Puerperal Eclampsia:

Its Symptomatology, Pathology and Treatment.

With Illustrative Cases.

Thesis for M. D. Degree

by

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Puerperal Eclampsia.

Definition

The term "Puerperal Eclampsia" is applied to tonic and clonic convulsions of an Epileptiform type coming on during, and being caused by the puerperal state. The fits are accompanied by loss of consciousness and are followed by Coma. Dr. Murphy of Dublin [17] restricts the term to cases in which Epileptiform convulsions are accompanied by Albuminuria and regards them as ~~symptoms~~ of albuminuria during pregnancy or labour. Convulsions occurring in the puerperal state arising from true Epilepsy, Apoplexy and Hysteria ought to be classed separately and regarded simply as accidental complications. Eclampsia might be regarded as a symptom of some other disease rather than as itself a disease, or we might look upon it as a group of symptoms due to various causes.

Percentage of cases of Eclampsia.

Although a ~~large~~ small proportion of pregnant women suffer from Albuminuria towards the end of pregnancy yet only a small number of the latter have Eclampsia. The proportion of cases of Eclampsia to deliveries is given by Lazeaux as 1 to 480; by Schroeder and by Lusk it is stated as 1 to 500. In 131 pregnant women Litzmann found Albuminuria in 3% and Eclampsia in 7 cases. [17] Of those patients who suffer from Eclampsia, a large number have

an abnormally small amount of Urea in the Urine, but a large corresponding excess of it in the blood - in some cases Fordyce Barker states it to be as high as 1 in 960. They also suffer from impaired vision and Albuminuric Retinitis.

Pathology. Pathology and Etiology:-

According to Galabin (1) twenty per cent. of all pregnant women suffer from Albuminuria during some period of the puerperium, but convulsions occur in only a very small proportion of those who have albumen in the urine. In Guy's Hospital Lying-in Charity, out of 23000 deliveries the ratio was only 0·12 per cent. Galabin asserts that, for accurate results, the urine should be drawn off with the catheter. The catheter is more frequently used on the Continent than in Britain, and hence the results in the former are more reliable than those in the latter. M. Blot found albumen in the urine of 20 per cent of cases observed during labour. M. Petit found Albuminuria in 20 per cent of cases during labour; just after labour, albuminuria was noted in 4 cases out of 17; in the ninth month, before the onset of labour, Albuminuria was found in 3 cases out of 22. M. Hippolette found Albuminuria in 17 out of 72 cases observed during labour; in the ninth month, albuminuria was detected in 10 cases out of 73; and after the termination of labour there was albumen in the urine of 5 patients out of 20 examined. The results of these statistics shew that before the onset

of labour Albuminuria occurs in one case in seven; during labour it is found in 1 case in 4½; while shortly after delivery albuminuria exists only in one case in four.

From his statistics[1] Galabin concludes that Albuminuria ~~is~~ more frequently exists during pregnancy than was usually supposed, and that it is much more common during or after labour than before its onset. Petit's statistics show Albuminuria is more often found in primiparae than in multiparae - in the ratio of 5 of the former to 4 of the latter. They also show that the tendency to Albuminuria decreases with the advance of age in the patients. These differences are still more marked in reference to Eclampsia

Dr. Lever[2] examined the urine of 50 patients during labour and in all of them albumen was absent from the urine except in those cases in which either actual convulsions had occurred, or there were premonitory symptoms of puerperal eclampsia. Dr. Hicks[3] in investigating the relation between Eclampsia and Albuminuria, found only one case of Albuminuria out of fifty cases observed during labour - and this patient had certainly suffered from old kidney disease. In the ninth month of pregnancy Dr. Galabin[1] found albumen entirely absent in 17 cases of multiparae; while in 26 cases of primiparae, albuminuria existed in only one instance, and that in small amount. French writers on the subject do not give the proportion of albumen present in the urine, nor

do they state whether tube-casts are found in their cases. The picric acid test used by many of them would enable them to detect a much smaller proportion of albumen than the Nitric Acid test used by British investigators.

In his cases, Galabin[1] found the proportion of Albumen in the urine large - varying from one-sixth to one half of the whole - and in most cases a very large deposit of tube-casts. Inasmuch as the same symptoms are found in cases of general dropsy during pregnancy as in patients who show the premonitory symptoms of Eclampsia along with Albuminuria, Galabin says that many of the cases of the French writers should not be put in the same category with those of British observers.

Although pregnancy occasionally leads, not only to a temporary albuminuria from mechanical pressure on the kidneys and ureters, but even to a true Inflammation of the kidneys causing permanent damage, - yet as a rule, this Nephritis passes off after delivery. In illustration of this,[1] Galabin records the case of a patient who had albuminuria accompanied by puerperal convulsions during the delivery of her first still-born child. During six labours albuminuria appeared, but entirely subsided after delivery until the next pregnancy when it again manifested itself. The children all died in utero and were born prematurely. The only cause which could be ascertained was the kidney disease. As each pregnancy advanced, the sight

became more and more impaired, and the haemorrhagic spots of Chronic Bright's disease were noted by the Ophthalmoscope. The patient ultimately died of some chest affection.

Causes of Albuminuria.

Albuminuria during pregnancy seems to depend upon a variety of causes - although some of them may be combined:-

1. Pressure on the Renal veins by the pregnant uterus may lead to increased tension in the venous circulation in the kidneys. This venous congestion leads to mechanical transudation of albumen through the walls of the capillary vessels into the tubules of the kidneys. In this condition, the kidneys will be easily inflamed by slight irritants and will less easily recover than in the normal state. This theory does not explain those cases in which tumours of a similar size to the pregnant uterus are not so liable to cause albuminuria. It also fails to account for those cases of albuminuria in the earlier months of pregnancy in which there is little pressure on the renal veins. Dr. Lever [2] held that albuminuria was due to pressure of the pregnant uterus on the kidneys and on their bloodvessels.

2. Increased Arterial tension throughout the body is caused by the hypertrophy of the heart which takes place during pregnancy. This tension in the Renal arteries causes a mechanical transudation of albumen through their walls. Some authors say that it results in Interstitial Nephritis. Both of these theories would explain cases of temporary albuminuria before labour.

3. During labour, the contractions of the Uterus are much stronger than before that period. These contractions cause great temporary changes in the renal circulation. When the uterus contracts there is a marked decrease in the quantity of blood passing through its walls; but at the same time there is a rapid rise in tension in the circulation through the renal arteries - since the latter arise from the aorta near the origin of the uterine arteries. The uterus, in contracting, presses out a large quantity of venous blood from itself, and this results, according to Galabin [1] in increased tension in the renal veins. The combined effect is increased tension in both arteries and veins of the kidneys. As this increased tension is much greater during the powerful contractions of labour than during the last months of pregnancy, we have a reason for the greater frequency of albuminuria during the former period when compared with the latter. In some cases albumen has been found in the first urine drawn off by the catheter immediately after delivery - and has then disappeared.

4. The intimate relationship between the uterus and the kidneys has led some authors to suppose that there is an active hyperaemia of the kidneys during the development of the Uterus - and that this is caused by a reflex influence of the nervous system. A parallel case to this is found ~~found~~ in the copious secretion of urine taking place in hysterical women with uterine disorders.

Although healthy kidneys are not injured by physiological active congestion, - yet if there is any source of irritation present, it may produce a state of true inflammation. Illustrations of this occur in the activity of the uterus during the menstrual period, or under the influence of sexual emotion. Disease in the pregnant uterus might set up reflex congestion of the kidneys.

5. The enlarged uterus may press on the ureters as well as on the renal arteries and veins. This may occur even early in pregnancy. The pressure of fibroids on the ureters will produce dilatation of the pelves of the kidneys. Although the temporary enlargement of the pregnant uterus does not necessarily cause permanent organic disease of the kidneys - yet at the same time, the obstruction in the ureters makes the secretory structures work under increased pressure, and to that extent makes the kidneys more liable to functional disorder. Halbertsma says that Albuminuria is caused by pressure on the ureters Some writers suppose that pressure on the ureters before delivery leads to the retention of decomposition products of the urine. But convulsions are absent in most of those cases in which there is pressure on the ureters. Bagot says that the theory of compression of the ureters does not apply to cases between the fourth and sixth months of pregnancy, because at that time the pressure is not excessive.

According to Lusk,[21] pressure from Ovarian and pelvic tumours affects the ureters rather than the renal veins.

6. Galabin[1] considers that one of the principal causes of albuminuria during pregnancy is the large increase in the amount of work to be done by the kidneys of the mother, due to the excretions of the foetus requiring to be got rid of in addition to those of the mother. In the foetus, tissue changes are more rapid than in the mother, and the products of these changes are probably much more irritating in their effects from the former than from the latter. While healthy organs might be able satisfactorily to overtake this increase of work, yet diseased structures are very liable to inflammation under these conditions, especially if they are at the same time subjected to mechanical congestion of their blood supply. In Granular kidney there is a similar cause at work in the form of the prolonged presence in the blood of an excess of uric acid or some irritant. The changes in the kidneys are often slight after the first attack of Albuminuria. But as the result of repeated attacks, organic changes take place similar to those in Chronic Bright's Disease associated with Granular Contracted Kidney.[1]

7. M. Gubler's theory of Superalbuminosis asserted that the Albuminuria of pregnancy was due to an excessive quantity of Albumen in the blood of the mother beyond that required for the nourishment of the foetus — and

that this excess of albumen, if not used up by the foetus found its way into the urine of the mother. Galabin[1] says that this view of the matter is not the true one, and it is overturned by the fact that the mother does not necessarily suffer from Albuminuria in every case in which the foetus dies or wastes.

Interference with the excreting functions of the kidneys will lead to a generalised dropsy of the trunk and limbs. The retention of solid waste products, such as Urea, will tend to cause Uraemic convulsions. In the earlier stages, this may lead to Acute Tubular Nephritis - but if it continues during several pregnancies it may result in Chronic Granular kidney.

8. Dr. Galabin[1] thinks that slight cases of Albuminuria may be accounted for by the presence of Cystitis following on Gonorrhoea rather than kidney disease. He says that this applied chiefly to Hospital patients found in the French hospitals.

9. In 1867, Dr. Braxton Hicks[4] read a paper before the Obstetrical Society of London in which he referred to four cases of Eclampsia where the urine before the fits or after the first fit was free from Albumen - and later on became albuminous. He accounted for the simultaneous appearance of Albuminuria and Convulsions in one or other of three ways:-

a. The Convulsions might be the cause of the Nephritis.

b. The Inflammation of the kidneys and the Convulsions might

be due to the same cause - such as some irritating waste product from the urine circulating in the blood - acting also on the Nervous system.
c. In Eclampsia, the spasm of the Glottis which leads to marked venous congestion of the whole system, would also cause congestion in the veins of the kidneys.

In reply to Hicks, Galabin[1] states that the functional derangement of the excreting cells during the early stages of congestion of the kidneys lessens their power of excreting solids. Even when kidney disease is present, albuminuria may be delayed till a later period. He suggests that one cause of Eclampsia might be the presence in the blood of poisonous excrementitious material. An analogous case to this occurs in Chronic Bright's Disease in which there may be secretion of an excessively large amount of urine of low specific gravity - with an unusually small amount of urea - some time before albuminuria is present. According to this theory, the convulsions tend to hasten the Nephritis by increasing the functional disturbance in the Renal circulation. Galabin's theory is supported by the fact that in one of the cases recorded by him, the albuminuria increased and continued for a fortnight after the fits stopped; also that in a second of his cases there was oedema without albuminuria before the onset of the convulsions - showing a defective excretion of water. The convulsions were followed by marked acute Nephritis in one case of Dr. Hicks.

