U N I V E R S I T Y O F G L A S G O W.

Degree of Doctor of Medicine (M.D.)

THESIS OF

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OBSERVATIONS ON ECLAMPSIA ESPECIALLY WITH REGARD TO AETIOLOGY and TREATMENT.

I hereby declare that the work comprised in this thesis has been done entirely by myself as also has the thesis been composed and written.

May 13th, 1913.

Midwifery may be said to differ in one great respect from its sister sciences Surgery and Medicine in that the questions raised in its practice and especially in the difficulties of its practice are very greatly questions of mechanics.

Such questions certainly are raised both in Medicine and Surgery but to nothing like the same degree as in Obstetrics. In normal labour, for instance, we have to consider the effect of uterine contraction on proper dilatation of the cervix and how this is much better and more evenly accomplished by a bag of waters exerting a pressure in every direction than by an unprotected head of foetus with hardly any elasticity: how the head descends in a flexed position in the axis of the pelvis: how at the vaginal outlet it is delivered by a process of extension and how by a lateral rotation of the head the pectoral girdle of the child assumes a position autero posterior as regards the mother and more adaptable for delivery.

These are essentially mechanical considerations and come to be considered in every normal case of labour. But in abnormal labour the mechanical question is perhaps a keener one because we have besides the above various other points to bear in mind in treatment. A pelvis is contracted; will the head come through at all? Will it require forceps? Or will it mould and come through of itself?

Contracted pelvis is so frequent an abnormality in

Glasgow that these are apt to be frequent considerations in our practice. Or again we find placenta praevia. What is to be done to bring down a part to exert pressure and so to stop haemorrhage?

Obviously then the mechanical points raised both in eutocia and in dystocia are many and important.

But there is quite another side than the mechanical to pregnancy and labour the importance of which we are perhaps apt to minimise. The ovum existing as it does in the living body of the mother exercises a keen constitutional effect on the parent organism and is in turn greatly influenced by various changes in the body of the host. In what we call a normal pregnancy the constitutional effect of the presence of the ovum is either absent, or if present not so gross as to be conspicuous. But under various conditions the effect on the mother is very marked constitutionally, and various degrees of what we consider toxaemia are observed, the most severe of which, and indeed forming the culminating acme of toxaemic conditions complicating pregnancy, we call Eclampsia or Puerperal Convulsions.

It is about this condition of eclampsia that I wish to speak in this paper.

While I was In-door Resident Obstetrician in the Glasgow Maternity Hospital there were in all 35 cases of eclampsia admitted and treated, 17 of which were under my own personal attention. My observations are drawn almost entirely from these cases which I observed in a systematic manner. It might be thought that I would have been wiser had I considered first the minor intoxications and perhaps

proceeded from these to the consideration of eclampsia.

My reason for adopting my present plan is that the signs and symptoms of toxaemia are more fully and characteristically exhibited in the major condition of eclampsia than in any of these minor states and that having considered such a major disease we might attempt to trace the relationship between it and various other states exhibiting fewer and less prominent signs of intoxication.

As the condition is obviously constitutional and as we soon find all the physical systems and also the mental mechanism more or less affected by the disease it behoves me to adopt some kind of method of procedure lest this paper become a general ramble and the points to be brought out be either blunted or their true significance lost.

I propose then to consider first the frequency and incidence of the disease, then the fit which is so characteristic a sign of the condition; later to take up the states found during life and after death in the various organs and fluids of the body: then to review the known pathology and aetiology of the disease. Diagnosis, prognosis and treatment will also be discussed.

It is obvious that to make my remarks of any value I would require to consult the writings of the various British, American and Continental Obstetric Authorities. I have done this, and in each case make a note of reference to the author and article quoted.

At the end will be found an appendix of cases referred to in the text of this paper.

FREQUENCY AND INCIDENCE.

In Hospital practice one is apt to get a wrong and exaggerated idea of the frequency of eclampsia in pregnant women. Men doing a big private midwifery practice report frequently about a dozen cases in ten years or so, while on the other hand we have one day in 1908 when five cases were admitted to the Glasgow Maternity Hospital. These figures are obviously very far apart and we must look somewhere in between them for a true estimate of the frequency of the condition.

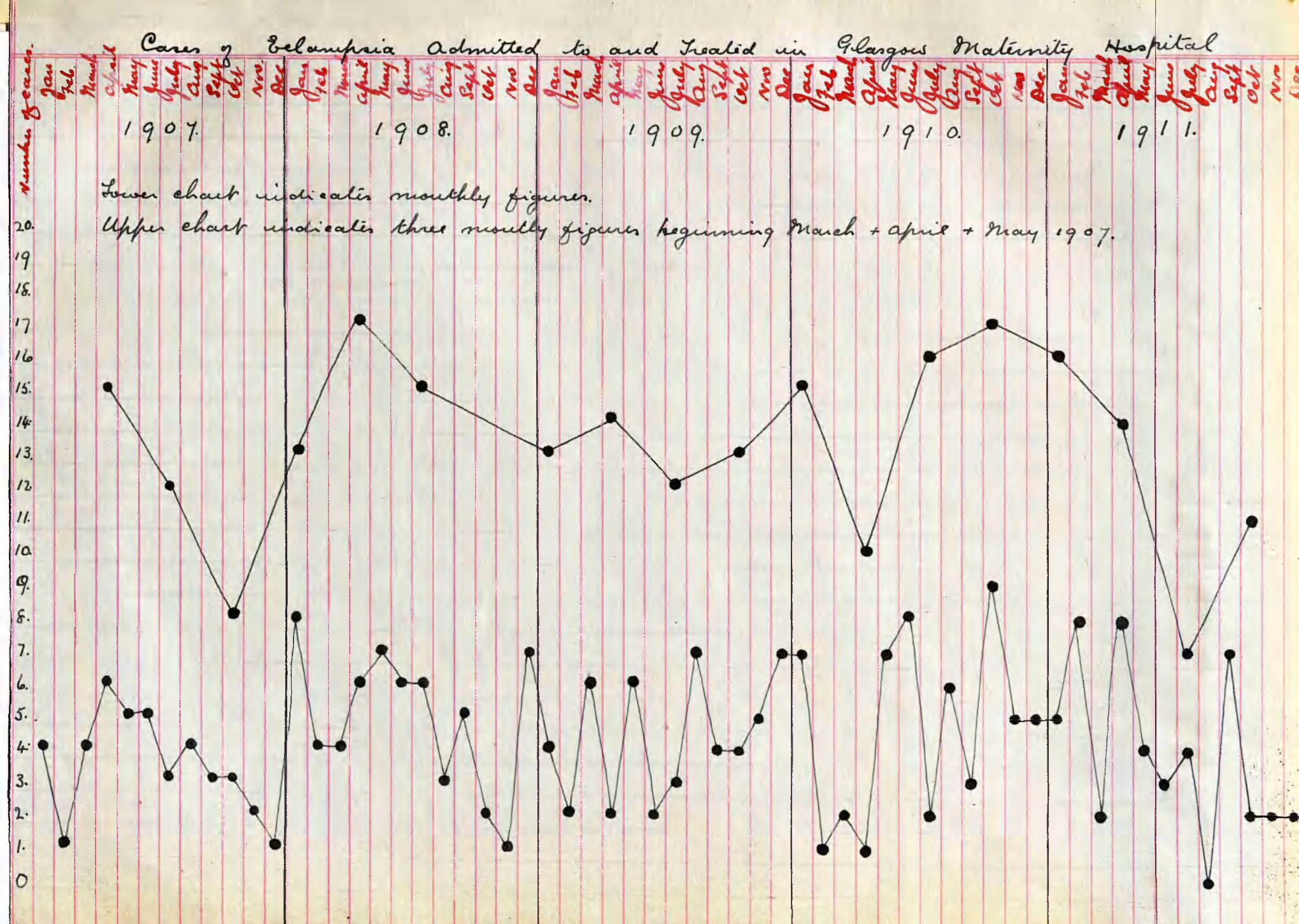
The figure mostly quoted and accepted is 1 in 500 cases and this is probably fairly correct.

Jardine[1] gives a frequency of 1 in 300 in private cases and quotes the frequency of the Glasgow Maternity Hospital as about 1 in 50, i.e. 10 times the usual all-round frequency. I have investigated the cases of eclampsia admitted and treated in that house from January 1907 until December 1911 - 5 years, and find that of 6,037 indoor cases 256 were eclamptics, that is over 4% or 2 in 50 cases. This is double the frequency of Jardine's figures, but his were taken over a much longer period.

Williams[2] gives the number in the Johns Hopkins Hospital, Baltimore, as 1 in 133 cases. He quotes the following Continental and American authorities giving their figures of incidence :-

Goldberg	(Dresden 1891)	10,717	cases with	81	Eclamptics ·75%
Cassamayn	(Paris 1892)	16,225	" "	99	" ·61%
Green	(Boston 1892)	3,500	" "	36	" 1%

Cases of Eclampsia Admitted to and Treated in Glasgow Maternity Hospital



Knapp	(Prag 1900)	7,636 cases	with	41 Eclamptics	·53%
Newell	(Boston 1900)	6,700 "	"	99 "	1·17%
Löhlein	(Germany 1891)	15,328 "	"	325 "	·62%
Veit	(Germany 1896)	149,366 "	"	905 "	·60%

The frequency shown there falls very far short then of the figures quoted above.

It is usually stated and believed that Eclampsia is more frequent in Autumn and Winter than in Summer, the reasons put forward being that in the former seasons people lead a more sedentary life and also do not perspire so freely as in the latter.

Little[3] of Montreal discusses 40 cases and notes that 32 of these occurred between October 1st and March 31st. He is inclined to attribute this mainly to the severity of the Winter causing difficulty of access to lavatory and outhouse with consequent tendency to constipation.

I have made a chart, which I enclose, showing the frequency in the Glasgow Maternity Hospital during these five years. It does not show any great or consistent rise during the Winter months.

This is somewhat surprising since it fits in so well with our present ideas of toxaemia that the condition should be more frequent when excretion is apt to be impeded. The figures require no vouching for: they are taken from the official register of the Hospital.

THE FIT.

Tweedy defines eclampsia as "the occurrence of fits in a pregnant woman which would not have occurred if she had

not been pregnant." Various definitions by other authors are to the same effect that the fit is the supreme clinical manifestation of the disease. This is true practically always, although recently it has been shown that the condition may exist without fits: I shall return to this later.

The fit may have two modes of onset: one sudden and unexpected, the patient being in apparently good health previously - this is very rare; the other after a varying period during which headache, epigastric pain, nausea, vomiting and swelling of limbs are complained of. She may gradually sink into unconsciousness and then fits appear, or the fits may come on previous to the coma. The previous history of eclamptic patients is full of interest and should be enquired into carefully and systematically in all cases.

I am convinced that heredity enters not at all into the causation of the condition. In one of my own series of cases the patient's mother had died of apoplexy following nephritis, but in no other case was there even so far fetched a connection between supposed cause and effect as there.

This is directly opposed to the views of Pinard, Olshausen and others who maintain that heredity may in some cases play a part.

It is well known of course that primiparae are more prone to the disease than multiparae. Of the cases referred to all were primigravidae except two - one a II-gravida and one a IV-gravida.

There is frequently a history that in early pregnancy the general health was poor or that morning sickness was

rather severe, but apart from that usually nothing abnormal is noticed until some time after the 6th month, when headahce, frontal and persistent, is complained of. After some time the woman notices that she is becoming pale and that her eyes are rather "puffy" or that her ankles are swelling. A difficulty in getting on shoes or boots in the morning may first cause this to be observed. Later she has disturbances of vision such as irregular flashes of light, attacks of sudden blindness or merely muscae volitantes. A characteristic symptom is the presence of epigastric pain. The cause of this is not quite clear. It has been suggested that it is due to minute multiple haemorrhages in the stomach wall, but confirmation is wanted ere we accept this explanation. I saw nothing in any case post mortem to support this.

Micturition frequently becomes scanty although occasionally it becomes more frequent during this premonitory period. I am inclined to think that the latter cases have previous nephritis. Constipation usually of long standing is almost always present and certainly plays a part in predisposing the patient to any toxaemic absorption.

When the fit does come on the patient usually falls down and passes through a condition like an epileptic seizure. I do not propose to describe the fit here. There are a sufficiently large number of other and fuller descriptions which may be referred to.

Eclamptic patients were admitted to the Maternity Hospital usually having fits, and the history required to be obtained either from friends or from the patient after

cessation of the convulsions. In the latter case usually one was told that she remembered nothing for a variable period, in some cases several days before fits came on, although she might have been going about all that time and acting quite normally.

It is generally believed that a cry is most uncommon at the outset of a fit and indeed it is put forward as a point in diagnosis. Galabin[4] states that it never occurs. In Case VI Appendix there was a history of two previous fits each having a cry and while she was being washed I heard her give a typical epileptic cry and fall into a fit. No personal or family history of epilepsy was to hand. I consider that only a modified importance should be put on the presence of a cry as excluding eclampsia. The mechanism of the fit so far as we know is similar to that of an epileptic fit, and as I have shown, a true uncomplicated eclampsia may exhibit a typical epileptic cry.

My own experience entirely supports the view that the onset of fits is mostly ante-partum. In 35 cases only 1 was post partum, i.e. 2·85%. Some observers are beginning to question the general application of this and some furnish figures in support of their views.

I give the figures below of the relative frequency of the types of cases by different observers.

	A.P.	I.P.	P.P.
Olshausen	40%	46%	14%
Knapp	24·5%	60·9%	14·6%
Goldberg	26%	57%	17%
Green	36%	22%	42%

These point to the onset being mainly intrapartum. However, I am inclined rather to the view that eclampsia sets up parturition than vice versa. It is a point yet to be fully decided.

An aura is not usually described although Olshausen remarks on its occasional presence. I noticed it in two cases. Löhlein and Knapp describe mental derangement as an occasional precursor to fits, the former quoting 5% and the latter 13% of cases showing this.

After the fit or fits subside the patient is at first comatose and then sleeps for a varying period. The mental state is usually very dull for a day or two and then gradually the normal level is reached.

But this is not invariable. Case VI Appendix showed great fits of excitability after cessation of Eclampsia proper and finally became acutely maniacal. Case III Appendix remained more or less comatose with an occasional stimulation and died quite quickly. No post mortem was allowed but the signs pointed to a cerebral haemorrhage. Other mental sequelæ such as permanent bluntness of intellect, loss of memory or irritability are described and also varying degrees of paralysis following cerebral haemorrhages. These last are usually very slight.

A few cases have been described of undoubted eclampsia in some cases verified post mortem where no fits at all occurred. Bouffe de Saint Blaise described one such in his Paris Thesis in 1891. Wendt described another in 1898, while Schmorl in 1902 reported 3 such cases. Subsequently Meyer-Wirtz and Esch each reported a case and recently two

others are described in the Johns Hopkins Hospital Bulletin. There are 10 cases in all, and while infinitesimal compared with the number of eclamptics having fits the record shows that this disease may occur and its main clinical sign be absent. I have never seen a case without convulsions.

CONDITION OF THE PATIENT.

DISTRIBUTION of OEDEMA.

Most but not all of our eclamptics exhibited varying degrees of oedema and in varying situations. The most common sites are the feet, ankles and legs; the face especially round about the eyelids; the vulva; the abdominal wall and the back. This of course includes most of the body, but particular cases exhibit particular places where oedema is most marked.

In a great number of cases I found very marked oedema in the lower part of the anterior abdominal wall. I found it persist here sometimes several days after it had disappeared elsewhere and am inclined to consider its appearance more frequent in uncomplicated cases than in cases with old-standing nephritis. I do not find this situation mentioned in most of the text books on the subject.

Oedema of the back is also to be noticed very frequently. It is not usually very marked but it is here that it disappears last and frequently pitting on pressure may be obtained in the lower lumbar region many days after it has completely disappeared elsewhere. The cause of this is

probably gravitation due to the patient in bed. It is observed in most general oedematous conditions and is frequently seen in Bright's Disease. Oedema of the face is seen. It is usually described as worse in the morning or after lying down. It is not so marked in eclampsia as in uraemia. Eden[5] considers that in such a case we should have some definite reason for excluding Bright's Disease before we diagnose eclampsia.

To follow such a course is obviously safe whether we agree with the idea or not that such oedema indicates Bright's Disease as at least a complication.

I have not often seen marked oedema of the vulva in eclampsia and am inclined to agree with Galabin[4] who states that marked oedema of the vulva usually indicates nephritis.

The most common site of all is the feet (mainly over the instep), ankles and legs and on recovery it disappears usually from these parts first.

Some cases, however, do not exhibit oedema anywhere. Two of my cases (Cases V and VI Appendix) were such and both were cases of very acute onset. One (Case V) was fatal and died shortly after admission. The other was dismissed three weeks after admission in a condition of mania. On sectio Case V showed extensive haemorrhage in brain and liver and presumably Case VI would also exhibit brain lesions. I am entirely at a loss to explain the total absence of oedema from these cases. I find in the literature 2 cases similar to Case V, one reported by Maygrier of Paris and the other by A. Randle. Neither author vouchsafes any explanation of the condition.

I shall return to the consideration of Case V when speaking of diagnosis.

Evidently then a patient may have eclampsia and be without either convulsions or oedema - two of the principal signs. Obviously a single case exhibiting absence of both signs would be most difficult to diagnose and one could hardly blame or be blamed for missing such a diagnosis.

While discussing oedema in eclampsia I must not omit to mention a very frequent situation, the amnion, i.e. hydramnios. I noticed when I first came on that a considerable proportion of the cases of albuminuria and eclampsia exhibited more or less hydramnios. I investigated and found the condition present although often not very marked in 8 cases out of 12 albuminurics and pernicious vomitings.

I am driven to the conclusion stated above that the condition is a manifestation of oedema seen in other situations, and think that it is probably the amnion which is affected in most cases being in a state of oedema.

I measured these cases at the level of the umbilicus and found the circumference always more than the average circumference at the end of a normal pregnancy.

The most marked of my cases measured 41" at that level and the measurements of the others ranged from 36" to 40". The average case free of hydramnios I found about 32" - 34". This may be important with regard to the theory put forward that pressure on the ureters with consequent supra-pelvic dilatation is a cause of the disease. Certainly such a uterus as I describe is bound to exert much more intra-abdominal pressure than a normal non-hydramniotic organ.

B L O O D.

As eclampsia is a generally toxaemic condition it is to be presumed that the toxin is present in and carried by the blood and that a careful examination of the blood of various cases would yield valuable information.

At that time I had no knowledge of the results of other work in the same line, and so my conclusions are free of possible bias which might have been produced by a knowledge of the work of such well known and reliable authorities as I propose to quote.

When our eclamptics were admitted in fits I always took a red and white cell count, a blood film and a haemoglobin percentage estimation as well as an estimation of the blood pressure. When the fits had ceased I repeated these procedures, repeated the blood pressure daily at about noon and repeated the blood counts, slides and haemoglobin estimation weekly and finally on the day of dismissal.

