ALBUMINURIA in PREGNANCY

with special reference to

PUERPERAL ECLAMPSIA,

with Cases.

THESIS for M.D.

by

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The occurrence of albumen in the urine of a pregnant woman has been considered by some writers on the subject as a trivial condition. The late Prof. Leishman, was of the opinion that the albuminuria of Pregnancy is "comparatively innocuous", ("A System of Midwifery", p.265).

Albuminuria in Pregnancy may be considered by some writers as a slight matter, but it cannot be so easily thrown aside without due consideration, and should always be regarded with feelings of anxiety.

No doubt, a large proportion of the pregnancies in which albuminuria is present, come to a happy termination, and it is fortunate that such is the case. There are cases, however, which do not attain a complete and successful issue, if so, recovery may result only after months of suffering and weakness. Such cases make the subject of Puerperal Albuminuria, of more than passing interest, and certainly of some importance.

Statistics vary greatly as to the frequency of albuminuria in pregnancy. In my obstetric practice, 2½% of the cases had albuminuria; others have found a much larger proportion. ("Science & Practice of Midwifery," Playfair, p.239, Vol.1.) Galabin in his "Manual of Midwifery" makes the following statement:-

"Out/

"Out of 200 cases inthe Guy's Hospital Charity in which the urine was tested, about the time of labour, albumen was found only in four, and two of these appeared to be of Chronic Bright's disease."

Allowing for the cases of Chronic Bright's disease Galabin's percentage is one. Albuminuria appears more frequently in primiparae than in multiparae.

Shroeder states that the urine of all pregnant women contains from 3% - 5% of albumen. A purely Physiological Albuminuria may be found in a great number of cases of pregnancy, but in these the Nephritic symptoms are absent, there being no tube casts. The presence of albumen in the urine during pregnancy is said to be very common during labour.

Five per cent is given as the frequency, with which albuminuria is met in primiparae. This frequency of albuminuria in primiparae would seem to throw light on the cause of the condition. This question will be considered further.

CAUSES:- 1. Mechanical Pressure of Gravid Uterus.-

That albuminuria in Pregnancy occurs late in the course of that condition; that it is the rule in twin pregnancies, and that it is more common in primiparae than multiparae, seems to give weight to the theory of mechanical pressure of the/

the gravid uterus as being the cause of puerperal albuminuria.

I have seen cases of Puerperal Albuminuria in multiparae, where owing to the pendulous condition of the abdomen the pressure of the gravid uterus on the renal veins could not be great, if any, and in neither of these cases, was there any history of a previous nephritis.

Graily Hewitt, in the London Obstet. Socy. Trans. Vol. XXVI, upholds the theory of mechanical pressure of the gravid uterus as the cause of Puerperal Albuminuria; so also does Halbertsma of Utrecht. Now if the ureters were compressed by the gravid uterus for 4 months, there would almost certainly be seen post-mortem, dilatation of these tubes, but according to Clifford Allbutt this is not the case. 'Lancet' 27 Feby. 1897.

Coats in his "Manual of Pathology" states that sudden relief of the obstruction of the ureter has been followed by an excessive secretion of urine containing albumen.

Halbertsma's theory is opposed by Winckel, who states that in cases of women dying in pregnancy he found the ureters greatly distended, and the women had not shewn any tendency to eclampsia.

Barbour states that "there is only one point on the Ischium where the ureters may be compressed and that the chances of compression are small, we must look elsewhere, therefore, than to a mechanical cause for the explanation of albuminuria."

Dr. Allbutt in the "British Medical Journal" of 27th. Feby. 1897, shews how the Theory of Mechanical pressure on the renal veins fails to account for the albuminuria. He states that swelling of the legs and other such signs of venous obstruction get more and more as the pregnancies increase in number, whereas albuminuria and eclampsia are evils of the primipara. With regard to the dilatation of the ureters as a proof of mechanical pressure on them as a complication of pregnancy, Allbutt in the same article, distinctly states, that only in rare cases does it happen.

Moreover, a great amount of Mechanical pressure is often exercised on the renal organs by abdominal tumours and yet there is no albuminuria.

The evidence in favour of the view that albuminuria in pregnancy is the result of renal congestion due to direct mechanical pressure of the gravid uterus is very conflicting and unsatisfactory, so much so that this theory is now given up.

Another theory is that Puerperal Albuminuria is due to the indirect compression caused by increased intra-abdominal pressure with subsequent stasis of the renal veins and resulting albuminuria. Here again there are fatal objections Albuminuria is sometimes found early in the course of a pregnancy, and this theory does not account for such a case.