10. In a paper on "Puerperal Albuminuria" (19) Dr. Robert Maguire asserts that those cases of puerperal albuminuria in which retenitis is present are associated with organic disease of the kidneys. These cases do not cure themselves, but pass into Chronic Bright's Disease. Those cases which cure themselves are functional in their origin. In most instances of puerperal albuminuria, there is a large quantity of albumen present in the urine in the ninth month of pregnancy - but this disappears after delivery.
In some cases a varying amount of Uraemia appears during pregnancy and labour and then disappears. When the urine remains albuminous after delivery, organic disease is present and the case becomes one of Chronic Bright's disease. This used to be considered as merely a hyperaemia caused by pressure on the renal veins, but it is really an organic disease of the kidneys. Dr. Maguire says that, on the contrary, anaemia with fatty degeneration of the kidneys is more often found - and it has been conjectured that this anaemia is due to spasm of the Renal arteries. He considers it very wonderful that, in those cases in which the patient gets better, such an extensive and chronic fatty degeneration of the kidneys should clear up without leaving any bad effects behind.
The other class to which Dr. Maguire refers are those milder cases of albuminuria which should be grouped under the

head of merely functional derangement of the kidneys rather than organic disease. On experiment, he found that the precipitate got from an albuminous urine by the action of Nitric Acid, or Heat with dilute Acetic Acid really consists of several substances viz:- various forms of Serum albumen (distinguished by their physical properties) - and globulin (which is chemically different from serum albumen). The globulin was precipitated in a non-coagulated form by saturating the neutralised urine with Sulphate of Magnesium in the cold. The serum albumen remained in solution.

In four cases of functional Albuminuria, Dr. Maguire (19) found that the precipitate with Nitric Acid or Heat consisted almost exclusively of Globulin - with hardly a trace of Serum Albumen. But in a case of Granular kidney 70 per cent. of the precipitate consisted of Serum Albumen - while the Globulin amounted to 30 per cent. The result also differed very much from that of a case of general anaemia with fatty degeneration of the kidneys in which he found 70 per cent. of Globulin and 30 per cent. of Serum albumen. The Clinical history of the last case showed that it began as a functional disorder, and ended as an organic lesion. In this respect it was analogous to what is found in some cases of puerperal albuminuria.

In one instance in which pregnancy and labour were both normal, the urine contained a large proportion of albumen

of the Globulin variety - but no serum albumen - just as in cases of functional albuminuria. The albumen rapidly disappeared after delivery. Dr. Maguire argues that (if this case is a fair type of the majority), those patients who recover from puerperal albuminuria are suffering from a purely functional type of the disease without any organic mischief; while those patients who do not recover have organic disease of the kidneys. Retinitis accompanies organic changes in the kidneys in severe cases in other diseases. Hence Dr. Maguire asks if Retinitis in Puerperal Albuminuria is as serious a matter as in other lesions; and also inquires if this condition is always prolonged after delivery as in cases of Chronic Bright's disease. Dr. Maguire sums up thus:- "There is undoubtedly a form of puerperal albuminuria which is associated with an organic lesion of the kidney, and which we may call Nephrogenic; but there is also another form in which there is probably no such kidney lesion, which is probably due, as I believe to the profound change of pregnancy, and which may hence be styled Hæmatogenic."

11. Traube suggested that convulsions were due to ordinary Uraemia, while Rosenstein also included Puerperal Eclampsia within the scope of Uraemic convulsions. This theory asserts that an excessive arterial pressure of highly watery blood leads to the transudation of serum from the blood vessels.

The result of this is an oedematous condition of the brain causing pressure on its own bloodvessels, and thereafter sudden anaemia of the brain sets in. This latter results in the production of convulsions. In criticising this theory, Galabin[1] asserts that while excess of blood pressure outside the vessels over that inside them would lead to excessive transudation of serous fluid, this would stop when the equilibrium of the pressure was restored. The excess of blood pressure could not cause Anaemia by pressure on the vessels - for in the latter case the pressure outside the vessels would be greater than that inside them - and an opposite result would be produced. Galabin rejects the Traube-Rosenstein theory because in Bright's Disease the liability to convulsions does not depend upon the extent of generalised dropsy; and he thinks that those causes which produce Oedema in the Brain should produce it in other organs also.

Spiegelberg holds[5] that the only definitely known cause of the convulsions is the kidney disease which gives rise to Uraemia, and that this is either brought on or aggravated by the puerperium. Those cases in which no kidney disease could be discovered might be due simply to the puerperal state apart from uraemia - although the worst forms are always Uraemic.

Lever showed[2] in 1843 that the Urine of patients suffering from Eclampsia nearly always contains a large proportion

of Albumen, and he found changes in the kidneys similar to those in Bright's disease. From this he concluded that the convulsions of Eclampsia were due to the retention of Ureain the blood, just as in the convulsions of Nephritis. In those cases in which the urea is not excreted, he held that it was decomposed into Carbonate of Ammonia, and that the latter causes convulsions when it collects in the blood. Lever's theory was opposed by Pathologists on the ground that no Ammonia could actually be discovered in the blood. Spiegelberg[5] found Ammonia in moderate amount in one case only - and in two others the merest trace was discovered. He hence regarded it as one of the rarest causes of convulsions, and considered Uraemic poisoning as caused by the retention in the blood of all the substances which should be excreted by the kidneys.

According to Rosenstein's theory, the cause of Uraemia is a sudden rise of bloodpressure in the Aorta, taking place in a patient with a very watery condition of the blood. The acute oedema of the brain thus produced causes compression of the Cerebral vessels, and leads to sudden anaemia of the brain. Anaemia limited to the Cerebrum results in Coma, but extension to the middle brain causes convulsions. It is supposed that the increase in bloodpressure might be due to pains, and this would explain many of the symptoms of Eclampsia.

Hecker asserts that something in addition to the ~~Eclampsia~~ hydraemia is required to account for the convulsions — especially if it is true that hydraemia is present in most pregnant women, whereas Eclampsia is a very rare disease.

According to Spiegelberg, [5] hydraemia is absent in most cases of Eclampsia; and most hydraemic patients escape from Eclampsia. In these latter instances, neither the hydraemia nor the increased arterial pressure produce oedema of the brain. Another objection is that dropsy is not always found post mortem in Eclampsia; and even when found, it might be due to some other cause. The Rosenstein theory does not account for convulsions before and after labour. The state of the pulse and pupils does not correspond to what is usually found in cases of pressure on the brain due to oedema. Spiegelberg says that the theory that Eclampsia is due to Uraemia, is the simplest explanation, agrees with all our experience, and accounts for all the symptoms of the disease.

The theory of Oedema of the Brain makes the predisposing cause of Eclampsia an undue watery condition of the blood, with loss of Albumen; while the exciting cause is raising of the arterial blood-pressure with the accompanying hypertrophy of the left ventricle of the heart. During labour, the great activity of the muscles concerned in parturition increases the high pressure in the arteries, and this is intensified by the patient holding her breath

when she bears down during the pains - thus preventing the proper oxygenation of the blood. (17)

By tying the Carotid Arteries in animals, Kussmaul and Tenner produced convulsions, and hence they inferred that Eclampsia might be due to Anaemia of the brain connected with contraction of the Arterioles. The loss of Consciousness in Eclampsia is caused by Anaemia of the Cerebrum such as occurs in Syncope; but Convulsions occur when the brain is removed, provided that the Medulla Oblongata and Pons Varolii are preserved intact.

Nothnagel (24) has proved that the convulsions arise from excitement of ganglia in the Pons Varolii. Schroeder van der Kolk states that the grey matter for the Cranial Nerves is located in the floor of the fourth Ventricle and in the Medulla Oblongata. Any stimulus of the Vaso-motor Nerves which would make the arterioles contract would cause Convulsions and Coma. Inasmuch as the convulsions may come on while the patient remains conscious Nothnagel believes that the same cause which could act indirectly through the vaso-motor nerves could also directly excite the centres of muscular movements.

Lusk (21) says that, on the theory of Nothnagel, convulsions ought to be divided into two groups viz:- (a) those due to irritation from the periphery, and (b) those due to Centric causes; and he holds that Cerebral anaemia is present in both

He maintains that in almost every case of Eclampsia there is both increased tension in the Renal arteries, and also retention in the blood of poisonous waste material of which urea is the most important. It matters little whether or not Carbonate of Ammonia or Cerebral Oedema is present in a few cases. Peripheral irritation, apart from uraemia, may sometimes cause Eclampsia

Since Chronic Bright's Disease was often connected with the retention of Urinary debris in the blood, and was often associated with Convulsions, it was inferred that the convulsions of Eclampsia were in the same manner due to a toxaemia caused by the retention of urea in the blood. This view was advocated by Braun and by Frerichs.

On the theory of Traube and Rosenstein, Eclampsia is due to acute cerebral anaemia brought about by the changes which occur in the blood during pregnancy. The watery condition of the blood is much more marked, albuminuria is also present. Hypertrophy of the heart intensifies the increased arterial tension. A temporary hydraemia of the brain is produced, followed by effusion of serum into the tissues of the Cerebrum - and this effusion causes cerebral anaemia by pressure on the small vessels.

Playfair (28) says that the theory accounts for the convulsions being intensified by labour because the cerebral arterial tension is much increased during the acme of the pains; but that

it does not entirely explain those cases with well defined precursory symptoms, and those with much albuminuria. The signs in the latter case are exactly the same as those preceding Uraemic convulsions in Chronic Bright's Disease.

Objections:- A strong objection to the theory is given by Lobleen (22) who found on post mortem examination that in the Brain there was neither oedema, anaemia, nor flattened convolutions such as are assumed by its supporters.

Macdonald (23) found on careful post mortem examination of two cases, that while there was marked anaemia of the Cerebro-Spinal Centres, with congestion of the membranes of the brain - there was entire absence of oedema. This result led him to regard Eclampsia as due to irritation of the Vaso-motor centre brought on by Anaemia caused by retention in the kidneys of waste products. He supposed the convulsions to be set up by the excessive irritation of this waste material which led to anaemia of the deep seated nerve centres. The abnormal irritability of the nervous system during pregnancy probably accounts for the peculiar liability of pregnant women to convulsions. But Playfair (28) holds that a definite morbid action requires a definite cause, and that this cause exists either in a very watery condition of the blood, or in a toxaemia associated with Albuminuria; and that sometimes there is strong emotional excitement either along with or apart from these. The extreme anaemia should be remembered in treatment.

Some writers maintain that the Uraemic variety is only one amongst many forms of convulsions. The albuminuria may be the effect of the convulsions rather than their cause, and may not be necessarily associated with Nephritis.
In the majority of cases Uraemia seems to be the chief cause of puerperal convulsions. Dr. Galabin[1] found Albuminuria in most of his hospital and private cases of Eclampsia, but in some cases albumen was either absent or only in minute quantity.

1. He records the case of a primipara, aged 14, who had a violent epileptiform convulsion seven hours after delivery - and this was succeeded by deep coma. The patient was anaemic On examination under chloroform the uterus was found to be relaxed and filled with clots. The latter were removed and the uterus syringed out with Solution of Perchloride Iron. Five more fits during the next eight hours were overcome by Chloroform and then the patient made a good recovery. Albumen was entirely absent from the Urine. The child had constant convulsions for two days and then died. Uraemia was quite absent in this case.
2. Another case was that of a Chronic Epileptic who, at forty years of age, became pregnant for the 12th time. She had been subject to Epileptic fits since she was 17 years old. After her second confinement she had convulsions for six days. In her twelfth pregnancy, convulsions began about the eighth month. Her friends did not send for the doctor until the fourth day of the convulsions when she had been unconscious for three days.

As the os was dilated, the membranes were at once ruptured, and the child born alive. Although Chloroform was given constantly for hours, the convulsions were only partially controlled, and the patient died in nineteen hours after delivery. Tube casts were quite absent from the urine, and there was only a trace of albumen. On P. M. Examination, the kidneys were hard and much congested. Galabin thinks that the trace of albuminuria in this case may have been secondary to the convulsions, and the effect aggravated by the pressure effects of pregnancy and labour, without any actual nephritis. I think this case might fairly be regarded as simply one of Epilepsy occurring during pregnancy.

3. In another case, aged 21, who had suffered from Epilepsy for six years but had been free from fits since her marriage nine months before - Eclampsia set in shortly before labour but was controlled by Chloroform inhalations. Even when the fits were most severe, the urine was free from albumen. The day after delivery, there was a trace of albumen in the urine and this increased slightly.

4. A fourth case had convulsions four days after delivery. Before the convulsions there was no albumen in the urine, but after the fits there was one eighth part of Albumen present.

Galabin records 18 more cases in which there was a very large amount of albumen in the urine - and in most of them tube-casts were also present in abundance. In 12 cases out of 13 in private practice a considerable amount

of albumen was present in the urine. In the thirteenth case the patient suffered from left hemiplegia after delivery and the child had hydrocephalus.