In the first place I quickly verified the well known fact that eclamptic blood is very prone to clot. I used Wright's coagulometer to estimate the coagulation time and found it work very satisfactorily. The coagulation time of normal blood is about 4 minutes or perhaps a shade longer. In blood of women at the end of normal pregnancy I found it generally slightly reduced, while in albuminurics it was distinctly so, being usually about 3 to 3¼ minutes. But in eclampsia the coagulation time was very greatly reduced and I may put the average time at 1¾ to 2½ minutes. In one case (Appendix Case IV) I found it as low as 1 minute.

This was a very severe case who was admitted in extremis and died 20 minutes after admission. I could not find that the coagulation period generally tended to be shorter in the more severe cases or vice versa.

This tendency to coagulation explains the rare occurrence of post partum haemorrhage. Internal haemorrhages are due to an entirely different set of causes.

On further investigation I found that as the cases improved and during convalescence the coagulation time visibly increased until in most cases it was quite normal on dismissal.

With regard to the red cell count I must admit that my observations and results are entirely opposed to those of other observers.

Zangemeister and others found the red cells during the period of fits to be greatly increased in number. The highest number quoted is 9,360,000. In two of his cases the fits ceased when the red cell count became normal, but he attaches no causal importance to this fact which I think is wise.

I found the red cell count at that time practically always much below the normal. Usually the number was between 2 and 3 millions with the exception of one case (Appendix Case III) which numbered 7 millions. I frequently did more than one count on a case for check purposes and these other counts always confirmed the previous figures quoted above.

I entirely agree with Zangemeister who observes as

peculiarities of the red cell counts (1) rapid variation within a short time and (2) great variations in different cases. The maximum difference observed by him was 5,120,000. My own greatest difference between two cases as I show above is about 5,000,000.

The explanation which Zangemeister puts forward for his findings is that the increase is a relative one due to the passage of plasma from the vessels to the soft tissues to cause oedema. My own view is that my own findings are explained by the fact that owing to retention of fluids there is a relative increase of plasma in the vessels (hydraemia) as well as an exudation to cause oedema: that the reduction in numbers noted is therefore a relative reduction. From other evidence I consider that some of the decrease in number is absolute being due to a lysis of red cells by the eclamptic toxin or toxines.

When the fits subsided and the patients improved I found the numbers of red cells increasing. On dismissal they usually numbered about 4,000,000 which in a female is not far short of normal.

In the charts accompanying the cases in the Appendix the blood counts are shown and their course may be followed.

On counting the red cells in cases at the end of a normal pregnancy I found mainly a slight increase of red cells to about 5,000,000.

Schroeder[6] and Wild[7] investigated the matter and came to similar findings.

Eden found in animals that the total volume of blood is increased during pregnancy. The proportion of water

increases while the proportion of red cells and haemoglobin diminishes. He found these conditions most prominent at midterm, but even at full term the red cells were usually under 4,000,000.

With regard to the white cells my findings are at one with most other observers to this effect that in eclampsia they are greatly increased and that as the condition passes off they gradually become reduced in number to the normal 8,000.

Much work has been done recently on the white cell and differential counts at the end of normal pregnancy and Carton[8] and Pankow[9] find practically always more or less of an increase. Generally stated their conclusions were that at the end of pregnancy there was an increase; in labour a further increase, and in twin pregnancy a still further increase.

These changes were more marked in primipara than in multipara where in about 50 per cent. no increase was observed during pregnancy although always some increase during and immediately after labour.

Pankow quotes rather higher figures than Carton. With regard to differential counting Arneth has found that during pregnancy the majority of leucocytes are multinuclear but that towards the end of pregnancy and during labour the ratio is changed and the mono and binuclear white cells preponderate.

Wild, remarking on the leucocytosis present during labour and early puerperium, advances as the cause the fact that the healing placental wound calls forth the large increase in white cells to act as phagocytes.

I counted the white cells of 5 patients in the 8th and 6 in the 9th month of normal pregnancy and can only say that my findings agree in the main with those quoted above. In the 8th month I found the average number of white cells about 10,000 while in the 9th about 12,000 to 13,000. The whites in the later month contained a relative excess of mononuclear cells with many large lymphocytes, but I could not find that these formed 70% of the whole as stated by Arneth.

In eclampsia, however, I found in every case a decided increase of white cells. While the patient was having fits I found a fair average of leucocytes to be about 19,000 or 20,000. The highest number I ever counted in these cases was 25,326. The increase consisted mainly of polymorpho nuclear cells with some relative increase in lymphocytes. This contrasts with the quality of blood count in a normal pregnancy. Many of the white cells were in various stages of degeneration.

Dienst writing on this subject found much higher figures than these. He put the average at 26,000 while in some cases he noted 40,000 white cells. He remarks on a frequent rapid diminution to normal during the puerperium and quotes one case where in 36 hours after admission to hospital the white cell count was normal. My own observations were that it was usually fully 10 days before the white cell count was normal and never before 1 week after cessation of fits. In more than one case I found that the white cells at first became reduced below the normal to about 6,000 and that a rise occurred to slightly above

normal before it finally settled to about 8,000.

Dienst lays stress on the leucocytosis as being important causally.

With regard to the microscopical characters of the cells as exhibited in stained blood films I found much of importance. The stains used were mainly Leishman, Jenner, Erlich's Tri-acid or Gram. The first two were the most satisfactory while Gram was used mainly to look for organisms.

In the first place I frequently found the red cells distinctly crenated. I may here state that my usual method of drying was to place the slides on a blanket laid on a radiator and that only a few were "flamed." I say this because of course severe flaming would crenate normal corpuscles: and for that possible reason I am not prepared to lay a great deal of stress on this change although I cannot see how it could easily be produced by such general and gentle drying as described.

Other changes, however, independent of any such possible cause were observed. Vacuolation of red cells was frequently observed. Sometimes single vacuoles were seen but more frequently they were multiple and when so were mainly in the periphery of the cells.

Where a number of cells are closely packed together they incline to lose their distinctive shapes and to become shaped according as the pressure is exerted from various sides just as multiple gall stones are faceted from mutual pressure. This was observed in these as in most blood films but alterations in shape apart from pressure were also common, i.e. poikilocytosis. Some cells were oval, others

were pointed at one or both ends, while others showed considerable flattening.

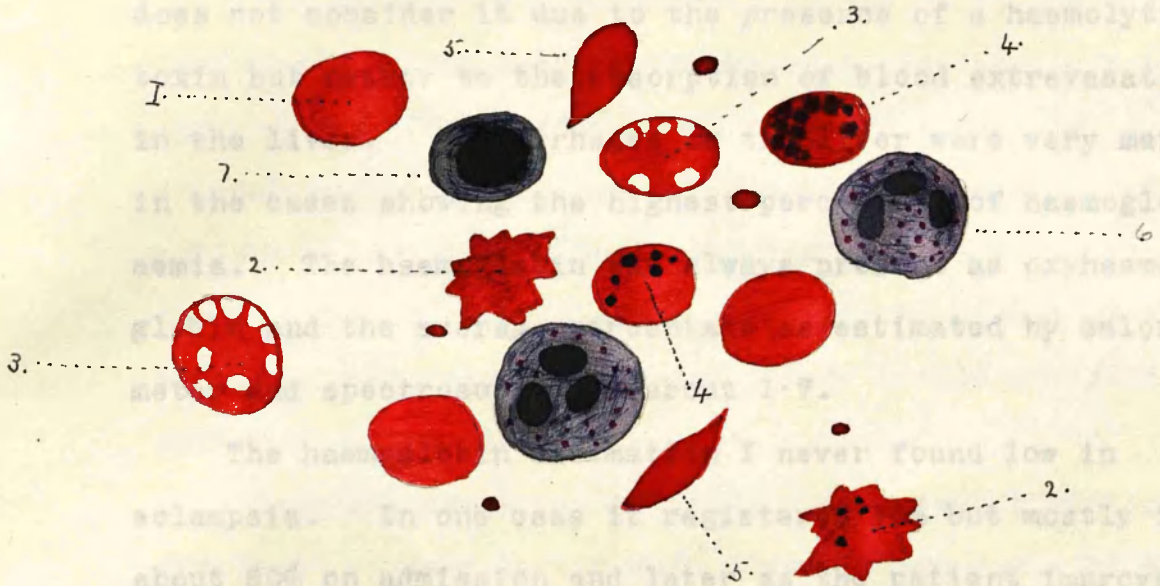
Again, in a considerable number of cases multiple black dots were seen in the cells. These like the vacuoles were present mainly in the circumference of the cells but not especially in cells in the circumference of the films.

The red cells took up the stain usually very well but some were seen to be distinctly pale while others showed very marked cupping and signs of disintegration.

The nuclei of the white cells stained very well. The main features to be observed in them was firstly their relative increase as noted above and secondly in a number of cases evident signs of lysis as shown by degeneration of protoplasm, failure of nuclei to take up the stain and in a few cases extravasation of nuclei into the blood plasma.

I repeatedly examined for organisms but must confess complete failure to find anything of that nature either intra or extracellularly. The black dots referred to in the red cells I cannot account for unless they were nuclei of leucocytes which had become engulfed by the red cells. A more likely idea is that they indicated some beginning degeneration. I had no opportunity of observing the results of blood cultures.

I show a diagrammatic field of a blood film exhibiting the changes I describe.



1. Normal Red cell
2. Red cell showing Crenation
3. Red cell showing Vacuolation.
4. Red cell showing true Black Spots
5. Poikilocytosis.
6. Polymuclear white cell
7. Mononuclear white cell.

I consider that these changes together with the low red cell count indicate the existence of some haemolytic toxin. It is interesting to note finally that these abnormalities gradually disappeared after cessation of fits and that usually within 14 days the films were those of normal blood.

In presence of a haemolytic toxin one would expect to find occasional haemoglobinaemia.

Zangemeister found distinct haemoglobinaemia in 5 out of 14 investigated cases and never in a normal case but he

does not consider it due to the presence of a haemolytic toxin but rather to the absorption of blood extravasations in the liver. Haemorrhages in the liver were very marked in the cases showing the highest percentage of haemoglobinaemia. The haemoglobin was always present as oxyhaemoglobin and the average percentage as estimated by calorimeter and spectroscope was about 1·7.

The haemoglobin estimation I never found low in eclampsia. In one case it registered 70% but mostly it was about 80% on admission and later as the patient improved rose to about 90%. These figures are of course practically normal.

I cannot find any literature to refer to on the question of microscopical changes in blood films in eclampsia and so have no means of checking my results and conclusions in this matter which I submit for consideration.

The plasma I found to almost always give a faintly acid reaction on admission, i.e. during fits, and as convalescence proceeded became first neutral and later alkaline. I had not the opportunity to estimate the alkalinity at different times: this has been done in some detail by Zangemeister[10] who found that during normal pregnancy the average alkalinity was equal to ·202 grms NaOH in 100 ccm of blood. In eclampsia he found very wide variations and frequently no divergence from normal. He puts the average estimate as equal to ·153 grms NaOH in 100 ccm of blood and accounts for the wide differences as due to differences in amount of urine excreted. Diminished diuresis means retention of acids in the blood and consequent low alkalinity.

The presence of lactic acid in the blood and urine has been observed and some are prepared to lay stress on this fact. Zweifel considers it a cause of eclampsia. He found in some cases more of the acid in blood from the cord than in maternal blood and in some other cases more in placental blood. He is of opinion that the origin of the lactic acid is foetal and not muscular and is the result of deficient oxygenation.

Cathcart as quoted by Jardine made some investigations and found no more lactic acid present than could be accounted for by imperfect oxygenation of blood.

An excellent resumé of Zweifel's work on this subject is to be found in an article by Holland[11] in the Journal of Obstetrics and Gynaecology. I shall refer to that very valuable article later on.

THE URINE.

Quantity. Often on admission we found the bladder completely empty and a history of scanty previous micturition and for several hours no urine would be passed. I never had a case of complete suppression although such are not rare as reported. Usually there was a small quantity, perhaps an ounce or so in the bladder. After suppression of fits usually about 12 to 20 oz. of urine passed in 24 hours but some cases pass much more, e.g. Case III Appendix, who passed 106 oz. In such a case the question of previous chronic renal disease must be strongly considered. She died with her urine full of albumen but no sectio was allowed.

Other cases voiding 70 and 80 oz. of urine went out well with no albumen showing, but in cases passing quantities over 50 oz. in the first 24 hours after cessation of fits the question of previous chronic nephritis must always be considered. After that time however the quantity voided rises rapidly in daily amount reaching 80, 90 or 100 oz. per day. Usually the acme is reached on the 5th or 6th day after which the daily amount begins to decline reaching the normal about the end of the second week. Frequently the drop was severe and the daily amount much below the normal for a day or two, after which it would rise again to perhaps 60 oz. and then decline to 40 or 50 oz. There is but little difference to be noted in the amounts passed by day and by night. The former quantity is a little larger but hardly ever more than 5 or 6 oz.

It is not easy to estimate the true quantity voided by these patients. Firstly when admitted and in fits the bladder may be emptied involuntarily and this may occur later in bed when the fits are over, but the woman is as yet in that low mental state which I have referred to. Again hydragogue cathartics take a prominent place in treatment and urine is frequently voided along with stools. Frequent catheterisation may be suggested as a remedy, but I do not think it justifiable as any female bladder is very apt to be contaminated by such procedures even when the greatest precautions are observed and in eclampsia sepsis of any kind is particularly prone to occur and must be correspondingly guarded against. The catheter was passed daily for a specimen for examination but not otherwise.

Specific Gravity. This varied greatly on admission and was anything from 1025 to 1045. A fair average was 1030 which is slightly higher than the normal. As the quantity increased the specific gravity usually declined reaching an average of about 1020 to 1025 which is normal. I did not consider the specific gravity a matter of any real importance.

Colour and Transparency. These qualities varied very much in different specimens from different patients but mainly the colour was either fairly deep amber brown or quite a light clear straw colour. The latter kind was usually transparent but the former variety was nearly always more or less smoky and translucent. I could not find the former variety associated with any worse fits or containing any more albumen than the latter. As the quantity passed increased the former variety usually rapidly apprpached the latter becoming in some cases very pale indeed and almost colourless. Frequently the difference between two consecutive daily specimens was very striking, the one being brown and muddy, the other clear and transparent. Only in one case - Case V Appendix - was the urine distinctly red. Here it was almost like pure blood and quite opaque. A feature which I noted several times was that after a case had been transfused the urine for several days would be cloudy and smoky but without major deposit. On heating the cloud would clear away and on centrifuging distinct uric acid crystals were evident. This suspension of urates after transfusion is remarked on by Jardine who considers the cause to be an albuminous suspension of urates in the urine. I never

observed the phenomenon in a case not transfused. In a few days usually either the urates disappeared entirely or were deposited in the usual manner.

Urinary Deposits. The most constant of these was a cloud of varying size and density consisting of mucus. Its presence was of no importance. Another very frequent deposit consisted of pink urates. When the quantity of these decreased the acidity of the urine frequently decreased and at times gave place to alkalinity so that it would appear that the acidity during fits which is frequently high would be due in many cases to the presence of uric acid. I have remarked above how under certain circumstances these urates may not deposit but remain suspended. Pale urates I observed only twice. They were transient.

Pus was present hardly ever on admission and in only one case - Case III Appendix - did it appear subsequently. The quantity was usually slight and readily gave way to urotropine. Sometimes a scanty brownish deposit of blood crystals was found. It was never present for more than a day.

Chemical examination of the urine yielded very definite results.

Before stating what I did find I may state that I did not find in any case of albuminuria or eclampsia any sugar or bile in the urine. The latter point is rather important in view of the frequency of liver implication. Blood was rarely present: when present except for Case III it was of small amount and usually disappeared within 24-36 hours.

In every one of my cases copious albumen was present in the urine on admission. I do not propose to spend much

time discussing this point as it is so universally known that albuminuria is an almost constant accompaniment of eclampsia. It is by no means invariably present as Schroeder has collected 62 cases from the literature, Ingersleo 112, and Charpentier 142 with urine free of albumen, while Van der Velde reports 2 cases of his own whose kidneys were markedly diseased and which yet had no albuminuria. Jardine is not convinced of this fact and inclines to the belief that most cases reported without albuminuria were not true eclamptics but suffered from other convulsive conditions, e.g. epilepsy.

Conversely to the above I had one case and know of other three where considerable albuminuria was present and yet on sectio no obvious kidney lesion was present. I measured the albumen daily by Esbach's standard. The daily amounts so estimated are to be found on the charts in the Appendix.

It mostly disappeared completely from the urine after a varying time. The shortest time I found was 72 hours after admission for it to clear up - Case VI Appendix. In quite a number of cases it did not clear up completely although the patients usually went out apparently well, and the question is raised in these cases whether it was eclampsia superadded on a previous nephritis or a previously healthy kidney damaged perhaps permanently by nephritis.

Three of my cases were such, not counting two who died shortly after admission.

The question of the nitrogen excretion in eclampsia and allied states has received much attention lately and

our knowledge of this matter has been greatly increased. In normal health the nitrogen is excreted in various combinations. Folin estimates the relative quantities of these are as

Urea	87·7%
Ammonia	3·3%
Creatinine	2·7%
Uric Acid	0·7%
Undetermined N_2	5·6%

The total nitrogen is usually about 15 grms. During eclampsia these proportions are greatly altered and Zweifel working on 32 cases found (1) a great decrease in the urea nitrogen, the percentage ranging from 27% to 55% of the total, while (2) the ammonia nitrogen is relatively greatly increased registering from 5% to 16·5% in different cases. The uric acid percentage remained about the same while a slight increase followed by decrease was found in the undetermined nitrogen.

Williams[12] has investigated this matter very fully and a complete statement of his latest opinions was given in a lecture before the Glasgow Obstetrical Society in June 1912. He not only agrees entirely with the above findings as true of eclampsia, pernicious vomiting and all other toxaemic conditions complicating pregnancy, but uses the fact in diagnosis. He emphatically stated that a pregnant woman having hyperemesis and a low percentage of ammonia in the urine is not suffering from true toxaemic hyperemesis gravidarum and that such a case after excluding medical and surgical causes of vomiting he was prepared to treat by

suggestion alone.

I consider that this is a most important point and while I do not propose to go into a lengthy account of tests which merely confirmed other findings I should like merely to mention my methods and results. I used always catheter specimens and adopted the course of treating a known quantity of urine with potassium oxalate adding a few drops of phenolphthalein and titrating with decinormal soda until the mixture turned faintly pink. Then 4% formalin was added to the mixture which was again titrated until faintly pink. The number of c.c. of soda for the two combined titrations was then read off and used in the formula.

$$\text{c.c. of } \frac{\text{NaOH}}{10} \times \cdot 0016 \times \text{total daily quantity of urine in ounces} =$$

grms of Ammon. Nitrogen in 24 hours.

