceps delivery. $CHCl_3$	Well	Well	C.D. 4½". Second stage much prolonged. Fit occurred suddenly when head was on perinaeum. Immediate delivery.

No	Age	Birth	Period	Onset of fits	Prodromals	Previous Health	Urine	No. of fits ante partum	No. of fits post partum	TIME BETWEEN last fit and delivery	TIME BETWEEN delivery and last fit	TREATMENT MEDICAL	TREATMENT OBSTETRICAL	RESULT Mother	RESULT Child	REMARKS.
23	22	1	Before full time.	Pregnancy	General oedema.	-	Copious albumin	10	5	-	29 hours	Sal.infus.Mag.sulph. Hot wet pack. Tr.Verat.Virid.	Accouchement forcé, Craniotomy.	Died	Died	C.D. 4¼". 10 fits: accouchement forcé: well for 1 day: 3 fits: Saline infusion: improvement for 6 hours: then two fits: nystagmus and conjugate deviation immediately before death which took place 8 hours after the last two fits.
24	20	1	Before full time.	Puerperium	Oedema	-	Albumin	-	15	-	19 hours	3 oz.mag.sulph. Hot wet pack.Tr.Veratri Virid. Morphia,KBr. Chloral hydrate.	Forceps delivery $CHCl_3$	Well	Well	Large child. First fit 4 hours post partum.
25	19	1	Full time.	Labour (1st)	Not reported.	Apex beat outside nipple line.	-	9	-	3 hours	-	Saline infusion, Steam bath.	Forceps to head in cavity.	Died	Dead	"Shortly after delivery of placenta pulse began to flag, patient became weaker and death supervened." No haemorrhage, no injury or laceration to any part. Chloroform was given for less than one hour.
26	22	1	Full time.	Labour (1st)	None	-	Albumin	5	-	3 hours	-	Saline infusion. Chloral, KBr.	Accouchement forcé	Well	Well	Fits began 1½ hr.after labour commenced:5 fits in 2 hrs: infusion: fits then stopped. C.V.3½".Acc.forcé 3 hrs.later.
27	23	1	8½ mths.	Pregnancy	Oedema for 1 month. No headache or vomitg.	-	Albumin	8	11	-	30 hours	Saline infusion. Mag.Sulph.	Accouchement forcé. Forceps to both children.	Died	?	8 ante partum fits in 5 hours. 11 post partum fits in 30 hours.
28	30	4	8 mths.	Pregnancy	None	Always miscarried at 5th.mth.	Very albuminous	4	-	3 days	-	Mag.sulph.Sal.infus. Hot wet pack.	Expectant.	Well	Dead	Natural labour. Slight oedema and dimness of vision on the day of delivery.
29	26	1	Full time.	Puerperium	Oedema, Headache, Amaurosis.	Renal disease for 3 years.	Albumin (solid)	-	2	-	17 hours	4 oz.mag.sulph. Steam bath. Saline infusion twice.	-	Well	Well	1st. fit 15 hours post partum. 2nd. fit 17 hours post partum. No retinitis.
30	19	1	Full time.	Puerperium	Slight oedema only.	"No cardiac disease"	Marked albumin.	-	1	-	20 minutes	Mag.sulph. Saline infusion.	-	Well	Well	Albuminuria disappeared in two days.
31	30	1	6 mths.	Pregnancy	Amaurosis, Sickness, Vomiting. Epigastric pain,for 2 days.	-	Albumin	5	-	30 hours	-	Mag.sulph. Saline infusion.	Expectant.	Well	Dead	Natural labour. 5 fits in 5 hours.
32	22	1	7 mths	Pregnancy	-	Fit two months ago.	Albumin	"Large No."	-	?	-	Mag.sulph. Hot wet pack.Saline infusion. $CHCl_3$	Accouchement forcé	Died	Dead	Diuresis never good although albuminuria lessened. Died on third day.

DESCRIPTION OF THE DISEASE.

Derivation and Definition.

The word eclampsia is derived from the Greek ἔκλαμψις, (a flashing out) and was adopted as the name of this disease to denote the sudden character, intensity and violence of its onset.

By "eclampsia" is meant the occurrence of one or more epileptiform convulsions during pregnancy, labour, or puerpery, intimately associated with pathological change in the kidneys of a nature peculiar to pregnancy.

Frequency.

Most writers give the frequency of eclampsia as one in five hundred labours. Churchill (1) in over two hundred thousand cases, found that convulsions occurred once in every 619 labours.

Predisposition.

Primiparae are more liable to the disease than multiparae. The percentage of primiparae is variously given by different observers as from seventy-two to eighty-four. Dr. Munro Kerr (2) found the percentage of primiparae in cases occurring in Glasgow Maternity Hospital to be seventy-two. In the last three years nearly eighty-four per cent of the cases have occurred in first pregnancies.

Age does not seem to have much influence in predisposition to the disease. In the three years 1900 - 1902, forty-nine per cent of the primiparous cases were over twenty-two years of age; thirty-four per cent were twenty-six or over. The average age was 23.8, the youngest 16 and the

oldest 38. In the six multiparae the youngest was twenty years of age, a IIpara; there were two cases at the age of thirty-two, a IVpara and VIpara respectively; the average age was 28.3.

Eclampsia is more frequent in twin than in single pregnancies.

Twin pregnancies occur, as a rule in this country about once in 87 cases (3).

Herman quotes 685 cases of Eclampsia from Schauta, Hofmeier, Löhlein, and Schreiber, with fifty-three cases of twins, i.e., one case of twins in every thirteen pregnancies. This makes the frequency of twin pregnancy in eclampsia about six and three-quarter times greater than ordinary.

It is rather a rare occurrence for a patient who has had eclampsia to fall a victim to it again in a subsequent pregnancy, although most obstetricians of large experience can quote one or more examples of second attacks.

Two cases are mentioned in Dr. Kerr's analysis, both Vparae,one of whom had eclamptic convulsions for the fifth time and recovered; the other had convulsions at her second, third, fourth and fifth labours and died. None of the cases in my list had had eclampsia before.

Many writers on this subject,mention as predisposing causes, anxiety, mental distress, shock, or sudden emotional disturbance. An ex-

ample of this condition frequently cited is the disgrace of illegitimate pregnancy. In eighty-nine cases occurring in Glasgow Maternity Hospital, twenty-nine were in unmarried women, i.e., about one-third; but as a large proportion of the first births in that institution are illegitimate, we must compare the numbers in primiparae only, when we find that in seventy cases of eclampsia in first pregnancies, twenty-eight are illegitimate, i.e., about two-fifths. This is certainly not a greater ratio of illegitimate to legitimate pregnancies than occurs usually in Glasgow Maternity Hospital.

Two cases are mentioned in my list, both in married primiparous women, as having had husbands at the war; and another (Case 41) is said to have been assaulted a week previously.

One of my own cases (No.3), an VIIIpara, had a great deal of worry over a drunken husband, but in her case a great many other circumstances conduced to her illness. Only one of my six primiparous cases was unmarried.

There is no doubt that epileptics are more liable to take fits when there has been any undue stimulus, emotional or otherwise, to the sensorium; but though the seizures of eclampsia are epileptiform, the diseases are not in any way analogous. The fits of an epileptic, though the disease is called "functional," are, as far as we

know at present, due to the undue liberation of nerve energy from centres in the cerebral cortex whose functions have in some way gradually become morbidly altered, and if the unusual external stimulus has brought about this liberation, it has only done so by acting on already impaired organs.

The mental anxiety, shock, or other irritation to which a woman at the end of a normal pregnancy might be subjected could hardly be expected to produce epileptiform seizures by acting on a a healthy brain.

If, however, a patient were in the preeclamptic state or had regained consciousness after one or more convulsions, we could easily conceive how such a reflex irritation, acting on partially poisoned centres, whose equilibrium is already unstable, might be the immediate, though not the essential cause of the seizures, and so cause the convulsions to be more frequent or to remain longer.

In the same way a long and severe labour might determine the onset of convulsions in a patient who was already in the preeclamptic condition; that is, in a state where the eclamptic poison has been circulating for some time and influencing the motor centres to such an extent that only some additional peripheral irritation (such as a severe labour pain) might be wanting to bring about a discharge of their nerve force.

In patients, however, who are not in this precarious condition a severe labour is not in itself sufficient to produce epileptiform seizures.

If a patient with existing renal disease become pregnant, one would suppose that she would run a greater risk of eclampsia than a woman entering pregnancy with her kidneys in a healthy condition, since the increased metabolism will throw a greater strain on already deranged organs. One of the principal excretory channels, or "defence organs," is thus already injured, and if there be any approach to a breakdown on the part of another such organ (e.g.the liver) the kidneys will be less able to deal with the excess of waste product remaining in the system. There can be no question that in some cases a chronic nephritis has been strongly conducive to the development of the acute toxaemia upon which eclampsia depends. In three of my eight cases there was a history of previous renal disease. Case 6 (Mrs. Chalmers), a primipara, aet. 22, had a scarlatinal nephritis when a young girl, had swelling of the legs at sixteen, and as early as the third month, oedema of the legs and feet began to appear. The two other cases (Nos. 3 and 5), both multiparae, had general oedema during most of their previous pregnancies, and the latter had inconstant oedema during many years even when not pregnant. In all these cases the history of the pregnancy shows a gradual sequence of (1) renal failure and in-

crease of oedema, (2) commencement and increase of toxaemia, (3) exaggeration of toxaemic symptoms (the occurrence of acute renal disease,)and finally (4) the development of eclamptic convulsions.

It is probable that preexistent chronic nephritis, more than any ordinary condition, lays the patient under a great handicap in her pregnancy and so makes for failure of the kidneys in dealing with the products of an increased metabolism.

In twelve post mortem examinations on patients dying of eclampsia in Glasgow Maternity Hospital, antecedent renal disease was found in three - twenty-five per cent.

As regards the influence of pregnancy on a chronic nephritis a very difficult question is raised, because in hospital practice one can seldom have the opportunity of examining a patient suffering from chronic nephritis prior to pregnancy, and keep her under observation during pregnancy and afterwards. All are agreed that pregnancy is a time of severer strain on the kidneys than usual, and it is only reasonable to suppose that any extra labour thrown upon diseased organs must be injurious to them.

Exactly how far the pregnancy is detrimental it is impossible to say, but I do not think it can be so much as one would at first expect, since in so many cases where there are symptoms of renal failure during pregnancy, whether eclampsia supervene or not,

the albuminuria and oedema disppear so quickly after delivery.

If, however, pregnancies succeed each other too rapidly, so that the patient for many years of her life is as frequently in the pregnant state as not, then the kidneys have no time to recuperate, and with each successive strain they must be more seriously damaged and the injury done to the whole system be more permanent.

Epilepsy is not regarded as a predisposing cause of eclampsia. In the forty-two cases tabulated, however, there are three where a history of epilepsy is given - a large proportion. One of these had fits for five years, usually one at each menstrual period; during the amenorrhoea of pregnancy there were no convulsions until eclampsia occurred. It is said that during pregnancy epileptics are less liable to take fits than at other times (4). Dr. W. A. Turner (5) gives some figures which rather throw doubt on this. Of twenty cases observed during pregnancy, nine were free from convulsions during this time, while in seven the seizures were more frequent.

Other predisposing causes mentioned by various authors are residence in large cities, deformed pelvis, tight garments, weather conditions, abuse of spirits, faulty positions of the child, overindulgence in sexual intercourse, want of exercise, constipation and prolonged retention of urine, irritation

from the intestines and bladder.

There is no end to the number and variety of such so called predisposing influences, and they are of some interest when not considered seriously as having much effect in the production of eclampsia, and when regarded only as a series of conditions differing from what ought to be and hence tending to make the patient more susceptible to disease in any form.

Time of Onset.

The convulsions may occur either in pregnancy or during any stage in the labour or puerperium. The average numbers given are twenty per cent before and after and sixty per cent during labour. It is often very difficult or even impossible to say whether the labour or the convulsions are antecedent, but these averages are taken over a great many cases and cannot be far wrong.

Convulsions occurring during pregnancy are generally in the later months, the seventh to the ninth being the most frequent. Some cases are mentioned much earlier than this. A case is mentioned by Cazeaux and Tarnier (4) of a young girl who had only reached the sixth week "in whom nothing but the extraction of the ovum could remove the symptoms." Eclampsia came on again at the same period in her next pregnancy and she aborted. Norris mentions a case at the third month, and Olshausen has seen cases at the third and fourth months (2). In the

period covered in Dr. Kerr's analysis there are several instances of convulsions occurring at the sixth month, and I find four at that period in the reports of the last three years. One case (No. 32) is reported in 1902; the patient had had one fit at the fifth month, and was admitted suffering from eclampsia at the seventh month;accouchement forcé was employed and she died.

In the great majority of cases the onset of convulsions brings on labour, if it has not already started; the result is that a great many of the pregnancies so interrupted end prematurely, probably thirty-five per cent.

Convulsions occurring during labour appear most frequently in the first stage, though there are several cases in the list where fits only began when the head was on the perinaeum (Nos. 13, 22, 26). It is important to note the differences in two such clinical types. In the former the patient has probably been in the preeclamptic condition for some time and the slight extra reflex irritation of the first pains of labour determines the onset of convulsions which were already impending. In the second type the toxaemia may not have been well marked, and the strong stimulus of the expulsive pains at the end of the second stage is required to bring about the loss of balance of the cerebral centres. If such a case be immediately chloroformed and delivered with forceps,

she will probably have few succeeding convulsions or none, but to apply the same treatment (immediate delivery) to a patient whose labour has not begun or is in an early stage would be most harmful.

Of the convulsions occurring during puerpery there is no difficulty in obtaining the percentage. Usually the fits ensue during the first twenty-four hours of the puerperium, but they may occur immediately on the birth of the child or not for a long period afterwards. My only puerperal case had convulsions beginning sixteen hours post partum. Parvin (6) mentions two cases of Bailly and Simpson occurring respectively twenty-nine and twenty-eight days after labour. It seems to be quite common for the convulsions to be delayed a week or so.

Premonitory Symptoms.

For a varying time before the onset of convulsions the patient is in the"preeclamptic state."

By this is meant that the poison circulating in her system has affected all the tissues and as its effects increase, only a determining cause, e.g.,the pains of commencing labour, is required to precipitate epileptiform convulsions.