In primiparae, this indirect compression would be greater/

greater than in multiparae in whom we sometimes have albuminuria. In cases of ovarian tumour albuminuria is usually absent, except when the pressure of an ovarian tumour exceeds that of the gravid uterus at full term. The theory of mechanical pressure certainly has something in its favouf, it may predispose to the disease, but that is quite a different thing from being the exciting cause. In cases of twin pregnancy, the albuminuria, when present, is greater than in cases where there is only a single child.

Defects in the cardiac valves, and pulmonary emphysema, in a case of pregnancy, would probably cause albuminuria, the result of inflammation of the parenchyma of the kidney. A woman suffering from Nephritis may become pregnant, but the albuminuria is not puerperal. The albuminuria is present in these cases prior to the pregnancy.

Another theory is that the kidneys are unable to bear the increased work, caused by the excretion of the waste products of the foetus and enlarged uterus. If this were the case, then multipara would be more liable the morbid condition, than primiparae, as each succeeding pregnancy would render the kidney less liable to exercise its excretory function.

There is also the theory of "reflex irritation." Here the explanation is that the irritation arising from the uterus/

uterus affects the renal organisms to such an extent, that albuminuria results. Now, "reflex irritation" may exist at an early period in the course of a pregnancy and so, albuminuria would be present also, according to this theory. Such is, however, not often the case.

Tyler Smith & Spiegelberg support this theory, stating that owing to the reflex irritation, resulting from the uterine contractions, causes a renal anaemia and subsequent albuminuria. My own opinion is that renal congestion results from the reflex irritation producing a venous stasis and albuminuria.

No doubt, albuminuria in pregnancy may be important clinically, but perhaps this importance is due to the fact, that in Acute Nephritis the prognosis is influenced greatly by the amount of the albumen in the urine.

There still remain two theories explaining, or partly so, the albuminuria of pregnancy.

The first of these two theories is that there is present in the blood of a pregnant woman, a particular form of micro-coccus. It is difficult to understand how anything of the nature of a micro-organism could enter the blood, unless there was some concurrent disease, in which micro-organisms might enter the system and cause untoward mischief.

The/

The theory which to my mind seems most plausible is, that there is circulating in the blood of a pregnant woman toxic material which causes the nephritis and eclampsia.

The presence of a placentitis might give rise to toxic material, but that such a disease exists is denied by many authorities. The poison seems to be an alkaloid capable of producing certain nervous symptoms, coupled with the presence of albuminuria.

We must not forget that vomiting is one of the earliest signs of pregnancy. Now, in many infectious diseases, the onset is characterised, amongst other symptoms by headache, nausea and vomiting. A case of Scarlet Fever, may up to a certain point proceed favourably. Suddenly, it may be, the patient complains of headache, accompanied with vomiting. The urine becomes more scanty than before and of a higher colour. On testing the urine, albumen is found. What is the conclusion we are forced to? It is that there is circulating in the blood some poison which causes the symptoms referred to.

In the kidney of Scarlet Fever, changes are to be detected in the Glomerules, the epithelium of which becomes enlarged and abundant, so much so, that considerable pressure is exercised on the Glomerule tuft. There are also haemorrhages into the convoluted tubules and sometimes the Glomerules themselves are found filled with blood.

Glomerulo-Nephritis is very common in the first week of Scarlet/

Scarlet Fever, so frequent is it, that the only permissible explanation is, that there is some poison present which affects the kidney, at the same time, it must be remembered that micrococci have been found in the Scarlet Fever kidney, gaining an entrance by the throat and setting up a septic Nephritis.

In Pregnancy, the kidney may shew any condition from congestion on the one hand, to anaemia on the other, and this, even when albuminuria is present. As a rule, there is during pregnancy a certain amount of renal congestion and hypertrophy. These conditions, apart from degenerative processes seen in Nephritis, have given rise to the term "Kidney of Pregnancy." It is an extremely different matter to draw the line between the Kidney of Pregnancy and commencing Nephritis. The presence of epithelial, granular or fatty casts however is decided proof.

In the kidney of Puerperal Albuminuria we have degenerative processes in addition to the congestion. The tubules are blocked with debris. In cardiac kidney, there is little degeneration and inflammation, the capsule is easily stripped and there is hyperaemia. Now, the changes in the kidney of Puerperal Albuminuria are more pronounced than in the kidney of pregnancy or of the Cardiac Kidney.

In considering the subject of Puerperal Albuminuria, it must/

must be remembered that there may be albumen in the urine of a pregnant woman, who is suffering from a catarrhal affection of the Urinary bladder or a vaginal secretion . In such cases the differential diagnosis must be made by means of the catheter. The microscopical examination of the urine, in all cases of albuminuria should be performed; the presence of renal epithelium and tube casts, at once shewing the renal origin. Of course, if the cystitis had passed up the ureters, and caused a Nephritis, then the diagnosis would require great care, as the epithelium of the bladder might be introduced into the ureter, by the catheter. The presence of the tube casts and renal epithelium or both, would, however, settle the diagnosis.