The grms of urea nitrogen were estimated by the following formula :-

$$\text{Grms of urea in 24 hours} \times \cdot 0303 = \text{grms of urea N.}$$

I carried out these tests in only 9 cases but in each of these on admission I found the percentage of ammonia or "Ammonia coefficient" high while the amount of urea was low. As convalescence progressed the figures altered and usually came back to somewhere near the normal, although in 2 cases while the ammonia came down low the urea failed to rise about 70% while the patients appeared healthy and well in every way and were dismissed so. In two cases admitted as hyperemesis gravidarum I found the ammonia coefficient low while the urea was about the normal. These cases were sent in with histories of severe and uncontrollable vomiting of several

weeks duration, but on no other treatment than purgation ceased vomiting practically at once.

In albuminurics again I found the ammonia high with low nitrogen, and may roughly say that the more severe the toxaemia the higher the ammonia coefficient and the lower the urea percentage. This is of course a generalisation and has exceptions to it. In normal pregnancy I incline to find the ammonia about 3·5% to 4% with urea about 80% to 83%. This is somewhat different from the figures quoted above, but I give them merely as my findings and not in any way to correct figures arrived at over a much larger number of cases and by much more exact methods than mine.

The highest ammonia coefficient I ever found was 13·5%. Zweifel reports 16·5%. The usual on admission was about 10% to 11%. My usual urea percentage was about 45% to 50% and the lowest was 38%. Zweifel's lowest case was 27%.

Similar work on this important subject has been done by Erving, Wolf, Leathes and Longridge. Their findings are to the same effect as Zweifel's and Williams' but they do not find such wide differences usually in the ammonia and urea percentages.

Zweifel ascribed the phenomenon to abnormal presence of some acid in the blood. He considered it to be lactic acid which he found in the urine of 17 cases of eclampsia. The idea is that the acids combine with the ammonia and prevent its conversion into urea. The lactic acid idea is not generally accepted and has been practically disproved. I shall discuss the matter more fully when considering aetiology. Other observers have analysed the urine for the presence of ultimate products of protein disintegration.

Hofbauer in 1907 published these results :-

Albumes	Found twice
Amnio Acids	Glycocoll was found once.
Lactic Acid	Found 3 times
Formic Acid	Found 3 times
Succinic Acid	Never found
Pentoses	Never found

I never made any attempt at such analyses and can only quote these figures and findings.

Microscopical examination of urinary deposits was done in all cases on admission and at intervals during convalescence. In most cases deposit was more or less plentiful and a drop sucked up in a pipette could be examined at once. If in any way scanty a specimen could always be centrifuged.

The most prominent single object was the cast. I found casts of every kind although perhaps the most plentiful of all were the simple hyaline casts. Their presence is probably of very little importance but they were practically never alone, being accompanied by epithelial and granular ones often in great numbers. The casts were hardly ever of any great length. Granular were more numerous than epithelial and I found that cases showing a large proportion of the latter were usually very severe. In the most of cases these casts disappeared as the condition improved but in a few cases this was not so. It was not usual however to find the casts lingering necessarily where the albumen was slow to disappear and indeed only 1 case showed the two elements remaining. I consider this a certain sign of previous nephritis. It of course indicates a present

nephritis. In one case - Case II Appendix - there was copious albumen on admission but no casts were to be found even after centrifuging the urine. My experience is that casts are much more numerous in eclampsia than in ordinary nephritis without pregnancy; that the presence of granular and especially epithelial casts is a sign of positive destruction of kidney tissue and therefore bad in prognosis: and that in uncomplicated cases the casts usually clear completely although in some they are slower in disappearing: that if with the patient well the casts remain fairly numerous especially granular and epithelial the patient probably has old standing nephritis and certainly has now permanent nephritis.

Besides casts uric acid crystals were frequently to be found. They presented their characteristic brown tint and differed nothing from their appearance elsewhere.

Pus cells and blood corpuscles were present in a certain number of cases. Blood cells were visible usually only for a day at a time. They were usually very scarce although in Case V. they were very plentiful.

In one case only - Case V. did I find tyrosin crystals. I never found leucin.

CHANGES IN THE ORGANS.

The Liver. Changes in the liver have been noticed from a very early time although it is only in recent times that we have found a conception of the relationship of these changes to the causation of the disease.

Of our cases 5 came to sectio and in every one of them

distinctive liver changes were present. In two cases there were extensive liver haemorrhages present, in one case some small haemorrhages and in the other two distinct fatty degeneration.

The main distribution of the haemorrhages was sub-capsular although in the more extensive cases patches were seen throughout the substance of the organ. I am convinced that the hepatic are the most constant lesions at least in fatal cases. Holland, although admitting the great frequency of hepatic lesions, considers renal lesions a shade more frequent.

In 1902 Schmorl[13] published a record of 73 sectios. In 71 cases liver changes were present, in the other 2 cases there was thrombosis of the main branches of the portal vein. Bouffe de Saint Blaise demonstrated hepatic lesions in 42 consecutive cases. Williams could demonstrate similar changes in all the livers examined but interference with hepatic function frequently could not be diagnosed during life. Slenious[14] reports that in 500 autopsies at the Johns Hopkins Hospital Opie noted these particular lesions to be present only in eclampsia.

Following haemorrhage necrosis may ensue. It was not seen in any of our cases.

Welch[15] writing on the findings in 12 cases of eclampsia diagnosed during life reports thus :-
Haemorrhagic changes in and about portal space - 6 cases: necrosis of centre of lobule - 3, cloudy swelling of parenchyma - 2: general swelling of cells and autolysis 1.

The most important work in this respect has been done

by Konstantinowitsch[16] who published the result of his work in 1907. An excellent account of his work is given by Holland. He examined microscopically the livers of 30 eclamptics and divided the lesions found into 3 classes according to their severity.

Class I. No Fibrinous thrombi in the capillaries, nor necrosis of liver cells nor haemorrhages. Degeneration of liver cells at periphery of the lobules.

Class II. Haemorrhage, fibrin formation, dilatation of capillaries, and degeneration of liver cells. These changes were entirely in the periphery of the lobules.

Class III. Necrosis diffuse and implicating besides liver cells and endothelium of capillaries the connective tissue of Glisson's capsule and its vessels throughout the liver.

He made an attempt to describe in terms of these changes the action of the toxin on the liver substance. He describes the changes met with in Class I as being the primary changes caused by the action of some toxin. The changes in Class II include thrombosis of vessels. This he considers due to the same toxin as causes necrosis of liver cells and points out that 2 factors aid greatly in causing thrombosis - the sluggishness of the hepatic circulation and the increased coagulability of the blood which I mentioned previously. The first changes are at the periphery but later the interlobular veins are affected. The necrosis of the liver cells is gradual and beginning from the periphery extends towards the centre of the lobule. It is due to the blood supply being cut off by the thrombosis of the vessels.

He noticed in two of these cases whose course was somewhat prolonged that there had been some reaction in these areas with formation of granulation tissue. In long standing cases therefore a process of cirrhosis may occur.

The sequence of events is thus supposed to be first degeneration of liver cells and endothelium of capillaries at the border of the lobule. This is followed by thrombosis in the peripheral capillaries which causes delatation of other parts of the capillaries and haemorrhages into the surrounding tissues; necrosis of liver cells follows on haemorrhage and thrombosis and then stasis of the circulation ensues causing thrombosis of the interlobular vessels with consequent necrosis of larger areas of liver cells.

The essential feature is the beginning of these changes at the periphery of the lobule: in hyperemesis and other toxic states the liver changes start in the centre of the lobule. Eclampsia may thus be recognised by the changes present in one organ.

It is evident that what the toxin of the disease may fail to do in some cases the results of its action may do: that a person may die solely of haemorrhage into the liver and not necessarily of eclamptic toxaemia. I am of the opinion that in no case having hepatic haemorrhage sufficiently extensive to be clearly seen by the naked eye does recovery ever occur.

The Kidneys. Renal changes have long been noted in eclampsia and for a long time the disease was thought to be merely uraemia occurring in pregnancy. Certainly a number of cases are such, yet the condition may occur and be

fatally severe with slight or no kidney changes.

Holland is of opinion that kidney changes are present in about 99% of cases. I consider this too high an estimate and base my opinion partly on my own experience and mainly on the writings of others. In our 5 sectios kidney changes were present in 3. The other two showed quite normal kidneys and in the affected cases the main changes were cloudy swelling and in one case a patch of necrosis under the capsule.

The changes found are of course degenerative and affect mostly the epithelium of the convoluted tubules where the cells show all changes from cloudy swelling to necrosis. In the glomemli fibrinous thrombi are found and sometimes proliferation of epithelium and exudation of leucocytes. Jardine likens the effect on the kidney to the effect of turpentine or cantharidine.

The same author reports two cases where changes were seen in the foetal kidneys. They were such as I describe in the maternal organ.

Jardine and Teacher reported (Journal of Path & Bacteriol Vol. XV.) in 1911 two cases of symmetrical necrosis of the entex of the kidneys associated with eclampsia and suppression of urine. Since then Jardine and Kennedy[16a] have reported 4 other similar cases. Grienard and Potocki administered methylene blue to 7 cases of eclampsia and found the drug always in the urine shortly afterwards, thus showing that although anatomical changes may be present the function was not greatly impaired. Prutz found kidney changes in 361 out of 368 cases from the literature.

The Brain. Frequently changes of various kinds are met with in the brain, the most important of which is haemorrhage. Holland suggests 90% as the frequency for brain changes to be found post mortem. I cannot form an estimate but can readily understand how the brain will frequently be affected in this disease. In 2 of our 5 cases distinct cerebral haemorrhage was present. In Case V it had torn up the whole left cerebrum and was pressing on the 4th ventricle. The other was subdural and extensive.

I am convinced that in a number of cases very minute haemorrhages occur and the damage done to brain tissue by these might account for the mania, undue stupidity and other mental signs following the disease.

The cerebral haemorrhages are thought by some to be due to the convulsions themselves. This is no doubt true in certain cases but it is not so likely to occur in a young and presumably healthy patient as in an old person with diseased vessels prone to rupture. In an ordinary case of eclampsia I consider that cerebral thrombosis is an invariable precursor to cerebral haemorrhage which obviously may then be relatively easily produced.

This is a further example of how a patient may be killed by the secondary results of a disease and not by the disease itself. Obviously no matter what treatment were instituted to meet the eclampsia such a cerebral haemorrhage as I describe would of itself cause death.

Other changes however are reported. Prutz reports oedema in 42% of cases: hyperaemia 35%; he finds apoplexy

in 13% and no change in 10% - just Holland's figures.

Some are of opinion that oedema and hyperaemia of the brain are the cause of the disease. Schmorl found thrombi of the smaller vessels in 58 out of 65 sectios and considers that the cause of the areas of necrosis occasionally seen.

It has been suggested that the brain cells may combine with and neutralise the toxin in some cases as happens with tetanus. No tangible evidence of such a combination is to hand but it is a good theory to explain eclampsia without albuminuria.

Blood Pressure. The blood pressure was taken always on admission, then on subsidence of the fits and then daily at noon till dismissal. During fits it was always high, a fair average being 180 mms of Hg. It usually subsides to 120-125 in convalescence in uncomplicated cases and a continued high blood pressure in a patient otherwise apparently well should at least lead one to look for other signs of chronic nephritis. The highest was in Case II Appendix and registered 210. She made a perfectly good recovery and was dismissed with b.p. 120. During fits the pressure was higher than between fits, being usually 190 - 195 as opposed to 180.

The normal blood pressure is usually about 120 while towards the end of a normal pregnancy the pressure is about 130-135.

In Case I Appendix which was a post partum eclamptic the blood pressure was 165 before the onset of convulsions. It never rose above 170 and declined quite normally.

I consider the arterial tension a most important matter in eclampsia and think that as it is probably the cause of cerebral haemorrhage and to some extent of liver haemorrhages also it is justifiable to treat it symptomatically e.g. by nitrites to prevent such consequences. Bar[17] states that increased arterial pressure is usually the first sign of eclampsia and considers it important from the point of view of prognosis also.

Changes in the thyroid, parathyroid, spleen, suprarenals, placenta and indeed in practically every organ in the body are described, but I do not intend to consider them here. I propose to discuss somewhat fully the present views regarding the aetiology of eclampsia and shall describe such changes as occur in the mentioned organs in this respect.

AETIOLOGY AND PATHOLOGY.

Eclampsia has been called the disease of theories and the name is apt when we consider the number of views put forward regarding its causation.

Many of these theories have been proved to be utterly wrong, others have not been substantiated by their originators, while of others we can as yet form no opinion.

As the disease is so general a condition practically all the ideas point to some condition of the blood or to some substance carried by the blood as being causal.

The exact cause is not yet known, yet one may say that we are coming nearer the light every day and with our improved modern methods of investigation we may hope in the near future to come on the exact cause and so to find an efficient preventive and cure.

The older views were to the effect that some chemical circulating in the blood was the cause and Spiegelberg in 1870 put forward the view that ammonium carbonate was that substance; analysis however has quite failed to substantiate this view.

Traube and Rosenstein later considered the cause to be oedema and anaemia of the brain. Often the brain is found normal post mortem and so this theory also had to be abandoned. On this theory it would follow that the brain taking part in the general oedema the more that oedema existed the more severe the eclampsia would be. This is not necessarily the case: again some severe cases occur as I have previously pointed out without any oedema.

With the advance of bacteriology it is not surprising that a bacterial origin has been suggested. It was first put forward by Delore and Rodet of Lyons in 1884. They cultivated various bacteria from the blood, urine and tissues but their results were very contradictory and others such as Döderlein and Schmorl who performed similar experiments found quite negative results.

The idea that the condition was a toxaemia then began to take hold of observers and it is the accepted view to-day. We are still left to discover the exact toxin or toxines however.

It was reasoned, especially perhaps by Fehling and Dienst, that under ordinary conditions of metabolism the liver was sufficient to act on and neutralise toxic products of metabolism which were then excreted by the various excretory organs, but that during pregnancy the maternal organism had the metabolic products of the foetus to deal with plus its own and that while this is in most cases quite successfully accomplished there are occasions when owing perhaps to injury to the excretory organs or to the liver some of the products are not neutralised and are not excreted, but remain as harmful substances circulating in the blood, i.e. a toxaemia is set up. This toxic condition of the blood further injures the excretory organs and so a vicious circle is set up. This proceeds until a certain degree of intoxication is reached when the nerve centres can stand no more and so begin to emit discharges of nerve energy recognised clinically as fits.

In support of this idea Baron and Castaigne showed

that substances injected into the foetus could be transmitted to the mother but that such transmission stopped immediately on the death of the foetus. Again the fact that convulsions similar to eclamptic fits may occur in newly born children would point to the fact that these toxic products could be transmitted from mother to foetus.

If the above theory be true it would invariably follow that eclampsia would never occur after death of the foetus and if present would cease on such an event. This however is by no means the case and Jardine reports 10 cases where in albuminurics or eclamptics he delivered a macerated foetus. Again eclampsia may develop where no foetus exists, e.g. with vesicular mole, a fact which to my mind entirely discounts the foetal intoxication theory.

Hitschmann in 1904 was the first to describe such a case and since then other cases have been recorded by Gross, Falk, Olshausen, Kroemer and Dienst.

The microscopical evidence to hand from the various maternal organs and blood all points to degeneration of cells, and the theory at present accepted is that such degeneration is of the nature of an autolysis. The results of chemical examination of the organs and fluids of the body point to the presence of various ultimate products of protein disintegration showing that a proteolysis is in progress.

While discussing the condition of the urine I pointed out that Hofbauer had found amido acids and other products of protein disintegration in the urine. The same observer made careful examination of eclamptic liver tissue for

similar substances and found various fatty acids-formic, lactic and succinic acid, certain amino acids-trypsin and glycocoll while albumoses were present in small quantities only. These experiments were to prove that autolysis of liver tissue occurred during life in eclampsia. He gave no quantitative figures and performed no control experiments on livers of other kinds of patients. His work and results are however extremely valuable.

Now every living cell contains ferments as a necessity to its existence. The normal action of these ferments is to break up substances brought to the cell into their ultimate products which are then used for cell nutrition. Again under proper conditions they would help to destroy a foreign cell coming within their sphere of action. Their action is thus normally entirely for the benefit of their containing cell. Now the cells of different organs contain a varying number and variety of ferments; the placental cells have been proved to contain by far the most and greatest variety of ferments; the liver and pancreatic cells contain a vast number of ferments also, but the cells of every organ although proved not to contain nearly so many or so virulent ferments as these mentioned yet all do contain ferments.

It is not to be wondered at that the liver ferments are so virulent and numerous when we consider what a vast amount of chemical work is performed by that organ in dealing with the products of digestion and neutralising the harmful toxic products into urea and other harmless bodies. But under certain conditions the ferments of the cells act not for the benefit of the cell as I have shown but actually to its

detriment breaking down the protein of the protoplasm into lactic and amino acids and other ultimate products of proteid disintegration. The conditions necessary may be briefly stated as two - one the presence of some substance or substances causing increased ferment activity, and the other some weakened condition of the cell itself. If the latter condition be present it is easy to understand how the presence and action of the ferment stimulant will produce further damage and weakness to the cells allowing of a more easy action of the contained ferment and so setting up a vicious circle. If the organ whose cells are so affected has an action in changing harmful and toxic products of metabolism elsewhere into harmless ones, e.g. the liver, it is obvious that these harmful products will be free to circulate unchanged and to produce a toxaemia.

This sketch of the process of autolysis is short but the main principle is shown that the essential feature is the destruction of protein of cell protoplasm by ferments in that particular cell. Autolysis may occur to some degree physioglogically. It forms the basis of the resolution of pneumonia in the main factor in the involution of the uterus after parturition. It occurs always after death and constitutes the primary stages of body decomposition.

The products of protein disintegration are many, and as has been previously noted they have been isolated in the liver, blood and urine of eclamptic patients. They have also been found very abundantly in the placenta.

However, not only are protein destroying - proteolytic - ferments present in these organs but also carbohydrate

destroying ferments - glycolytic - and in some cases fat destroying - lipolytic - ferments. The main products of protein disintegration are many and at present a great number have not been identified. The main known ones are

Amino Acids

Amino Acetic Acid - glycocoll

Amino proprionic acid - alanin

Amino - valerianic acid

Dibasic Amino Acids

Aspartic and Glutanimic acids

Lysin and Arginim

Aromatic constituents, the most important of which is Tyrosin

The above process of toxaemia caused by toxines formed within the body is known as an autointoxication and according to this theory eclampsia must be considered as such.

The main feature of such a condition is the saturating of the body with end products of protein disintegration, with toxic ferments causing such disintegration, with the results of imperfect function of such an organ as the liver or kidney and also with the substance causing the original stimulation of the intracellular ferments.