As a rule this condition is marked by unequivocal symptoms. They are of varying importance - some only of interest in confirming our suspicions of a toxaemia, others of the gravest import, and heralding the immediate onset of convulsions. It is a rare thing for a patient to take eclamptic fits

without their being preceded by some evidences of toxaemia, but in a few cases the premonitory symptoms have been so little remarkable that the first symptom of the grave condition of the patient has been an eclamptic seizure.

The commonest, as well as the most important of all the prodromals, are headache, oedema, epigastric pain, and disturbance of vision.

Headache is very frequently present; all my cases had it.

It is most generally frontal in situation; indeed Hamilton (6) referred to frontal pain as especially characteristic. Sometimes the headache is confined to the occipital region (Case 9), more rarely to the vertex, and occasionally to one or other side of the head.

It is at first comparatively slight, but as the patient becomes more intoxicated, the pain becomes excruciating and is continually present. It may be present in the former way for weeks but when the pain becomes very severe and continuous the toxaemia is well advanced and fits are not far off.

Oedema is present in more than half the cases. It may be slight or well marked but the amount of oedema is no criterion of the imminence of convulsions, or of the violence they will exhibit. Oedema may be present for weeks or months - a symptom of chronic renal disease, but the acute

parenchymatous change in the kidneys may bring about an intense toxaemia and convulsions so rapidly that these symptoms forestall the oedema.

The oedema may affect the lower extremities only, or with them the cellular tissues of the face.

The swelling is, I think, more significant in the latter situation. It is a fairly frequent history that the patient has had oedema of the legs and feet for some weeks; then, without increase of this swelling, the face also becomes oedematous and eclamptic seizures follow within forty-eight hours.

This is well illustrated in my cases Nos.8 and 9. In the former, oedema of the lower extremities was present for eight days; the eyelids then became swollen and eclampsia supervened in twenty-four hours. In the latter case, oedema of the lower extremities commenced three weeks prior to the attack, became gradually worse and was succeeded by swelling of the eyelids and face. Only eight hours after the first appearance of puffiness of the face the seizures ensued.

One of the most important because one of the most suggestive and unequivocal of the prodromals is pain in the epigastrium.

The pain is at first slight or there may only be a sense of discomfort, but in those patients who have this symptom well marked, it becomes later so

intense that the woman is rendered prostrate with it, and cries out with pain. It may be associated with pain in other regions of the abdomen or in the loins, and may be accompanied by vomiting, or difficulty and oppression in breathing. These concomitants are most frequent when the pain has become intense and continuous, and betoken the imminence of convulsions.

Epigastric pain was present in six of my eight cases; but either this is a larger proportion than usual, or else in other reports less attention has been paid to this symptom as an important premonitory sign.

Dr. Kerr (2) found in his eighty cases that it was only mentioned thrice, and he mentions that Dührssen only found it in one case in his analysis of two hundred. On the other hand Olshausen considered the symptom "fairly general." Parvin says that it is the "least frequent of the prodromata" (6). It is similarly mentioned in other works on obstetrics as one of the least frequent and least important of the premonitory symptoms. 25 of the 42 cases in my table are well reported as to the presence or absence of premonitory symptoms. In 11 of these epigastric pain is mentioned as present, and in 14 is either not mentioned among the other prodromals or is said to have been absent.

This makes a much higher proportion of cases with this symptom than what is given above; but I

am not able from a comparison of so few cases to give a definite percentage, though it would appear from these cases to be much more frequent than is usually supposed.

In all my cases where it was present it became continuous and very severe shortly before the onset of convulsions; and it appears to me that its significance is very similar to that attached to headache - both may be present for some time intermittently and not very acute but when either becomes severe and constant it is an unmistakeable warning that the patient is far advanced in toxaemia.

This symptom, therefore, even if not a very constant one, is I think a most important one.

Disturbances of vision of variable nature are present to some degree in a fair number of the cases. Like the other symptoms of the preeclamptic state they are at first slight, but as the toxaemia becomes more marked they become aggravated. These visual disturbances are not produced by any changes in the eye, but are manifestations of a poisoning of the cerebral centres.

There may be only a mist before the eyes so that the patient cannot read or sew and this may be present only at intervals (Case 6). There may be complete blindness for many hours before convulsions ensue (Case 5).

The patient previously mentioned (No.41) who

was assaulted a week prior to the development of eclampsia had "loss of vision" during that time, and in another case (No.31) there was complete amaurosis for three days before the first fit.

Between these extremes there are many varieties and gradations of vision changes. Amongst these are mentioned flashes of light and spots before the eyes, amblyopia, diplopia and hemianopia.

Meigs (7) relates a case where the patient could only see one half of the objects she looked at, but on being bled she immediately regained normal vision. Another case is narrated by Ramsbotham (8) where the patient declared that the room was "studded with diamonds" immediately before taking a convulsion.

Disturbance of vision when well marked as a rule heralds the near approach of convulsions (Case 5) and this is therefore an important symptom as indicating an advanced state of toxaemia, but slight alterations of visual acuity may be present for a long time before anything happens, as in Case 6 where for about five months the patient had at intervals a "mist before the eyes."

It is moreover a fairly frequent symptom according to the statements of most authors. Only two of my eight cases, however, had disturbance of vision prior to the convulsions, and I find in the forty-two cases tabulated that where the pro-

dromals are well reported this symptom is not more frequent than epigastric pain. The record of these cases would place epigastric pain as a rather more frequent and hence more dependable symptom than disturbance of vision.

Many of the patients during the earlier part of the preeclamptic condition have a well defined sense of general malaise; this was present in nearly all my cases. A sense of fear may accompany the feeling of illbeing.

Lightheadedness or giddiness are often present before the headache becomes very severe.

Tinnitus aurium or other alteration in the sense of hearing may be present.

Other altered sensory impressions brought about by the toxaemia and noticeable in the early part of the preeclamptic state are "pricking sensations" or "formication" in legs or elsewhere and blunting of the sense of touch (4).

Impairment of the intellectual faculties accompanies the sensory disturbances and the patient may become irritable, easily excited, agitated, impatient and restless or she may lapse into a state of hebetude only to be aroused with difficulty, easily distracted and answering incoherently when spoken to.

Often she will fall into a state of stupor for some hours before the convulsions and lie with

fixed expression, quite inert, impossible to rouse (Case 3).

Just as frequently, however, the patient retains consciousness until the onset of convulsions and appears to the bystanders quite sensible, although she will remember nothing of this period when the disease is cured and she is again conscious (Cases 4 and 8).

The Urine. The urine at some stage of the disease is always altered in some morbid way qualitatively and almost always quantitatively also.

Albumin. Lever in 1843 first showed that the urine in eclamptic patients was generally albuminous (3). Herman (9) says that in all cases at some stage of the disease the urine when boiled becomes solid with albumin. In seven of my eight cases the first specimen of urine obtained turned solid on boiling: this was usually shortly after several fits. In the remaining case (No.5) the fits had come on after labour, the patient was passing a great deal of urine and the first specimen was obtained fifteen hours after the last fit: it contained three and a half parts per thousand of albumin.

It is impossible to say in most cases when the albuminuria appeared first, but cases are recorded in which the albumin was not present until after the convulsions (3). Braxton Hicks says

that the nearly simultaneous appearance of albuminuria and convulsions.....must then be explained in one of three ways:-

1st. That the convulsions are the cause of the nephritis.

2nd. That the convulsions and the nephritis are produced by the same cause, e.g., some detrimental ingredient circulating in the blood irritating both the cerebro-spinal and the other organs at the same time.

3rd. that the highly congested state of the venous system induced by spasm of the glottis in eclampsia is able to produce the kidney lesion.

Herman quotes a case reported by Dr. Handfield Jones (9) where there was no albuminuria three and a half hours before the first fit,but apart from this he knows of "no case of eclampsia where the urine had been examined immediately before the onset of fits."

As regards Hicks' first explanation he says that in disease other than renal which causes fits - in epilepsy, cerebral tumour, etc.- the fits are either not followed by albuminuria, or the urine next passed contains only a trace of albumin. "Such fits even when occurring in pregnant women are not followed by the passage of urine solid with albumin." At the same time he admits that there is no <u>direct</u>

evidence to negative this first supposition.

The same objection might be raised to the third explanation that in spasm of the glottis produced in other ways albuminuria is not a frequent sequel. In a little girl, aged three and a half years, a patient of mine who died from convulsions in pertussis and who was in the status epilepticus for two hours, a catheter specimen of the urine contained no albumin.

The second explanation is certainly the most probable and is most in keeping with our present notions of the aetiology of eclampsia.

It falls into line with the toxaemic theory of eclampsia, the poison affecting at the same time the parenchyma of the kidney and the nerve cells, but in cases where convulsions would appear to precede albuminuria, being so violent as to affect the brain cells before the renal epithelium has suffered equally. The usual rule is that a less severe poison first causes a preeclamptic condition with well marked nervous symptoms.

Notwithstanding the likelihood of the second explanation being the correct one, it is probable that, albuminuria being present and convulsions supervening, the <u>increase</u> of albumin may be due to the convulsions, probably by heightening the renal congestion. In my case (No.7) of threatened

eclampsia where almost every symptom of impending convulsions was present, the albumin was only two parts per thousand on admission.

Herman (9) says that in patients who have had albuminuria before either from chronic nephritis or from pregnancy kidney, the urine does not solidify on boiling until the patient has had fits.

In cases which recover the albumin decreases very rapidly after the cessation of the fits and the birth of the child. Case 4 illustrates this well; on admission the urine turned solid on boiling, yet twenty-four hours later there was only .5 part albumin per thousand. The albuminuria frequently quite disappears within a week or ten days.

If, however, the fits cease and labour passes off, although diuresis is established and the percentage of albumin falls, the improvement is not nearly so marked until the uterus is emptied.

Cf. Case 6.

20th.Jan.	(after 10 fits)	-	Albumin solid.
21st. "	(after treatment)	-	" .4%
22nd. "		-	" .4%
23rd. "	(after delivery)	-	" .1%
24th. "		-	" .1%

Both serum albumin and paraglobulin are present in the urine and their relative proportion varies (9). As the latter is more diffusible than the former it

follows that if the proportion of serum albumin is greater than that of paraglobulin more damage must have been done to the renal epithelium to allow of its presence in quantity; hence if serum albumin is present in excess the prognosis is graver.

It has been said (Balfour) that the death of the foetus is succeeded by diminution in the degree of albuminuria. It is impossible to estimate exactly how much effect the death of the foetus had on the albuminuria in my cases.

In only one (No.6) was the labour delayed after the stoppage of the fits. In that case the child probably died during the fits on 20th. Jan'y (see pp.53,54),but there was not a very marked fall in the percentage of albumin until after delivery

Urea.

Equal in constancy to the presence of albumin in the urine is the decrease in the proportion of urea.

In each of my cases where the urea was estimated the percentage was largely decreased even when the output of urine was at its lowest, and as the diuresis increased during convalesence the elimination of urea rose both relatively and absolutely.

The same difficulty arises as in the case of albuminuria in determining <u>when</u> the urea becomes diminished as we cannot often get patients who are on the eve of eclampsia and estimate the changes in

the urine.

In my Case(No.7) we had this opportunity and found that the percentage of urea on admission was normal, but on the following day the urea fell to 7 gr. per oz., with aggravation of the other symptoms.

If the amount of the urea goes on decreasing after the fits have stopped the prognosis becomes grave (9).

Sugar. Sugar is said to be occasionally present, but Herman accounts for it by the reabsorption of sugar from the breasts and calls it "a transient galactosuria." In none of my cases had the urine any effect on Fehling's solution, but in some cases the blue colour is changed to purple or there is a slight reduction; this is due to creatinin (10).

Bile. In only one of my cases was bile present in the urine (No.8); this appeared along with jaundice about twenty-four hours after the last of a long series of severe seizures.

The jaundice and the presence of bile in the urine continued for four days; she died two days later.

In only one case in the table (No.2) was there bile in the urine: it was accompanied by jaundice and clay motions for twelve days: she recovered.

I do not know whether it has any significance

in prognosis.

Casts. Tube casts are present in the majority of eclamptic cases. In seven of my eight cases they were present: in the case of threatened eclampsia there were none.

Where casts were present the granular variety were always in excess. Epithelial casts were present in five of the cases.

It was noticed that in most of the cases the epithelial casts underwent granular degeneration very quickly and this accounts for the great excess of the granular variety. Examined as soon as a slight deposit had settled in the urine glass, granules were apparent in the epithelial cells and the cells were losing their outline. This is the condition one would expect from the microscopic appearance of the kidney.

Blood casts were always present with haematuria.

In all cases the casts disappeared very quickly from the urine when general improvement began. Even in the two Cases (Nos. 3 and 5) where eclampsia had supervened on chronic renal disease casts were absent on dismissal.

Blood. In seven of my eight cases blood was present in the urine. The quantity was greater in the more severe cases and Case 1, a mild one, was the only one in which it was absent. In the case of threaten-

:ed eclampsia no blood was present in the urine.

Haematuria always disappeared quickly after treatment, quicker even than the decrease in albuminuria or the increase in urea.

Urates. Urates are generally present in quantity even when the patient is convalescing and diuresis is established. There is generally a very copious suspension as well as a large deposit of pink urates.

Dr. Jardine (10) mentions this peculiarity and suggests that probably the suspension is due to the large amount of albumin in the urine.

Specific Gravity. The specific gravity is high while the diuresis is small, but falls very quickly along with the decrease in albumin when much urine is passed. In one of my cases (No.5), this fall was almost as striking as the rapid decrease in albumin. On admission the specific gravity of the urine was 1020, albumin .35 per cent: sixteen hours after saline infusion the specific gravity was 1002, albumin .15 per cent.

Reaction. The reaction is generally acid. If much blood be present, it may be neutral or alkaline. Usually after infusion of saline solution the reaction was either alkaline, neutral, or faintly acid.

In Case 1 where there was no haematuria and

where saline infusion was not used the urine was strongly acid in reaction.

Bacteruria. In this case bacteruria was present from admission till dismissal. The microbes were large, rod-shaped and motile and were present in great quantity.

Plate cultures were made, but nothing grew on incubation.

The numbers of bacilli were only slightly decreased on dismissal. The patient had never had any symptoms pointing to the presence of this condition.

Quantity. In every case the quantity of urine excreted is markedly diminished, indeed in not a few cases there is complete suppression.

The diminution usually begins two or three days before the onset of convulsions, and at the same time the urine becomes high-coloured. The amount gradually decreases until with the onset of convulsions there may be complete suppression.

During the time that the fits are at the height of their intensity there is very little or no urine secreted, no matter what treatment is adopted; but within four to eight hours after their cessation urine begins to be secreted in greater quantity, the amount gradually increasing until

within twelve to twenty-four hours a profuse diuresis is established.