Galabin[1] has four objections to the Uraemic theory as explaining all cases of Eclampsia:-

1. While puerperal convulsions are generally the sign of a very early stage of kidney disease, other forms of uraemic convulsions as a rule occur in advanced cases.
2. The convulsions are more marked than the coma.
3. The fits are very severe, and follow each other in rapid succession.
4. While in ordinary uraemia the temperature is subnormal; in eclampsia it is sometimes much elevated - even as high as 110°F.

Galabin believes that in every case there are several causes at work, and that no one theory accounts for all the cases. In the increased excitability of the nerve centres during pregnancy a poison in the blood more easily sets up convulsions than at other times. In Chronic Nephritis the blood has acquired such a tolerance for the poison, that Eclampsia is less liable to develop. The influence of Reflex irritation is proved by the fact that most cases of Eclampsia begin during labour, and that the fits accompany each labour pain, and may be excited by a digital examination. In most cases, an early stage of Inflammation of the kidneys is followed by a poison in the blood, and this seems to be proved by the frequent death of the foetus even when protected from pressure by the Liquor Amnii

Spiegelberg (5) refers to ~~four~~ three objections which have been urged against the Uraemic theory and tries to answer them:-

1. A patient who may have had Bright's disease for a long time previously, may escape from Eclampsia during pregnancy. He says that the reason of this is that the healthy portions of the kidneys may secrete sufficient urine to prevent the onset of Uraemic poisoning. Another explanation is that Eclampsia is caused by a sudden inflammation of the whole parenchyma of the kidneys, leading to an almost entire suppression of urine. If chronic inflammation prevents a sufficient secretion of urine, or if pregnancy causes a suppression of urine, then there may be convulsions during pregnancy. The eclampsia is accounted for by the injury caused to the system by the retention of urinary debris in the blood which ensues when suppression of Urine takes place. There is either entire suppression of urine or only a very small quantity secreted. In the former cases there is a great increase of urea in the blood.

2. Another objection stated is that very often the changes in the kidneys post mortem are either absent or very slight. The reason of this is that sudden suppression of urine in persons formerly quite healthy may leave little or no changes in the kidneys. The non-secretion of urine when absolute may be due either to disturbance of the Renal circulation or to some affection of their vessels both coming on and disappearing so suddenly as not to leave any organic lesions behind.

Spiegelberg believes that convulsions occurring in those women who have been free from previous Nephritis are due to changes in the renal circulation. In most cases the venous engorgement in the kidneys could not be caused by simple pressure of the pregnant uterus on the renal veins. When severe pressure arises from abdominal tumours, the ureters are more liable to be compressed than the renal veins. From the fact that the kidneys are more frequently pale and anaemic than congested when examined post mortem, he holds that the alteration is in the walls of the bloodvessels in the form of a degeneration which presents transudation through them. This would account for premonitory symptoms.

Another explanation might be that sudden arrest of the arterial renal circulation might be caused by spasm of the vessels under the influence of the vaso-motor nerves. The sensitiveness of the renal epithelium is so marked that brief interruptions of the blood flow are liable to make the secretion of urine cease from very slight causes, and according to Overbeck, it may even cease for three quarters of an hour. The epithelium of the kidneys needs a regular and steady supply of blood for doing its own work. Frequent spasm of the arteries first stops the secretion and then destroys the epithelium - leading to retention of urinary debris in the blood. There is anaemia simultaneously in the brain and kidneys. The uraemia explains the irritation of the brain, even apart from anaemia.

The relation of the contractions of the Uterus to the convulsions is probably accounted for by the reflex stimulation by the uterus of the sensory nerves of the kidneys. Convulsions are more frequent in primiparae than in multiparae because the nervous system is more irritable in the former than in the latter. Lohlein & Hecker make the proportion of the former 85 per cent. Increased arterial pressure in Eclampsia is not the cause of the disease, but it intensifies the attacks. The arterial tension is increased by the labour pains during delivery.

3. In reply to the objection that Eclampsia may occur apart from Albuminuria, or may only be followed by it, Spiegelberg says that these cases are rare, and some may be accounted for by Uraemic poisoning. Cases of sudden renal incompetency might show an insidious onset of Albuminuria even in urine formerly quite free from Albumen. The Albuminuria is here caused by interference in the renal circulation due to an alteration in the renal bloodvessels. Cases in which the Albuminuria is either entirely absent, or only a trace of albumen is present for a short time, (with the exception of those due to Organic brain disease) Spiegelberg would put into a separate class as "Eclampteform", and would regard as due to reflex irritation of the Vasomotor and convulsive centres by some stimulus at the surface of the body. These are of the nature of Epileptic attacks brought on by artificial stimuli. Attacks of this nature were set up by Brown Sequard

by irritating the Sciatic Nerve; and by Hecker by stimulating the organs of generation on the surface of the body.

Spiegelberg records a case in which very great distension of the bladder from prolonged pressure of the head on the pelvis set up the fits. The head was so low in the pelvis that it compressed the Urethra and prevented the urine being drawn off by the catheter. The head was delivered by forceps. There was no albumen in the Urine - even in the portion drawn off by the catheter. The fits did not return and the patient made a good recovery.

Seyfert of Prague opposed the Uraemic theory in 1865, for the following reasons:- (21)

1. That there may be Convulsions without Albuminuria.
2. That Albuminuria is more often the effect than the cause of the Convulsions
3. That the kidney lesions in many fatal cases of Eclampsia are either absent or very slight.
4. That Convulsions are rare in those cases of Chronic Bright's disease which had existed prior to pregnancy.
5. Convulsions do not occur in cases of true Uraemia where the suppression of urine was caused by invasion of the ureters in cases of uterine Cancer.

According to Lusk (21) too much importance is attached to the presence or absence of Albuminuria, whereas the convulsions and Uraemia are really due to suppression of urine.

Braun (33) has noted fatal cases of Eclampsia in which post mortem examination showed amyloid degeneration of the kidneys and heart while no albuminuria had been observed during life. In other cases the kidneys were atrophied, and the albuminuria, tube casts and dropsy of the earlier stage of pregnancy had all disappeared.

Albuminuria may sometimes fail to be detected because it may disappear first, and then reappear after some hours. Seyfert found convulsions in only two women out of 70 suffering from Bright's disease, who became pregnant. Of 46 cases of Chronic Nephritis reported by Hofmeier, one half died - but only one third of them had convulsions. Out of a total of 5000 births there were 137 cases of Nephritis; and of these 104 had Eclampsia. According to Braun, only sixty per cent of all the acute and chronic cases of Nephritis suffer from Eclampsia.

In consequence of having discovered dilatation of one or both ureters in 8 out of 32 cases examined by him, Löhlein (22) raises the question as to whether cases of Eclampsia accompanied by Albuminuria may be explained by mechanical obstruction.

Although Uraemia is absent in a few exceptional cases of Eclampsia, still it is so markedly present in such a huge majority of cases that we must regard it as one of the most important, if not the most important factor in its production even although it seems not to explain every case satisfactorily.

Frerichs supposed that a ferment in the Blood changed urea into Carbonate of Ammonia. This view was at first supported by Spiegelberg [5] who in 1870 discovered in one case of Eclampsia an appreciable quantity of Carbonate of Ammonia in the blood of the patient; but as this view was not confirmed by further experiments, he thought Ammoniaemia one of the rarest causes of convulsions. Hammond [30] by a series of experiments showed ~~by a series of experiments~~ that Urea never becomes decomposed in the blood into Carbonate of Ammonia, and denied that any symptoms of Uraemia were ever caused by this process. Frerichs produced convulsions of the nature of Eclampsia by injecting Carbonate of Ammonia into the veins of animals.

Some authorities believe that there is some poison in the blood different from Urea, and that this irritates the brain. This was supposed to be due either to Bacteria in the Brain or to Acetone in the blood of the mother absorbed from the foetus. The latter theory is supposed to explain those cases which recover after the death of the foetus. The disease is due rather to the Ptomaines which are the products of the Bacteria than to the Bacteria themselves. The smell of Acetone has been observed in the breath and vomited matters of Eclamptic patients. Hence it was supposed to be associated with the sugar found in the urine in some cases of Eclampsia.

Post Mortem appearances. Kidneys.

Post Mortem Examination :- Usually there is slight organic disease in the kidneys - in a few cases no renal lesion is present. The kidneys are more often pale and anaemic than congested. In the "pregnancy kidney" there is marked anaemia with fatty degeneration of the glomeruli. Sometimes there is atrophy of the kidneys. In 8 out of 32 cases examined by him, Lohlein [27] found dilatation of one or both ureters and of the pelves of the kidneys.

Brain

The Brain usually shows marked anaemia, and has a copious transudation of serum into its substance and between its membranes. There are sometimes extravasations of blood similar to those found in Apoplexy. In fatal cases, the brain is found to be oedematous, with its membranes flattened out. In some cases, the lesions in the brain are very slight. Lever [2] had a fatal case in which death was due to Acute Meningitis. In 1878, Angus Macdonald [23] found the membranes congested and the venous sinuses filled with blood; but the deeper layers of the brain were markedly anaemic. The ventricles were filled with serum instead of being empty as was formerly supposed. He thought that the anaemia was due to contraction of the arteries following on the irritation of the vaso-motor centres brought about by waste products from the urine retained in the circulation - when the kidneys were not excreting properly.

Liver

The Liver occasionally undergoes Acute Yellow Atrophy.

Blood.

The Blood contains Urea, and excess of Carbonate of Ammonia

in a few cases - and occasionally Acetone. Schroeder held that the Anaemia of the Brain was caused by some poison in the Blood which should have been excreted. Probably the nature of the poison varies in different cases.

Heart. The Heart has been found hypertrophied by Lohlein, and this would account for the increased Arterial tension.

Lungs. Hypostatic congestion of the Lungs has also been found.

Immediate Cause of Death. The Immediate Cause of Death is usually from suffocation caused by spasm of the muscles of Respiration. In some cases it is due to Apoplexy; in others, death is caused by exhaustion from an over-excited brain.

Many patients die during the attack, either shortly before or soon after the birth of the child.

Symptoms and Progress:-

Predisposing causes. The Predisposing causes of the development of Eclampsia are the primiparous condition, twin pregnancy, contracted state of the Pelvis, over-distension of the Uterus, severe bruising of the cervix uteri, the presence of antecedent kidney disease. Epidemics of Eclampsia are supposed to be due to atmospheric changes, which by interfering with the functions of the skin increase the amount of work requiring to be done by the kidneys. This view is supported by Lusellie, Rosenstein, and Simpson.

Time of Onset. The Time of Onset of the Convulsions is usually during the first half of labour. It is seldom before the end of the sixth month of pregnancy. But Spiegelberg[5] and Paetsch have each had a fatal case in the fifth month; Hecker and Willis each had a case in the fourth month; and Damyan had one as early as the sixth week, in which the patient recovered after the Ovum had been removed. The fits do not necessarily always come on in connection with labour pains. In many cases the attacks come on during pregnancy, and during absolute inactivity of the uterus. If the convulsions come on during pregnancy they are quickly followed by labour pains, but the latter may again pass off under favourable conditions. If the patient has Uraemia, the contractions of the uterus may be the exciting cause of the attack. When the

Uterus is stimulated to contract by the friction of the hand, the convulsions may come on after the birth of the child or after the expulsion of the placenta. The fits, in these cases had no direct effect on the pains. Spiegelberg ⑤ has never observed the same kind of tonic spasms in the Uterus as in the muscular system as a whole during Eclampsia.

Bagot says that when Eclampsia occurs during pregnancy it is most common between the sixth and tenth months. When it begins after delivery it is most frequently within 48 hours of the birth of the child. The onset of the fits is usually succeeded by labour, but sometimes there may be an interval of several days between them. Occasionally the fits may stop and the pregnancy continue. Eclampsia coming on after the termination of labour is usually rapid in its onset. But Legroux has observed it as late as the 14th day, and Simpson even in the eighth week after delivery. These late cases are usually of a milder type.

Premonitory symptoms.