What this last substance is is not at present definitely known. But we know that the most potent stimulant of ferment activity in the cells of an organ is the presence of a ferment from another organ. What is the other organ responsible in this case?

Eclampsia never occurs in a non-pregnant woman although, as previously noted, it may occur in a molar pregnancy.

The organ therefore furnishing the autolytic ferment stimulant must be present in pregnancy but not otherwise.

Obviously then we must consider the uterine contents, the most important of which are foetus and placenta. Eclampsia may occur after foetal death as has been proved. This and the discovery of molar pregnancy with eclampsia would seem to exclude the foetal origin.

The other organ therefore, the placenta, has been thought and is by most observers of to-day considered to be the origin of this proteo-lytic ferment stimulant.

The placenta has its whole maternal aspect in contact with the maternal blood, therefore any substance found in the placenta would very easily find its way into the maternal blood stream. Again, the placenta is known to be the seat of many and powerful ferments: free placental cells have been found in various organs in eclampsia. This suggests cytolysis but the value of this observation is somewhat nullified when we find similar free placental cells in the maternal circulation in normal pregnancy. We shall return to the consideration of the placenta. For the present let us consider how changes in the organs and in the fluids of the body are to be explained in terms of the above observations. The changes in the organs are mainly then necrosis of cells and thrombosis of vessels. Which is the primary change?

Schmorl was of opinion that thrombosis was primary and caused cell degeneration followed by autolysis. He injected emulsions of placenta and found these to be followed by widespread coagulation and other eclamptic changes and concluded that eclampsia probably was an intoxication with

placental cells causing coagulation.

Konstantinowitch however is of opinion that the primary change is degeneration of cells and that the end products of such along with the intracellular ferments percolate through the endothelium of the capillaries and so cause thrombosis secondarily. Later the process extends and then haemorrhages occur.

Dienst[17a] looks on the increased amount of fibrinogen in the blood with the consequent increased tendency to coagulation as the chief factor. He considers that the origin of the fibrinogen is the white cells which are greatly increased in eclampsia and which he considers are broken down in the placenta so liberating the fibrinogen.

These products of protein disintegration are of the nature of organic acids and by their circulation they cause an acidosis of the blood. The low ammonia coefficient of the urine is explained by the supposition that the acids combine with the ammonia and so prevent its conversion into urea as occurs usually.

Most of the changes found therefore may be explained in terms of this theory. What is supposed to happen is this. Normally many ferments pass from the placenta into the blood stream. In most cases they are met and neutralised by the liver cells but in certain cases where the integrity of these cells is impaired, e.g., by constipation, gastritis or hepatitis, or where their vitality is lowered, this does not occur and the ferment then stimulates the intra-cellular hepatic ferments to start autolysis. Once started the condition is progressive unless very soon checked. However

the kidney is presumably still intact and is of known value in eliminating toxic substances. But the circulating toxin damages the renal parenchyma causing limitation of function and still further retention of toxines and waste products in the body. This probably causes a reflex spasm of arteries which is responsible for the high blood pressure and which, plus the thrombosis of vessels and degeneration of cells of vessel walls, is responsible for the haemorrhages seen in various organs. Again the circulating toxin comes into constant contact with the elements of the blood which it stimulates, attacks and injures, producing the condition described and impairing the normal function of the red cells.

This theory seems to explain most of the facts of the case. It explains why the condition is more frequent in primiparaewhose systems are not "tuned" to the presence of the results of pregnancy: it explains why the condition is more frequent in the later months of pregnancy when the placenta is obviously most actively functionating: it explains how eclampsia is relatively frequent in twin pregnancy where the placental function is also relatively greater than normal.

It also explains how if the hepatic function - the first line of defence - be healthy and consequently active in changing harmful into harmless substances a patient may yet have deficiently functionating kidneys and still escape eclampsia.

I am inclined to believe that such a state of affairs is the cause of the condition. It however does not explain all the signs met with. What causes cerebral haemorrhage?

Is it thrombosis plus fits or is it that the toxic results of intracellular fermentation percolate here as in the liver damaging the vessel walls and so predisposing to haemorrhage. This would indicate that unless for some reason the brain were relatively seldom affected thrombosis or haemorrhage or in less severe cases degeneration of brain cells would occur very frequently. In the last state there would follow permanent or at least long lasting mental changes or paralysis. It is possible that some such sequence of events occurs.

Much work has been done to find out the substances coming from the placenta to cause the changes described. When it was discovered that placental cells were found in the circulation Veit, Ascoli and Weichardt concluded that these were actually responsible or were secondarily so by causing the production of antibodies. Experiments were made by these and other observers by making emulsions of normal or eclamptic placentas and by injecting such emulsions into various animals and noting the effect.

Leipmann[18] did some very valuable work in this respect. Jardine quotes him and gives an excellent summary of his work and results.

He injected powdered normal placentas into rabbits and got no results; he then injected powdered eclamptic placentas and found frequent coma and convulsions. In all 113 experiments were made.

When the placentas were compressed and the juice expressed no bad results followed injection while powdered pulp caused death. He reasoned from this that the toxin

was contained in the placental cells. In some cases a slight amount of toxin was found in the juice; it was removed by reagents precipitating albumen. Again while the powdered pulp was very toxic one day it might be quite inocuous on the following day, showing it to be very volatile and easily disintegrated.

He injected 6 animals with pulp from 3 patients, each of whom had only one fit - all died.

He injected 4 animals with pulp from patients with 20 fits - one died. He then injected 3 animals with placenta pulp from a case having 27 fits - one died. It would appear from that that the toxin gradually passes out of the placenta during the fits.

He explains post partum eclampsia by supposing the liver to act as a filter to the toxin and stores some of it up to ultimately render it inocuous, but in some cases having a special predisposition this stored toxin passes post partum into the system and causes fits.

He made a powder from the brains of 3 eclamptics and injected 9 animals. No result followed. Thus either there is no toxin in the brain or if there it is neutralised.

Leipmann's work has been very severely criticised and Lichstenstein, Frank and Dryfuss performing similar experiments could find no difference of action between normal and eclamptic placentae.

Many ferments have been found in the placenta. There are glycolytic, proteolytic and lipolytic ferments and a blood coagulation ferment discovered.

It has lately been shown by Mathes, Basso and Dryfus

that autolysis probably takes place during life in eclamptic placentas but if we admit this we still have to face the fact that such autolysis is only an isolated fact along with others in the disease, in that it may be the seat of the origin of eclampsia. With all this work we are not yet at the actual cause of the disease and my own opinion from clinical observation is that the causation of eclampsia is a multiple state of affairs: that the actual fits are caused by discharges of nerve energy from the poisoned nerve centres, especially the motor centres: that the coma is due to brain implication: that the actual causal toxines are multiple and include ferments from the placenta, ferments from cells of organs, results of disintegration of cells, results of lack of hepatic, renal, intestinal and entaneous action in neutralising and eliminating noxious products of metabolism and usually results of intestinal putrefaction or stasis or at least constipation.

Theories however quite apart from the placenta have been put forward.

Vassale[10] is of opinion that the cause of the condition is disease or at least loss of function of the parathyroids. He adduces as reasons the fact that congenital absence of these organs or various morbid changes in them have been found in many cases: from the beneficent effect of parathyroid treatment in eclampsia reported by Stradivari and Zanfrogmier: from experiments on gravid cats and rats reported by Thaler, Adler and others and showing that in latent parathyroid insufficiency there often occurs in during the last third of pregnancy severe para-thyroideoprival convulsions known as

experimental eclampsia. Three bitches were used and the parathyroids removed. All went well until the last few days of pregnancy. In two cases experimental eclampsia developed two days before parturition. In one case parathyroid extract was given orally in large doses: the convulsions ceased and a normal labour followed. The other case was not given parathyroid and died. The third case took convulsions a few minutes before labour. A large dose of parathyroid was given and 6 pups were born - 4 alive. This case and the first one made a normal recovery. The urine in these cases contained albumen.

Massalia and Sparapani[20] report on having removed the parathyroids from 2 dogs and 1 cat before or during pregnancy and eclampsia followed each time. In two cases a complete cure was effected by administering parathyroidin. In post partum eclampsia occurring in a setter a cure was effected after giving parathyroidin. They conclude that eclampsia is an autointoxication which fulminates in the fit and which is due to deficiency of parathyroid secretion and that this secretion is antitoxic to certain products given by the foetus to the mother.

Albech and Lohse[21] consider the liquor amini to be important causally and report that the liquor of 5 bad but not fatal cases of eclampsia was drawn off into sterile glass tubes by puncturing the membranes high up. Guineapigs and cats were taken and 30 c.cm of this sterile liquor was injected into the peritoneum. One pig died in convulsions in 32 hours and the others were killed in 6 days.

Typical eclamptic liver changes were found. In some

cases the liquor was filtered before injection. Similar but slightly less marked effects were produced. Cloudy swelling and fatty degeneration was found in the kidney but the liver changes were more prominent. Control experiments were done with healthy liquor and no toxic action was observed.

Leith Murray considers eclampsia due to a toxin having haemolytic, haemagglutinative and endotheliolytic elements. These characters have been sufficiently emphasised earlier in this paper. Murray however compares eclamptic lesions to those produced by snake poisons and finds them similar. This is not an accidental resemblance as Burgess has demonstrated in the maternal serum a cobra inhibiting factor which Murray found to be absent in eclampsia. He suggested the experimental use of Calmette's antivenine to be given subcutaneously in the pre-eclamptic stage and intravenously during eclamptic fits.

So far as I know, no such treatment has ever been attempted and certainly I have never seen any account of such.

In 1906 Silvestre[22] put forward the suggestion that eclampsia, tetany and similar convulsive conditions are due to a deficiency of calcium in the blood. He points out that at periods of life such as early infancy, between 6 months and 12 years and the puerperal period when spasmodic affections are apt to appear this deficiency of calcium occurs.

He bases his hypothesis on these facts :-

(1) An increased elimination of calcium occurs in various psychopathies associated with much mental work.

(2) An excess of calcium in the body causes cerebral depression.

(3) The brains of children the subject of tetany have been found to contain less calcium than normal.

(4) Therapeutic benefit is obtained by administering calcium salts in various nervous affections of rickets.

(5) The stimulation of the nervous system caused by administering lecithin or phosphorus has been thought to be due to their favouring the assimilation of calcium.

(6) In pregnancy and lactation the mother loses an appreciable amount of calcium.

I have been able to find nothing fresh on this subject nor can I speak on any results of treatment based on this supposition.

When discussing the placental theory I remarked that the foetus and placenta were bodies present only in pregnancy: there is however another organ - the breast, which although present always is greatly hypertrophied in structure and function during pregnancy and lactation and some incline to the view that it is in the breast that the exciting toxin of eclampsia takes origin.

Hugo Sellheim[23] holds this view and supports it by pointing out that eclampsia occurs usually late in pregnancy and not when the placenta begins to functionate, and that its occurrence post partum cannot be explained on a placental basis. He theorises that various antagonistic bodies may be produced normally in the breast and eclampsia may be caused by the suppression of some which of course allows others to act unchecked and to cause toxaemia and eclampsia. He suggests accordingly the use of breast gland extract in eclampsia and reports 2 cases both post partum, one of which recovered

after injecting pot. iod. into the breast while the other also got well and after removing the breast.

Gottfried Persson[24] compares eclampsia with a pareses occurring at times in cows after calfing and finds many analogies. He then tried in 4 cases manual stimulation and milking of the breasts with good results. He supposes the cause to be a disproportion between the amounts of the constituent elements of the milk circulating in the maternal blood and the capability of the foetus to make use of them.

Van Hoogenhwyze and Ten Doeschate[25] are of opinion that the high percentage of creatin during pregnancy is very important. They found it increased during eclampsia and conclude that this increase should be regarded as a sign of hepatic deficiency, showing that the liver is unable to convert it into creatinine. Thus its presence is a sign of toxaemia even although the kidney be functionating properly.

Now all these various views are obviously very far apart and reasoning from one point of view we might ask which, if any, of these is the correct theory of causation that we might discount and exclude the others as erroneous. Another possibility of course is that all of them are wrong and that the cause or causes are some as yet unconsidered factors and it is to settle this very question that the vast amount of experimental research work at present being undertaken is being done.

But there is another consideration for us to bear in mind and it opens up the whole question. It is this. Is the condition that we at present call Eclampsia a single disease or is it not possible that various separate conditions

all characterised clinically by fits are being named indiscriminately eclampsia? This of course involves the question of diagnosis in any particular case, but I am referring here to cases admittedly of what we call eclampsia. We know that it is practically impossible to differentiate eclampsia from uraemic convulsions in pregnancy and we have no guarantee that various other conditions may not simulate it as well.

The above theories of eclampsia respectively hold the condition to be due to the presence of ammonium carbonate in the blood, oedema and anaemia of the brain, bacterial infection, the presence in the maternal blood of products of foetal metabolism, autolysis of organic cells stimulated by placental ferments, absence or disease of parathyroids, abnormal liquor amini, deficiency of calcium in the blood and mammary toxaemia. These views are very far apart, and I submit for consideration the theory that one day we may require to entirely recast our ideas of the disease and that in place of the name Eclampsia signifying a single definite condition we may substitute the term "fits during pregnancy" to be classified according to their causation. The one point clear is that Eclampsia occurs so far as we know only during pregnancy or immediately post partum so that it would appear that either the uterine contents have a direct effect causally or they so influence various organs and systems of the body that the effect is caused.

Again there is the question of post partum eclampsia. All our theories point to the cause being present during gestation and post partum eclampsia has been somewhat clumsily

explained by Leipmann. Sellheim's theory fits in rather better with facts. Yet it is not difficult to imagine how the effect of a toxin may not be fully exerted until shortly after the absence of its cause: that however does not explain eclampsia occurring 10 days post partum (Brenot), or 9 days (Naegele) or 12 days (Cazeaux) or 16 days (Legroux.)

A case has been reported occurring 8 weeks post partum (B.M.J. 1908 Vol. I Page 1220). It is difficult to reconcile these facts to any of our accepted theories and perhaps Sellheim's views fit in best here. But it also opens the question as to whether post partum eclampsia is not due to somewhat different set of causes to those that cause the condition ante partum.

Shortly then on our present basis of knowledge I favour the placental ferment stimulant theory but look to the future not to be surprised if our views of the condition are totally changed and that we come to some such findings as I indicate above.

The question of causation of eclampsia is obviously a vital one and I consider that a sufficient reason for my loitering so long discussing it.

D I A G N O S I S.

Regarding eclampsia as we do as a toxaemic condition complicating pregnancy and evidenced clinically by fits, obviously the conditions to be excluded in making a diagnosis are other conditions causing convulsions or coma which may

appear in a pregnant woman, although not necessarily so as in the case of eclampsia.

The state of the ammonia coefficient of the urine is a valuable thing in this respect and although certain other causes for a high coefficient must be borne in mind, e.g. prolonged starvation or any cause of acidosis, it is in my opinion an excellent help in diagnosis. It is invaluable negatively because if the coefficient be low I think that one may fairly exclude eclampsia and look to epilepsy, hysteria, cerebral haemorrhage and to other causes for the condition.

Mayer of Koingsberg reports a case of fits during pregnancy diagnosed eclampsia and which turned out later to be dementia paralytica: he quotes Jolly who reported a similar case. Both cases had albuminuria.

The Wassermann reaction has been tried in eclampsia with most useless results diagnostically.

Gross and Bunzel in 1909 reported positive results at the height of the disease only - 5 cases.

Nadory had 3 positive results but Alsberg and Thomsen in 21 cases found all negative. Semon tried it in 20 cases: 13 severe, 6 slight, and 1 with post partum functional amaurosis; all were examined at the height of the disease and of the severe cases 3 were positive: all the rest - 17 - were negative.

On these findings one cannot consider the Wassermann test of any diagnostic value in the condition.

Knapp mentions strychnine poisoning, while Schild mentions nitro-benzol as causing symptoms the same as eclampsia and requiring differentiation. They also point to the effects

of lead, phosphorus, carbolic acid, perchloride of mercury and mushrooms as causing a toxic state accompanied by convulsions and coma.

I have previously pointed out how cases have been recorded of eclampsia without convulsions.

Of eclampsia without oedema I had two cases - Cases V and VI Appendix - and one of them (Case V) had to be differentiated from chlorodyne poisoning as she was found unconscious with contracted pupils and with a bottle of chlorodyne beside her. I have fully reported her condition in the Appendix. She had extensive cerebral and hepatic haemorrhage and was 7 months pregnant. Similar cases are published by Randle[26] Maygrier[27] and Desmoues[28]. Case VI gave typical epileptic cries but the lack of epileptic personal or family history, the albuminous urine with high ammonia coefficient and the final development of the case settled the question.

The above remarks serve to point the main common and some uncommon conditions to be differentiated from eclampsia. The whole question of differential diagnosis is discussed at length in many text books and I do not propose to pursue the matter more fully. I consider the best single diagnostic sign then is a high ammonia coefficient in urine likely but not necessarily albuminous. Other causes for this must be excluded. It is a perfect negative sign. I recognise that this is dogmatic but I am convinced that it is true and I put forward the statement for consideration.

PROGNOSIS.

It is difficult to generalise on the outlook in eclampsia as this is decided by so many and so varying circumstances: also, as I have previously maintained, eclampsia itself is not the primary cause of death in a great percentage of cases but rather the effects of eclampsia e.g. cerebral or hepatic haemorrhage, which may secondarily cause the fatal termination.

The maternal mortality is usually stated at about 20% to 25% with a foetal mortality of 35% to 50%. The consideration of various published mortalities of course brings us to consider the relative values of different treatment, but I wish to refrain entirely from doing so until I consider in the following section the whole question of treatment. In the Birmingham Maternity Hospital the usual mortality of recent years is 33%. Tweedy reports in 7 years in the Rotunda Hospital 74 cases with a mortality of 8·1%.

Lush[29] states the mortality in New York from 1867 to 1875 to be 1 in 700 cases, i.e. 1/7th per cent. It would appear from these figures either that New York is exceptionally lucky in that eclampsia there assumes a very mild form, or that the figures quoted were not quite correct. From my experience it is impossible to conceive so low a mortality in unselected cases.

From 1907 to 1911 we had in the Glasgow Maternity Hospital 256 cases of eclampsia with a maternal death rate of 29% and a foetal death rate of 45%; 22 of these cases died un-

delivered, while 13 were dismissed undelivered to come back at labour. While I was House Surgeon there we had 35 cases and 11 deaths - about 32%. Of course with one exception all our cases were admitted in convulsions while 5 were in extremis and died in periods from 20 minutes to 2 hours after admission. Naturally then Hospital figures are bound to be relatively higher than those of private practice because not only has one usually no chance of preventing the condition but also so many are sent into the big clinics because of their gravity, while in some cases they come in merely to die irrespective of any treatment employed.