This diuresis does not always begin so early as this, and particularly when the birth of the child is delayed after the fits have stopped.

If the secretion of urine does not speedily become established after the fits have ceased, and delivery is affected, every hour of delay makes the prognosis graver.

In two cases (Nos. 3 and 5) the urine was not diminished in quantity before the convulsions, but these patients were both the subjects of chronic renal disease.

Herman (9) says that such patients are "Cases in which the acute renal disease of pregnancy has attacked kidneys already" affected with "disease such as may occur independently of pregnancy."

In another case (No.4) the patient, a Ipara, with no history of previous renal disease, said that for three days before the onset of fits she was passing more urine than usual. In this case, however, albuminuria never quite disappeared, although there was only a faint trace twelve days later. It may have been only a frequency of micturition.

As a general rule certain of the premonitory symptoms are sufficiently in evidence to arouse one's suspicions of a threatened attack of eclampsia.

After a longer or shorter time (usually from one to three days) in this preeclamptic condition, a convulsion occurs. Its onset is always sudden though for about a minute before the tonic spasm occurs it is sometimes possible to see that a convulsion is imminent. During this time the eyes are fixed and staring, the pupils become dilated, and the patient is utterly unconscious; the carotids throb vigorously; the patient lies quite still.

The eyelids then begin to twitch rapidly and the eyeballs to roll. The head is thrown to one or other side and respiration ceases; the jaws become firmly clenched; the arms and body and sometimes the legs go into a condition of tonic spasm, the hands firmly closed most frequently with the thumb in the palm. This stage lasts as a rule about fifteen seconds during which time the face has become purple and the lips livid.

A wave of clonic spasm now succeeds, beginning also in the face which becomes horribly distorted; the head is then thrown from side to side and the arms become alternately flexed and extended at a rapid rate.

Respiration of a kind goes on during this stage with the contraction of the chest muscles; it is accompanied either by hissing or by a short sharp 'ah' sound. Saliva is thrown from the mouth and may be

blood-stained if, as often happens, the tongue has been bitten.

The clonic spasm may then invade the muscles of the legs and back and with each contraction I have seen the pelvis raised nearly a foot from the bed. Just as often, however, the muscles of the back and lower limbs seem to escape. This state of clonic spasm lasts most often about two minutes.

Herman (9) says that the duration of a fit is seldom longer than a minute and that the interference with respiration is such that a fit which lasted longer than two or three minutes must be fatal from asphyxia, and further that fits reported to have lasted for many minutes must be really instances of a succession of rapidly following paroxysms.

Herman is so exact in all his statements that one feels very diffident about denying them, but I have no hesitation in reporting a case (No.8) where a fit lasted for eight minutes and the patient did not die of asphyxia.

During that time oxygenation of the blood must have gone on to some extent. Respiration was short and gasping but there was a hiss at every contraction which meant that air was issuing from the chest. Besides this, after we had watched the convulsion for eight minutes by the clock, during which time the movements were quite continuous and regular, chloroform was administered and they at once ceased.

This does not quite prove that the chloroform was inhaled (though I think it was) for the fit may have been on the point of stopping in any case, but I am quite convinced that efficacious respiration must have gone on during that time to some extent, otherwise the patient could not have lived.

Regarding the time the movements lasted there can be no question, for we watched it on a clock, and I am equally certain that during the whole period of clonic spasm, the movements were quite regular, and that there was no interval at any time long enough to allow of natural breathing.

Another point which renders improbable Herman's explanation of long-continued fits being in reality a series with respirations active between, is that after an ordinary eclamptic seizure the patient does not immediately breathe but for a good many seconds (sometimes ten or fifteen) lies absolutely motionless with the face pale or cyanotic.

If then the patient were to have a <u>series</u> of convulsions so rapid in their succession as to be mistaken for one continuous paroxysm, the interval could not be made use of for respiration.

I think it is almost certain that during such a seizure oxygenation of the blood must go on to some extent and if that be the case there is nothing to prevent our acceptance of the cases which are reported

to have had very prolonged convulsions lasting even to "twenty minutes." (4).

During the convulsion the involuntary muscles are said to participate in the spasm, and it is not an uncommon occurrence for the bladder and rectum to be emptied but "this may be due to the convulsive action of the diaphragm and abdominal muscles" (6). Gowers (11) however, in describing the frequent voidance of urine in epileptic fits says that it is not due to the action of the muscles of the abdomen but to strong spasm of the bladder wall for "the urine is discharged with violence."

Braxton Hicks says that the uterus sometimes contracts vigorously during the fit.

The clonic movements cease gradually, the cessation sometimes extending over a minute and a half. The patient then lies pale and livid or motionless for a few seconds before respiration commences; the lividity then passes off gradually. She sometimes groans a little at this time, but is quite insensible to all external stimuli.

At no time during the period of convulsions or afterwards did I ever find the tendon reflexes altered.

The pulse which before a convulsion is usually hard and firm frequently becomes weak or barely perceptible at the end of a seizure; the rate is generally increased a little.

The temperature may have risen a degree or a fraction of a degree, but this is not constant. It never was higher in my cases than 102.6°. Herman thinks that cases without pyrexia are those in which kidney disease was present before pregnancy. Of my eight cases all except one had temperatures of 100° or more, including three cases with a history of previous nephritis; the remaining case never had a temperature higher than 99°and there was no history of antecedent renal mischief in her case; the highest temperature (102.6°)was in a patient with chronic renal disease.

As a rule during the whole period of convulsions the pulse is of high tension and generallly over 100 in frequency; if the fits do not stop and improvement begin the pulse later becomes soft and compressible.

It has frequently been noted that immediately prior to an attack the carotids throb vigorously and digital compression of the arteries has therefore been suggested as a means of warding off an impending paroxysm. I tried this several times in my last case, but was unable to form any conclusion as to its efficacy.

In all my cases the vascular tension was greater than normal, and in the cases where bleeding was practised this was markedly so, the blood rising a foot

high in a steady stream when the median basilic vein was incised.

After the convulsion has passed off the patient remains in a state of coma for a variable time. This is a distinct manifestation of the disease and not a result of the fits, for a patient may have six or seven fits at intervals of half an hour or so and yet recover consciousness before the advent of each, and on the other hand she may only have one convulsion and yet remain comatose for hours. This is explainable if the substances isolated from the urine by Bouchard (9) are the specific toxins of eclampsia.

A frequent history with my patients was that consciousness was regained after the first few seizures, the periods of coma becoming more prolonged after each fit, then it was quite lost and coma continued for many hours after the last fit.

As a rule the fits are not more than one to six in number and often there is only one, but as many as one hundred and sixty have been counted (12). They frequently occur about every twenty minutes to half an hour, but there may be no regularity and the status epilepticus may even obtain. The most rapid succession of fits among my patients was in the case of the Polish woman (No.2), where there were sixteen fits in nine hours, many of these lasting five minutes, and in

Case 8, where there were twenty-eight convulsions in twelve hours. In Case 6 there were seven fits in one hour.

During this time there is almost complete anuria; any specimen of urine obtained becomes solid on boiling, often contains blood,and the percentage of urea is at a minimum.

During the interparoxysmal coma the pupils are moderately contracted; immediately before a convulsion they may be observed to dilate, and they continue dilated for a few minutes after the seizure is over.

Subconjunctival ecchymoses sometimes occurs during a convulsion. This did not happen with any of my patients in hospital, but Case 8 when admitted had very much injected conjunctivae and in one place a slight haemorrhage was visible.

When retinitis is present it is that of chronic renal disease, but there may be retinal haemorrhage in eclampsia after the fits with no inflammatory affection of the disc.

In only two (Nos.4 and 6) of my cases-which were all examined-were any fundal changes found. They were both primiparae, and in each there was a small recent haemorrhage in the left macular region; in each the haemorrhage was almost invisible from absorption within a week.

In case 4, besides the small haemorrhage,there was a peculiar glistening appearance over the whole fundus which was thought to be due to an effusion of serum.

Squint and nystagmus may be present during the convulsions and may persist for some time afterwards (Case 3). Even in cases where there is no definite amaurosis, the visual acuity or field of vision may be altered; by the time the patient recovers consciousness the amaurosis has usually passed off. It was most pronounced in my fifth case who was able to distinguish objects clearly about sixty hours after the last convulsion - more than twenty-four hours after intelligence had returned.

In all my cases the blood was of a very dark colour ,and the specific gravity was always raised although in normal pregnancy it is lower than usual. During the first day or two of convalescence the specific gravity usually fell to about normal, but not below 1056.

The haemoglobin, contrary to one's expectation at the end of pregnancy (13), was always increased. Hawksley's haemoglobinometer was used so that only haemoglobin could have given the high reading.

The effect of the convulsions on the uterus is sooner or later to bring on labour, if labour has not already commenced; this makes it often very difficult to say whether labour or convulsions were antecedent.

It is mentioned in most text books as an occasional occurrence that with frequent and violent seizures labour may be rapid, owing to the uterus sharing in the strong muscular contractions, to the increase of intra-abdominal pressure, and to the lax state of the perinaeal muscles after spasm has passed off. This happening, however, seems to be rare, and it is pointed out that if the uterus go into spasm during a convulsion the cervix will at the same time become spasmodically contracted.

It would seem probable, taking both circumstances into account, that, if we have the convulsions beginning early in the first stage or before labour has commenced, the duration of labour will be prolonged or not affected, but if convulsions are frequent in the second stage, expulsion will be hastened.

It was impossible to determine this point from a study of my cases because we could not be sure whether the labour or the fits were antecedent in most of them, and in all cases except one, instrumental delivery under chloroform was had recourse to when the patient reached the second stage.

The following table shows that at any rate in cases 1,3,6,8 and 9 dilatation of the os was not brought about rapidly by the convulsions, whatever effect they may have had in Nos. 2 and 4.

No.of case.	Approximate No.of hrs. in labour before admission.	No.of hrs. from first fit till admission.	Size of os on admission.	Time from first fit till full dilatation.	2nd.Stage.
1	5 (?)	5½	Half a crown	-	$CHCl_3$ Instrumental.
2	6 (?)	4	Fully dilated	4 hours	$CHCl_3$ do.
3	?	3½	Half a crown	-	$CHCl_3$ do.
4	12	6½	Fully dilated	6½ hours	$CHCl_3$ do.
6	?	30	Half a crown	Nearly 4 days	¾ hour.
8	7 (?)	9	Crown piece	17 hours	$CHCl_3$ Instrumental.
9	?	1½	Sixpence	15½ hrs.	$CHCl_3$ do.

It is an exceptional occurrence that labour is long delayed after convulsions have occurred; even if it should pass off with the stoppage of the fits (Case 6) it will return ere long; the frequent death of the child may be in great measure the occasion of this. The longest intervals in my cases were in Nos. 6 and 9. In the former labour passed off and did not return for three days during which time neither feotal heart nor foetal movements could be made out; the child was stillborn, having apparently been dead for some days. In the latter case (No.9) labour pains recommenced about nine hours after the last fit and the patient was delivered of stillborn twins two and a half hours later. Dr. Munro Kerr (2) quotes several cases where eclampsia was cured during pregnancy, and the patient was delivered some months later. He also draws attention

to the fact that in such arrested cases it is rare for convulsions to recur when labour ensues. A case is reported in the journal of Glasgow Maternity Hospital for 1901 (No. 12 in my list) where a patient had six fits on Oct. 2nd. The convulsions passed off though albuminuria persisted until her delivery a month later. Another case is reported wherein labour was delayed for five days after the stoppage of the fits (No. 39).

As a general rule eclampsia has run its course under forty-eight hours.

In cases which recover the convulsions become less frequent and less severe and finally stop; diuresis is established; the coma lessens; and finally the patient regains intelligence. She is usually dull and somnolent for at least two days after the fits have stopped. Even when quite intelligent again and able to speak of her condition previous to the onset of the disease, there is a striking loss of memory for events occurring immediately before the attack, although during that time she may have spoken with her friends and acted quite rationally.

Herman (9) says that the"secretion of urine begins to be reestablished a few hours after the cessation of the fits; but it takes from two to four days for the secretion to become as abundant as in health."

This is not exemplified at all in my patients, but they were all treated by saline infusion except the first. Indeed in those cases where diuresis did not become pretty free about eight or ten hours after a saline infusion, stimulation of the kidneys was further attempted by drycupping, mustard poulticing, etc.

From the following table it will be seen that diuresis was in excess of health in every case within the first twenty-four hours.

No. of case.	Hours after cessation of fits.	Amount of urine.
1	1st. 24 hours	2 pints
2	1st. 24 hours	2 pints
3	2nd. 12 hours	Could not be measured. "Passing urine copiously."
4	1st. 18 hours	3 pints
5(	1st. 12 hours	"Fair quantity."
)	2nd. 12 hours	"Passing urine freely, probably about 2 pints."
(		
)		
(	2nd. 24 hours	"Fully 2 pints."
6	2nd. 12 hours	1 pint.
	2nd. 24 hours	"Diureses abundant."
8	1st. 24 hours	Nearly 2 pints.
	2nd. 24 hours	1½ pints.
9	1st. 12 hours	About 10 oz.
	2nd. 12 hours	About 2 pints.

Convalesence is usually rapid.

Headache, eyesymptoms, pains in the limbs, melancholy, and loss of memory may be present for a few days after the fits have stopped, but at the end of a week the patient is generally as well as any other woman on the seventh day of puerpery.

A noticeable and striking phenomenon is the rapid disappearance of the albumin, blood and casts from the urine; within a week the urine will probably be healthy or containing only a trace of albumin.

A condition I noticed in my cases was the rapid involution of the uterus. This will be seen on the charts. I do not know what is the cause of this or whether it occurs in all eclamptic patients; I have never seen it mentioned anywhere. Parvin (6) says that the eclamptic is more liable to post partum haemorrhage, but this does not seem to be the general experience. Jardine (10) reports a case where there was an "alarming" post partum haemorrhage, and says "In my experience of eclampsias, which has been a fairly large one, I have only seen one other case of post partum haemorrhage, although many of them have been under chloroform for a long time, and the uterus has been purposely left to relax if it would." In this case"a twin pregnancy was the cause."

This was remarked in all my patients; even without controlling the fundus manually during the third

stage and after the expulsion of the placenta it contracted firmly itself and the bleeding was less than usual. This natural "good management" of the placental stage may account for the customary rapid involution.