The Premonitory Symptoms of Eclampsia vary greatly in severity. Oedema of the hands and face is of marked significance. There may also be oedema of the limbs and labia majora. Sometimes the dropsy is only observed in the morning after the rest of the night, and disappears on walking about. It may sometimes be limited to the feet and ankles. The presence of albumen and casts

in the Urine is the most important symptom. There may be dyspepsia, nausea and vomiting. Hemicrania, giddiness, loss of memory, melancholia; partial or complete blindness, either in the form of amblyopia or of amaurosis, flashes of light before the eyes, contracted pupils may be present, as also tinnitus aurium. The intellectual faculties may also be impaired. If there is oedema of the face, hands or forearms, the urine should be examined for albumen. In slighter cases the symptoms are general malaise, irritability of the nervous system and slight headache or stupor.

Onset. The Onset of the Disease resembles a severe Epileptic attack but there is no introductory cry. Although tonic and clonic Symptoms. spasms are both found in a marked degree, yet they are not present in distinct stages. Consciousness is entirely lost. During labour, the attack is preceded by a calm period, in the course of which the patient stops complaining and falls into a quiet sleep of short duration. The calm is succeeded by greater restlessness than before. The Orbicularis Oris exhibits convulsive movements which give a smiling appearance to the face. The eyelids open and close with great rapidity. At first, the pupils contract, and the eyeballs stare in a fixed position. A few seconds later, the eyes oscillate from side to side or roll upwards, and the pupils dilate and cease to respond to light. Sometimes the eyeballs are excitedly rolled about in every direction, and there may be external strabismus. Next,

the other muscles of the face are thrown into convulsions. The mouth is opened and becomes markedly drawn to one side. By being violently tossed about, the tongue is liable to be bitten by the teeth, and often bleeds. The head is either bent violently backwards as in opisthotonos, or rapidly moved from shoulder to shoulder, or bent to one side. Often, at the onset, there is only pronation and supination of the forearm and clenching of the fingers into the palm of the hand. The arms and legs are violently jerked in various directions, or are extended in a state of tonic spasm. As a rule, the upper limbs show more marked movements than the lower. The lower limbs are sometimes rigidly fixed in a state of tetanus; while on other occasions, they are bent at the knee and drop of their own weight. There is well-marked throbbing of the carotids and swelling of the superficial veins of the neck due to the disturbance of the circulation and respiration. At first the face is pale, but afterwards becomes livid from engorgement of the veins. The capillaries of the conjunctivae become injected. At the height of a fit, the breathing stops for a moment on account of spasm of the muscles of respiration. The breathing is irregular, and stertorous in character. The pulse is intermittent, rapid, small, and easily compressed. The teeth are often clenched together. A profuse, cold, clammy perspiration breaks over the body. The patient may lose control over the bladder and rectum. The temperature is raised.

The convulsions may stop either gradually or suddenly. When the fits pass off, the features return to their normal state, bloody and frothy saliva escapes from the mouth and nostrils. There is swelling of the tongue and lips. The patient falls into a state of stupor accompanied by stertorous respiration. When spoken to, he can at first be roused from the stupor. The depth of the stupor depends upon the frequency and violence of the attacks. Sometimes the breathing is accompanied by deep sighs, and the inspirations are separated by long pauses like those in Cheyne-Stokes' Respiration. The pulse becomes slower and fuller, but the temperature rises. The face becomes livid and cyanosed, and assumes a vacant expression. In favourable cases, the patient falls into a partial sleep, broken by a few uterine contractions. The sleep is light in mild cases, but in more severe attacks, deep coma may come on, and the patient may never return to full consciousness and is quite unable to speak between the fits - especially if the fits frequently recur. This deep coma is due to congestion of the brain caused by interference with the circulation in the large veins in the neck. The groaning of the patient during the uterine contractions shows that the coma is not absolute, and besides this, the patient shows signs of sensibility when irritated. The patient may die in a state of great torpor in very severe cases. Playfair (28) says that consciousness soon returns after the first attack, but that the patient remains in a half

sleepy condition and has a very hazy recollection of the fit. But if the fits frequently recur, there is deep coma in the intervals between them. After the patient has been restored to consciousness - in cases ending in recovery - the patient may have entirely forgotten the events which have occurred just before, and during the attack. The whole period of illness may be quite forgotten - in the same way as those who have been rescued from drowning lose all recollection of the events just preceding, and those during the immersion. In attacks coming on during pregnancy, the nervous shock and general disturbance of the system are almost certain to bring on labour. If the convulsions come on for the first time during labour, there is such an increase in the force and frequency of the pains, that the child may be born very quickly and unexpectedly during one of the convulsions brought on by a fresh pain.

Duration of the Paroxysms.

The duration of the Paroxysms has been variously estimated by different authors. Spiegelberg[5] says that the paroxysm itself rarely lasts more than sixty seconds - and that, as a rule, its duration is from ten to thirty seconds. The error of thinking that some paroxysms continue longer, he asserts, arises from regarding two or more really successive attacks as a single one. Since the respiration is momentarily arrested during a fit, a prolonged paroxysm must necessarily destroy the life of the patient. Lusk[21] also holds

that a single attack on an average lasts from ten to thirty seconds, and that the longest attack is one minute. Playfair, (28) however, differs from Spiegelberg and Lusk in saying that the duration of the convulsion may be as long as three or four minutes - although it is usually less. After a single seizure, the stupor passes off in about two hours. It seldom lasts more than one day.

Interval between attacks. The interval between the attacks varies from a few minutes to several hours - according to the severity of the cases. The rapidity of fresh attacks increases with the severity of the first fit.

Number of attacks. The number of attacks varies from two or three to fifty or sixty - a single attack is seldom met with. It is sometimes very large - as many as 81 have been observed by Brummerstadt ("Bericht aus der Central Hebammenanstalt zu Rostock," 1866), and Depaul has noted 160 ("Leçons de Clin: Obstet." 1872, p. 288).

The Urine. The Urine, in a very large proportion of cases, contains a considerable amount of albumen - much renal epithelium, which is often in the form of fatty tube casts - also fibrinous casts, and blood. In very severe cases, the urine is either scanty or totally suppressed - and the suppression may last for some hours. Sometimes there is a great decrease in the amount of urea excreted. Fatal cases show great diminution in the total amount of urine secreted, and the urine is very albuminous - is smoky from the presence of blood and contains tube-casts.

Terminations of Eclampsia.

The Terminations of Eclampsia:- Cases occurring before delivery are usually fatal from the supervention of Oedema of the Lungs associated with a weak heart. Or death may be due either to Carbonic Acid poison from spasm of the respiratory muscles, or to nervous exhaustion. (21) A case is recorded by Bailly of death from Asphyxia produced by a swollen tongue which had been severely bitten. During and after delivery the convulsions are less severe - of shorter duration - and occur less frequently than before delivery. After delivery the convulsions may entirely stop; the breathing becomes natural; the pulse becomes regular and less frequent. Profuse perspirations break out. The patient falls into a gentle natural sleep after the expulsion of the Ovum and afterwards slowly regains consciousness. After awaking from sleep, the patient has headache and partial loss of memory. The whole period of the convulsions becomes a blank in the memory, and sometimes the patient even remembers nothing of the pregnancy. The tongue is often sore from having been bitten. Eclampsia may leave behind a feeble condition of the mind and even Mania (although this passes off usually in a few days) Dropsy and albuminuria usually pass off rapidly.

Sequelae.

The Sequelae of Eclampsia:- Even after consciousness has returned, eclampsia renders the patient specially liable to Post Partum haemorrhage and to Puerperal inflammations.

The Post Partum Haemorrhage may be due either to the albuminuria or to the venous condition of the blood after the fits. Patients are also liable to suffer from very great poverty of blood. Those convalescent from Eclampsia are more liable than others to die - probably on account of the frequency of operations required during labour. (5) Spiegelberg has seen Aphasia in a patient with a brain which was otherwise healthy; but in a fatal case of Mendel, (6) a cerebral lesion was noted at the post mortem.

Mortality. The Mortality a good many years ago was as high as 36 per cent. but at present, on account of improved treatment and the rigid use of antiseptics, it is stated by some authorities to be reduced to about ten per cent. The accuracy of diagnosis in the early stage has helped to reduce the death rate. Playfair (28) says the death rate has been reduced to between 33 and 25 per cent. In 1885, Barker ("The Puerperal Diseases" p. 125) found a mortality of 32 per cent. in cases before and during labour - while it was 22 per cent. in cases after labour. But since that date it has fallen to 14 per cent. Dr. Phillips (29) in 1870 recorded the same conclusion, showing that the mortality has considerably decreased since the substitution of chloroform inhalations for indiscriminate bleeding. Dohrn gives 29 per cent. of deaths in 747 cases. Hofmeier gives a death rate of 32·4 per cent. in 104 cases. Lusk (21) gives the total mortality from Eclampsia alone as compared

with death from all causes during pregnancy as nearly 1 to 8. The total mortality from all cases during pregnancy is 1 in 700.

Prognosis. The Prognosis of Eclampsia:- The danger is greatest the earlier in pregnancy or labour the convulsions break out. It is intensified by delay in the expulsion of the child, and by difficulty in delivery. The danger is greater, the more numerous are the attacks and the more rapid and deep the coma. It is also greater as the renal inadequacy increases. Thomas (8) proves from his statistics that cases are more fatal in multiparae than in primiparae - and are more serious in the robust and plethoric, than ~~the~~ the weaker and spare built. As death is often due to Oedema of the lungs associated with a weak condition of the heart, the prognosis depends greatly on the state of the heart and lungs. Inflammation of the kidneys is a grave complication. Sometimes Apoplexy of the Brain is the cause of death. If the patient recovers consciousness between the fits it is a favourable sign. The prognosis is more favourable before the onset of labour than after it has set in. In cases occurring before the onset of labour, if the foetus dies, the prognosis for the mother is good. If the urine is freely excreted in large amount, the prognosis is good - but if there is suppression of urine, the patient will probably die. Premature delivery of the child is unfavourable. The earlier in labour the fits set in, the more unfavourable is the prognosis. Out of 83 cases

before or during the first stage of labour, Loblein (22) reported 40·5 per cent. of deaths. But of fifteen cases occurring after the first stage of labour had terminated there was only one death. In the most favourable cases, the fits come on for the first time during labour. As a rule, the fits do not stop until the child is born. C. Braun says that, after delivery, the convulsions stop in thirty-seven per cent.; become less violent in thirty-one per cent.; but that in thirty-two per cent. they continue as severe as ever, after delivery. A return of free secretion of urine, followed by the subsidence of the oedema, and the disappearance of albuminuria is favourable, on account of accumulation of carbonic acid gas in their mothers' blood, about half of the children of eclamptic women are dead born. There is much less danger to the child after the completion of the first stage of labour. Hall Davis records 10 still-born children, out of a total of 36 cases. Lazeaux records several cases of convulsions attacking children in utero. Death of the children from suffocation has been proved by post mortem examination. The danger to the child is in proportion to the number and severity of the attacks.

If a tonic spasm is unduly prolonged, the mother may die of Asphyxia. In this case, the respirations are suspended in the same way as during the convulsions of Laryngismus Stridulus in children. The heart may also be affected

by convulsive contractions. Later on in the attack death may be due to a combination of exhaustion with asphyxia. (28)
A child of an Eclamptic patient, apparently healthy at birth, may subsequently suffer from convulsions.

Diagnosis. Diagnosis of Eclampsia :- Eclampsia may be diagnosed when there is a combination of the fits described above with accompanying renal disease. In some instances it may not be possible to examine the urine at once. Occasionally there may be Eclampsia without Albuminuria. These cases might be mistaken for Epilepsy or Hysteria or other forms of convulsions. Dr Murphy (17) regards Eclampsia as a symptom of Albuminuria during pregnancy or labour, and he thinks that the term should be restricted to cases in which albuminuria accompanies the convulsions.

1. In Epilepsy, there is usually a history of the patient having had Epileptic fits for many years before marriage, and of the occurrence of them at frequent regular intervals. Probably the other members of the family have been subject to Epilepsy or to other forms of diseases of the nervous system. There is only one fit in Epilepsy during the illness, whereas in Eclampsia there are a series of fits in rapid succession. In Epilepsy there is ~~only~~ one cry before each fit, but none in Eclampsia - the tongue is usually bitten; the breathing is stertorous; the pupils are insensible to light. After Epilepsy, the period of Coma is shorter; the intervals between the fits are greater; and there is

a shorter period of unconsciousness. In Epilepsy the pulse rate is almost normal; and the temperature is either quite normal or only slightly elevated - whereas in Eclampsia the temperature gets higher with every attack, and even remains somewhat elevated between the attacks - until the fits cease. There are no kidney symptoms throughout the disease, and neither albuminuria nor Uraemia in Epilepsy - whereas these are the rule in Eclampsia. The Respiration in Eclampsia has a distinctly hissing character.