The best authentic statistics of recent years are those of Stroganoff of St. Petersburg. He published first a series of 200 cases with a mortality of 5% and later 400 cases with a 6·5% death rate.

It is usually stated that the more numerous the fits the worse the prognosis. While I do not quite agree with this statement in toto I think that it is a sound thing to bear in mind in order to institute treatment as soon as possible. Before the Edinburgh Obstetrical Society, March 9th 1906, Jardine reported a case having over 200 fits which recovered. I consider that numerous fits while serious are less so than short and decreasing intervals between the fits. This last I consider very grave as the condition soon passes into coma and then death unless speedily relieved.

I consider the ammonia coefficient of the urine however as the best single sign of the gravity of an individual case. The higher the coefficient the more serious the case and vice versa. It is of course a rule with exceptions, but I

usually found the cases with high ammonia coefficient having more frequent convulsions than the others. Both of my own cases admitted in extremis had high ammonia and low urea with high albuminuria.

I may say however that clinically I found the general look of the patient the best prognostic sign. This of course means nothing in print, but I fear that I cannot find words to express my meaning more clearly. Some cases having quite a lot of fits might cause one very little anxiety, while others perhaps not nearly so convulsed caused misgivings - usually justified - as to the result. I am convinced from this that there is a type of case which, were it left entirely untreated would recover quite well. This of course means a slight degree of the condition, but such cases as I think of frequently exhibited quite numerous and decided convulsions.

High and especially continued or increasingly high blood pressure also forms a grave prognostic point. Not only does it point to a more severe action of the toxin but it also indicates increased probability of cerebral or hepatic haemorrhage.

With regard to the amount of albumen present I have not quite made up my mind as to its prognostic value. It would appear that the more albumen present the more injury to kidney tissue and consequently the greater poisoning of the system with effete metabolic products and I incline perhaps to look on marked albuminuria with suspicion prognostically. It would indicate greater difficulty on the part of the kidney in returning to the normal state and a greater tendency to future derangement, therefore it is likely that

the ultimate prognosis may be badly influenced by severe albuminuria even though the immediate outlook be unaffected.

I know that many, perhaps most, observers lay no grave stress on "heavy albuminuria" and W.F. Shaw[30] actually lays stress on scanty albumen as bad. He considers as bad prognostic signs (1) a small amount of albumen during the convulsive stage, (2) a high temperature and (3) post partum onset of convulsions. 1. Taking 10 grms. of albumen per litre of urine as a fair standard he reports 21 cases with quantities below it - 16 deaths - and mortality 61·9%: 18 cases with quantities over it and 3 deaths showing 16·6% mortality. 2. 100° was taken as a standard here and of 26 cases with a temperature over 100° 15 died giving 57·7% death rate: 19 cases had a temperature below 100° and 1 died. Death rate was thus 5·2%.

He also reports 12 cases with less than 10 grms per litre and temperature over 100° with 100% death rate. He had 7 cases with more than 10 grms and temperature below 100° and a death rate of 0%.

So far as these figures go they absolutely prove his contention but a more extended series of cases is perhaps to be desired.

Bar[31] lays stress on a continued high blood pressure as a bad sign.

Reddy[32] considers the amount of diminution of urea as the best sign of the degree of intoxication present and consequently as a prognostic point. Usually of course the less the urea the higher the ammonia coefficient so that this view agrees with my own ideas as stated above.

With regard to post partum eclampsia I cannot give a worthy prognostic opinion having had only 1 case: theoretically I would think that it would not be so severe as the ante or intra partum condition. Previous opinion was to this effect but nowadays it is coming to be considered as seriously as the other. Some indeed consider it the graver form and Shaw as quoted above considers post partum onset as of itself a bad prognostic sign.

However, apart from mortality the morbidity prognosis is worse in eclampsia than in normal labour for post partum sepsis is particularly liable to occur. Some report great tendency to post partum haemorrhage but my experience is quite the reverse. I am convinced that not infrequently a previously healthy kidney is permanently damaged by this disease. Such damage may not be extensive but it will form a nucleus for more advanced nephritis later in life.

TREATMENT.

The methods of treating eclampsia as suggested and practised are indeed multitude but I cannot think any more numerous than in the case of any other disease of so obscure origin.

Of no condition of affairs can it be more fittingly said that prevention is better than cure: obviously it is a very well done thing to prevent the onset of any disease having a death rate of 20% to 25% and it is the object at which all those practising obstetrics in private should aim. I am not quite prepared to uphold the view of Edgar and others that under proper treatment eclampsia is entirely preventable, but I consider that fully 80% of cases could be prevented if proper prophylactic treatment were instituted. In Maternity Hospital practice one sees a good deal of the results of such lack of prevention: these results are always serious and not infrequently most grave.

I intend first to discuss the prevention of the condition and then the various methods of cure.

PROPHYLAXIS.

As this should extend over several months it is obviously a part of treatment that practically always falls to be carried out by the practitioner attending: later if eclamptic fits develop the specialist is called in or the patient sent to hospital according to circumstances.

The possibility of eclampsia developing in any particular case should be borne in mind by the practitioner when engaged to attend any woman, especially a primipara. I am glad to think that the men are perhaps more alive to this now than was previously the case: it is only by impressing men with the seriousness of the disease and the urgent need for early treatment that we can ever hope to reach minimal incidence or complete prevention.

Whenever a practitioner is engaged then he should appoint a very early date when he can completely examine the patient in bed. General condition; state of tongue and teeth; condition of the bowels; presence or absence of oedema; duration of pregnancy; condition of heart and of pulse especially with regard to high pressure: these things should all be noted and a specimen - not necessarily at this time by catheter - of urine examined. The points in the urine are of course presence of absence of albumen and amount of urea and of ammonia. But a man in practice cannot be expected to have time or apparatus to test for ammonia coefficient or hardly even for urea, although such tests are simple in hospital. He must then fall back on the presence of albumen as his main objective sign of danger.

If all is found normal and healthy the patient should be advised as to her food - to eat light and easily digested articles which must be varied according to individual tastes: to ensure a quiet but thorough daily evacuation of the bowels - drastic purges are to be avoided - for which calomel, cascara or aloin will be found best: to be out in the open air some part of every day: to take suitable but

not severe exercise; quiet walking or driving in a rubber tyred vehicle are suitable forms of exercise: to have at least 8 hours sound sleep nightly and a nap in the afternoon if required: and lastly, and very important, to immediately notify the Doctor on any divergence from the normal good health and especially on the onset of headache, giddiness, epigastric pain, troubles of vision, or oedema anywhere.

If this is about the 6th month she may be left alone for 4 weeks when a second and similar examination should be made. If things are still well the instructions should be reinforced and another and similar visit paid in 3 weeks time. There should then be only 2 weeks between the visits and for the last month they should be made weekly and more frequently still when term is at hand.

Obviously by such visiting and examining of person and urine the accoucheur is warned quickly of any threatening toxaemia and can institute treatment at a time when it is most likely to be beneficial. The specimens of urine need not be catheter ones at first. If no albumen be present they need never be but if it be present a catheter should be passed to determine whether the albuminuria be due to fouling by vaginal discharge or to kidney impairment. However if things do not go well and symptoms of toxaemia develop active and immediate treatment must be instituted to combat it. The mildest form of treatment is restriction of diet and purgation while the most extreme is the inducing of labour or hysterectomy and any intermediate steps may be taken.

On the appearance of the slightest sign of toxaemia

then the patient should be at once confined to bed and a saline purge administered. An excellent purge is calomel g V in the evening with mag. sulph. the following morning. The action of calomel on the liver makes it peculiarly indicated here: it acts as a liver stimulant and such action is greatly required here to strengthen the first line of defence. The diet also must be considered. The usual treatment is to enforce a rigid milk diet entirely cutting off meat of any kind. Fully 2 or, if possible, 3 quarts of milk should be taken in 24 hours. Some observers, and among them Tweedy, however regard milk as positively harmful and hold out starvation as the best dietetic treatment. In view however of the known liver condition and of the saturation of the system with the results of proteolysis it has recently been suggested to feed such patients on glucose alone. My own knowledge of this treatment is, I confess, only second hand, but I hear it excellently spoken of in places where it is now routine practice. 14 to 15 oz. of glucose may be given daily. Longridge [32a] advocates such treatment and points out that although large quantities of sugar were given none appeared in the urine. Such starvation treatment need not be feared as an ordinary person, especially when confined to bed, can quite well stand complete withdrawal of food for several days and often for a week. Glucose too acts as quite efficient nutriment. I am inclined to favor the withdrawal of food and for several reasons. Milk is not nearly so easily digested as we are apt to think and the curds formed may require quite powerful ferments to digest them. Such curds are apt to

act as pabula for bacterial growth with consequent intestinal putrefaction. Now by withdrawing solid or milk food we obviously leave the stomach and bowel clear as a channel by which excretion can take place but from which little or no toxic absorption can occur.

The stringency of treatment employed will of course depend on the time of onset and severity of the symptoms, e.g. if only a trace of albumen be present and the patient be feeling well enough such a rigid diet as the above would not be required: fish and chicken may be allowed but meat must always be cut out. I prefer however to regulate prophylactic treatment according to the general condition of the patient more than by the amount of albumen present.

Other excretory channels must be attended to also. Free perspiration is induced at this stage best perhaps by frequent large draughts of tepid water followed by wrapping in blankets and surrounding by hot water bottles properly guarded. This is not depressing treatment in any way.

If under such treatment the toxaemic symptoms pass off we may allow a return to a more natural state of life allowing fuller diet with possibly later meat if greatly desired. It must not be taken more than once daily. Exercise may again be indulged in. But above all the bowels must act daily and the urine must be frequently examined for any trace of albumen. If treated in such a way toxaemia may never return and the pregnancy continue to full term.

But if the symptoms become worse, the albumen increased in amount and any sign of convulsions appear or if after

improvement the toxaemic signs return, then we are dealing with an intractable case requiring severe treatment. The treatment medically will be on similar lines to the above, food being entirely withheld or if milk be given it must be skimmed. Mag. Sulph. must be freely administered with pot. cit. and pot. acet. $\bar{aa}$ g 20 4 hourly for diaphoretic purposes. If under such treatment improvement does not take place the question must arise of terminating the pregnancy which is the cause of the whole condition.

In deciding on the inducing of labour several factors must be borne in mind. Firstly then the general condition of the patient must be seen to as to whether she will stand the procedure. In this respect I maintain that a considerable number of these cases are left far too long before induction is considered, often until they are fatally ill when the operation will merely hasten the end. If it be done in proper time it is accompanied by no risk. This merely emphasises the point that in treating the slightest degree of toxaemia of pregnancy we must always remember that we may require later to induce abortion, and that such induction should be done with these definite indications, namely that medical treatment is failing to arrest the toxaemia and that in the ordinary course of events eclamptic convulsions are likely to supervene. The best way is by inserting a bougie No. 20 about 8" up between membranes and uterine wall and leaving it to be expelled when contractions set in. This can be done quite painlessly without any anaesthetic. Secondly, the period of gestation must be considered but only very secondarily from a professional

point of view. If the foetus be over 7 months and consequently viable we would perhaps have less hesitation in inducing labour than with a foetus before that time but we must bear in mind this fact that the mother is here the main consideration and our treatment to a great extent must be to disregard the child entirely. Again with a dead foetus we would have no hesitation in inducing, but I maintain that we should have as little in dealing with a live foetus.

Against induction it has been urged that the uterine contraction might reflexly set up eclamptic convulsions. But obviously if this might happen at any stated time before term it would be much more likely to happen the longer we delayed until at normal full term convulsions would probably occur. At this time the labour will be more severe while the patient is less able to stand it, being poisoned by the prolonged toxaemia. The rational treatment then is if induction be necessary to perform it in good time. However if the earlier cases were more carefully treated there would be much less need for induction later and if observation and treatment as suggested above could be started early in all cases the eclampsia incidence would fall tremendously.

The reader is referred to an excellent article by Hardie [33] on the subject of prophylaxis of eclampsia. If however in spite of all our treatment even induction fits do develop we must then deal with eclampsia in the curative sense. I shall discuss this aspect of treatment later.

Reddy [32] of Montreal lays stress on the diminished

urea excretion as a guide to the amount of toxaemia present and states that by paying attention to this factor and treating it eclampsia has been abolished in the Montreal Women's Hospital.

The treatment recommended above is obviously on general lines rather than in minute detail: the detail must be decided on in every individual case by the special severity and other circumstances of that case. I am painfully aware that as perhaps the most of eclampsia occurs in the very poor classes such individual attention as I suggest cannot be obtained under present circumstances. From middle class upwards however when the accoucheur is engaged early there can be no difficulty and where the conditions are bad we should try and come as near the ideal as circumstances will allow.

I make no apology for dallying so long in this discussion of prophylaxis. It is in some ways the most important part of treatment.

CURATIVE TREATMENT.

It is frequently said that the treatment of eclampsia is to control the fits and to empty the uterus. Most of us agree in the main with this dictum although many are now in favour of leaving the uterus alone and treating the disease almost entirely symptomatically by sedatives. There is a third line of treatment however not included in the above, namely, promoting elimination. Perhaps the main

point of dissension between various obstetricians is not what principles of treatment are to be employed but how they are to be put into practice, e.g. what sedative to use - chloral, bromides, morphia: how to eliminate - transfusion, hot pack or hot air bath: how to empty the uterus - induction of labour, accouchement force or Caesarian Section. Besides these points treatment e.g. for severe haemorrhage or collapse - on general principles must be employed when necessary.

When a patient is admitted in eclamptic convulsions I consider that the immediate primary indication is to quieten the convulsion, although the immediate secondary, unless the fits show signs of ceasing quickly, is to empty the uterus as rapidly as may be safe for the patient.

Sedatives have been used from an early time in treating eclampsia and I fear that their indiscriminate use must have been productive of great harm. I favour the use of sedatives but only as a temporary measure in order that reflex stimulation be abolished while we empty the uterus or if labour be sufficiently far advanced while the uterus empties itself. I agree in the main with Jardine, who says "to give drugs to control the convulsions, which are after all only a symptom, seems to me somewhat irrational".

The main sedatives employed are morphia, chloroform, chloral, bromides and paraldehyde. Morphia is greatly used by many including Williams, Tweedy, Galabin, Eden, Stroganoff, while Bumm, Jardine and Little are averse to its use. The advice is usually to give a good dose gn.$\frac{1}{4}$ or gn.$\frac{1}{2}$ at first repeated after 2 hours if necessary, and again

if necessary but no more than 3 doses to be given. Galabin advises not more than 2 grains in 24 hours. Tweedy continues to administer it until the respirations are 4-7 per minute.

The reasons put forward for its use are that it decreases metabolism and so partly puts the metabolic sources out of action: it decreases the cerebral irritability and so controls the fits: it does not depress the heart while it lowers the blood pressure: while the reasons against its use are that it damages the kidneys and impairs excretion of all kinds.

Morphia I consider undoubtedly the finest sedative which we have, but in eclampsia while $\frac{1}{4}$ or in a case with severe fits $\frac{1}{2}$ grain may be given initially its repeated use is to be avoided owing to its accumulative action and its prevention of excretion.

The use of chloroform in eclampsia is rather a vexed question, some considering its continual administration ideal treatment, while others avoid it owing to its action on the liver.

Galabin advises its use if morphia fails, while Williams gives it during the attack to cut it short and morphia afterwards to lessen the tendency to future attacks. Tweedy gives it lightly and only as an aid to manipulation. He and Ward [34] and others find a similarity between liver lesions caused by chloroform and by eclampsia. Ward likens its administration to the giving of antipyretics for fever without looking to the cause of the condition.

Eden and Jardine strongly favour its use to control

the fits while Stroganoff uses it with good result.

Shortly, my own ideas as to its administration are that it can be safely used as an anaesthetic during manipulation but that its continual use during many fits is to be avoided lest it cause hepatic lesions. If labour be well advanced and the head well down it might be justifiable to give it repeatedly and of course lightly during pains, but a better plan is to put on low forceps and take the head out of course without undue speed, chloroform being used then for a single and shorter period.

Chloral hydrate with pot. bromide is a much used sedative and all are agreed as to their safety however much their sedative value may be doubted. Stroganoff gives them alternately with morphia and reports magnificent results. Chloral gr.20 ċ pot. bromide gr.40 may be given rectally hourly if necessary for 6 doses after which it ought to be discontinued.

It is better for the general restlessness often seen between fits than for the actual fit itself, but certainly the mixture has a powerful effect in blunting the sensibility sufficiently to reduce the tendency to fits.

Paraldehyde is a very valuable drug in this respect and I consider that it is not sufficiently used. I have never known of it doing harm and almost always it does good. It is perhaps best given rectally and 2 drams so given to a case of average severity will frequently entirely subdue the fits. It may be repeated as required in 1 dram doses at least 1½ hours between each dose. I find it having frequently as much effect in eclampsia as morphia has and I

cannot think of any contra indications to its use.

Most authors do not mention it or barely do so and obviously do not use it much.

Drugs however are not the only means by which sedatives can be administered. Attention should be given to all the surroundings of the patient and here of course good nursing is invaluable. In the first place when a sufficiently complete examination has been made treatment should be decided on and no further examining done as it is well known that peripheral stimulation of any kind e.g. vaginal examinations, is very apt to reflexly set up fits. The windows should be darkened although the room need not be in utter blackness, there should be as little movement in the room as possible and heavy shoes must never be worn by anyone in attendance. Loud voiced conversation should not be indulged in and indeed anything acting on the tactile, aural, visual or other sensory nerves should be excluded. It is a good thing to administer chloroform while the patient is being washed and examined and then she should be put into a room containing no other patient with 2 experienced nurses and a doctor in attendance. These are obviously ideal conditions which are obtainable practically only in hospital and at times not even there.

Such treatment as I outline has been worked out in considerable detail by Stroganoff of St. Petersburg whose marvellous mortality rate of 5% I quoted above. The essence of his treatment is this :- The patient is put in a darkened and absolutely quiet room and the head of the bed is elevated. Chloroform is given very lightly during the fits

but not between times. Oxygen also is administered during the fits and at any time when any degree of cyanosis appear. Morphia is alternated with chloral and bromide as a sedative. The patient is placed lying on one side and after an hour is turned over to the other side to avoid pulmonary congestion while the head is hung if necessary face downwards over the end of the bed to allow free escape of all mucus from the throat and chest. The stomach and bowel are washed out under chloroform and the patient left as much alone as possible. All this is expectant treatment perfected and the quoted results seem to justify it.

E. Roth of Dresden tried 50 cases of all kinds under this treatment: 37 were intrapartum, 6 antepartum and 7 post partum. He had 4 deaths, giving 8% as against 19% by previous treatment. The foetal mortality also was 18·6% as against a former 55%. If expectant treatment is to be tried it should be only under such perfect conditions. The essence of the treatment is the exclusion of any peripheral stimulation or irritation.