In some cases recovery is not so rapid or so complete. The symptoms already mentioned may remain for many weeks, or permanent damage may have been done to the brain during the convulsions and hemiplegia may persist. Only one such case is mentioned in the journals of the Maternity Hospital during the last three years and hers is rather an interesting history. She had fits for five years preceding her first pregnancy, usually one at each menstrual period. From the commencement of amenorrhoea, eight months before, she had no fits until labour commenced. She had no premonitory symptoms of eclampsia save slight oedema of the legs. There was marked albuminuria.

At 8 a.m. 6th. August, the os admitted one finger; between 11 a.m. and 5.30 p.m. nine convulsions occurred. On the patient regaining consciousness hemiparesis of the right side was noticeable which gradually disappeared, full power returning.

Puerperal insanity is more frequent after eclampsia than after normal labour(3). In the forty-two cases mentioned there are two instances of insanity following eclampsia (Nos.4 and 41), one a Ipara and

the other a VIpara.

In cases which do not recover, death may take place in several ways.

A. Of these the commonest is in COMA. Parvin (6) talks of this as a "slow asphyxia" - a phrase which is somewhat unintelligible.

Herman (9) divides cases dying in Coma into two groups, (a) one in which the Coma becomes deeper and deeper with or without cessation of fits, and the patient dies of hyperpyrexia, and the other (b) where the patient has continuous Coma with a falling temperature or subnormal throughout. Patients in the latter class he thinks were the subjects of renal disease before pregnancy occurred.

B. Many cases die of CARDIAC FAILURE. Fluid is often found post mortem both in pleural and pericardial sacs, and oedema of the lungs is an almost constant condition.

The venous congestion and engorgement of the right heart along with the high arterial tension are alone sufficient to cause these conditions, and the frequency of death from Cardiac Failure is hence not difficult to understand. It is said (4) that sudden stoppage of the cardiac action often causes death, and a footnote suggests that this may be due to spasm of the heart, since it is a muscle under nervous control and hence liable to share in the par-

oxysm.

C. Nearly every case of eclampsia has a degree of bronchitis after the convulsions, and when we remember the oedematous condition of the lungs and the already enfeebled state of the heart, it is not surprising how many patients die from capillary bronchitis or lobar pneumonia. My second case was a very sad example of this mode of death. She was considered practically out of danger after a very severe attack of eclampsia when pneumonia (lobar) supervened and she died in two days. Double lobar pneumonia and pericardial effusion were found post mortem.

D. Haemorrhage into the brain may take place so abundantly as to cause rapid death.

E. A rare form of death but mentioned by most writers, is asphyxia from prolonged fixation of the chest wall due to spasm of the respiratory muscles in a convulsion.

F. In his "Lectures on Haemorrhage and Eclampsia"(10) Jardine reports a case of which a hopeful view was taken four days after the cessation of convulsions. She was dead within twenty-four hours from perforation of a duodenal ulcer which had given rise to no symptoms even when free purgation was being employed.

G. He also reports a case (op.cit.) where oedema glottidis occurred, but subsided under treatment, the patient making a good recovery. He also mentions a

case where death occurred from this complication. H. Herman quotes a case published by Bailly(9) in which a patient died from asphyxia brought about by swelling of the tongue; it had become swollen from the haemorrhage into its substance which the bites inflicted during the fits had produced.

These latter are rarer forms of death.

SYNOPSIS OF 8 AUTOPSIES

performed in the

GLASGOW MATERNITY HOSPITAL

141.

Case.	KIDNEYS.	LIVER.	LUNGS.	H E A R T.	SPLEEN	OTHER ORGANS &c.
1 (Case No.2)	Left-Congested, hyperaemic, and almost haemorrhagic. Right-Little affected save for some diminution in the cortex.	Healthy.	Left-adherent, showing distinct engorgement & actual consolidation. Right-Partly adherent, marked consolidation of upper & middle lobes.	Pericardium contained a considerable amount of straw-coloured fluid. Heart small, right ventricle thin, dilated and fatty. Left small hypertrophied. Auricles normal. Tricuspid valve dilated, incompetent.	Healthy.	Pancreas and intestine normal throughout. Uterus well contracted and q uite healthy. "Death was due to pneumonia and cardiac failure subsequent to eclampsia."
2	Chronic parenchymatous change in both.	Normal	-	Ante-mortem clot in right ventricle, extending into pulmonary artery.	Normal size. Sago grain.	-
3	Normal except that cortex was thinner and paler	Friable. Mottled appearance. Purple, red or bluish petechiae in surface layers.	Slight hypostasis of both bases.	Diastolic. 3 oz. pericardial effusion. Large ante-mortem clot in right auricle; smaller clots in both ventricles. The valves were healthy.	Normal.	-
4	Left-Small capsule adherent; cortex thin and pale Right-Same characters.	Normal size. Pale brown. Num :erous haemorrhagic spots in capsule.	Left-Great oedema. Right-Moderate oedema and congestion of	Healthy.	Normal.	Icterus marked. Large calculus in gall bladder. Pancreas normal. Marked engorgement of pia-arachnoid; no meningeal haemorrhage. No oedema or haemorrhage in brain structure, (healthy).
5	Cortex pale, otherwise healthy.	Large size but apparently normal.	Left-"Diseased." Right-Oedematous.	3 oz. pericard. effusion. Right auricle filled with ante-mortem clot. Long A.M. clot in right ventricle extending into pulmonary artery & attached to musculi papillares. Valves healthy.	Weight 8 ozs. Normal.	-
6 (No.17)	Left-Fatty, several cysts. Right-whole substance replaced by cysts.	-	-	-	-	-
7 (No.23)	-	-	-	Nothing abnormal found save slight anaemia of brain.		
8 (No.27)	-	-	Oedematous. Fluid in both pleurae.	-	-	Cortical veins engorged over tem :poro-sphenoidal lobe. Nocerebral lesion. Fluid in peritoneum.

Pathology. The pathology of puerperal eclampsia is one of the most abstruse and difficult, as well as one of the most interesting questions in the whole domain of medicine.

Even with all the material that our Maternity Hospitals offer and with all the investigation that has been going on, it is still only the clinical aspect of the disease with which we are familiar. Indeed, so ignorant of its pathology are we that of all the phenomena we observe in treating a case, there is hardly one which can be accurately accounted for, and which we can therefore state definitely to be harmful or beneficial to the patient.

Let us look first at some of the older explanations of the disease, which for a long time have been mentioned in all text-books. These ascribed the convulsions to anaemia, hyperaemia, or oedema of the brain.

As regards the first explanation, anaemia of the brain with some "oedematous infiltration" has been a common condition observed post mortem (3). Acute anaemia, as in severe post partum haemorrhage, may cause epileptiform seizures, but there can never be any condition apart from external haemorrhage capable of producing such a degree of cerebral anaemia as to account for the convulsions of eclampsia.

In animals ligature of the carotid and vertebral

arteries produces convulsions (14) but it has been shown by Moxon (15) that vasomotor nerves cannot cause vessels to contract anything like so much as a ligature.

Hyperaemia of the brain associated with or producing an oedema was the theory promulgated by Traube and Rosenstein. The hyperaemic condition of the blood present in pregnancy, the usually hypertrophied heart, and the increased vascular tension, bring about a temporary hyperaemia of the brain and serous effusion. This effusion soon becomes so great as to press on the smaller vessels of the brain, hinder an efficient blood supply and so bring about an acute anaemia which in turn causes convulsions.

Playfair (3) mentions as objections to this theory (a) that "it does not account for those cases which are preceded by well marked precursory symptoms," (b) that "on post mortem examinations the brain does not, as a rule, exhibit the oedema, anaemia, and flattened convolutions which this theory assumes." Herman (9) first of all questions an extra hydraemic condition of the blood in eclamptics, and then quashes the whole theory by appealing to a simple rule in physics - when the transudation had gone far enough to cause pressure on the vessels, then it would cease, otherwise there would be a flow of fluid from a less pressure to a greater. He further shows that if this theory were correct, transudation would go on in

other parts of the body, even more freely than within the unyielding bony cavity of the cranium, and we should find the tendency to eclampsia increased with the amount of oedema: the very opposite is the case.

Suppose we even accepted the theory that eclampsia was caused by anaemia or hyperaemia of the brain, we are not any nearer the solution of the problem, for the question immediately arises "What is the cause of the anaemia or hyperaemia?"

Braun and Frerichs assumed that in common with the convulsions of uraemia, eclamptic seizures were caused by the circulation in the blood of urea; this was assumed to be decomposed into ammonium carbonate and that the new product was the direct intoxicant. This theory is not now received for there is no evidence that urea is ever decomposed in the blood into carbonate of ammonium, (3).

The view now most accepted is, to some extent, an advance on the lines of the former theory, in as much as the cause of the convulsions is believed to be a toxaemia, though what the toxin or toxines are is still unexplained.

This view is supported by a great many facts and observations.

First of all, the sequence of the preeclamptic state, epileptiform seizures and coma and the ultimate recovery or death is a natural order of events if we assume that during the same time there has been an

ever-increasing amount of toxin generated and circulating in the bloodstream, and an ever-decreasing elimination of this poison by the kidneys. Moreover, in a case which recovers, improvement is always synchronous with diuresis, and the amount of urea and presumably other poisons in the urine is increased thus getting rid of a certain proportion of the toxaemia.

It is not assuming too much to say that the condition of the blood must be markedly altered, for as all animal tissues are continually manufacturing and being rid of waste products-the result of metabolism-the blood must continually be cleansed of these; if then these poisons are not separated and excreted by the kidneys (or satisfactorily dealt with by the other "defence organs") as is presumably the case in eclampsia it follows that a condition of toxaemia must result since tissue - change is ever present.

In eclampsia an attempt has been made to prove this supposition experimentally to be a fact.

Tarnier, Chambrelent and many other French workers concluded that not only is the toxicity of the blood serum very considerable, but further, that the toxicity of the blood is in inverse proportion to the toxicity of the urine (16).

Many other experimenters have wrought at this subject, i.e., the toxicity of the urine in eclampsia and during normal pregnancy, labour and puerperium. The method in general has been to inject the urine

intravenously into rabbits, and the number of cubic centimetres per kilogramme of the body weight necessary to kill the animal is taken as the "urotoxic dose."

Stewart has published (17) the results of two sets of experiments undertaken to verify or disprove these results. Observing strict aseptic precautions, particularly in the second series, he injected urine at 100° F. into the peritoneal cavity to avoid all risk of clotting in the veins. He found that fresh unboiled urine caused death in twenty per cent of the rabbits, that fresh boiled urine was innocuous, and that urine kept for twenty-four hours without boiling caused a mortality of one hundred per cent.

In a further series of experiments he found that, where the urine proved experimentally to be toxic, microbes were always found by culture to be present in it, and that when the animals died a condition of septicaemia was always found post mortem.

These results show that the conclusions of Chambrelent, Bouchard, and Tarnier, who did not use the precautions which Stewart found necessary, are practically worthless as evidence of a special toxicity of the urine in eclamptics.

If the disease be a toxaemia it must be produced by the excessive formation of toxic matter, or by its diminished excretion. The great questions then arise, "What is the nature of the poison or poisons?" "Where do they come from?" "What causes their excessive

formation, or diminished excretion?" If these questions were satisfactorily answered we would have the pathology of eclampsia.

The answer to the first question has surely been long delayed, notwithstanding its difficulty. The blood is no doubt a very large haystack from which to pick the needle; but still we know that the needle must be there, and surely when the stimulus is so great and the opportunities so frequent, modern physiological chemistry should not be long baffled.

That the poison was at one time believed to be urea is not to be wondered at. A patient in the pre-eclamptic condition passes urine containing an ever-decreasing percentage of urea; when the convulsions are at their height there is almost complete anuria and a specimen of urine obtained contains a very small proportion of urea.

When the fits cease and the urinary output rises, there is an ever increasing percentage of urea, the excretion being thus raised both relatively and absolutely. There is thus a very definite relation between the excretion of urea and the manifestations of toxaemia.

It has been shown, however, by Bernard, experimentally, that it is not possible to produce eclamptic fits by the intravenous injection of urea and Bouchard (9) found "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 in the "urine during the twenty-four hours."

In cholera, where an increase of urea is found in the blood, there are no convulsions and the temperature is subnormal.

These facts show that urea cannot be the essential eclamptic poison, although its excretion has such a close relationship to the phenomena of the disease. The diminished proportion of urea in the urine is now explained not by its retention in the blood, but by its non-formation as the ultimate product of tissue metabolism.

Substances have been isolated from the blood, organs or urine from time to time which are probably to some extent causal of the symptoms, but as yet there is no definite toxin or toxines which can be blamed for every case of eclampsia.

Doleris and Butte published in 1886 the results of observations made by them on the blood of eclamptic patients (16). They succeeded in isolating "a crystalline inorganic substance" from the blood, which, injected under the skin of rabbits in a dilute solution, caused convulsions and death.

Bouchard has also isolated from the urine two substances which produce convulsions and one which produces coma (9); this would account very satisfactorily for the differences in the degree of coma and

fits in different cases.

Such research would appear to be a very rational mode of inquiry, and one would expect a greater interest to be taken in such work, either to homologate or confute these discoveries. If it could be shown that in normal human blood serum there was no such poisonous substance as that claimed to have been discovered by Doleris and Butte, a strong prima facie case could be made out for the specificity of the crystalline toxin obtained by them.

Even then further proof would be necessary, however, for such a principle might be lethal to certain animals and yet not at all poisonous in the human subject.

A micro-organism isolated from the urine has been blamed for the production of the toxines (9), but it has not been shown experimentally to be the cause of the disease.

Another bacterial explanation is that micro-organisms, not of any specific character, are present in the uterine mucosa during pregnancy; that in the later months these reach a higher degree of virulency; and that a sudden inpouring of their toxines to the bloodstream causes the disease (18).

Müller, the promulgator of this doctrine, holds that as the disease only attacks patients who are pregnant or lying in, the uterus must be the seat of the disease; and further that as the convulsions may ensue for some time after labour, neither foetus nor secun-

: dines can be blamed for it.

As puerperal fever arises from like conditions he explains the occurrence, at one time of eclampsia, at another of puerperal fever, by supposing a difference in the amount and rapidity of toxines absorbed. A gradual introduction of toxines into the system produces puerperal fever, but a sudden and large introduction of microbic poison into the circulation produces eclampsia. Each disease, he argues, produces in different degree and proportion the same symptoms, (1) nervous disturbances, (2) injury to the kidneys, (3) fever.