2. In the convulsions of Hysteria the patient never becomes absolutely unconscious. There is an increase of the activity of the senses and of the irritability of the reflexes. (5) No paralysis sets in, and the worst effect of the fits is fatigue.
3. The stupor of Apoplexy is very like the coma succeeding Eclampsia, but the coma of Apoplexy is prolonged after the fit - and there are localised twitchings present, followed by hemiplegia.
4. The convulsions due to profuse Internal Haemorrhage would be distinguished by the physical signs, and by a careful examination of all the other symptoms present.
5. The nature of the vomited matters and the smell of the breath should distinguish the coma of Drunkenness from Eclampsia.
6. Meningitis is often difficult to distinguish from Eclampsia. But in the former, the symptoms come on gradually; there is no albuminuria present; and there is a smaller number of muscles involved in the convulsions than in Eclampsia.

Treatment. ## Treatment :-

Prophylactic treatment :- If there is a history of former attacks of kidney disease, the functions of the kidneys should
Diuretics. be regulated by alkaline diuretics or vegetable acids.
The pores of the skin should be opened, and free perspiration
Hot Pack. encouraged by the use of a hot pack or warm baths. Free
action of the bowels should be secured by the use of such
Purgatives. purgatives as Compound Salap Powder, Sulphate of Magnesia,
Compound Scammony Powder to produce watery evacuations.
Iron Iron and tonics should be given if there is Anaemia.
Milk diet. Charpentier [9] regards Milk diet as the best preventive
treatment and orders it at the very outset of the detection
of albumen in the urine during pregnancy. Tarnier also
upholds a milk diet. He says that headaches and pains
over the Epigastrium, as well as the presence of albumen
in the urine, often precede Eclampsia. In all cases of
pregnancy in which convulsions are to be feared, the
urine should be regularly and carefully examined for
albumen, and special note should be made as to whether
or not the albumen is persistent. [21] Although Nephritis
does not necessarily cause convulsions, yet the presence of
disease of the kidneys intensifies the danger of sudden suppression
of urine. Pregnancy aggravates inflammation of the kidneys
when it is present. Hofmeier has shown that many kidney
lesions which appear for the first time during pregnancy

do not disappear after delivery. Flaischen (25) disputes this statement and asserts that the anaemia of the kidneys is of reflex origin - altering the epithelium of the glomeruli and producing albuminuria without tube casts. At a later stage, about the middle or end of pregnancy, albuminuria is associated with tube casts. The urine is passed in small quantity and is of high specific gravity - thus distinguishing it from cases of Chronic Nefferites.

Warm clothing. Patients suffering from Albuminuria should wear flannel next the skin to avoid cold. They should be removed from any causes of mental excitement. The digestive system should be put in working order. If there are such premonitory symptoms as oedema of the face and limbs - partial suppression of urine - headache - giddiness - vomiting - muscae volitantes floating before the eyes - then give

Hot baths. steam or warm water baths with mustard at 100° F for half an hour, followed by a Dover's powder, or some

Diaphoretics diaphoretic and diuretic mixture such as 30 grs. of Citrate of Potash well diluted with water every four hours. Lusk (21) advocates

Iron. advocates large doses of the Tincture of Perchloride of Iron every four hours as he says this is a good diuretic, tonic to the bloodvessels, and enricher of the blood. The patient should

Milk diet. be placed on milk diet, or if she does not agree with that, she may use large quantities of such alkaline diuretic mineral

Mineral Waters waters as Seltzer, Vichy, Hunyadi Janos or Lithia Water.

Hot Pack. Free perspiration should be promoted either by a Hot pack or by a Turkish bath in order to get rid of fluid accumulations.

Laxatives. The bowels should be regulated by mild laxatives.

If brain symptoms develop, the irritability of the nervous system should be overcome by rectal injections of thirty grains each of Chloral & Bromides. Chloral Hydrate and Bromide of Potassium. The bowels should be freely moved by 5 grains of Calomel and forty or sixty grains Purgatives. of Compound Jalap powder - as this free purgation purifies the blood from Urea, relaces the arterioles, and decreases the tension in the arteries. This treatment may remove the headache and sickness, soothe the patient, and bring on refreshing sleep.

Premonitory stage. Treatment of Premonitory stage:- If the patient has oedema of the face and limbs, albuminuria and tube casts, Murphy (17) recommends the use of such starchy foods as Diet. tapioca and arrowroot, a spare vegetable diet, with sugar and cream. A rich animal diet increases the amount Benzoic Acid. of urea. Frerichs advises Lemon juice or Benzoic Acid to be given, in order to neutralise the Carbonate of Ammonia. When exudation has taken place into the Malpighian tufts and Uriniferous tubules by alkaline diluents followed by Seltzer or Vichy water. Frerichs also gives tonic pills of Aloes & Tannin Aloes and Tannin in order to restore the tone of the kidneys. Colchicum Byford regards Colchicum as very useful. Murphy has Jalap. got good results from a purge of Jalap followed by a mixture containing Sweet Spirits of Nitre and Tincture of Steel. The

Objection to induction of premature labour + Chloroform

objects to the Induction of premature labour as advised by Tarnier, - except in very urgent cases. He does not approve of the suggestion of Blailly to give Chloroform to all patients suffering from Albuminuria - whenever labour begins.

Venesection Chloral Hydrate Morphia

Spiegelberg (5) recommends a moderate amount of Venesection, and the use of such narcotics as Chloral Hydrate per rectum, or the hypodermic injection of Morphia. He also supports the suggestion of Frerichs to give such vegetable acids as Benzoic Acid and Citric Acid to neutralise the Carbonate of Ammonia in the blood.

Vegetable Acids

The objection to the induction of premature labour at this stage is that we are never quite sure when the paroxysms will come on. Besides, the induction of labour may set up such activity in the uterus as to bring on the fits. Cases of very marked albuminuria might necessitate induction of premature labour even when fits were absent.

Treatment during attack

Treatment during the Attack :- The patient may either be kept in bed (care being taken to watch that she does not roll over its edge); or she may be placed on a mattress on the floor. Her arms and legs should not be forcibly held down lest she suffer from shock, but should be gently guided with the hands to prevent her injuring herself.

Prevent tongue being bitten

Prevent the tongue being bitten by inserting between the teeth the handle of a spoon round which surgeon's lint has been wound. Murphy (17) recommends that, instead of using the guarded handle of a spoon, the tongue should first be pressed

well back, and that then the end of a towel should be inserted between the teeth. He says the handle is liable to injure the teeth.

Venesection. Venesection is strongly advocated by Spiegelberg[5] as the quickest and most effective method of lowering the blood-pressure and restoring the kidneys to their normal functions, and soothing the vaso-motor nerves. He advises seven ounces of blood to be drawn from the veins of the arm if necessary - the effect in each case being carefully watched. If the first bleeding fails to accomplish its purpose, or if its effect is only temporary, he repeats the bleeding after an interval. From extra robust women he has drawn as much as seventeen ounces of blood without harm. In attacks which are only of an Eclamptiform character, he uses Narcotics instead of resorting to Venesection.

Narcotics.

Venesection followed by Chloral.

Chloroform followed by venesection. Quinine & Calomel.

Charpentier[9] advises venesection to the extent of the withdrawal of sixteen ounces of blood - followed by the use of Chloral - in the case of robust patients who show signs of marked Cyanosis; but in feeble persons, he prefers to use Chloral alone. Dr. King of Ohio[15] successfully treated a case of Eclampsia in the first stage of labour by the free use of Venesection preceded by Chloroform inhalations and followed by Quinine and Calomel powders. After its use, the breathing became more natural and the convulsions stopped, but the pupils still continued insensible to light. Dr. Wilson[16] records a case of Eclampsia, two hours after labour, in a stout, plethoric woman, where repeated venesection at

intervals of three hours - followed by a purge of Calomel and Croton Oil, failed to check the convulsions, and the patient died in 12 hours after the onset of the fits.

Objections to Venesection. Although formerly, free venesection was practised habitually it is much less frequently resorted to now, because, while the removal of a large quantity of blood at first relieves the blood-pressure, yet the amount so removed is speedily restored to the circulation by a draining off of serum from the tissues of the rest of the body. This produces a very watery condition of the blood, which as a rule does harm. Occasionally Dr. Murphy (17) has produced a good effect by a moderate amount of cupping over the kidneys. Professor Fordyce Barker is in favour of venesection in suitable cases in Eclampsia. Lusk (21) has been favourably impressed with the good effects following venesection in the Hôpital des Cliniques in Paris. He thinks that its principal merit lies in its rapidity of action and in its preparing the patient to benefit more fully from the use of other remedies afterwards. It promotes absorption of fluids in the brain. He recommends the withdrawal of from eight to sixteen fluid ounces of blood from the patient - the amount to depend upon the size and vigour of the patient. The good results formerly got in cases in which patients were rapidly restored from coma to consciousness after copious bleeding were often only of a

Cupping over Loins

Advantages of Venesection.

temporary character, and the convulsions returned again with renewed vigour. Schroeder believes that venesection often helps to increase the convulsions. He says that if the theory of Traube & Rosenstein is correct, then the sudden diminution of the pressure by the depletion of the blood-vessels must stop the attacks. He asserts that although the quantity of blood removed is soon restored to the circulation, yet its quality is greatly deteriorated, and the blood is far more watery than before. The removal of blood would theoretically seem to cut short the convulsions at first. But since the blood-pressure, after a time resumes its former height, while its quality has been greatly deteriorated, then it follows that the danger of the disease will be much increased. While Venesection did much good in suitable cases, yet its reckless use greatly raised the death-rate from Eclampsia. Playfair (28) regards blood-letting as a valuable aid to treatment when wisely employed in properly selected cases. Its chief use is in lessening the severity of the first violence of the attack, and thus giving time for other remedies to be used later on. It gives special relief in marked congestion of the brain, and in cases of increased arterial tension shown by lividity of the face, full bounding pulse, and pulsation of the carotids. Its use is advocated in strong and healthy women, but it should be avoided in the feeble and weak. Study the constitution of each patient. It is only a temporary remedy

to stave off immediate danger to the brain, and must never be regarded as the principal part of the treatment. In most cases, the bleeding is sufficient. The amount of blood to be drawn must depend on the effect produced.

Chloroform. Chloroform is very effective in quickly lowering the arterial tension, soothing the nerve centres of the convulsions, and in subduing those causes which act reflexly. It will not accomplish these results unless the patient is fully under its influence; and hence its use is sometimes accompanied by danger, especially if it is given after Blood-letting. In the latter cases it has sometimes caused sudden death. The danger is less if it is inhaled in the early stage of the convulsions during the fits. The effect of this will be either to cut short the aura or to make it less severe. There is little risk of the chloroform accumulating in the blood, if the inhaler is removed when the fits cease, and in the intervals between the attacks. Spiegelberg (5) advises the combination of hypodermic injections of Morphia or of rectal enemata of Chloral with the Chloroform - between the fits. These keep up a continuous anaesthesia by intensifying the effects of the chloroform and prolonging its influence. I have seen excellent results from the adoption of this method in cases of Eclampsia in the Rotunda Hospital, Dublin. Even if given alone, Chloroform, Chloral, and Morphia decrease the number of the fits and lessen their intensity, but do not stop the pains.

Morphia or Chloral with Chloroform.