Medication is employed however with other ideas than solely to allay convulsions. The high blood pressure is an almost invariable accompaniment of the condition and is responsible for perhaps most of the haemorrhages. A circulatory sedative and vaso dilator is thus indicated and I consider it sound treatment to administer such in proper doses.

Veratrium viride is well spoken of in this respect and Zinke [35] reports 90 cases, 26 of which were treated with Norwoods tincture: ms 20 was given hypodermically at first

and repeated hourly until the pulse fell to about 60. When the pulse rose ms 10-15 were given and repeated hourly until the pulse again fell to 60. The maternal death rate was 15·38% and the foetal 53·38%. With the 64 cases not so treated the respective death rates were 34% and 45%. The drug while appearing from this to improve the mother's chances appears also to prejudice the child's.

Mangiagalli [36] of Milan used the drug on 94 cases with a maternal death rate of under 6% and a foetal death rate of 43·37%. The dosage was small ms 5-8 repeated frequently until the fits stopped, the indication for the next dose being a tendency of the blood pressure to rise.

Bailey [37] speaking of veratrium points out that if given to its full physiological effect it may cause a drop of no less than 145 mms of Hg. with very severe shock.

This is a dangerous drug and is much better administered frequently in small doses when its action is much more under control than otherwise.

Nitro-glycerine and the nitrites may be administered here as elsewhere as vaso dilators. Their action is evanescent but if other means be taken to relieve the condition they help by keeping low the tension until it ceases its tendency to go high.

Hydrate of amyl is a drug strongly advocated by Härle, Marasch, Naab and Kobert. Kobert showed that large doses could be borne without permanent ill effect. Härle gave grs 3-4 intramuscularly in 5 cases; all fits stopped and sleep came on. They were delivered normally and the albumen disappeared post partum.

Thyroid extract is advocated especially by Nicholson [37a] as an ideal vaso dilator. He recommends large doses, 30 to 40 grains at first and 20 to 30 in six or eight hours if no improvement occurs. He considers that usually thyroid secretion acts as an antagonist to suprarenal secretion which acts as a vaso constrictor especially to the renal circulation, and that in eclampsia this check action is lost and so the symptoms are produced.

He reports excellent results from the treatment but I cannot find that it is universally adopted. Morphia also acts as a vaso dilator and so may be given with a dual intention.

Another method of treatment is by elimination by the various excretory organs. It has been practised long and has given very satisfactory results. The rationale is of course that since the condition is a toxaemia as at present held and due to retention in the body of substances usually excreted if the various excretory organs could be got to act obviously a proportion of the toxin would be eliminated from the body; the remainder would be further combated if diluted e.g. by putting fluid into the body.

However before elimination be instituted it is wise to abolish any source of further toxaemia and it is for this reason that I hold that the main place for eliminative treatment is post partum when the placenta has been got rid of. Another source of toxines however is the stomach and bowel. Every case in fits should have the stomach washed out. This should be done primarily while she is under chloroform and at the end of the process mag. sulph. 2 or

3 oz. may be left in the stomach. The bowel also should be washed out and Tweedy advises continual lavage with a high tube until the return flow be quite clean. Under such treatment the bowels will probably be evacuated thoroughly shortly. Such treatment is absolutely safe and rational. Various other purgatives have been used, among them Croton Oil; I have tried it in doses of 1 and 2 minims and have not been impressed by it. Jardine reports oedema glottidis twice after its use and remarks that whatever purge is given must be given in big doses. He also remarks that a hypodermic purgative would be valuable here. I know of none.

Castor oil 2 oz. may be substituted for the mag. sulph. if desired. If she be conscious perhaps the best purge is Pulv. Jalap. Co. Galabin and Tweedy strongly recommend it. Grains 90 may be given. I have found such a dose advantageously combined with calomel gr. V. Castor Oil 1 oz. is sound or 3 oz. black draught. Besides emptying the bowel these act as hydragogues and so lower arterial tension. Even after recovery purging should be kept up for a time and a mixture containing mag. sulph. g. 90, mag. carb. lev. g X. and aq. menth. pir. to 1 oz. should be given every morning with cascara and glycerine 1 dram of each the previous evening for a week after consciousness returns.

Varying opinions are held as to the value of eliminating by the skin - diaphoresis: Tweedy maintains that at best the skin is a poor excretory organ and that the amount of excretion does not compensate for the shock caused by severe diaphoresis. Many, perhaps most, observers however favour

diaphoresis and personally I can speak only well of it having seen it many times produce improvement and have never seen any of the severe collapse described by Tweedy. The hot pack and hot air bath are the two best methods here. The pulse must be carefully watched and should be observed every 10 minutes. She should be wrapped in blankets and guarded hot water bottles put round. Pilocarpine I have never used. I fear to use it so consistently have I been warned against its lowering and reducing action.

But the main organ of elimination which we wish to stimulate and to restore to function is the kidney and in recent years a vast amount of work has been done in this respect. It may fairly be assumed that the kidney being affected similarly to the other organs in this disease its normal action is interfered with and that in it there accumulate the results of its own metabolism: that if it be flushed through with fluid these results of metabolism are washed away into the blood streams and that consequently the kidney being left clean is stimulated to reassume its normal function. Now the normal renal function is secretion of urine so that if the kidneys can be washed out it is to be assumed that more urine would be secreted and elimination greatly improved. Jardine is the man in this country who has done most work in this matter. He strongly recommends intravenous transfusion of a solution of sod. acetate 1 drachm to the pint of sterile water at a temperature of 104°F. The sodium acetate is used for its kidney stimulating properties and I rather think has now been put aside by Jardine.

Certainly most other obstetricians who transfuse use normal saline with equal result. The treatment is undoubtedly efficacious in increasing the output of urine in my experience and is a valuable and rational form of treatment. Various contradictions to it are held: oedema is one the assumption being that if the kidneys cannot get rid of what water is in the body it is dangerous to further introduce fluids. But if other methods of elimination - purgation, diaphoresis - be concurrently employed a lot of the oedema will be relieved while if transfusion acts according to theory the kidneys should be stimulated to further remove the oedema. I cannot see that oedema per se is a contra indication. Rales in the chest in eclampsia if not due to bronchitis are usually due to oedema of the lungs and are also held to be a contraindication to transfusion as the patient is apt to be literally drowned. My own experience is that severe oedema of the lungs occurs usually in cases ill beyond aid where no treatment is going to be of avail. However it is perhaps safer to regard this as a general contra-indication and so be on the safe side although I have several times transfused cases of moderate severity where pulmonary rales existed with entirely beneficial results.

I do not propose to describe the operation of transfusion but merely observe that eclampsia is an ideal condition for it from the operator's point of view for the veins stand out and are easily isolated. I have more than once found a sudden elevation of temperature e.g. Case III. appendix after transfusing. The same has been observed by others and some consider it due to too hot saline and use it

at 100°F. I found no bad effects following the rise and the temperature itself usually subsided in a few hours.

Transfusion is the method to use in the stage of fits, but after the patient be quieted and the bowel emptied saline may equally well be introduced rectally. 10 oz. may be put in two hourly for 24 hours if necessary with benefit and without discomfort. It is of course not so rapidly acting as when given intravenously but it acts sufficiently quickly. Given intracellularly into the breasts and axillas it is absorbed very quickly and may be so given after an initial intravenous administration.

Other and more radical methods however of treating the kidney in eclampsia have been advocated and practised. Hot packs, fomentations and even cupping of the loins are simple procedures and whatever good they do not do they can hardly do harm.

Now the kidneys like most other organs become congested and oedematous during eclampsia and one result of congestion is swelling of the organ. But the kidneys are encased in a firm fibrous capsule so that they cannot expand to any appreciable extent so that the pressure within the organ is increased and it may be higher than the intra-glomerular pressure so that anuria is produced. Edebohls of America going on these suppositions suggested that to remove the capsule - decapsulate - from the kidney would reduce this pressure and restore the output of urine while the operative procedure required for this would stimulate the renal plexus and so reflexly set up micturition.

Good results are reported by Edebohls but various other

writers take differing views.

De Bovis [38] reports 3 cases, 1 of them a post partum case, all recovered.

Stoeckel [39] reports 2 cases both recovered. A. Sippel of Frankfort records no less than 46 cases of which 30 are now alive.

Lichtenstein of Leipsig tells of 3 cases all of whom recovered. There was considerable increase in urination after operation in each case.

Baisch [40] reasons that only in 50% of cases do the fits cease after the uterus be empty and of these about 20% are fatal while where the fits do not cease the death rate is 36% and so more than rapid delivery solus is required. Kidney excretion must be kept up and he considers decapsulation the best diuretic. He reports immediate and great improvement in micturition in 81% of cases. Reifferscheid [41] of Bonn also reports 3 cases, 2 for eclampsia. In the first case the fits continued and anuria persisted. In the second case the fits ceased and anuria persisted for 48 hours. Both were fatal. Of 4 cases reported by Alsberg of Charlottenberg 1 died and 3 recovered. In 2 cases the convulsions ceased at once after operation while in the other 2 they became milder although present some time. Diuresis was increased in all cases.

Ehrenfset [42] in a series of experiments can find no increased diuresis after operation, while Zangemeister can see no curative effect re mortality or diuresis.

The indications for the operations are : 1. The uterus should be empty. 2. Oliguria or anuria should be present.

3. There must be no pulmonary, cerebral, hepatic or cardiac complications present. 4. Deep coma. 5. Deterioration of the pulse. 6. Acerbation of fits. The first 3 are the main indications.

The operation should be done bilaterally to produce its full effect.

Some however and among them Nicholson of Edinburgh advocate mere puncturing of the kidneys to relieve congestion, while Chambrelent and Pousson of Paris advocate incision of the kidney - nephrotomy - plus decapsulation. I have no first hand experience of the operation as it was not done in Glasgow in my time.

The result aimed at is good but I consider that such an operation would probably take too much out of a woman already very ill. However, cases with anuria are obviously desperately ill and if after other treatment diuresis did not rapidly set in it would be justifiable to decapsulate. If performed only in such very bad cases obviously the operation mortality will be very high. It should only be done under such unusual conditions. If performed indiscriminately it will certainly do more harm than good.

Now as the toxin circulates in the blood it is obvious that to remove a certain percentage of blood from the body would be to remove a certain percentage of toxin and if transfusion be practised at the same time the remaining toxin would be diluted. For these reasons many now perform venesection and so far as my experiences goes with excellent results. A bounding pulse is frequently held to be an essential to the procedure but I have several times

venesceted when the pulse was quite faint and the result has been eminently satisfactory. Some discrimination wants to be used here however to distinguish between a faint pulse due to toxaemic collapse and a faint pulse due to an engorged right ventricle. In the latter case venesection is indicated, whereas in the former stimulation is required.

In an ordinary case oz. 15 to 20 may be removed and then 1½ pints of sterile normal saline may be put into the vein: the combination of treatment is often most beneficial for the reasons mentioned. Tweedy is not keen on transfusion and is actively opposed to bleeding.

Lichrenstein [43] from Zweifel's clinic maintains that the good results of emptying the uterus do not so much depend on that fact as on how it is accomplished and in a word the more blood lost the better up to a point. He takes 400 c.cm. as the average amount of blood lost and he finds that amount exceeded in 52% of cases delivered by operation and only in 5% of cases delivered spontaneously. 40% lost more than 500 c.cm. when delivered by operation and none by normal delivery. Women in whom fits passed off after delivery lost about half as much blood again as those in whom they continued and about four times as much as those in whom it started post partum. 45 patients were treated on these principles with a maternal death rate of 11·1% and a foetal death rate of 40%. The amount of blood extracted per patient was 710 c.cm. Such evidence goes to show some real benefit from judicious bleeding.

Elimination via cerebro spinal fluid is not often done but Hebb [44] reports a case with cessation of symptoms after

lumbar puncture. 38 c.cm. were taken off and the fluid came out in a stream. Jardine in another case found the fluid in a stream during the fit but coming in drops in between times. He injected cocaine gr 1/5 into the canal with benefit. Bataski also has practised lumbar puncture and considers it more rational than venesection. He withdraws 20-35 c.cm. Other forms of medical treatment may be mentioned here. Bellotti [45] recommends the use of paraganglin (Vassall). 2 c.cm. may be given subcutaneously and repeated if required 3 hourly. The action of the drug is similar to parathyroidin. Mayer of Tubinger advocates the injection of normal serum from pregnant women but I do not know what the results of this are.

As one great feature of the disease is quick coagulation of the blood, hirudin - leech extract - has been advocated by Englemann, Bumm and others. I have no knowledge of the results of such treatment, but am in favour of anything which will tend to prevent thrombosis and to cause post partum haemorrhage. I favour the idea of using hirudin.

I said previously that every source of toxaemia should be treated before elimination be instituted and Sellheim [46] who considers the mammary glands the sources of toxaemia advocates amputation of the breast as treatment. He records cases improving after this procedure. Wilson of Birmingham reports one case where double amputation preceded cure. Mammary interference short of amputation has been done and Licht reports cases treated by intramammary injection of air, while Sellheim himself reports injecting pot. iod. with beneficial results. I consider that any beneficial effect

of such treatment is probably due, as Martin suggests, not so much to the operation as to the loss of blood during operation and that the whole procedure is rather like the ancient Chinese manoeuvre of putting a pig inside a hut and then burning down the hut in order to get roast pork. In my opinion the operation is quite unjustifiable and will do more harm than good to anyone far less a toxaemic and convulsed woman.

As acidosis is present it is quite a rational thing to administer alkalies either orally or in rectal saline. Pot. cit. grs 30 and pot. acet. grs 40 may be given in each 10 oz. of saline or by mouth 4 hourly. The tendency will be to neutralise the amino acids which by combining with ammonia prevent its transformation into urea.

If the pulse be failing subcutaneous ether 2 c.cm. $\bar{c}$ strych. sulph. g 1/60 may be given as required, or better still pituitary fluid 1 c.cm. which besides stimulating the heart and toning up the vessels helps uterine contraction. However a weak pulse indicates cardiac failure and when that appears especially before delivery the case is in my experience mostly fatal.

I now wish to consider the question of emptying the uterus and how and when it is to be done if at all. As eclampsia without pregnancy has never been seen it may be taken that the pregnant state is the foundation on which eclampsia is built, and it would seem to follow that to terminate pregnancy would be to remove the cause of the condition and so be productive of cure.

This is the view I may say generally held in Britain,

America, and the Continent, and most obstetricians are in favour of emptying the uterus as speedily and safely as possible. Bumm [47] quotes Seitz in favour of immediate delivery. After early delivery the mortality with Seitz was 6·5%, after later delivery 17·2% and without delivering at all - expectant treatment - 28·6%. Bumm himself found 30% death rate with expectant treatment but since 1901 he has adopted prompt delivery as soon as possible after the first fit with a mortality of 2·8% which later fell to 1·8%. This is a minimal death rate.

Zweifel stated that in 66% of cases the fits cease after delivery while Dührssen puts the percentage at 93, and Olshausen at 85%. Zweifel reports a mortality of 28·5% with expectant and 11·25% with active treatment. Horn [48] writing from the Christiania Lying-In Hospital reports a reduction of 50% in death rate by immediate delivery.

Some other observers however do not agree with these figures and Hermann states that in his experience fits continue after delivery in 57% of cases.

Again Stroganoff with purely expectant treatment has a death rate of 5% while Ballantyne [49] of Edinburgh reports much decreased mortality since he gave up forced delivery and adopted purely medical treatment. Veit and Charpentier also are averse to hasty delivery. Lichtenstein [43] points out that death rates compiled after delivery omit to include the post partum cases of eclampsia which in his experience represents some 20% of all cases and which has a death rate of 20% which is as high as with late delivery, which he considers strange if delivery per se has so beneficial an

effect. When he added the post partum mortality to his early delivery mortality he got quite as high a death rate as with late delivery.

Little of Montreal reporting on 31 cases of which 15 were delivered at once and the rest treated expectantly, gives about 60% of cases with cessation of fits for each class, but points out that in the former case the foetal mortality was 8% and in the latter case 77%.

While most obstetricians favour delivery some advocate early delivery - as soon as possible after the first fit - while others prefer to wait until a sufficient degree of dilatation of the os has occurred before interfering. My own impression is that since most of the fits begin intra-partum or that at least labour pains set in along with the convulsions it is better to allow the uterus to empty itself unless some pressing indication for interference e.g. cardiac failure, be present, but that if she show no signs of going into labour the uterus should be emptied by one of the later described methods. I consider the indications for immediate interference to be (1) absence of rapid cessation of fits under other treatment: (2) the patient not at once going into labour: (3) the slightest indication of failing respiration or circulation - at this stage the prognosis is very bad: (4) the presence of a foetal heart; if no foetal heart be present the mother only need be considered but if the child be alive the sooner it be extracted the better its chance. In this disease however the mother's interests must be almost the only thing considered. (5) Any impediment to labour - rigid oedematous cervix,

contracted pelvis, rigid perineum - also necessitates immediate interference. When the fits show signs of quick cessation purely sedative and eliminative treatment is quite justifiable and I consider it the best course. Case VII. appendix was so treated and was delivered 2 days after admission and after cessation of all fits of a live child and she herself made an excellent recovery.

The methods of delivering are many: the main ones are:- dilatation of the cervix, version, forceps and vaginal or abdominal Caesarian Section. The usual ways of dilating the cervix are manually (Harris), by Bossi's steel dilators, by Dr. Ribe's bag or by multiple incisions.

I consider that Harris's manual dilatation is a very dangerous procedure and is accompanied by a very great deal of shock. I have seen a woman who was certainly however fatally ill die instanter during manual dilatation and in other cases have always seen post operative shock. If the cervix be almost fully dilated it is justifiable but never in my opinion under an external os equal to a teacup. Jardine recommends and practises it much. Williams advocates the use of Bossi's dilators. I have never used them or seen them used but can see no good object in employing them, as by indiscriminate tearing they are more likely to do harm than good. De Ribe's bag is safe but rather slow. Multiple incisions of the cervix as recommended by Dührssen I am greatly in favour of if the cervix be intact or rigid or dilating too slowly. We know much better what we are doing here: it does not cause the shock of manual dilatation and the blood lost is beneficial. It should be done without

hesitation when the above indications are present and 4 cuts may be made one into each vaginal fornix. The cervix is at once fully patent and any desired method of extraction may be at once employed. I consider it the best way of dilating a cervix in any way rigid. After dilating the cervix the foetus may be extracted by version or forceps. The former is the more rapid while the latter produces rather less shock, therefore if speed be indicated e.g. when a foetal heart is heard, version is the procedure of election, whereas if the foetus be dead and the mother in as yet good general condition either forceps might be applied and the head extracted or the patient might be left under keen observation to deliver herself, and if at any time during parturition a danger sign appeared forceps might then be applied. The advantage of this last method is of course more preparation and less tendency to rupture of the perineum, and this is a most vital point when we remember that most eclamptics are primiparae.