Albert has written papers advocating the same theory (18). As further evidence he points out that the uterus is well drained except during pregnancy; toxines are then collected and in the later months poured suddenly into the bloodstream, eclampsia resulting. He mentions two cases of eclampsia who died undelivered, post mortem examination of the deciduae showing bacterial infection. From patients who recover it is often possible to obtain a history of dysmenorrhoea, endometritis, or leucorrhoea.

If this view be correct why is it that between two homologous diseases (as eclampsia and puerperal fever are assumed to be) there should not be more frequent gradations? Yet it is not a common occurrence for eclamptic patients to develope puerperal sepsis in the usual sense, although from the frequency of

operative interference in delivery one would imagine that the percentage of cases becoming septic would be higher than usual.

Further explanation is also required of why the microbes only become so extremely virulent towards the end of pregnancy.

In the post mortem examinations tabulated not a single case is mentioned of the uterine mucosa being in an unhealthy condition, and on post morten examination of my second case the uterine condition was perfectly satisfactory. Now if microbes had been present whose toxines were so virulent as to be capable of producing eclampsia, there would certainly be some local evidence of their action if the uterus had been the infected part.

In only one of my cases (No.3 - a forceps delivery) was there ever any anxiety as to the condition of the uterus; in that case slightly offensive lochia appeared on the third day, but douching for three days soon rendered the discharge quite healthy. In every other case the lochia were healthy during the whole puerperium and in no case was there evidence of a more chronic and subsequent absorption of toxines such as one might expect under this hypothesis. The spleen is either mentioned as being healthy in these post mortems or is not said to be affected: yet we know that in cases of acute septicaemia the spleen is always congested and swollen and frequently is al-

most diffluent.

In none of my cases was there any history to be obtained pointing to a previous endometritis.

Bouchard(quoted by Herman (9)) believes that the poisons are formed in different cases (1) in the tissues, (2) in the secreting glands, and (3) in the intestine either (a) ingested with food or (b) formed by decomposition in the bowel. Assuming this to be true, it does not take us very much further, for the elements of the body not included in this category would be the last we would suspect as directly causal of the disease.

Herman (9) seems to favour the idea that the disease is really due to a poison from without, and he is led to this belief from the analogy of this disease to other acute poisonings like those of lead and phosphorus, which are also sudden in their onset and produce a rapid degeneration of the renal epithelium.

The third question "What is the cause of the excessive formation or diminished excretion of toxines?" is probably the most important of all for on the answer to this question our scheme of treatment ought to be based.

A woman who is pregnant must be more readily affected by any fresh change in her economy, for she has not only the products of her own metabolism to deal with but also those of the foetus and this must be all the more the case towards the end of pregnancy.

If then from some unknown cause any of her "defence" organs (e.g. the liver) be suddenly called upon to deal even with a small dose of some new or altered tissue product or of some extraneous poison, the task may be more than it is able for, as its powers are already taxed to the utmost by the strain of pregnancy. The result is that a substance circulates in the blood which at another time would have been changed into some inert product and been excreted by the kidneys. By this poison the renal epithelium is gradually affected and the kidneys fail to separate the toxines-products of metabolism-which they are specially called upon to deal with. A vicious cycle is thus established at first gradually affecting certain organs but more and more rapidly throwing the whole bodily economy out of gear, the last phase of the breakdown being eclampsia.

This theory perfectly explains the usual sequence of events - the preeclamptic condition and all its symptoms culminating in convulsions and coma, but how does it account for the rapid cure of the condition? The convulsions hardly ever last longer than forty-eight hours and as a rule are not prolonged beyond twelve to twenty hours. How is it then that in seventy per cent of all women who suffer from this disease the convulsions cease, the coma disappears, the bodily functions are resumed, intelligence returns, and the patient recovers perfectly, and in

subsequent pregnancies suffers no unusual inconvenience?

A definite answer cannot be given to this question. If the poison were bacterial in origin we could understand the cause better, for then, as in other acute infective diseases, the invasion is met by a reaction and antitoxines are formed which gradually become equal to and then in excess of the toxines. Crisis occurs and the patient becomes convalescent. But the bacterial theory of eclampsia is built on such a slender foundation that we cannot be satisfied with such an explanation as this.

If we consider that eclampsia has arisen from want of a proper completion of the metabolic processes, and that instead of the ultimate product of tissue change (urea) being formed, intermediate substances circulate in the blood with which the "defence" organs are incapable of dealing, we would naturally look for some occurrence during the progress of the disease which would have a profound effect in stimulating and increasing tissue change and hence bring about a cessation of the toxine production.

Of all the phenomena of eclampsia nothing is so striking as the severity of the convulsions and their general character; hardly a muscle of the body escapes in a severe convulsion. Besides this, it is

a matter of clinical observation that if the disease be not severe we have fewer fits, and if the toxaemia be extreme we have a correspondingly greater number. The temperature is also frequently raised and this is most marked when the convulsions are numerous; it is often noticed that there is a rise of temperature after each seizure (Case 8).

From what physiology teaches us is the result of muscular action, and from this rise of temperature after a paroxysm, it is evident that the convulsions must in themselves greatly increase tissue change;hence it is more than possible that the fits, by promoting a more complete metabolism, have a beneficial effect on the disease. There is nothing else which can have the same profound effect as the convulsions, and although in themselves a serious risk to the patient, it is possible that if they did not occur, a greater number of patients would die in coma from the prolonged toxaemia.

This may be the explanation of the short course which eclampsia runs, but whatever theory is approved, as being most likely to account for the inception of the disease, should be equally required to explain the briefness of its manifestations and the early return to convalesence.

One of the most recent explanations of the cause of the toxin-production is that of Nicholson of Edinburgh (19) who states that "The symptoms of the eclamptic state can be expressed in terms of thyroid inadequacy."

His argument is shortly this: Thyroid secretion is required for proper tissue metabolism. In pregnancy more "thyroidin" is therefore required, and normally the thyroid gland hypertrophies during the pregnant state. In eighty per cent of the cases where this hypertrophy does not accompany pregnancy, albuminuria and sometimes eclampsia result (Lange). If thyroidin is not supplied in sufficient quantity, urea is not formed, but intermediate poisonous bodies are the result of metabolism. Thyroidin is a powerful vaso-dilator, and if there be a diminution in its production, the unbalanced secretion of the supra-renal glands brings about a contraction of the arterioles and the urinary secretion is lessened. In these two ways - the absence of urea a strong diuretic, and the contraction of the arterioles - renal activity is much hindered, and there is still less excretion of the intermediate products. "In this way the degree of the toxaemia of pregnancy comes to be dependent,directly or indirectly,upon the quantity and activity of the thyroid secretion: the thyroid gland may therefore be given a primary role in the causation of eclampsia." (19). He also reports four cases in which thyroid extract was used either in the treatment or prophylaxis of eclampsia with excellent results, making a very interesting and convincing paper.

There was nothing to direct attention to the

thyroid except in one of my cases, that in which the patient was admitted apparently entering the pre-eclamptic condition. She had a large goitre which had been present for twelve years: during the time that she was pregnant and particularly in the last two months she thought it had increased more rapidly in size. (Case 7).

Nicholson's explanation certainly goes a step nearer the fundamental question of the pathology, for it seeks to answer the question "What is the cause of the increased formation and diminished excretion of toxines?" It must finally be admitted that however much we know of the clinical aspect of eclampsia, and however ingenious and satisfactory as regards the explanation of the phenomena of the disease are our theories of its causation, we are quite ignorant of the great questions of its pathology, and not even the most frequently assumed hypothesis, the toxaemic condition, has been indubitably proved.

Morbid Anatomy.

The post mortem appearances of the organs in women who have died of eclampsia are not uniform. In some cases very little pathological change is to be found (No.7); in others, morbid conditions are more marked.

The most constant changes found are in the liver and kidneys.

Liver. The liver shows evidence of an acute toxaemia, there being usually haemorrhages both in its deep substance and on the surface. These haemorrhages appear to the naked eye as purplish or dark-red spots and are usually surrounded by yellow areae, in which the liver cells are in a state of fatty degeneration or acute necrosis. This condition of the liver, found in the majority of autopsies, is very like the appearance of that organ in the early stages of acute yellow atrophy - also a general toxic disease, but with the local manifestations most expressed in the liver.

In it the liver is at first enlarged and is of a yellowish colour, this being due to the condition of the liver cells which are in a state of "true fatty degeneration (20), although the fat is in comparatively large drops." The toxaemia which brings about acute yellow atrophy is, however, not so acute as the eclamptic poisoning; hence after the liver has passed through this early stage of enlargement and fatty degeneration, a second stage ensues in which the liver becomes smaller than normal, and mixed with the yellow areae are patches of reddish colour. Examined microscopically the yellow parts are found to be made up of granular debris with much fat in droplets and the liver cells hardly recognisable as such. The red parts do not contain the same amount of fatty

debris (although fat granules are still present) but are composed mostly of fibrous tissue.

Such a condition of the liver as this second stage of acute yellow atrophy does not occur in eclampsia. "Phosphorus poisoning produces changes so similar that some observers have suggested that acute yellow atrophy is always due to poisoning with phosphorus" (20).

In a case reported by R. H. Bell (21), where a careful thorough autopsy was made, two wedge-shaped infarctions were found on the anterior margin of the right lobe of the liver; they were recent with a definite margin and no trace of suppuration. So rare is this condition of the liver that the writer hesitates to declare definitely that these areae were true infarctions, and all the more so as no thrombosis or embolism could be demonstrated either in the hepatic artery or portal vein.

The points in favour of their being infarctions are their wedge-shape, and the fact that they extended to the surface of the liver; microscopically too the cells in the interstices of the haemorrhage were in a state of "Coagulative necrosis."

In this case too, besides the conditionsalready mentioned, "there was a desquamative catarrh of the bile-ducts, the epithelium being shed and in places blocking the lumen." This is only further evidence of a very virulent poison passing through the liver

and causing a necrosis of the hepatic epithelium analogous to the acute parenchymatous change observable in the kidneys.

In the post-mortem examinations tabulated the liver was said to be "healthy" and "normal" in the first two, but microscopic examinations were not made. In the third and fourth cases changes are mentioned which are evidently such as are already described, viz. the presence of haemorrhages amid yellowish areae giving a"mottled"appearance.

In none of the reports is there any mention of the microscopical appearances.

Kidneys. Perhaps even more constantly affected than the liver are the kidneys.

In Dr. Kerr's series (2) there were eleven autopsies; the liver was found altered in three, and the kidneys in seven cases. In the table at the end of his article the kidney condition in these patients is shortly stated; in two of these seven there was evidence of previous renal disease. In two of my eight cases there were signs of antecedent renal disease (Nos.2 and 6).

Diseased conditions of the kidneys are more frequently found in patients dying of eclampsia than in those dying from other diseases. Among the lesions found are amyloid disease, hydro- and pyonephrosis, pyelitis, cystic disease (No.6), chronic interstitial,

and chronic parenchymatous nephritis (No.2). Dilatation of the ureters and renal pelves seems to be a frequent condition, for in a hundred and twenty-two autopsies by Schauta and Lohlein (quoted by Herman (9)) twelve cases were found to have this morbid change. So frequent indeed is this condition that Herzfeld (18) (who found in eighty-one autopsies on eclamptic patients that the ureters and parts of the kidneys were dilated in 18) believes it to be causal of the disease.

He found that in these eighteen cases (all primiparae) instead of the common iliac artery dividing at the usual position that it divided either higher or lower than normally and so exposed the ureters to the pressure of the uterus.

The commonest condition of the kidney found post-mortem is that of hyperaemia, often very intense (Case 2). This state of hyperaemia was present in two of Dr. Kerr's seven cases, and in two of my eight. There were no haemorrhages in any of the cases. Frequently the cortex is paler than usual and this is due to fatty change; this was the condition in three of the eight post-mortem examinations tabulated.

It often happens that on naked-eye observation only, nothing at all abnormal is to be noticed in the kidneys. On microscopic examination, however, changes are found - most frequently in the epithelium of the tubules - which would escape notice on macroscopic

examination.

In these instances the epithelial cells are separated from the basement membrane, and are to be found in the urine undergoing granular degeneration (markedly in Case 8). In other cases the cells are not so acutely affected but may show granular or fatty droplets in their protoplasm. These changes occur mostly in the cortex, and to a much less degree in the medulla.

In the autopsy reported by R. H. Bell (21) already mentioned, microscopic investigation showed that the renal changes were most marked in the cortex. The protoplasm of the cells of the convoluted tubules was clouded, there being frequently no nuclei visible. The glomeruli were little affected; the loops of Henle in the medullary rays were practically normal. The connective tissue was not increased and there was no infiltration of leucocytes as there would have been in a true nephritis.

Herman (9) summarises the condition of the kidneys generally met with thus:- "Although the kidneys look "like inflamed kidneys, yet the lesion of puerperal "eclampsia is not nephritis, but an acute degeneration, "such as is caused by blood poisons."

Heart. In four of these eight post-mortem reports there was pericardial effusion, from three to six ounces in amount.

In three also there was antemortem clot in the heart; this may be found in any of the chambers, or in the pulmonary artery (Nos. 2 and 4), but it seems to occur oftener and to a greater extent in the right side of the heart. There is, however, nothing distinctive of eclampsia in this condition, nor in the valvular lesions often found. Haemorrhage may be found in the endo- or pericardium.

Lungs. The lungs are always found oedematous in some degree, and bronchitis is always present; either lobar pneumonia or patches of pneumonic consolidation are frequently met with.

Pleural effusion is not uncommon (No.8) and there may be haemorrhage beneath the pleura or in the substance of the lung.

Spleen. In four of these eight cases the spleen is said to be normal and in one case (No.2) a "sago-grain" condition was present.

In R. H. Bell's case the spleen was much swollen, soft and diffulent - a condition usually found after death from septicaemia; this is the only case where I have seen this condition of the spleen mentioned as being present in eclampsia.

Brain. In one of these reports (No.4) it is stated that there was marked engorgement of the pia-arachnoid, but

no meningeal haemorrhage; this, however, may be present and be the immediate cause of death.

In another case (No.8) there is said to have been engorgement of the veins of the cortex over the temporo-sphenoidal lobes, which were pink on section from the extravasation of the colouring matter of the blood. Haemorrhage into the substance of the brain may be found.

Oedema, hyperaemia, and anaemia of the brain may be present (No.7), but it would appear that the two latter conditions depend in a great measure on whether the thoracic or cranial cavity be first opened post-mortem.

Other changes.

Changes are found in other organs of varying character which are dependent on distinct diseases or accidental causes and have little significance in the pathology of the disease.