Narcotics should be given soon after the patient has been bled, if there are any more fits; or if there are no fits, they may anticipate their recurrence. Murphy[17] advocates the use of Chloroform followed up by hypodermic injections of Morphia or by Enemata of Chloral Hydrate, and insists on these being pushed until the patient is quite insensible - no regard being paid to the amount given, since the effect of a given dose varies in different cases. The Chloroform completely controls the convulsions when given during the fits. There is little danger to the mother from its use; but the child is liable to be injured by it. Lusk[21] restricts the use of Chloroform to the time of the pains and to the restlessness preceding fresh attacks; but he objects to its prolonged use in full anaesthetic doses (such as were formerly given) as in itself a source of danger. He first puts the patient under Chloroform and then keeps up its action by giving a rectal injection of 30 grains each of Chloral Hydrate and Bromide of Potassium stopping the Chloroform inhalations whenever the injection has begun to take effect. The frequency of repetition of the Chloral should depend on the recurrence and severity of the attacks. In one case, a single dose may check the fits for hours, while in other instances, the effect passes off in a very short time. Chloroform can be given at all stages of the disease, whether the patient is conscious or not. It always greatly decreases the frequency and violence

Chloroform Morphia or Chloroform pushed to insensibility.

Chloroform during pains, followed by Chloral & Bromide.

of the convulsions. Clearpentier[9] found that, in 48 cases out of 63, it either diminished or arrested the attacks, and that there was only one fatal case. Playfair gives the chloroform inhalations at the beginning of fresh attacks in order to restrain its intensity. He sometimes has cases in which chloroform is unable to arrest the convulsions or in which it produces extreme cyanosis in the patient and in these cases he gets good results from chloral.

Chloral Hydrate

Chloral Hydrate in the form of an enema of forty grains may be given in two ounces of starch per Rectum, and repeated in four or six hours if necessary. Lusk combines thirty grains of Chloral Hydrate with an equal quantity of Bromide of Potassium and gives it as an enema in starch after the patient has been put under chloroform. Playfair[28] has got good results from a combination of 20 grains of Chloral Hydrate with thirty grains of Bromide of Potassium—repeated at intervals of four or six hours as long as necessary. He has given the Chloral and Bromide either by the mouth, or in the form of an enema when the patient could not swallow. In some cases he has given a hypodermic injection of six grains of Chloral dissolved in sixty minims of water. The Chloral is very useful in cases in which the chloroform is insufficient, by itself, to arrest the paroxysms, or in which the patient becomes very much cyanosed. It requires less constant supervision than chloroform, and is more continuous in its action than

Chloral with Bromide of Potassium.

Advantages of Chloral Hydrate.

the latter. But Fordyce Barker (31) maintains that Chloral Hydrate excites reflex irritability instead of lessening it. The Chloral may be given in doses of thirty grains in the form of Syrup of Chloral by the mouth. Duchamp (10) gives it by the stomach pump in preference to enemata.

Morphia. Hypodermic injections of a quarter of a grain of Morphia repeated at intervals of half an hour until a total of one and a half grains have been given is another method of treatment. Dr. Smyly late Master of the Rotunda says that the Chloroform followed by Morphia. inhalations followed by the Morphia injections is the best treatment in these cases, both as ~~the~~ regards the mother and child. Morphia is less dangerous to the foetus than the Chloroform, but is more dangerous to the mother on account of the large doses which must be given to produce a decided effect. It is best to give small injections of a quarter or half a grain of Morphia, and assist its action by repeated inhalations of Chloroform when the fits come on - keeping the patient slightly under its influence. Good results have also been got by giving 3/8 grain Morphia after each fit until the fits stop*. In this way it has been given by White, up to a total of four grains in six hours. Lusk advocates injections of 1/6 to 1/4 grain - repeated in an hour if necessary. Playfair (28) uses 1/4 to 1/3 grain every three or four hours, so as to keep up such deep and continuous action as to entirely prevent the convulsions. He approves of the intermittent anaesthesia of Chloroform with the continuous action of other narcotics.

* Note:- In his latest statement on this subject (Jellet's "Practice of Midwifery") Dr. Smyly advises the use of Morphia in repeated doses without any Chloroform.

Hot bath followed by Hot Pack. The hot bath followed by a hot pack has been strongly advocated by Breus (27) as both a good prophylactic measure against Eclampsia in the kidney affections of pregnancy - and as a good method of treatment during the convulsions when used in association with the Chloral Hydrate. The patient should be immersed up to the neck in a bath beginning with a temperature of 102°F. which is to be gradually raised to 112°. After being in the bath for half an hour, the patient is to be dried thoroughly, then wrapped in a warm sheet, and put to bed. The bed is to be previously spread with two blankets, on the top of which is placed a warm sheet. After the bath, the blankets are to be rolled round the patient - the face only being left uncovered, and she is then covered with two more dry blankets. She is kept in the pack for two or three hours, and during this time profuse perspirations break out. The blankets are then removed, one by one, and she is allowed to cool down slowly. Thirst is relieved by Seltzer or Soda Water. After perspiring, the patient sleeps for several hours, and then awakes feeling much better. The perspiration removes the dropsical symptoms, and diminishes the amount of Albumen in the urine. Another method of causing perspiration is to place the patient in a vapour bath for an hour or longer.

Vapour bath

Diaphoretics. Diaphoretics to act vigorously on the skin have been used

by Porter.(13) These are mild measures, and are only of use in slight attacks. They are best adapted to the lying-in period and to the comatose stage of the illness. Their action can be kept up during the whole illness. The wet pack may be applied to the whole body at a temperature of 72° F.

Wet Pack

Pilocarpin

Pilocarpin is only safe when given at the beginning of the attack, before the onset of coma. If given during the stage of unconsciousness, it is liable to set up suffocation from the excessive flow of saliva, and the great increase in the bronchial secretion, thus favouring the development of oedema of the lungs. It has been given to diminish arterial tension by producing free perspiration and salivation. Braun(33) had successful cases from hypodermic injections of 3 centigrammes of Muriate of Pilocarpine. But Fordyce Barker(34) holds that its use is dangerous from the extreme depression it causes. However, Dr. T. Cooke Squance(20) records a successful case of treatment of Eclampsia by hypodermic injections of gr. ¼ Pilocarpine. After the first injection, the patient seemed almost suffocated, the respiration had a hissing sound - the movements of the larynx were "convulsive" in character. A large quantity of frothy mucus tinged with blood was expelled from the mouth. After three minutes the patient perspired freely - the convulsions stopped, and the breathing became natural. This attack was only half as long as the preceding, and she had only a slight convulsion the next morning. After two more injections, the fits

stopped, although she was unconscious for three days longer, and during that time she passed a large quantity of urine. In addition to the Pilocarpine injections, the patient had a mixture of Chloral Hydrate and Bromide of Potassium, and used vaginal pessaries of Eucalyptus and Perchloride of Mercury. She made a good recovery in about a week.

Purgatives. Purgatives in the form of hydragogue cathartics, by producing watery evacuations from the bowels, indirectly relieve the congestion of the kidneys. They also lower arterial tension, and remove any irritant matter from the intestinal canal. If the patient can swallow, you may give a dose of four or five grains of Calomel along with forty or sixty grains of Compound Jalap Powder or twenty grains of Compound Scammony powder by the mouth. If she is not able to swallow, then a drop of Croton Oil made into a pill with bread crumb, or a quarter of a grain of Elaterium may be placed on the back of the tongue. A Rectal injection of Turpentine, Castor Oil, and soft soap is very efficient in giving rapid relief to the Rectum and Colon.

Veratrum Viride. Veratrum Viride has been highly praised by Dr. Fearn (14) as a substitute for blood-letting in Eclampsia. He advised the use of 15 to 60 minims of the Tincture repeated at intervals of five to ten minutes until the pulse became soft and vomiting set in. Even after the convulsions were subdued, he urged that the drug should be continued

for several hours, in smaller doses, in order to keep the pulse-rate below fifty per minute. He said that the large doses did no harm while the convulsions continued. Kenyon (26) also, in his investigations, found that Veratrum entered the circulation rapidly and was soon absorbed. He found that by weakening the sensibility of the vaso-motor nerves, the bloodvessels lost their power of tonic contraction. These arguments are strongly in its favour, if the drug can be used with safety.

Bromide of Potassium

Bromide of Potassium, on account of its success in subduing the convulsions of infancy has been advocated by some authorities. Lusk and others give it in combination with Chloral Hydrate in various proportions. Fearn proposes to substitute it for Chloral or Morphia.

Blisters, Stimulants.

Blisters are liable to do harm by setting up convulsions of a reflex character. Stimulants are often given in the stage of Paralysis, but seldom do much good.

Diuretics

Diuretics such as Citrate of Potassium, and Preparations of Benzoin are chiefly of use in the convalescent stage to stimulate the secretions of the kidneys. Thirty grains of Citrate of Potassium may be given well diluted every four hours.

Transfusion of Blood

Transfusion of Blood has been used with success in cases in which both Venesection and the use of Narcotics have failed to give relief. Dr. W. Lange (18) records a case of Eclampsia in the early stage of labour, with oedema and albuminuria, in

which Venesection, Chloroform, morphia injections, and the application of cold to the head, all failed to give relief, and the patient fell into a deep stupor after thirty two fits. After seven ounces of defibrinated blood were injected by transfusion the respiration became freer, the pulse stronger, and the patient went into a quiet sleep, and afterwards recovered. Murphy (17) strongly advocates the adoption of Transfusion in those cases in which Narcotics and Venesection fail, on the ground that one of the chief causes of Eclampsia is the altered condition of the blood. He regards transfusion as both safe and efficient.

Cupping over the loins.

Moderate cupping over the kidneys has sometimes given good results in the hands of Dr. Murphy.

Nitrite of Amyl. Oxygen Strontium Lactate.

Inhalation of Nitrite of Amyl has occasionally been successful; and Schmidt of St. Petersburg (32) has had good results from the inhalation of Oxygen gas in some cases.

Strontium Lactate has been used successfully, along with other treatment, in one case reported by Ménager. It produced well marked diuresis, but did not much influence the convulsions.

Gustave Lang (12) holds that Syphilis is a predisposing and aggravating cause of Eclampsia. If this is the case, then its treatment would need to be borne in mind in the stage of convalescence.

Murphy had good results from a purge of Jalap followed by a mixture of Tincture of Steel & Sweet Spirits of Nitre in the early stage

Compression of the Carotids

Compression of the Carotids has been used by Playfair (28) with good results, as a temporary expedient during the convulsions (following the method of Trousseau for the convulsions of Infants). He pleads in its favour that it avoids the deterioration of the blood connected with venesection.

Induction of Premature Labour

The Induction of Premature Labour:-

1. The most severe forms of Eclampsia are those which occur for the first time during labour. Since labour makes the case worse than before, it is better not to induce premature labour in those cases which occur during pregnancy. If there are no labour pains, the convulsions may subside, and the pregnancy go on uninterrupted. On the other hand, if the fits are at all severe, labour will set in spontaneously without operative interference. Another reason against the induction of premature labour during pregnancy is that, gentle means of provoking labour pains are too slow in their effects; while those that act quickly would tend to make the fits worse by irritating the uterus. As long as the cervix uteri is undilated and remains rigid, forced delivery would both injure the parturient canal of the mother, and run the risk of sacrificing the life of the child. If labour pains are threatening to come on, try to stop them by opium or morphia. Lusk (21) objects to the common method of waiting for the onset of natural labour in cases during pregnancy, as he found it always disappointing - while he has had a number of

recoveries in cases in which premature labour was induced. In cases between the fourth and sixth months, Braun met with only one recovery among the cases in which spontaneous abortion did not take place.

2. In cases far advanced in pregnancy, in which the symptoms have so far advanced as to involve the Central Nervous System, the question arises as to whether or not we should induce premature labour. The most eminent authorities favour delay - on the ground that to interfere with pregnancy is such an extreme measure as only to be called for in cases of exceptional danger. Lusk,(21) on the contrary, holds that induction of premature labour is called for whenever grave cerebral symptoms develop themselves, because the good effects of cathartics and chloral soon pass off; and besides, their prolonged use would not be safe. Another reason for prompt interference is that the prolonged circulation of Urea in the mother's blood is liable to cause the death of the foetus. According to Lusk, there is little danger in induction of labour by the bougie, aided by the Vaginal douche and by Barnes' bags - provided that these are used after the Uraemic symptoms have been controlled. It is held to be justifiable as a last resort, after other methods of treatment have failed - although not at all likely to be successful in the latter condition.