Vaginal Caesarian Section is advocated by many authorities. Möller [50] reports 7 cases with good result. Paterson [51] also recommends it and describes the operation as being based on sound surgical principles, quickly performed and the technique easily acquired. Humber of Strasburg also advocates it. Abdominal Caesarian Section has also been done in eclampsia as it is considered by some the quickest way, consistent with safety, of emptying the uterus. McCann [52] describes one successful case and reports from the literature a death rate of 50%. He considers as indications for the operation (1) frequent fits:

(2) labour not commenced: (3) difficulty of dilating cervix from elongation, hypertrophy or rigidity: (4) a moribund mother and a living or viable foetus: (5) labour set in but disproportion between size of foetus and pelvis.

Van der Abber in 1875 was the first man to have a successful result. In 1897 Kettlitz collected 28 cases with 14 deaths - 50%. In 1899 Hillman collected 40 cases showing 21 deaths - 51%, and in 1903 Streicherein reported 26 cases with 8 deaths - 31%.

Boldt reports one case, a child of 12 years, where the mother died but the child lived.

Caesarian Section evidently aims at saving the child if necessary at the expense of the mother and I maintain that the reverse should be our aim in this disease. I have never seen section performed for eclampsia and can see no real indication for it.

These then are the main practices employed to-day in treating eclampsia and my views on them. In conclusion I wish to say just a word regarding the after treatment of such cases. When consciousness returns then nourishment can be given orally. This should consist entirely of milk at least 2 quarts being taken daily. Imperial drink and water may be given ad lib the more the better. Mag. sulph. should be given daily for a week at least.

Milk diet should be kept up certainly until the urine is albumen free - Williams says for a week longer - after which soft food may be added to the diet. In 10 days after the albumen disappears some solid food may be given and the patient allowed on the couch for an hour. She must return

very slowly to her normal habits of life and diet however and whenever possible a change of air at the coast or in the country should be ordered for at least a fortnight and better still a month. I fear that the impossibility of enforcing such after treatment in such a vast majority of cases is responsible for a deal of chronic renal trouble later on.

These are my observations on eclampsia which with their results I now put forward for your consideration. I have had to consult many articles and magazines and have obtained much valuable help from these sources. Of special value have I found the writings of Williams, Jardine and Holland.

May 1913.

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APPENDIX OF CASES REFERRED TO IN THE TEXT.

History and Condition on admission stated.

Treatment and Progress stated on the Charts.

CASE I. Post Partum Eclampsia.

Mrs. G. Aged 20. Primipara.

Admitted 6. 5. 12.

History. The patient was in good health in every way until 2 months before admission when the feet and legs began to swell. This was seen more especially immediately after she would lie down. The face also began to swell up especially round about the eyes and it has become paler lately. These symptoms have steadily increased but none other have appeared.

These conditions were verified on admission and patient who was in labour was delivered normally 3.55 a.m. 7. 5. 12. About 2 hours afterwards severe eclamptic fits began to come on. On admission her manner was rather wild and she was very restless. She remembered nothing for 4 days before admission.

The patient's mother died aet 47 years of apoplexy following dropsy.

Urine. Dark brown colour and smoky. S.G.1030. Acid reaction. Contains much albumen (18 parts) and a faint trace of blood.

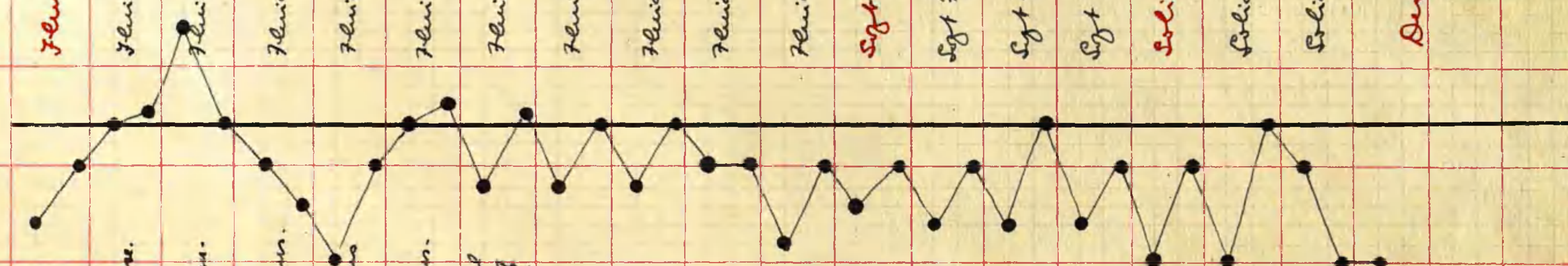
MOTHER'S CHART.

Name, Mrs 9. No.

Admitted, 6. 5. 12. Delivered, 7. 5. 12.

Day of Puerperium	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Date of Month	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.	25.	26.
Notes	On Admission.	Delivered. Child alive. Placenta 1¼ lbs. Hot Packs. Croton oil m 2 on tongue. Enema. Chloral 20 c bromide 40-4 hourly. Morph gr ¼.	Salts	Pulv.		Chloral & bromide stopped.		Pulv.	Enema. Salts			Pulv.	Pulv.	Enema Pulv.		Allowed up.		Salts.			
Diet	Fluids	Fluids	Fluids	Fluids	Fluids	Fluids	Fluids	Fluids	Fluids	Fluids	Fluids	Lgt Food	Lgt Food	Lgt Food.	Lgt Food	Solid Food	Solid Food.	Solid Food.	Discharged Well.		

Height of Fundus. 7 inches, 6 inches, 5 inches, 4 inches, 3 inches, 2 inches, 1 inch, Level of Symphysis. — 107, 106, 105, 104, 103, 102, 101, 100, 99, 98, 97, 96



	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Condition	Restless No Fits.	8 Fits Comatose.	0 Fits Semiconscious.	Semi-conscious.	Fairly Conscious No Fits.	Conscious.	Normal Frame of Mind.	"	"	"	"	"	"	"	"	"	"	"	"		
Albumen (Esbach)		84.	93.	61.	12.	13.	9.4	8.	7.3	5.	2.4	Trace.	Trace.	Free	Free	Free	Free	Free	Free.		
Pulse	68. 70.	84. 92.	92. 106.	100. 100	102. 102	88. 96.	100. 84.	94. 83.	86. 90.	80. 96.	82. 94.	76. 88.	74. 88.	70. 88.	66. 80.	70. 66.	75 70	69 73	70.		
~~Respiration~~ Haemoglobin %	90.										90.								85.		
Urine (Quantity) ʒ	None.	12.	46.	74.	70.	45.	69.	79.	34.	40.	66.	68.	68.	61.	60.	56	58	48	45		
Bowels	1. 1.	0. 1.	0. 1.	2. 1.	0. 2.	1. 2.	0. 1.	0. 0.	1. 0.	1. 4.	3. 1.	0. 0.	1. 0	1. 0.	2. 2.	1. 1.	1. 1.	0. 0.	2.		
~~Lochia~~ Blood Count	3 million reds. 8,000 whites										3½ million 15,000 whites.								4 million 9,000.		
Blood Pressure.	170.	165.	165.	145.	140.	130.	125.	123	120	120	122	120	124	122	120	125	123	120	120.		

CASE II. Case Running Ordinary Course.

Mrs. N. Aged 23. Primipara.

Admitted 6. 5. 12.

Previous health was always good and the present condition started 2 weeks before admission when sickness and vomiting set in. At first it was not very bad but later became very severe occurring after the ingestion of any kind of food. About the same time the feet and legs were noticed to be swollen especially after lying down for a time. They were always worse on getting up in the morning. The patient lost consciousness about 5 a.m. on the day of admission and recollected nothing until the evening of the following day. The last thing remembered is severe sickness.

On admission the patient was seen to be very wild and restless and could be controlled only with difficulty. The face was pale and puffy and the feet, ankles and lower parts of the legs were very oedematous as was the vulva.

Patient was in labour with a semidilated os. The tongue was very dirty and the bowels loaded and very constipated.

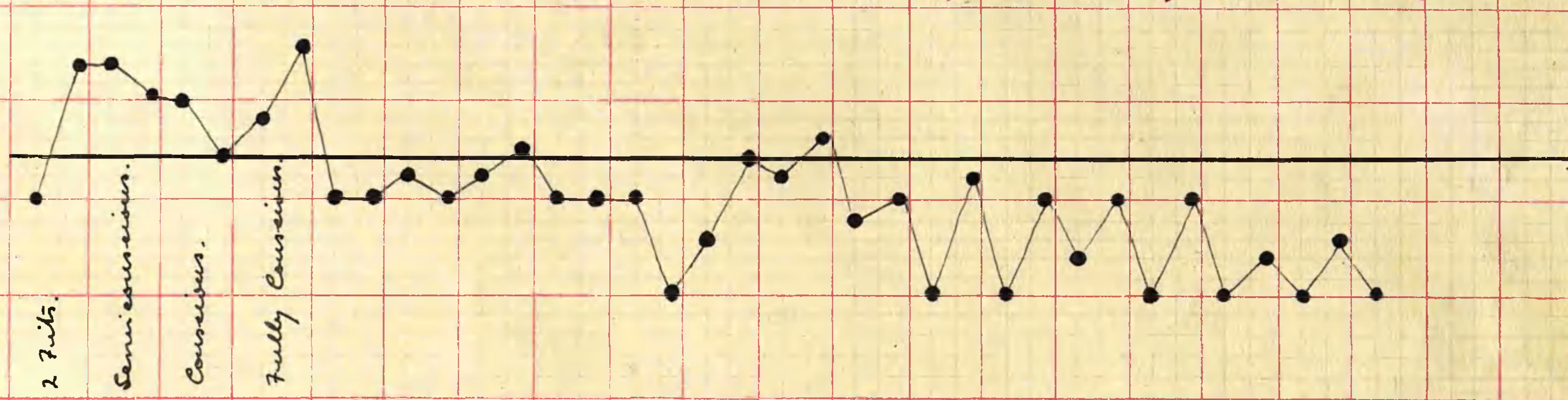
Urine. Clear amber colour. Deposit of mucus. S.G.1010. It contains albumen (4.5) and a faint trace of blood.

MOTHER'S CHART.

Name, Mrs N. No.

Admitted, 6. 5. 12. Delivered, 6. 5. 12.

Day of Puerperium	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Date of Month	6	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.	25.	26.
Height of Fundus / Notes	On Admission. Calomel gr iii c compd Jalap Powder gr xxx. Fluids. Delivered. Low Forceps. Placenta 1½ lbs.	Fluids. Salts	Fluids	Fluids Rubs.	Fluids	Fluids Rubs.	Fluids.	Fluids.	Fluids Salts.	Fluids	Fluids	Fluids Rubs.	Soft Food.	Soft Food Rubs.	Soft Food.	Solid Food. Allowed up.	Solid Food.	Solid Food. Salts.	Dismissed well.		
Temperature notes	2 Fits.	Semiconscious.	Conscious.	Fully Conscious.																	
Albumen (Esbach)	69.4	43	24.	20	7.2.	4	Trace	Trace	Trace	Trace	Trace.	Free.	Free	Free	Free	Free	Free	Free	Free.		
Pulse	100. 100.	120. 108.	86. 84.	98. 84	80. 86.	96. 90.	84. 80.	92. 92	78. 72	80. 96.	78. 90	82. 78.	66. 84.	80. 72.	74. 80.	64. 84.	60. 80.	76. 82.	72.		
~~Respiration~~ Haemoglobin %	75										80								80+		
Urine (Quantity) ℥	72.	72.	75.	83.	80.	42.	35	45.	35.	70.	25.	50	41.	50.	51	?	?	42	50.		
Bowels		2. 0.	3. 2.	2. 0.	2. 2.	1. 0.	1. 1.	0. 1.	0. 0.	4. 4.	2. 1.	0. 0.	1. 2.	0. 0.	2. 2.	2. 1.	1. 1.	0. 0.	2.		
~~Lochia~~ Blood Count	18,000										12,000								9,200		
Blood Pressure	210.	165.	160.	152.	132.	125.	125.	120.	123	125	122	120	126	125.	120	123.	122	123	120		



CASE III. Post Partum Mania, Coma and Death.

Mrs. D. Primipara. Aetat 18 years.

Admitted in labour and having eclamptic fits 9.7.12.

Previous History. The patient had Enteric Fever 6 years before admission and went to Ruchill Hospital. She had a severe attack of acute gastritis one year before admission.

History of the present condition is that about 6 hours before admission the patient, who had previously been complaining of some swelling of the feet and ankles, suddenly became convulsed and then comatose and that about 1½ hours later a second and similar fit occurred, after which the patient remained unconscious.

On admission she was unconscious with a pale and oedematous face and froth round the lips.

The legs and feet were distinctly oedematous as was the vulva and lower abdominal wall.

The pulse rate was 130 per minute and the tension was high.

The tongue was furred and was somewhat lacerated. There was a history of continual difficulty in moving the bowels which lately were become very constipated. Faecal accumulation in colon and sigmoid was to be felt.

No rales were heard in the chest.

The fundus uteri was 1" from the ziphoid and the position was cranial.

The cervix was taken up and dilated equal to a shilling.

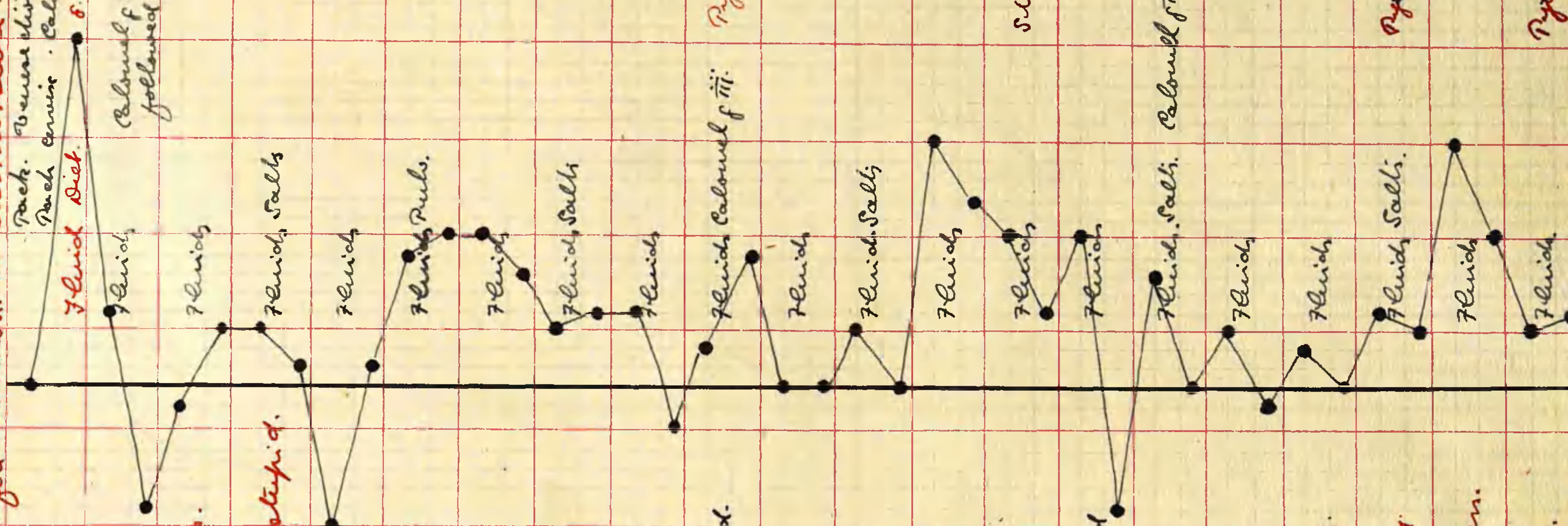
MOTHER'S CHART.

Name, Mrs D.　　No.

Admitted, 9. 7. 12.　　Delivered, 9. 7. 12.

Day of Puerperium	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Date of Month	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.	25.	26.	27.	28.	29.
Treatment / notes	On Admission. Pack. Venesection ℥xxi. Intravenous saline OT. Pack cervix. Calomel gr iii c Jalap gr xxx. Salts 1 enema. 8.30 p.m. Delivered Macerated Placenta 1¼ lbs.	Calomel gr iii c Jalap gr xxx followed by Mag. Sulph. Chloral 40 Bromide 20. 4 hourly	Chloral & bromide	Chloral & bromide	Chloral & bromide	Chloral & bromide	Chloral & bromide stopped			Calomel gr iii. Pyuria. Urotropin gr 20 daily.				Still pyuria. Urotropine.		Calomel gr iii c Jalap gr xxx.			Pyuria still. Urotropin.		Pyuria. Urotropin.
Diet	Fluid Diet.	Fluids	Fluids	Fluids, Salts	Fluids	Fluids, Pulv.	Fluids	Fluids, Salts	Fluids	Fluids	Fluids	Fluids. Salts	Fluids	Fluids	Fluids	Fluids. Salts.	Fluids	Fluids	Fluids, Salts.	Fluids	Fluids.
Condition (2 fits before admission)	12 fits		Restless.	Dull & stupid.	Dull	Dazed	Dull	Dull	Dazed	Stupid.	Dull.	Dull	Dull	Dull.	Stupid	Dull	Dull.	Noisy.	Noisy.	Restless.	Noisy.
Albumen (Esbach)		220	37	40	50	24	23	30		42	35			24	20	12		18	?	?	16
Pulse	130. 120.	124. 90.	96. 104	114. 112	108. 96.	120. 120	118. 114	104. 112	130. 116	104. 114	112. 114	108. 114	128. 122	120. 120.	116. 120	94. 120.	124. 140	100. 134	124. 128.	136. 124	132. 120.
~~Respiration~~ Haemoglobin %.	90																90.				
Urine (Quantity) ℥		106.	50.	90.	100.	64.	59	48	24.	50	73	60	40	13	25	32.	40	32	Lost	Lost	50
Bowels		9. 0.	0. 2.	2. 0.	3. 2.	3. 1.	4. 1.	0. 1.	1. 2.	1. 3.	1. 2.	1. 0.	4. 0.	1. 1.	4. 0.	0. 0.	1. 2.	1. 0.	0. 0.	1. 1.	2. 1.

Height of Fundus. 7 inches / 6 inches / 5 inches / 4 inches / 3 inches / 2 inches / 1 inch / Level of Symphysis.