Diagnosis.

The diagnosis of eclampsia during the period of convulsions is easy.

In Playfair's "Science and Practice of Midwifery" Vol.II. it is stated that "considerable confusion exists in the description of puerperal convulsions, from the confounding of several essentially distinct diseases under the same name," and that "in most obstetric works it has been customary to describe three distinct

classes of convulsions; the epileptic, the hysterical, and the apoplectic."

This confusion may have existed in the older textbooks, but in the modern works on obstetrics no such confusion exists, although in most it is pointed out that the epileptiform convulsions of eclampsia have to be diagnosed from the convulsions of true epilepsy, from hysterical seizures, and from the fits resulting from organic brain disease.

Epilepsy. In epilepsy there is probably a history of previous fits, frequently there is a cry preceding the convulsion, the coma after the fit is not so profound or so long, the urine will probably be clear of albumin (if there be albumin the urine will not become solid on boiling), and there is not the suppression of urine characteristic of the paroxysmal stage of eclampsia.

Hysteria. Hysterical seizures usually occur in the earlier months of pregnancy. The convulsive movements are different from those of eclampsia as they do not pursue a regular order; during the fit the patient is not so completely devoid of consciousness, she does not bite her tongue, the conjunctival reflex is present, and urine or faeces are not voided involuntarily. It is said that the fingers do not grasp the thumb in hysteria and that they usually do so in eclampsia; in a case of pernicious vomiting three months pregnant, who had a hysterical seizure in Glasgow Maternity

Hospital, I particularly noticed that the thumb was in the palm grasped by the fingers, and in two of my eclamptic cases the thumb was flexed over the fingers during the convulsions.

After the hysterical seizure is over there is no coma and "the attack may subside with the patient in tears or laughter" (6). There is no suppression of urine, and, as a rule no albuminuria.

Fits from Organic Brain Disease.

If organic brain disease, such as a cerebral tumour, be present, there will probably be a history of previous fits; there will be a longer history of headache and vomiting, and there may be localising symptoms, or optic neuritis.

Apoplexy.

Apoplexy can only be mistaken for eclampsia or vice versa in the comatose stages of the latter disease, when, if a previous history were not obtainable, the diagnosis might be more difficult, but in apoplectic coma evidences of paralysis are still present particularly in the face, and one side of the body would be more lax than the other. Suppression of urine is not to be expected in apoplexy but there may be marked albuminuria. It must not be forgotten that apoplexy may follow an eclamptic seizure.

The great essential, however, is the early diagnosis, not of eclampsia when convulsions have occurred, but of the preeclamptic condition - the pre-

sence of the acute renal disease. If a woman in the latter months of pregnancy complain of any of the premonitory symptoms, an immediate examination of the urine should be made. If epigastric pain, associated with headache, eye-symptoms, or other nervous manifestations be present, and especially if there be a decrease in the percentage of urea excreted we may feel sure that eclamptic convulsions are imminent.

Prognosis. The death-rate over all cases of eclampsia is a high one - probably about twenty to twenty-five per cent.

Death is said to occur oftener in multiparae than in primiparae. Schauta (9) in 306 cases found the death-rate in primiparae to be 37.3 per cent and in multiparae 44.9 per cent. Dr. Kerr (2) in his analysis of eighty cases found the opposite to be the case, his percentage death-rates being forty-two in primiparae and twenty-three in multiparae.

He quotes Dührssen and Goldberg who found that the death-rates were nineteen and twenty-one per cent respectively in primiparae and twenty-eight and forty-five per cent in multiparae. "Olshausen found the mortality the same in both" (2).

The age seems to have but little influence on the prognosis.

All authorities are agreed that the greater the number of fits the graver the outlook. Herman puts it

"When the fits exceed twenty in number, more than "half the patients die. Of those in which the fits "exceed fifty in number almost all so end" (9).

Charpentier, quoted by Parvin (6), says that "If the fits average one in ten, one-fourth of the "patients die; sixteen to twenty, one-third die; "twenty-one to fifty, one-half die."

Bidder (quoted in Kerr's Analysis") found that where the fits were more than sixteen in number, forty-three per cent of the patients died, while in those with less than sixteen fits, the mortality was only 7.5 per cent. Kerr found that when there were twelve fits or more the mortality was fifty-five per cent, and when less than twelve, thirty-one per cent.

Of the fifty cases in my list the patients who recovered had an average of nine convulsions, and those who died of twelve. The highest number of fits amongst those who recovered was twenty-eight; the lowest number amongst those who died was four. Of my cases the two patients who died had twenty and thirty-one convulsions respectively.

Apart from the number of fits, however, one must look also to the severity of the seizures in prognosis, for it is not infrequent for cases in which there is a comparatively small number of fits, to have a fatal issue, these fits being very severe, (i.e., Cases 8, 17, 38) while other patients having a greater

number of seizures (each lasting probably about a minute and affecting only the head and arms) recover completely (cf. Case 20).

Of still more import in the prognosis are the depth and duration of the coma. It is convenient, as well as probably correct, to consider the coma as a manifestation of a distinct toxin. This explains the frequent occurrence of a fatal issue in cases where the convulsions were few in number but the coma profound and continuous.

If the coma be deep and especially if in the interparoxysmal period the patient show little tendency to escape from it and still more if the coma continue for many hours after the fits have ceased, we must consider it as a sign of the very gravest import (e.g. Case 8). If on the other hand, there be an indication of returning consciousness after each seizure, or if the patient become fully conscious between the paroxysms, and especially if she regain consciousness soon after the final cessation of the fits, we may take a much more hopeful view of her condition.

It sometimes happens that after the fits have stopped for many hours and the patient is not deeply comatose or only dazed that an isolated seizure may occur (cf. No.41 in table and Case 8). I do not know whether this should be looked upon as especially

unfavourable in the prognosis; one of the cases cited died, the other recovered. In the table at the end of Dr. Kerr's"Analysis," such a case is mentioned. The patient, a primipara, aged twenty-eight, had had ten fits before delivery which was accomplished after forcible dilatation. She had seven fits post partum but these did not occur until the fourth and fifth days of the puerperium; on the latter day the patient died.

Another case had eight fits in labour, then two more eighteen hours after treatment and recovered.

When the convulsions begin before labour the outlook is graver than when labour is antecedent. In my list the death rate in cases where the fits commenced in pregnancy was about thirty-three per cent; when the fits began during labour it was twenty-five per cent. These figures correspond with Dr. Kerr's forty-three per cent in antepartum cases, twenty-eight per cent in intra partum cases, and 0 per cent in post partum cases.

When the convulsions begin early in labour it would seem that the prognosis is slightly worse than when they commence during the second stage. Where the facts are stated in these fifty cases we find that there were no deaths in cases where the convulsions began during the second stage; there were three recoveries. Where the fits began in the first stage,

five recovered and three died. Coeteris paribus, the most favourable of the cases occurring during pregnancy or labour are those in which the convulsions cease after or are mitigated by the birth of the child. Unfortunately we cannot be sure that the birth of the child will bring about this mitigation; hence the unwisdom of resorting to accouchement forcé before other means of treatment have been tried and found wanting.

The most hopeful of all cases are those which occur during the puerperium . Statistics from all writers coincide in this. Olshausen says that if the seizures occur late in the puerperium the chances are not so good as when they occur early (2).

The death of the child in utero is said to have a favourable influence on the course of the disease, the fits tending to cease and the degree of albuminuria to become less. It must be difficult to be sure of this because after a certain time during which there has been a series of fits, the child is almost sure to die if it be not expelled, and its death must often occur at the very time when the disease would be beginning to show an improvement in any case. It is certainly often found on reading these reports that where the treatment has been expectant, the mother has recovered and the child been stillborn, but I think it would be extremely difficult to conclude from this that the death of the foetus was the cause of the

betterment. As regards the influence of the death of the child on the albuminuria, I am unable to draw any conclusion from the notes I made of these cases. In Case 6 the child was apparently dead on admission and the patient had no more fits after that, but the usual improvement as regards diuresis and albuminuria did not follow the cessation of convulsions, but began immediately post partum. The output of urea, however, improved before delivery, there being 6 gr. per oz. on admission, about eighteen hours after the last fit, and 8 gr. per oz. the following day.

I do not think that any stress can be laid on the quantity of albumin, blood, or casts present in the urine; but there can be no question that the amount of urine excreted has a very decided influence on the prognosis.

Jardine (10) says that the main thing in treatment is to "get the kidneys to act," and if this can be done the outlook is much brighter. In cases where there is complete suppression, the prognosis is almost hopeless, and even if the resumption of the renal function be long delayed, the chances of recovery are more remote.

The temperature in itself is practically useless as a guide to prognosis for patients may die from hyperpyrexia, or die with a temperature which has never been febrile.

The influence of delivery upon the convulsions will be discussed under "Treatment."

The foetal mortality in eclampsia is very high, and this for several reasons.

First of all we must bear in mind that in a goodly percentage of cases the child has not reached a viable age; in a still greater number it has not reached full time. Besides this, twin pregnancy is more frequent in cases which develop eclampsia, and the children are therefore smaller and less well nourished.

But putting aside these causes a great many healthy children die in utero as a result of the convulsions. There can be little doubt that the convulsions are the cause of death, for we generally find that where the child is born alive, either the convulsions have come on when labour was well advanced, or else accouchement forcé has been resorted to.

In several of my cases the foetal heart could be heard on admission, but after a series of convulsions, neither foetal heart sounds nor foetal movements could be detected. In Case 8, however, the foetal heart sounds were distinct after twenty-four seizures. The death of the child must result from the interference with the blood supply to the placenta and from the want of proper oxygenation of the blood which is supplied. In a certain proportion of cases - I am sure a fairly large proportion - death occurs from haemorrhage behind and partial separation of the placenta.

In some of my cases a quantity of blackish clot was found adherent to the uterine surface of the placenta. This clot may be seen to be the result of a haemorrhage which has separated a part of the placenta from the uterine wall; indeed the placental tissue itself may be in part disintegrated by the haemorrhage. In Case 6 this "apoplectic" placenta was well marked, the rest of the organ being pale, and here and there fatty: the child had been dead for some days.

The foetal mortality in Dr. Kerr's cases was fifty-seven per cent. In these fifty cases there were thirty-one children stillborn, twins in two cases: this gives a mortality of sixty-two per cent. Eight of these, however, had not reached a viable age and craniotomy was performed in one case which reduces the death rate considerably.

Where the treatment was expectant, seventeen died; seven died with accouchement forcé; craniotomy was performed in one case and in seven the obstetrical treatment is not stated.

It will be seen from these figures that the foetal mortality will vary much in the hands of different obstetricians, for if accouchement forcé be practised it will be much lower than if we adopt an expectant treatment.

Even when children are born alive they frequently succumb in a few days. Dr. Kerr cites two cases where the children died of convulsions on the fourth and

tenth days respectively, and he mentions a case reported by Fitzgerald (23), where the child had thirty-nine convulsions in the first few days but recovered perfectly.

Jardine (10) mentions two cases in one of which the child was stillborn with both its arms flexed and rigid; in the other case the child was born "perfectly rigid all over." Jardine (10) in reporting a case (No.15) says, "When the child was two days old its urine contained a considerable amount of albumin, and it was loaded with urates." Kerr, in speaking of children which develope convulsions, says (2) "Several observers have found albumin present in such infants' urine."

In Hirst's Midwifery (22) there is a statement that albuminuria is as a rule present for the first few days of any child's life.

I had the urines collected of thirty-four newly born infants, male and female, and found that this was the case. In the urines of all the babies from which we obtained a specimen during the first two days, albumin was present in varying amount - generally a "distinct"quantity. From the fifth day onwards it was not constant, and in the only two cases from which we obtained a specimen after ten daysalbuminuria was absent.

Treatment. As so little is accurately known of the exact

pathology of eclampsia, it behoves us to keep an open mind regarding the efficacy of any scheme of treatment, for on this very account the treatment is largely empirical. Nevertheless, although we cannot employ a method of treatment, rational in so far as it is exactly calculated to influence the first cause of the disease, our treatment may be so far rational that we can count on improving some of the morbid conditions present, which, although not the first step in the causation of the disease yet tend to increase the general breakdown of the economy which results in coma and epileptiform seizures. For example, the failure of the kidneys to secrete urine is not the original causa morbi, but we know that if suppression of urine cannot be relieved speedily, the patient will die; hence a treatment directed to the promotion of diuresis may be termed rational to some extent, and certainly it is scientific.

Although, then, from want of knowledge, we cannot treat eclampsia on the same rational and scientific basis as we can treat diphtheria for example, we can at any rate lay claim to having certain methods of dealing with this disease which are helpful to recovery.

Herman (9), says that "No treatment has been proved to do good." Now in the Glasgow Maternity Hospital the death rate from eclampsia, prior to 1898, was forty-seven per cent. Since then it has been less

than twenty-four per cent. The class of patients is the same; the obstetrical treatment is practically the same, and yet the death-rate has fallen to one-half of what it was. The only great change in the treatment has been the introduction of saline infusion, and where one sees these figures and at the same time has some experience of the effect of this treatment on individual cases, one cannot but feel convinced that to say that this method has not been "proved to do good" is unfair.

If in the same class of cases and in the same general circumstances, a great fall in the death-rate be established, and that over a large number of cases, surely it is unjust to assume that a new method introduced for the first time just prior to the improvement in the results, and continued ever since, has not been largely instrumental in the improvement.

Prophylaxis. Before considering the curative treatment of eclampsia, it would be well to consider shortly, what measures may be adopted in its prophylaxis and how far these are successful. We are assuming that the pre-eclamptic condition has been diagnosed and that the patient is beginning to suffer from the effects of a morbid metabolism.

First of all the patient ought to be put to bed and kept as nearly as possible at rest. This will

diminish the amount of tissue change and there will be less call on the "defence" organs, one or more of which are on the eve of a breakdown. For the same reason highly nitrogenous diet should be avoided and the patient put on milk only or on milk diet. In order that any toxic products may be eliminated from the system as rapidly as possible, we give abundance of fluid to drink. The kidneys at this stage may have difficulty in dealing with the products of metabolism but the renal tubules are not blocked with degenerated and desquamated epithelium and there will be no hindrance to the passage of water. More than this, the waste products are not presented to the renal epithelium in such concentrated form and hence the cells will suffer less, and at the same time a greater quantity of these toxic bodies will be excreted.

For the same reason-the elimination of toxic waste products-we endeavour to keep the bowels acting freely, and one or two watery evacuations should be ensured every day by the exhibition of salines (perferably), or hydragogue cathartics.