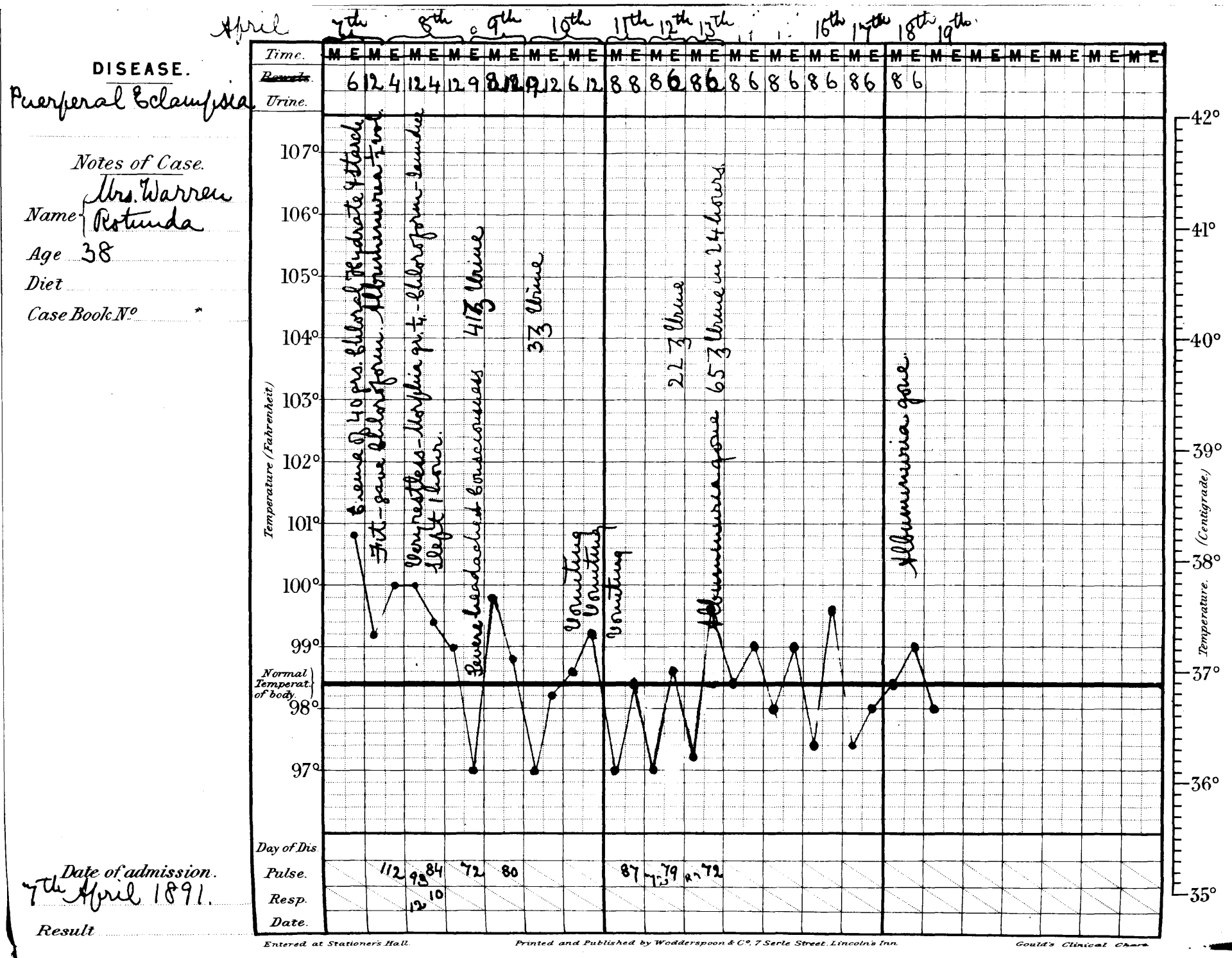
3. When labour has actually commenced, every means which would not interfere with the safety of the mother should be taken to

hasten delivery - because the convulsions tend to return again as long as the labour lasts, but subside after the birth of the child. In the beginning of the first stage, if the pains are feeble and slow the membranes should be ruptured and the amniotic fluid allowed to escape. This diminishes the frequency and violence of the convulsions. Barnes' bags may be used to dilate the cervix; or Tarnier's bags may be introduced into the lower segment of the Uterus. Spiegelberg objects to Vaginal douches on account of their difficulty in application. The effect of allowing the Amniotic fluid to escape is to lessen pressure on the Uterus, ureters and adjacent organs. Spiegelberg asserts that if the fits become worse before the cervix is fully dilated we would be justified in artificial dilatation of the cervix, and then the child could be delivered either by the forceps - if the cranium were presenting - or we could turn and bring down a foot. Forcible stretching of the cervix by the fingers of the hand formed into a cone is to be preferred to the making of radial incisions round the cervix, as the latter are liable to tear further back than is necessary. In order to avoid bringing on a fresh attack, the dilatation should be performed either in the stage of stupor, or soon after one of the fits. If the forceps are used, the os must be first fully dilated. Great care is required to avoid injury to the soft parts of the mother. In cases in which the head is high up in the pelvis, chloroform has not stood

results from the use of Tarnier's forceps - replacing them by Simpson's forceps when the head approaches the outlet. As the labour advances the danger of artificial induction of labour decreases. Gooch says:- "Attend to the convulsions and leave the labour to take care of itself." Schroeder also asserts that no kind of obstetric manipulation is necessary for the safety of the mother; but that sometimes the safety of the child necessitates the hastening of labour. Playfair (28) says that when the fits come on during labour, the pains are often strong and regular, and as long as the labour makes good progress, interference is not required. Although, in other cases, hastening of delivery might be of great benefit to the patient, the irritation of this interference might excite fresh attacks. This is illustrated by the recurrence of the paroxysms along with the pains. He thinks that if the os is not dilated and labour has not begun, we should avoid active induction of labour, but should rupture the membranes. Tyler Smith recommends the treatment least likely to irritate the mother. If the convulsions are caused by the pressure of the foetus, and the head is fairly low down in the pelvis, use the forceps; but if either using the forceps or turning would do harm to the mother by renewing the fits, we should not use them. Pelvic deformity and complications require suitable modifications to the nature of the special case.

If the labour is in the second stage and is making satisfactory progress at the time the fits come on, we should leave the case to nature. But at the end of the second stage, the forceps may be necessary, either in the interests of the mother or to save the life of the child.

The treatment of Eclampsia after Delivery :- If there are any clots left behind in the Uterus, they should speedily be removed. The chief treatment consists of Chloral, Opium, Digitalis, or Veratrum Viride - also purgatives and diaphoretics Venesection and Chloroform should be used with great care if ever employed at all.



Date of admission.
7th April 1891.
Result

Notes of Illustrative Cases of Puerperal Eclampsia.

Case I. (See Temperature Chart) Mrs. W. aet. 38, was admitted into the Rotunda Hospital on 7th April 1891 at 3.45 p.m. in the eighth month of her fourth pregnancy. Her family history was good. She had Typhus fever fourteen years ago, and Scarlet fever when a child.

State of health during present pregnancy:- She had vomiting twice daily during the whole of her pregnancy - but it became much worse during the three weeks preceding admission - so much so that latterly she could retain nothing on her stomach. For the past three months her ankles and legs have been swollen. During the past four months she has been slightly deaf. She has had twitchings in the limbs for two months. Her urine was scanty, and high coloured for several days before admission.

Present Illness:- Began on 4th April with twitchings of the muscles in the limbs. On the 5th, she had frontal headache, dimness of vision, and severe twitchings. On rising from bed on the 6th, she had severe headache, dimness of sight, and partial deafness. She had two fits on the morning of the 6th. Dr Ufford and I visited the patient in the afternoon. We found her in a dazed condition, and just recovering from a fit. On examination, she was found to be 8 months pregnant - and the child was alive. The patient

was quite robust. During the examination she had a fit with continuous vomiting. The pupils were dilated - the respirations normal - the pulse full, and about 76 - the ankles and eyelids were slightly oedematous.
Character of the Fit :- The patient uttered no cry. At first a thrill seemed to pass through her frame, and she had a slight clonic spasm or fibrillary twitching of all the voluntary muscles - of momentary duration before the fit actually started. She was very restless and tossed about. The fit itself was characterised by a rigidity of all the voluntary muscles - a tonic spasm - eyes open - tongue protruded - mouth pulled to one side - breathing arrested - pupils dilated - great lividity - mouth firmly closed - exudation of froth from the mouth - no urine passed involuntarily. This lasted for about 8 seconds, and then she had a clonic spasm.
Clonic Stage :- All the voluntary muscles were in a state of clonic spasm - breathing became natural again - lividity disappeared - the tongue was bitten - this lasted for about 50 seconds. The arms remained flexed at the elbow for some time after the fit was over. After the fit was over the patient remained quite unconscious - consciousness gradually returning, and with it severe vomiting. The patient had three of these fits before she was admitted into the Hospital, and nine during

the next 18 hours. The patient was admitted to the Hospital at 3-45 pm on 7th April. She had fits at 3-45 pm 4-15, 4-30, and these were controlled by Chloroform inhalations. At 4.30 pm she got a purge consisting of 4 grains of Calomel and 60 grains of Compound Jalap Powder - and she was then put into a Vapour bath for 1½ hours. At 6 pm her temperature was 100.8°, and she had another fit for which she had an enema of 40 grains of Chloral Hydrate and 3ss of Mucilage of Starch At 7-10 pm she had another fit which was controlled by a hypodermic injection of Morphia (gr ⅛) - after which the patient slept till 11-50 pm The next fits came on 8th April at 12-15 am and 1-25 am, and Chloroform was again administered. At 2 a.m. the child was born dead - enveloped in the membranes - the patient being quite unconscious of it. At 2-35 am ~~the~~ the patient was very restless, and gr ¼ Morphia was injected hypodermically. At 5-35 am and 7-50 am she had more fits which were controlled by Chloroform. She had another fit at 8-5 a.m. At 10 am as the patient continued restless, she had another enema of 40 grains of Chloral Hydrate in Starch, and also Chloroform inhalation - after which she slept quietly till 12 noon. Becoming very restless at that hour, she had an injection of gr ¼ of Morphia - the temperature was 100° -

the eyelids were oedematous - and there was slight jaundice (probably due to Chloroform). From noon till 4-15 pm she was very restless - after which she had broken sleep until 6 pm (about an hour at a time). At 8-55 pm she was restless for 15 minutes, after which she slept well. She had 6 oz. of Milk and Barley Water every 3 hours, and 30 grains Citrate of Potash well diluted every 3 hours. On April 8th at 10 pm ℥XXI Urine was drawn off by the Catheter. When tested for Albumen, the urine showed the presence of Albumen to the extent of half its volume

On April 9th the patient was unconscious, though somewhat dazed, most of the day from 9-15 am till 8-20 pm She vomited several times during the day - and the bowels acted twice

On April 10th she had vomiting at 3 am - 11.30 am and 6 pm. She had slept for 5 hours at intervals during the preceding night. The bowels moved at 3-30 pm

On April 11th there was vomiting at 12-30 am, 8.30 am and 6 pm. She slept fairly well during the preceding night. At 12-30 pm she had ℥V of Beef Tea The bowels acted at 9-30 am and she passed urine at 3-15 pm

On April 13th she continued to improve. On 14th April the Urine passed in 24 hours amounted to ℥LXV, and it was entirely free from albumen. The amount of Urea was not estimated. The patient left the Hospital quite better.

This case may be regarded as a fairly typical case of Eclampsia

QUEEN VICTORIA'S JUBILEE INSTITUTE FOR NURSES.