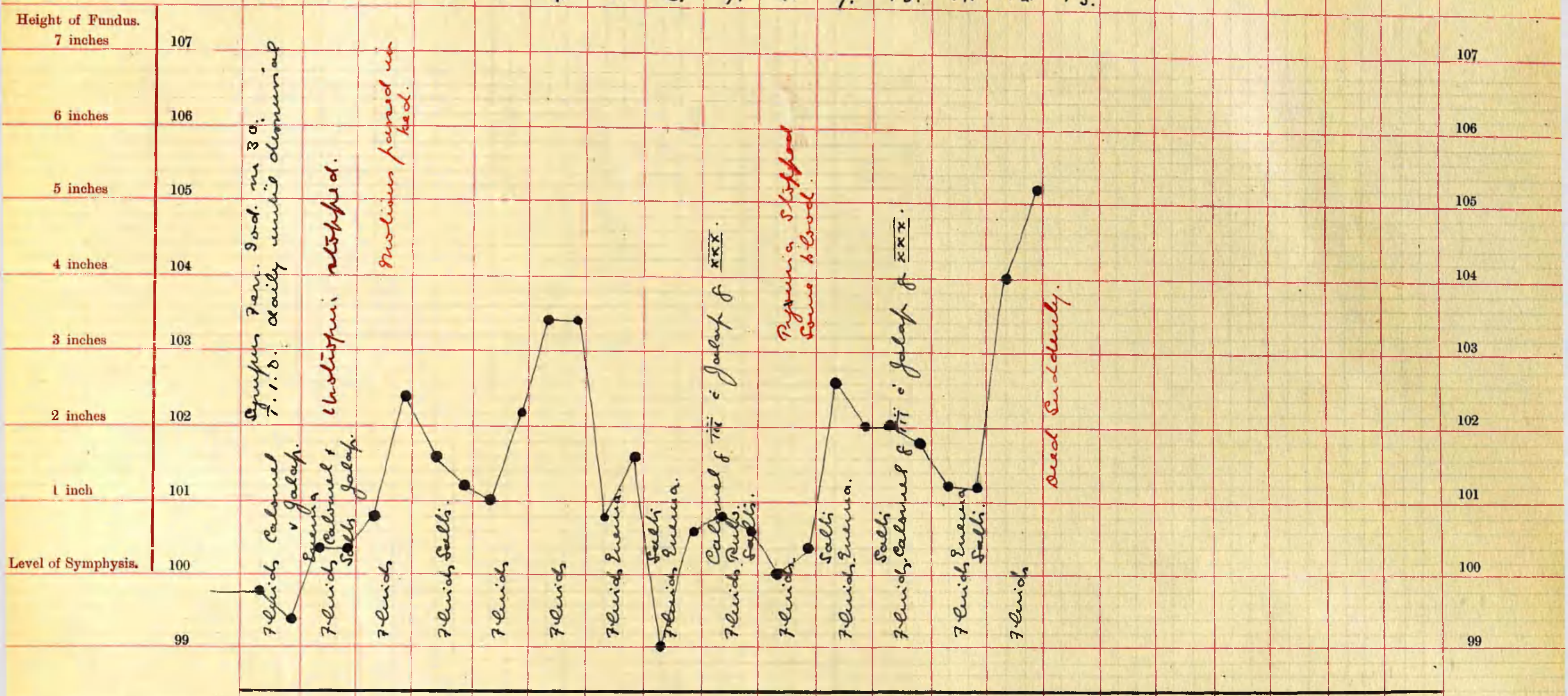


MOTHER'S CHART.

Name, Mrs D. No.

Admitted, 9 · 7 · 12. Delivered, 9 · 7 · 12.

Day of Puerperium	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Date of Month	30.	31.	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.						



	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
	Dull	Restless	Dull	Dull	Dull	Dull	Noisy	Dull	Dull	Dull	Stupid.	Dull	apathetic	Comatose	Comatose.						
albumen (Esbach)	?	20.	?	?	13.	?	16.	12.	?	?	21.	?	10.	?							
Pulse	142. 134.	136. 138.	120. 136.	150. 136.	140. 144.	150. 136.	136. 140.	132. 134.	124. 140.	122. 128.	132. 130	128. 128.	132. 130.	136 128	130.						
~~Respiration~~ Haemoglobin %								80.													
Urine (Quantity) ℥	Lost	45	Lost	Lost	60	Lost	54	80	Lost	Lost	60	Lost	72	Lost							
Bowels	0. 0.	0. 3.	1. 0.	0. 0.	0. 3.	1. 0.	0. 0.	0. 0.	0. 1.	3. 0.	0. 0.	0. 0.	0. 0.	0. 0.	0.						

CASE V. Difficulty in Diagnosis.

Mrs. T. Aged 32 years. IV. - gravida.

Patient admitted 23. 7. 12 unconscious and comatose. She was found in this condition at 9 a.m. on the day of admission. Her previous health was reported good and her labour normal. A bottle of chlorodyne was found beside the patient.

She lies absolutely unconscious and comatose with stertorous breathing and frothing at the mouth. There is no evident unilateral paralysis.

The legs and knees are somewhat stiff and are difficult to flex. Both the arms are quite lax.

The pupils are both slightly contracted and equal. They do not react to light.

No oedema is found anywhere.

The tongue is dirty and the bowel loaded.

The pulse is very fast and feeble and increasingly so.

Medium rales are heard all over the chest but their detection is not easy on account of the stertorous breathing.

The breasts are secreting.

The fundus uteri is felt 1½" above the umbilicus, i.e. she is about 7-7½ months pregnant. The presentation is cranial and no foetal heart may be heard. The cervix is quite undilated.

The Urine is dark red with blood and thick with albumen. S.G.1025. Acid reaction.

Pulse 72 on admission: later 104.
Temperature 97°
Respirations 48 per minute.

Treatment. The stomach was washed out with a weak solution of Pot. Permang. Mag. Sulph. ½ dr. was left in. Pituirin (P.D. & Co.) 1·5 c.c. given hypodermically. Head of bed was elevated.

The patient sank and died at 6.20 p.m.

Sectio. Kidney - No definite change.

Liver - Throughout all the organ were many various sized recent haemorrhages from a pea to a billiard ball in size. No necrosis was seen. Condition markedly subcapsular but also profuse throughout the substance of the organ.

Brain - There was a large haemorrhage occupying most of the mid-part of the right cerebrum and forcing its way into the 4th ventricle.

No changes were seen in any other organ.

CASE VI. Post Partum Mania.

J.G. Primipara. Aetat 23 years.

Admitted 21. 7. 12, having eclamptic fits and in labour.

The previous health had always been satisfactory and the patient was apparently quite well until about 2 hours before admission when she suddenly gave a cry and then fell into a convulsive fit followed by coma. Medical aid was summoned and the patient's removal to the Maternity Hospital ordered, but just before she left the house she had another and similar fit also preceded by a cry. There was no previous personal or family history of epilepsy.

On admission the patient had another fit preceded by a cry and while being washed 2 others similar in every way. The two latter were typical eclamptic seizures.

There was no oedema evident in any part of the body.

The tongue was very dirty, the dorsum being caked with a yellowish fur. The bowels had been very constipated for some weeks previously.

The pulse rate was 154 per minute and the vessel walls healthy. High tension was present.

No rales were to be heard in the chest.

The breasts were secreting colostrum.

The fundus uteri was 1½" from the ziphoid.

The head was down on the perineum.

No foetal heart could be heard and no foetal movements elicited.

Urine was of acid reaction. The quantity was insufficient to take the specific gravity. It was dark amber in colour and clear with a flocculent deposit. It was almost solid on boiling and contained blood. There was no sugar and no bile.

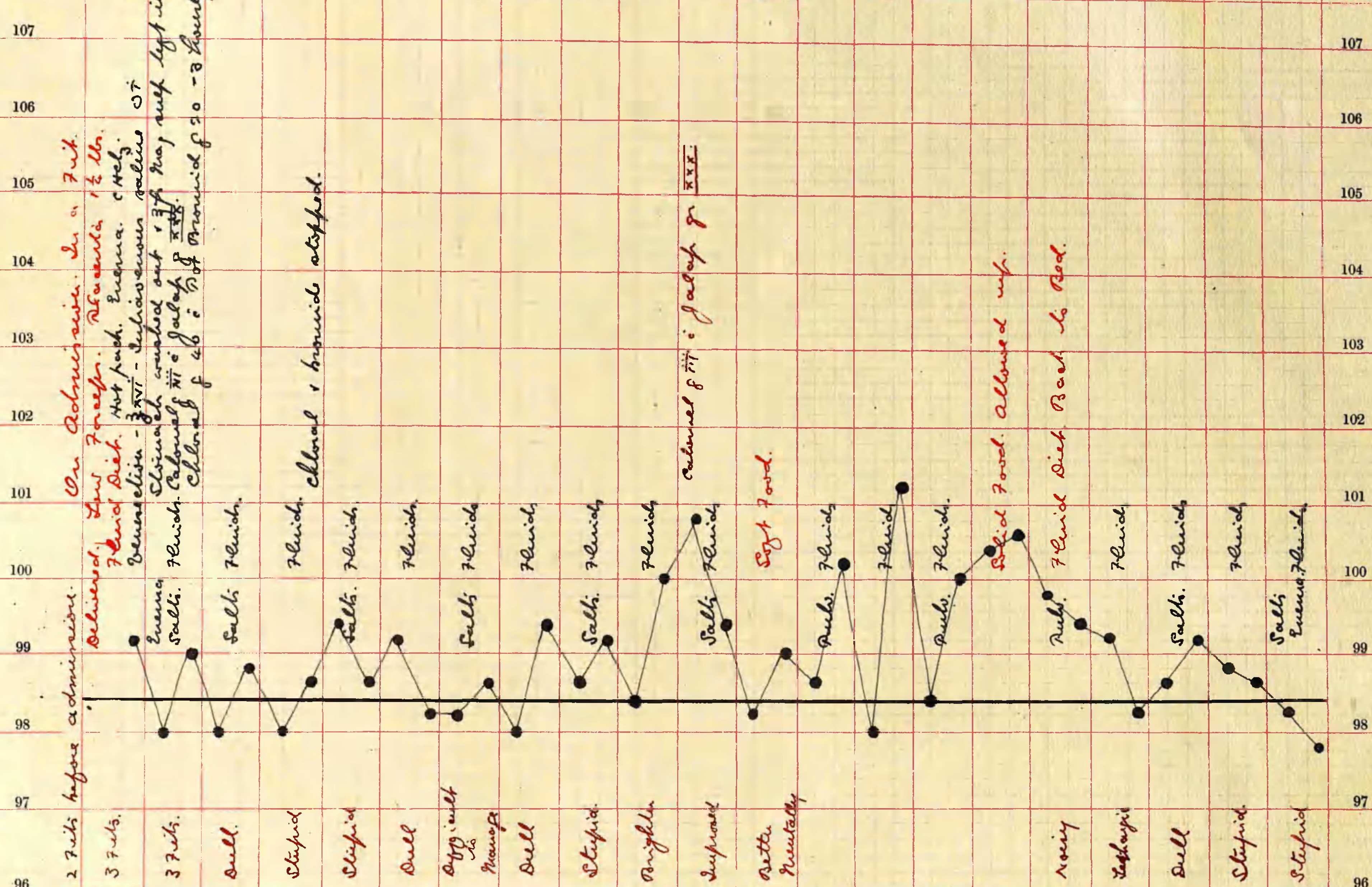
MOTHER'S CHART.

Name, J. C. No.

Admitted, 21. 7. 12. Delivered, 21. 7. 12.

Day of Puerperium	Date of Month	Notes	Albumen (Esbach)	Pulse	Haemoglobin %	Urine (Quantity) ℥	Bowels
1	21.	On admission. In a Fit. Delivered. Low Forceps. Placenta 1½ lbs. Fluid Diet. Hot pack. Enema. c Hcl. Venesection – ℥xvi – Intravenous saline O¹. 2 Fits before admission. 3 Fits.	93	154	70.		
2	22.	Stomach washed out & ℥ʒ Mag. sulf. left in. Calomel gr iii c Jalap gr xxx. Chloral gr 40 c Pot Bromid gr 20 – 3 hourly. Enema Salts. Fluids. 3 Fits.	28	128. 100.		27	2
3	23.	Salts. Fluids. Dull.	Trace	98. 96.		46	0. 2.
4	24.	Fluids. Chloral & bromide stopped. Stupid	Free.	100. 108.		64	2. 2.
5	25.	Salts. Fluids. Stupid	Free	104. 102		60.	1. 0.
6	26.	Fluids. Dull	Free	108. 94		80.	2. 1.
7	27.	Salts. Fluids. Difficult to Manage	Free	100. 104			4. 0.
8	28.	Fluids. Dull	Free	108. 104			2. 1.
9	29.	Salts. Fluids. Stupid.	Free	96. 88.			1. 0.
10	30.	Fluids. Brighter	Free	100. 110			3. 1.
11	31.	Salts. Fluids. Calomel gr iii c Jalap gr xxx. Improved	Free	100. 100.		69.	2. 0.
12	1.	Soft Food. Better Mentally	Free	104. 100.			3. 3.
13	2.	Pulv. Fluids.	Free	78. 104			3. 0.
14	3.	Fluids.	Free	100. 110.			4. 2.
15	4.	Pulv. Fluids.	Free	104. 116.		41.	1. 0.
16	5.	Solid Food. Allowed up.	Free	104. 112.			0. 2.
17	6.	Pulv. Fluid Diet. Back to Bed. Noisy	Free	98. 98.	80.	42.	1⅓. 0.
18	7.	Fluids. Lethargic	Free	100. 98.			3. 2.
19	8.	Salts. Fluids. Dull	Trace	100. 96.			2. 0.
20	9.	Fluids. Stupid.	Free	100. 96.		31.	0. 0.
21	10.	Salts. Enema. Fluids. Stupid	Free.	76. 80.		28.	0. 0.

Height of Fundus. 7 inches, 6 inches, 5 inches, 4 inches, 3 inches, 2 inches, 1 inch, Level of Symphysis.



MOTHER'S CHART.

Name, J. G. *No.*

Admitted, 21. 7. 12. *Delivered,* 21. 7. 12.

Day of Puerperium	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Date of Month	11.	12.	13.	14.																	

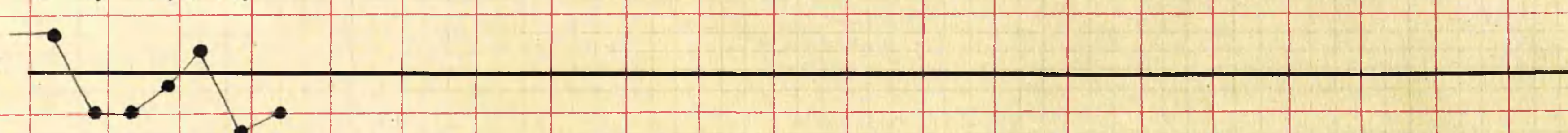
Height of Fundus. 7 inches — 107; 6 inches — 106; 5 inches — 105; 4 inches — 104; 3 inches — 103; 2 inches — 102; 1 inch — 101; Level of Symphysis. — 100; 99; 98; 97; 96

Day 1: Enema. Fluids. Salts. Chloral & bromide as required.

Day 2: Fluids. Calomel gr iii c Jalap gr xix. Salts.

Day 3: Enema. Fluids. Calomel gr iii c Jalap gr xxx. Salts.

Day 4: Fluids. Dismissed to Parish Council Observation wards. Condition of Mania.



Day 1: very noisy. Day 2: Delusions. Day 3: Incoherent.

	1	2	3	4
Albumen (Esbach)	Free	Free	Free	Free.
Pulse	92. 84.	98. 84.	100. 92.	92.
~~Respiration~~ Haemoglobin %		85.		
Urine (Quantity) ℥	26.	11	30.	8+.
Bowels	0. 0.	0. 0.	1. 3.	1.

CASE VII. Subsidence of Fits under Expectant Treatment.

Mrs. S. Primipara. Aetat 26 years.

Admitted 13. 7. 12 in eclamptic convulsions.

None of the friends could speak English and the patient herself was a Pole so that no clinical history could be obtained.

There was oedema of the face, lower abdominal wall, legs and feet. The tongue was caked with fur and lacerated.

The bowels were loaded with faecal material and purgation and enema produced an enormous result.

The pulse was fast 120 and of tension 180 mgs. of Hg.

No rales were to be heard in the chest.

The breasts were secreting.

The fundus uteri was 2" from the ziphoid.

The foetal heart was heard best $2\frac{1}{2}$" above and to the left of the umbilicus, the presentation being breech. The cervix was not dilated at all and the patient was not in labour.

MOTHER'S CHART.

Name, Mrs S. No.

Admitted, 13. 7. 12. Delivered, 16. 7. 12.

Day of Puerperium	1	2	3	4	5	6	7	8	9	10	11
Date of Month	13.	14.	15.	16.	17.	18.	19.	20.	21.	23.	24.
Height of Fundus	On Admission. Packed. Enema. Calomel gr iii c Jalap gr xxx. Chloral gr 40 c bromide gr 20-3 hourly	Chloral + bromide.	Chloral + bromide	Delivered Spontaneously	Breech. 5lb. Placenta 1 ¾ lb. Chloral + bromide stopped.						
Diet	Fluid Diet.	Fluids	Fluids	Fluids	Fluids	Fluids	Fluids	Fluids	Fluids	Fluids	Soft Food.
	Enema	Salts.	Salts		Salts						Salts
	Fits	Restless	Restless	Normal.							
Albumen (Esbach)	·175%	·15%	·3%	·6%	·4%	·3%	·5%	·4%	·4%	Trace.	Trace
Pulse		106. 84.	70. 68.	70. 80.	70. 70.	70. 76.	80. 88.	76. 80.	60. 80.	64. 68.	62. 80.
~~Respiration~~ Haemoglobin %		80.									
Urine (Quantity) ℥			80.	Lost	Lost	Lost	Lost	Lost	Lost	Lost	Lost
Bowels	0.	1. 1.	1. 1.	2. 1.	3. 2.	1. 1.	1. 2.	2. 2.	1. 1.	1. 0.	1. 2.

Day of Puerperium	12	13	14	15	16	17	18	19	20	21	
Date of Month	25.	26.	27.	28.	29.	30.	31.	1.	2.	3.	4.
Height of Fundus							allowed up.				Dismissed well.
Diet	Soft	Soft	Soft	Soft	Soft	Soft	Solid Food.	Solid	Solid.	Solid.	
			Puls.	Salts.						Salts.	
Albumen (Esbach)	Trace	Sl. T.	Sl. T.	Free.	Free	Free	Free	Free	Free	Free	Free.
Pulse	76. 76.	68. 80.	60. 80.	60. 64.	60. 64.	60. 64.	64. 60.	68. 60.	60. 72	65. 70.	62
~~Respiration~~ Haemoglobin %	90										90.
Urine (Quantity) ℥	Lost	Lost	Lost	27.	30.	Lost	26.	Lost	Lost	Lost	
Bowels	0. 1.	1. 1.	0. 2.	1. 0.	0. 0.	2. 2.	0. 1.	0. 2.	1. 2.	0. 0.	0.