Even in a moderately severe case this will be sufficient and improvement will immediately ensue, as evidenced by the disappearance of headache, nausea, epigastric pain or other subjective symptoms, and by the decrease in the amount of oedema and albuminuria if these be present (Case 7).

Should this treatment not prove sufficient and the subjective and objective symptoms continue or increase, further means must be taken to increase the elimination of toxines by free purgation and diaphoresis. At the same time we may appeal more strongly to the kidneys and give diuretics or even saline infusion.

I had three patients admitted to the Maternity Hospital in this preeclamptic condition, but, unfortunately, I took careful notes of only one - No.7. In each case the patient improved rapidly under the above treatment. Two were delivered in hospital without untoward symptoms, and the third improved so much that she was sent home after three weeks, about a month before full time, foetal heart sounds and foetal movements being distinct. In each of these cases, the patients on admission appeared to be in a far advanced state of toxaemia, most of the symptoms being present which threaten an early appearance of convulsions. Rectal lavage is also recommended as part of a system of prophylactic treatment (18).

If these measures do not bring about an amelioration of the symptoms, the question of induction of premature labour must be raised.

As it is practically only the child's life which can weigh against the risk of allowing the pregnancy to continue, we must remember that the child is already probably viable, and that in allowing the pregnancy to

go on the risk to the child's life is probably greater, for the percentage of foetal deaths is very high when convulsions have occurred.

If, then, no improvement follow medicinal and hygienic treatment, induction of premature labour ought to be undertaken.

I have already referred under "Pathology" to the prophylaxis of eclampsia by the exhibition of thyroid extract.

For all the different methods of treatment, after convulsions have occurred, there are strong advocates. Amongst the principal schemes for combating the fits are the administration of chloroform, of morphia, of chloral, and the employment of venesection and saline infusion. Besides these there are the questions of the obstetrical treatment, the management of the patient during the convulsions, the use of purgatives and diaphoretics, and the hot pack. There are other medicinal measures in less frequent use.

Bleeding. Bleeding was formerly regarded as the "sheet-anchor" (8) in the treatment of eclampsia, but the reasons for its employment were different from those now held.

Meigs (7) said "The remedy" for convulsions "is "to moderate the excitement by venesection and evacu - "ants.....By the abstraction of blood we can "weaken the force of the circulation of the whole "system; we can make the heart beat gently and cause it

"to send the blood in a milder current into the "vessels of the brain; we can thus diminish the "innervative function of that organ and control the "muscular excitement."

Depaul (4), says, "I have no hesitation in ex- "pressing my conviction that venesection should be "regarded as of primary value."

Bleeding is also recommended in Cazeaux and Tarnier's "Obstetrics," but the objections to it and the dangers accompanying its indiscriminate use are fully commented upon.

Spiegelberg places bleeding "First in the treat- "ment of eclampsia."

Parvin (6) says that bleeding is not universally applicable but that the abstraction of blood gains time for the use of other therapeutic means, and prevents the consequences of congestion. "The extraction "of ten to fifteen ounces of blood can only in excep- "tional cases be immediately or remotely injurious. "Profuse bleeding is not best, for the statistics of "Charpentier prove that recovery is less frequent after "copious than after moderate depletion."

Trousseau (24) says that paralysis is one of the most frequent sequelae of eclampsia, and that this may be due to an organic cerebral lesion - haemorrhage into the meninges or the substance of the brain. The cerebral congestion which is so frequently present is no more the cause of puerperal convulsions than it is of

the convulsions of epilepsy; in each case it is an effect of the seizures. "I therefore do not include "in the treatment of eclampsia general or local "bleeding intended to do away with the pretended cause "of puerperal convulsions, no more than I advise it in "epilepsy or in the eclampsia of children."

Herman (9) says, "Bleeding as a routine treatment "and in large quantities is no longer practised in "eclampsia. But in a plethoric patient with a full "hard pulse, much lividity, and pulmonary congestion, "I think a moderate venesection may do good by relieving the right heart."

Playfair (3) says, "I have no doubt that, in properly selected cases, and judiciously employed, "venesection is a valuable aid in the treatment of "eclampsia, and that it is specially likely to be "useful in mitigating the first violence of the attack, "and in giving time for other remedies to come into "action. It will be specially indicated when there is "marked evidence of great cerebral congestion and vascular tension."

To summarize, the arguments in favour of bleeding are, (a) that the congestion of the lungs and right heart is relieved, and (b) that time is gained for other remedial measures to produce their effect, the patient in the meantime (c) being relieved in regard to the frequency and severity of the fits and the depth of the coma. The objections to venesection are,

shortly, that (a) we do not know that high arterial pressure is a bad thing for the patient (Herman), that (b) the tension in any case soon becomes what it was before from absorption of fluid from the cellular tissues, that (c) the composition of the blood is deteriorated, (3), that (d) even at the best it is only a temporary expedient. From a study of the reports of the cases I have tabulated, and from the very beneficial result which venesection exercised on my two cases (Nos. 8 and 9), I think bleeding is to be strongly recommended, especially in plethoric patients where the loss of a pint of blood can be well borne, and all the more if the fits occur early in labour or before it has commenced.

The rationale is not only that the right heart is relieved and the congestion of the lungs lessened and so lividity decreased, but also that by abstracting so much of the toxine from the circulatory system (particularly if we dilute the remaining poison by a copious saline infusion or transfusion) we are immediately acting beneficially on the cerebral centres in supplying them with less poisonous blood, and the coma and convulsions will tend to decrease. There can be very little risk in taking about fifteen to twenty ounces of blood from most patients, for there are few women who could not survive a post partum haemorrhage of this amount, and in eclampsia we can generally take it for granted that the uterus will retract well after the expulsion of the placenta. It may also have an

effect in allowing a more rapid dilatation of the os uteri.

Phlebotomy was only practised in two of my cases (Nos. 8 and 9), and in each of these I thought it had an excellent effect. In Case 8 the patient was having very frequent and intense paroxysms, there being three in the hour previous to bleeding. She was then bled to fifteen ounces and another seizure followed, though not so severe as the preceding ones. An hour later there was a comparatively slight fit and no more followed until sixteen hours later.

In Case 9 the patient had had ten fits in three and a half hours, there being three in the hour previous to bleeding. Eighteen ounces of blood was then withdrawn from the median basilic vein and there were no more seizures for three hours, when the last, a slight fit, occurred. In this case, besides the benefit in regard to the convulsions, there was a very marked and immediate improvement in the patient's colour and breathing.

In neither case, however, was there any distinct change in the coma, but in the latter, the patient before being bled was either restless or profoundly comatose between the fits, while after the bleeding she appeared to fall into a more natural sleep, which lasted uninterruptedly for three hours. Among the forty-two cases tabulated there are only three where phlebotomy was practised, Nos. 33, 34 and 35. In each

case the patient recovered, and in two there were no fits after the operation, while the third case became conscious nine hours after. In the first two cases the fits stopped four days and twenty hours respectively before delivery.

Saline Infusion.

There has probably never been a greater aid added to our resources in the treatment of eclampsia than the intercellular infusion of saline solution.

This was introduced into the Glasgow Maternity Hospital by Dr. Jardine, and by his writings on the subject he has brought it prominently before the profession.

After using different diuretic salts in different proportions he has finally settled on acetate of soda and sodium chloride as being the most suitable, being both safe and strongly diuretic.

One dr. of each is dissolved in a pint of sterilised water, and the solution at 100° F. is run into the areolar tissue either beneath the breast, or, after delivery, into the lax abdominal wall. In choosing the former site the great essential is to direct the point of the cannula right <u>under</u> the breast. If the instrument be introduced <u>into</u> the breast the solution will not run in so rapidly; the operation will be painful; the absorption of the fluid will be slower, and for an hour or more afterwards there will be considerable suffering.

If the right plane be struck, which is ensured by grasping the entire breast with the left hand and lifting it from the chest-wall as far as possible, the trocar and cannula being then introduced well beneath it, the fluid will run into the loose tissue almost as quickly as if it were running into a basin; there will be comparatively little pain (at any rate until nearly a pint has been introduced) and absorption will occur in about twenty minutes to half an hour. In a patient who had had infusions under the breast twice before, two pints were injected in six minutes.

Care must also be used to secure asepsis, but with this I think the risk of inflammation is very slight. I have never seen any sign of inflammation in any patient in more than a hundred injections.

The object of the treatment is primarily "to get the kidneys to act." "If we knew what the poison was, we might be able to neutralise it by the injection of an antidote, but in the meantime until we discover what it is we can only use means to get it expelled from the body" (10). The suppression of urine is one of the most marked clinical features of eclampsia, the continuation of which will make the prognosis graver and graver. If then this method will help to stimulate the kidneys throughout, our resources are greatly increased. That it does so in

most cases is easily seen from reading the reports of cases treated with it and without it.

As an example of the progress of the disease when not treated by saline infusion, we may take the following statement from the most complete work on the subject - the article on Eclampsia by Herman in Clifford Allbutt's"System of Medicine," Vol.VII. "The secretion of urine begins to be reestablished in from six to twenty-four hours after the cessation of the fits.....but it takes from two to four days for the secretion to become as abundant as in health." Compare this statement with the table on page 135; this in itself is a sufficient proof of the active diuretic effect of the treatment.

Analyses of the urines of the cases reported by Jardine in his "Lectures on Haemorrhage and Eclampsia" have shown conclusively "that there is a marked in- "crease in the excretion of urea and uric acid, and "considering that the urine is tremendously increased "after the injection, the amount of urea and uric acid "expelled from the system must be very great..... "The presence of large quantities of them in the "system cannot be held to be beneficial to the patient "and they are certainly better cleared out. What else "may have been carried out I cannot say" (10). This great increase in the elimination of urea is also shown in my cases Nos. 4 and 6.

Besides this eliminant effect, saline infusion

has the property of diluting the toxines in the blood-stream. The fluid is quickly absorbed, and, as there is shown to be an action on the kidneys there must be a general dilution of the blood. That it has this diluent action I think there can be no doubt for it is to be observed in other diseases. A boy aged twelve was admitted to the Western Infirmary with diabetes and soon became comatose. I twice infused a pint of warm water containing 1 dr. of bi-carbonate of soda when he seemed almost on the point of death. The rallying effect was most marked, especially on the first occasion, and it was unnecessary to repeat the infusion for twelve hours. He died about six hours after the second operation.

Jardine prefers the intercellular infusion of saline solution to the intravenous transfusion because the absorption is more gradual from the areolar tissue to the circulation while the actual operation does not take any longer. When one is opening a vein, however, in any case the latter objection does not hold, and if the bleeding be adopted for the purpose solely of stopping the fits and not for relief of the right heart, it is just as well not to delay the dilution of the poison in the blood but to run in the saline at the same time as we withdraw the blood.

There is still another benefit to the patient obtained by infusing saline solution, and that is the

stimulation which follows this treatment when the patient is collapsed. This stimulating effect of infusion or transfusion is of course best seen when the collapse is due to loss of blood,but even in other cases of collapse it is often marked, for at such times the disposition of the quantity of blood in the body makes the same circumstances for the heart as an external haemorrhage. I have frequently seen this in the collapse subsequent to a lengthy operation when there had been practically no bleeding.

Infusion of saline solution may, however, be overdone. If the patient be very oedematous, if the heart be failing, and there be much pulmonary congestion I think infusion should be only moderately practised (perhaps to the extent of one pint) unless the patient be bled at the same time. In the case of eclampsia before mentioned (21) eight pints of saline solution was infused in four hours, and later on ten pints was injected into the vein at once, (it is not stated what salts were in solution). This is stated to have caused a temporary rally from a state of collapse; the patient however died some five or six hours later. To increase the amount of fluid the failing heart had to deal with by ten pints,probably given within half an hour,was to add the last straw to its burden.

There is more than a slight risk of increasing

the work of the heart by too large infusions, and the result will be, if not an immediate cardiac failure, an oedema of the lungs which will be no less certainly fatal.

Chloroform. "The great indication in the management of "eclampsia,"says Playfair (3),"is the controlling of "convulsive action by sedatives. Foremost amongst "them must be placed the inhalation of chloroform..... "Practically no one who has used it can doubt its "great value in diminishing the force and frequency of "the convulsive paroxysm."

"At the first threatening of another paroxysm "chloroform inhalation should be at once begun, and "continued until the convulsive attack ends; even then "it is best to continue the inhalation though using "only a small quantity of chloroform for some hours, "but the quantity must be increased at the approach "of a convulsive paroxysm." Parvin (6).

"When eclampsia comes on during either pregnancy "or labour and the closure or undilatability of the "cervix makes it impossible to effect delivery, or "when the attacks, having resisted bleeding and re- "vulsives, are very frequent and by their steadily "increasing severity threaten the lives of both mother "and child, then we are convinced the use of chloroform "may be of some service." Cazeaux and Tarnier (4).

"As fits are provoked by peripheral stimuli, it

"would seem good practice so to treat the patient as to "protect her nervous centres from such stimuli. This "can be done by keeping her anaesthetised by chloro- "form." Herman (9).

Trousseau says that antispasmodics are the treatment indicated in puerperal convulsions and of these chloroform is the first. (24).

The older objections to the use of chloroform were, that it increased cerebral congestion, and added another risk to the life of the patient. The most enthuiastic of the supporters of this method of treatment is Playfair. He recommends the interrupted use of the anaesthetic, "carefully watching the patient "and exhibiting the chloroform as soon as there were "any indications of a recurring paroxysm with the view "of controlling its intensity." Herman says "It is "absurd to talk about giving chloroform when the fits "come on; a fit is not preceded by warning symptoms "giving sure and timely notice of its onset. When a "fit comes, it is over long before the patient can be "anaesthetised; besides, during the first part of a fit "the chest is fixed so that the patient cannot inhale." He recommends using it thus:- "If the patient be coma- "tose chloroform is superfluous; if she be restless it "must be pushed until she is fully under its influence "and kept up. If fits do not recur after an hour or "two the drug may be withheld; and if when this is done

"the patient continues tranquilly sleeping, it may "be put aside. If on the contrary the patient become "restless, it must be resumed."

I have very little experience in these cases of the effects of chloroform alone on the convulsions, but it was noticeable in Cases 1, 3, and 4, in which chloroform was administered for a considerable time during the delivery, that the interval till the next fit was longer than it had been between any of the previous seizures.

In Case 6 the patient had no more convulsions after the administration of chloroform by her doctor for one hour, but there had been no fits for at least an hour previous to its exhibition, though in the hour before that there had been seven.