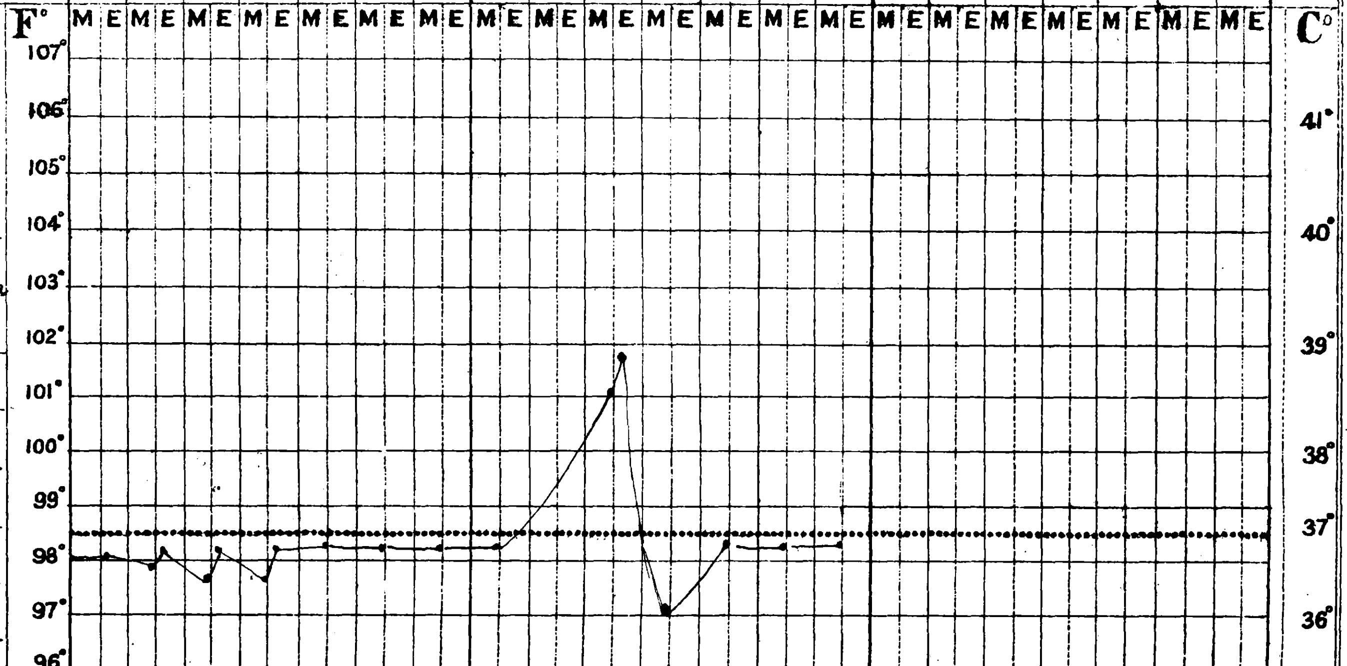
No.

MONTH	April			May										
DAY OF MONTH	28	29	30	1st	2nd	3rd	4th	5th	6th	7	8th	9	10	11
DAY OF DISEASE														

NAME: Alice Newman

AGE: ~~[illegible]~~ 40

DISEASE: Puerperal Eclampsia



		28	29	30	1st	2nd	3rd	4th	5th	6th	7	8th	9	10	11
PULSE	M		42	76	86	80					100				
	E	84	58	84							100				
RESPIRATION	M														
	E														
VOMIT															
AMOUNT OF URINE															
MOTIONS		2	1	0	0	0	1	1	0	0	0	1	2	1	1

MEMORANDA

Case 2. (See Temp. Chart) Mrs. A.N., Dublin, 40 years of age - was delivered of her second child at term at 2 a.m. on 28th April 1891, before the arrival of the doctor in charge of her case. I was called to see her in consultation (when Clinical Assistant pro tem. at the Rotunda Hospital), and found her in a state of unconscious stupor following a fit of Eclampsia, of which she had had several during the preceding night. She had never been subject to fits of any kind before her confinement - and seems always to have enjoyed fairly good health.

1. Her first fit began at 9 p.m. on 27th April, and lasted half an hour. During that time she bit her lips - violently contracted her fingers into the palms of the hands - had loud snoring respirations - talked incoherently - showed no signs of violence. After the fit she became quite unconscious, and then fell into a dazed condition in which she remained until 2 a.m. on the 28th.

2. At 2 a.m. on the 28th she had a second fit, during the end of the second stage of labour when the child was being born. The fit lasted half an hour and then passed off - but she remained unconscious for some time afterwards. The symptoms were much the same as during the first fit, except that the snoring was more marked. The neck and trunk were violently bent back, and the face became very purple.

3. A third fit of much the same character came on at 6-45 a.m. on the 28th and lasted for 10 minutes. She then

got into a condition of stupor in which she remained until 9-30 a.m.

4. At 9-45 a.m she had a fourth fit lasting 15 minutes. During this time she clenched her teeth firmly together - had loud stertorous breathing - and remained unconscious for some time afterwards. The pulse was 84 and of fair tension.

I ordered the following treatment:- During the fits, a cork or guarded spoon to be placed between the teeth - and after the fits: Ammoniae Aromaticus to be applied to the Nostrils & lips. Between the fits:- ℞ Pulv: Jalapae Co grs XXX Hydrarg: Subchlor: grs IV Mitte tales ij. Sig. Take one powder at once, and the other at 8 p.m. if necessary. (1st pulvis)

℞ Chloral Hydratis ℥iij
Potass: Bromidi ℥iv
Syr: Tolutani ℥i
Aquae Mentth: Pip: ad ℥vi (1st)
Sig. Take two tablespoonfuls at once - and then one tablespoonful after each fit - in half a cupful of water
Diet:- Milk - gruel - beef-tea

28th April 7.45 p.m:- The patient regained consciousness about 2.30 p.m - one hour after receiving the purge, and the sedative mixture. The bowels acted freely. There was no return of the fits. I ordered the second Jalap & Calomel powder to be taken at 8 p.m - also the Chloral & Bromide Mixture if the fits recurred. Diet of Beef Tea and Milk. The Temperature was 98° F. and the Pulse 84.

29th April 1 p.m :- Patient quite conscious. No return of the fits. No oedema of feet or legs. Urine abundant, with copious deposit of Urates, and a slight trace of Albumen. I got two trained nurses to attend the patient from the Queen Victoria

Institute. As the lochia had a foul odour, I gave a Vaginal douche with antiseptic precautions. Temp. 98°. Pulse 56. I drew off 40 oz. urine with the catheter. Beef Tea and Milk diet were ordered, and a Mixture of Nitrate of Potash Glycerine & Water every four hours.

30th April 1/pm :- Pulse 72 - rather rigid and incompressible. Temp. 97.6° Slept well during the night. No return of the fits. For the constipation I ordered a powder of 4 grs. of Calomel with 40 grains of Compound Jalap Powder.

1st May, 1.30/pm :- Pulse 60, good tension. Temp. 97.4° Sleepless during night.

2nd May 5/pm :- Pulse 72, good tension. Temp. 98.2° Bowels still constipated. Patient feels much stronger. I ordered a Purgative powder of Calomel & Compound Jalap powder - also a Mixture of Nitrate of Potash (grs 30 doses) thrice daily - and a Mixture of Digitalis & Aromatic Spirit of Ammonia to be taken thrice daily.

3rd May 5/pm :- Pulse 72 very good tension. Patient slept better.

4th May 1/pm :- Pulse 92 good tension. Temp. 98.2° F. Patient gaining strength. Ordered Castor Oil & Essence of Peppermint for constipation.

5th May 5.30/pm :- Patient feeling much better.

7th May 12.40/pm :- Pulse 96. Temp. 102° F. Patient complains of headache, weakness, and slight abdominal pain. Lochia slightly fœtid - slight redness of skin over buttocks. I douched out the Vagina. I ordered Beef Tea & Milk diet - also grs. IV doses of Quinine every four hours, and a Mixture of ℞ Digitalis, Aromatic Spirits of Ammonia and Infusion of Calumba.

8th May, 1·35 p.m. :- Temp. 97·2°, Pulse 84 with good tension. Abdominal pain gone - headache better - lochia free, and with no foul odour. Bowels regular. Patient feels stronger.
10th May 3·30 p.m. :- Pulse 84 - tension good. Temp. 97.6° F. Patient stronger.
28th May :- Patient has quite resumed her usual health. Last visit.

Case 3. Case 3. Mrs. E. R. Dublin, 26 years of age - was delivered of a female stillborn child in the 8th Month of her 6th Pregnancy on 6th July 1891 at 7·40 a.m. The presentation was 1st Vertex. The second stage of labour lasted only about half an hour. In her 5th pregnancy she had an abortion at the 3rd Month.
5th July, 5·30 p.m. :- The patient had an Eclamptic fit lasting half an hour. The muscles of the hands, limbs, and face were alternately contracted and relaxed, and exhibited twitching movements the whole time. The face was flushed - the teeth were clenched, but she did not bite her tongue. She screamed out, as if in great agony. Her respirations were not noisy. She complained of severe headache.
6th July 1 a.m. :- She had a second fit similar to the first. The pupils were dilated. The head was very heated, and she had frontal headache. Her extremities were cold. She had violent twitchings of the muscles. There was severe pain over the loins. No history of Epilepsy or of Apoplexy.
3·30 a.m. :- Ordered Purgative powder of Calomel & Jalap.
3·45 a.m. :- I gave grs 40 Chloral Hydrate in Syrup form.

4 a.m:- Had Haemorrhage of about ℥xv Blood and clots, Abdominal palpation showed a first Vertex presentation. No foetal heart could be heard.

4-15 a.m.:- I gave an enema of hot water, soap & turpentine for the bowels.

4.30 a.m:- I made a P. V. examination, and found the Os dilated to the size of half a crown, with very irregular, ragged and bleeding overlapping edge which seemed to be due to Placenta Praevia. The membranes had not ruptured, and were very tense. The bones of the head appeared to be very loosely connected together, as if the child were dead. No movements of the child were observed by the Mother since 4th July.

5-25 a.m:- The patient had a third fit which lasted 15 minutes. She had very slight tonic convulsions, followed by clonic convulsions.

6-15 a.m:- I gave a hypodermic injection of Morphia Sulph. gr ¼ with Atrop: Sulph: gr $\frac{1}{200}$; and repeated the dose at 6-45 a.m.

6-0 a.m. I ruptured the membranes, and let the head descend

7.40 a.m:- Child born dead in L.O.A. position of the Vertex.

8 a.m:- I gave a Vaginal & Intra-uterine douche to remove clots.

8.35 a.m:- Uterus well contracted. Pulse 84. Temp. 98.2

3-45 p.m:- No return of the fits. Pulse 68. Temp. 97.2. Ordered Tablespoonful doses of Brandy in water every four hours.—and diet of Milk and Beef Tea. The patient then progressed very favourably till the morning of 9th July.

9th July 1.5 a.m.:- Patient sent for me on account of a severe pain in the Hypogastrium, and in the right & left Lons- especially in the latter. She had a Rigor on 8th July at 12 midnight. Temp. 99.8° Pulse 92. Lochia scanty and foetid. No milk in the breasts. Bowels well moved at 5 p.m. on the 8th. Micturition normal. No chest symptoms or physical signs. I douched out the Vagina - ordered cold compresses for the hypogastrium, and gave m XX Laudanum.
1 p.m. Pulse 105. Temp 99.5° F. Abdominal pain gone. Lochia free and healthy.
11th July 1 p.m.:- Pulse 85. Temp 98.3° Patient feeling well
12th July 9 p.m.:- Pulse 85. Temp 99.2 Vaginal douche. Headache
15th July 12 noon:- Temp 98.6. Headache gone. Much stronger Last visit

Case 4. Case 4. Mrs. I Govan - 24 years of age primipara - was delivered of a still born male child weighing 8½ lbs on 5th Oct. 1899 at 11.45 am Eclamptic convulsions began at 4 a.m. on the 5th simultaneously with the onset of labour pains, and they continued at frequent intervals until the birth of the child at 11.45 a.m. As the convulsions were very severe in character and came on with very great frequency I sent for the assistance of a chloroformist to put her fully under the influence of Chloroform. I then dilated the cervix fully by means of the fingers put into the form of a cone, and afterwards applied the midwifery forceps and delivered the patient with a good deal of difficulty. The presentation was Vertex.

The convulsions were checked by the Chloroform at intervals until after the birth of the child. She was then put on a mixture of Citrate of Potash, Acetate of Potash, Bromide of Potash etc every four hours as a diuretic and sedative. Poultices were ordered over the loins to help the action of the kidneys - as the amount of Urine was scanty - and large quantities of Barley water and Solution of Cream of Tartar & Lemon were ordered - as well as milk. The perinaeum was lacerated, and required to have several sutures inserted into it after the Vagina was douched. The patient died on 7th Oct. at 8.30 a.m - the immediate cause of death being suppression of Urine and extreme weakness following the convulsions. No P.M. Exam. was obtained.

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