The interval was also delayed in Cases 8 and 9 after giving chloroform, but these patients were bled and transfused at the same time.

In Case 8 I have already referred to the long-continued fit in which respiration was active all the time and which appeared to be stopped by the inhalation of chloroform.

Is it not very possible that the benefit said to accrue from delivery of the child by accouchement forcé is as much due to the long-continued inhalation of the anaesthetic as to the expulsion of the foetus? For it is a common enough occurrence in reading the reports of cases to see stated that after accouchement

forcé, the fits stopped for two or three hours and then returned again.

Chloral. As the prolonged administration of chloroform requires constant watchfulness and the continual presence of a medical man, chloral is advocated as a means of procuring the same effect uninterruptedly and without this drawback. Very brilliant results have been obtained by the use of this drug in the hands of Charpentier and Winckel, the latter having treated ninety-two cases with seven deaths. It should be given in large doses and if not retained by the mouth, be injected as an enema suspended in mucilage.

Morphia. Even a smaller death-rate has been secured by Veit with the use of morphia hypodermically, he having treated sixty cases with two deaths (2). This drug is also to be given in large doses and repeated as often as there is any restlessness.

Morphia is supposed to have not only a sedative action on the nerve centres, but to act as a diuretic by diminishing spasm in the renal arterioles which it is said to do when given in large doses (Nicholson).

Veratrum viride. Veratrum viride is a drug used largely in America in the treatment of eclampsia. Its hypodermic use is made part of a routine treatment by Hirst (22). It slows the pulse, causes sweating, and reduces the temperature. It has not been so much used in this

country.

Pilocarpin. Pilocarpin has been given to produce sweating and so aid in the elimination of toxin. It is a dangerous drug in such a condition as it produces oedema of the lungs, the tendency to which in eclampsia is already great enough. In a late number (27) of the British Medical Journal there was a report by a New Zealand doctor of "A case of Eclampsia fatal after delivery." He saw the patient after she had had one fit post partum, and injected half a grain each of pilocarpin and morphia. This seems to have been all he did. No fits recurred within an hour and he left, to be again summoned, as the patient had had another fit. He again injected the same quantity of pilocarpin and morphia but notwithstanding this the patient died. He is not to be held altogether excusable for the result.

Herman advises that pilocarpin "be not given in puerperal eclampsia"as it makes the bronchial tubes sweat as well as the skin.

Of all these drugs I have no experience in eclampsia.

In the forty-two cases tabulated there is not one where any of these drugs was used alone. Five cases were treated with veratrum viride as well as by saline infusion, and four recovered - about the usual percentage.

Purgation. It is the routine treatment in Glasgow Maternity Hospital to administer two to four ounces of Epsom salts at the commencement; if this cannot be swallowed it is given by the stomach tube. It is advised by most authors to give a purge, and croton oil or jalap are most frequently recommended. Jardine has found croton oil "uncertain" in its action in these patients even when given in large doses and that nothing starts purgation sooner than large doses of salines.

The object of giving hydragogue cathartics is not, as Herman makes out, to remove scybala which are presumed to be in some measure causative of the convulsions, but to increase the "water-circulation" and hence to eliminate toxin. Herman (9), knows "of no reason for thinking that purgation does good "in eclampsia," ignoring the very obvious one just mentioned. He thinks it "more likely to do harm; and it has the grave practical disadvantage that it interferes with the collection of the urine and thus deprives us of the best means of prognosis." He therefore advises against the administration of a purgative as he thinks that reflex irritation might set up fresh fits.

From observation of my own cases I think that there is a great deal of truth in this contention. It will be noticed in these reports that the giving of the salts was often immediately followed by a

convulsion and in two cases (Nos. 2 and 4) by vomiting, and one can easily conceive that such large doses of salts must set up rather severe colic and hence militate against the cessation of the fits. On the other hand we must try to rid the system of the eclamptic poison and this is a sure means of increasing the "water-circulation" and so almost certainly expelling some of the toxin.

It would therefore be better to give a more moderate dose of salts (say 1 oz. of mag.sulph) followed soon by a large warm enema gently given than to give up the advantage which purgation probably ensures, or on the other hand to run the risk of setting up fresh seizures by causing too great intestinal irritation.

Hot Packs. Another means of expelling some of the eclamptic poison is diaphoresis. The use of pilocarpin is not necessary for this as we have a safer and at the same time a soothing remedy in the hot wet pack. This has been employed in many of the cases tabulated, but as it is only an adjuvant it would be useless to compare the death-rate with and without its use. It was given in six of my cases and generally had a quietening effect if the patient were restless.

The skin does not always act well, but often, if the pack be repeated a profuse sweating will occur. The pulse and temperature should be watched

during the time the patient is in the pack. As a rule a good effect is produced in from twenty minutes to half an hour.

If restlessness, or very profound coma continue the hot wet pack should be repeated again and again.

Drycupping, etc.

Drycupping and poulticing over the kidneys seemed to hasten diuresis in three of my cases where it was employed (Nos. 2, 4, and 8). Spirit of nitrous aether was also given hypodermically in large doses.

Continued pyrexia should be met with cold baths or cold sponging.

Nursing.

As regards the management of the patient during the period of convulsions constant and unvarying watchfulness on the part of the nurse is essential.

As soon as there is any evidence of the appearance of a fresh seizure, some soft resistant substance should be placed within the teeth for the double purpose of preventing the forcible closure of the jaws and of preventing the extrusion of the tongue. As a rule the patient does not greatly alter her position in bed during the seizure, but there is a possibility of this and too violent or too extensive movements would require restraint.

Froth and mucus must be quickly cleared from the lips and mouth after a fit that they be not inspired. Sips of milk and imperial drink should be

given frequently whenever the patient is able to swallow.

The skin must be kept healthy and active by washing with soap and hot water morning and evening, and more frequent sponging with tepid water will be necessary if there be much sweating.

The same attentions must be given by the nurse as is usual to any lying-in woman. It may be necessary to catheterise frequently for a day or two.

The patient should be kept for a day or two lying on her side or semiprone to prevent hypostatic congestion of the lungs.

The nurse should pay particular attention to (a) the amount of urine passed, (b) the number and character of the evacuations, (c) the condition of the skin, (d) the amount of natural sleep or the presence of restlessness, (e) the amount of nourishment taken, (f) the time at which the patient begins to regain consciousness and make known her wants.

Fluid nourishment should be regularly and freely given.

Obstetric treatment.

The obstetrical treatment of eclampsia is as much debated as is the medical.

The extremes of opinion are:-
Allow the labour to go on naturally, directing your whole attention to the stoppage of the fits and the medical treatment of the case: and, Deliver the

patient as soon as you possibly can even if you have to rapidly dilate the os or incise it.

The advocates of the first opinion hold that any interference with the progress of the labour in the way of obstetric manipulation will tend to set up convulsions, that accouchement forcé is a shock and extra risk to the patient, and that in any case no good is to be done to the patient by the delivery of the child.

The advocates of the second opinion hold that the pains of labour tend in themselves to set up reflex convulsions, that the delivery of the child is followed by a decrease in the severity and frequency of the convulsions if not by an actual cessation.

Certain of these beliefs may be reconciled, but there are some of the statements diametrically opposed, and, as it is only by accepting the truth of one or other opinion that we can base a correct obstetrical treatment, it becomes difficult to decide what is best. First of all then it is admitted by both sides that peripheral irritation is a bad thing in eclampsia, whether this be due to uterine contractions or to obstetrical operations.

The point therefore, to be deduced from this is "Adopt that method of treatment which will least tend "to harass the patient."

If the labour be well advanced and the os sufficiently dilated, chloroform should be administered

and forceps applied. This will certainly give the child a better chance, and it cannot be said that a forceps delivery under these conditions would be as great a source of irritation as a succession of labour pains in the second stage becoming more and more frequent and forceful which might last for an hour or more.

I think we may take it for granted that the real difficulty in deciding on the proper obstetric treatment will only arise when the convulsions have occurred before or during early labour. The point where the two opinions clash is whether delivery will bring about a cessation or mitigation of the convulsions, or whether the course of the disease is influenced at all by the birth of the child.

Enormous quantities of statistics are to be had showing cases where there were fits before delivery and none after. It is nearly always found in any writer's collection that there were more fits before delivery than afterwards, there being always a large percentage of patients who had no fits after delivery. Such statistics (i.e. those showing only the number of fits before and after delivery) are most misleading if we try to found upon them any opinion as to the influence of delivery upon the convulsions. Statistics intended to show the influence of the birth upon the fits must tell <u>how long</u> before delivery the convulsions ceased if there were none post partum,

and also whether or not they seemed to be coming less frequently for some time ante partum. Otherwise the delivery will receive the credit of having caused the stoppage of the fits in the large proportion of cases in which they had ceased long before delivery. Take, for example, Case 6. Put down in the usual way, she would be said to have had eleven fits antepartum and none post partum, but it was three and a half days between the occurrence of the last fit and delivery. This case shows the fallacy of founding any belief on a series of statistics where the stage of the disease with relation to the fits is not expressly shown. In a tabulated series of cases we ought to see in a separate column the time which has elapsed between the last fit and delivery. In the "Lancet" of 26th. April, 1902, there is an article by Herman which conclusively proves that after delivery the fits do not cease. He gives statistics from a great many authors, the average figures appearing to show that in about forty-four per cent of the cases fits continue after delivery, and in fifty-six per cent they cease with the birth of the child. Of those cases in which the fits continue in about fifty per cent they are mitigated in intensity. He points out that eclampsia is a disease running its course in less than forty-eight hours, and that in a great many of the cases where convulsions ensued in pregnancy or in early

labour a cessation of the fits would occur about the time of delivery independently of whether the uterus be empty or not.

"Four-fifths of the cases of eclampsia end in "recovery within a day or two. If the disease be- "gins before labour, fits will generally cease before "delivery. If it begins early in the first stage "of labour, the last fit may be before delivery or "soon after delivery. The fact that recovery follows "delivery does not prove that it is the result of "delivery"(25).

I saw this paper while house surgeon in the Glasgow Maternity Hospital, and in subsequent cases I tried to form as clear an impression as possible of the effects of delivery on the fits.

I will briefly state what it appeared to be in each case.

Case 1.

Ante partum - 5 fits occurring between 6 and 8 p.m.
Patient could be roused on stimulation by 9 p.m.
5th. fit at 11.40 p.m.
Delivery a la Bossi at 1.20 a.m.

Post partum - 2 slight fits 2 hours and 3½ hours respectively after delivery. Able to swallow within an hour of delivery.

This was undoubtedly a mild case as evidenced by the

slight coma and the number of fits. There was a long interval between the 4th. and 5th. and more than an hour and a half between the 5th. and delivery.

I could not, therefore, form a definite opinion i n this case, but it appeared as if accouchement forcé had helped and it certainly had no bad effect.

Case 2.
Ante partum - 4 severe fits.
Post partum -16 severe fits.
Delivery had not the slightest effect in mitigating their intensity.

Case 3.
Ante partum - 5 fits between 4 and 7.20.
Under $CHCl_3$ for one hour, delivery at 8.40.
Post partum - 4 fits, the first 4½ hours after delivery. The fits did not come in such quick succession after delivery but otherwise there seemed to be no effect on the disease.

Case 4.
Ante partum - 9 fits.
Post partum - None.
Except that patient had a good deal of treatment at the time of delivery (chloroform for one hour, 2 pints saline infusion, hot wet pack) it would appear that the

cessation of the labour pains post partum had a beneficial effect.

Case 5. Fits began during the puerperium.

Case 6. Already quoted. No fits for 3½ days before delivery.

Case 7. Case of threatened eclampsia.

Case 8. Ante partum - 29 fits the last 4½ hours ante partum and the last 4 less severe and coming at longer intervals.

Post partum - 2 fits at long intervals.

The delivery in this case did not in any way affect the stoppage of the fits.

Case 9. Fits stopped 9 hours ante partum. Delivery had nothing to do with the cure.

In these seven cases (Cases 5 and 7 not being countable) it would appear that in one case delivery stopped the fits, in two cases delivery mitigated the fits, and in four cases had either no effect or a bad one.

Of the forty-two cases tabulated, two died undelivered, seven occurred in the puerperium, so that there are thirty-three remaining on whom the effect of delivery can be seen.

No effect, or fits ceased three hours before delivery.	Fits ceased at delivery tho' frequent up till delivery.	Fits mitigated after delivery tho' severe just before delivery.	Fits more severe & frequent after delivery than before delivery.
21	4	3	5

These figures strongly support Herman's contention that in the great majority of cases the disease is uninfluenced by the birth of the child, and that in those cases where the delivery seems to have had an effect, it is nearly as often a bad one as a good one.

But besides the effect on the convulsions we must carefully consider the effect on the patient.

It is no good adopting a line of treatment because it benefits the fits, even although it were allowed that accouchement forcé did so, if it were found to be harmful to the patient.

Herman found that the chances were about 2 per cent better if delivery were hurried than if an expectant attitude were maintained, and he asks "What surgeon would recommend an operation if all "he could say in its favour was that his patient's "chance of recovery was about two per cent better if

"he had the operation done than if he were let "alone?" (25).

In my table I find that the death-rates with accouchement forcé and with expectant obstetrical treatment are exactly equal. Of seven patients where accouchement forcé had a good effect upon the convulsions either in stopping them or in lessening their force or frequency, six ultimately died.

Surely this is a very striking proof that the good effect on the fits may have been secured at the expense of the patient, and it is sufficient to show that accouchement forcé, even when the patient is "deeply" under chloroform, is a very severe shock to her.

As regards the child, however, the treatment by rapid delivery has a much smaller mortality. The death-rate with expectant treatment is 2.4 times greater than where accouchement forcé is adopted. This fact, however, should hardly be considered if the mother's life can be saved by adopting an expectant attitude or if it would be endangered by active obstetrical interference in an early stage of labour.

Jardine (26), in his "Clinical Obstetrics", advocates what appears to be a reasonable treatment, as least likely to do harm by undue haste, and most likely to do good when delivery is called for. If

medical treatment has been given a fair trial, and the convulsions continue and cannot be controlled, only then should accouchement forcé be employed.

That almost every question in the study of this disease is a debatable one is in many ways unfortunate, and especially is this so for the student. The diversity of opinion among authorities on so many points in its pathology and treatment makes it a necessity that all should keep an open mind to the proposed solutions of its intricacies, and maintain a keen interest in any work done that will tend to increase our knowledge of its origin or further our resources in its successful treatment.

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