PROGNOSIS.-

The cause of Puerperal Albuminuria is the true guide to the prognosis. When due to a vaginal secretion or catarrh of the bladder, the prognosis is good, and in many cases where the albuminuria is associated with Nephritis or even Eclampsia, the prognosis is fairly good, though one must be guarded in pronouncing on the case.

In cases where there is albuminuric retinitis and blindness, the prognosis is not at all bright as the conditions would indicate an old standing Nephritis.

We must, however, remember that haemorrhage may cause

temporary blindness, which in such cases ensues after delivery, and therefore may have no connection with the retinitis.

The amount of urea excreted by the kidneys during Puerperal Albuminuria is said to be lessened, but the relation between the two does not correspond. There may be much albuminuria and little uraemia or vice-versa.

In any case, the persistent presence of albuminuria, in large quantity during pregnancy, must always be regarded with anxiety.

SYMPTOMS:-

Very often Puerperal Albuminuria may exist for a time, without there being any marked symptoms. The patient may feel little wrong, and yet the albuminuria may be present. Attention is generally drawn to the existence of a slight swelling in the ankles, increasing as night approaches, while in the morning that may be absent or at any rate greatly lessened. On rising after a night's rest, the eyelids are swollen. As time goes on, the swelling of the ankles gradually increases, until in well-marked cases, the whole body may become oedematous.

When a pregnant woman feels out of sorts, and nothing can be given as the cause, and if this condition exists in the latter half of a pregnancy, then the urine should be examined/

examined for albumen, and urea. It is not sufficient to test the urine from the bladder; the catheter should be passed into the ureter and the urine thus obtained, examined.

The urine insuch a case is scanty, concentrated, of high specific gravity, and when boiled or even heated in hot water may become solid, and perhaps contains renal epithelium and casts. There is often headache, giddiness and visual disturbances, also nausea and vomiting. In many cases there is mental confusion, e.g. inability to recollect the day of the week. Insomnia is often pronounced and such patients often shew great irritability of temper.

Very often the foregoing phenomena may be absent, or present in such a slight degree, as to attract little, if any, attention, and the first thing which shows itself may be Eclampsia.

Paralysis is apt to occur, the most common form being paraplegia. There may also be haemorrhage and inflammation of the retinae, causing blindness, while the auditory nerves may be involved resulting in deafness.

In cases where there is Chronic Nephritis, there is, very often, abortion or premature labour.

In one of my cases, there was abortion in two pregnancies one following the other, and also albuminuria with retinitis. In the interval between the two pregnancies the albuminuria and eye affections disappeared.

TREATMENT:-

The treatment of Puerperal Albuminuria depends wholly on the cause. If it is due to fever cachexia, Cardiac disease, then we must if possible, remove the cause or at least moderate its action.

If the albuminuria is slight, and the microscope reveals nothing in the way of tube casts, or if there is absence of oedema, the case will likely have a happy termination, when the pregnancy is completed and the mother able for household duties. In such a slight case, great care should be taken to prevent the exposure of the mother to cold, for even a slight albuminuria may be the danger signal to serious damaging of the kidneys, if there is the slightest exposure to cold.

The action of the kidneys, in a case of Puerperal Albuminuria is generally sluggish. The urine is scanty, highly coloured, albuminous, and of high specific gravity. Another thing in connection with the urine, I have noticed, is its distinctly "fishy" odour, which, in one case was very pronounced. Micturition is often painful.

The action of the kidneys, not being what it ought to be, gives us a clue to the treatment. We must endeavour to get the kidneys into quicker action, otherwise they will become blocked with renal debris. For this end, the kidneys should be flushed, by means of diuretics or agents, which

will/

will remove as far as possible any irritant acting on the kidneys. Of all the diuretics employed, water is the best, having no irritating action on the kidneys. The patient should be encouraged to drink plenty of water. One is surprised to find out, how little water some females drink.

Skim-milk and also butter-milk are good diuretics, Salines, e.g. Salts of Potash or Lithia may be given:-

R. . Tr. Hyoscyami, ℥vi
Liq. Potassae ℥ss
Ext. Pareirae Liq. ℥ii
aq. ad ℥viii
ft

Liq. A dessertspoonful every 3 hours in a little water.

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or R.. Lithii Carb. grs. xxiv
Ammon. Carb. grs. 40
Tr. Chiratae ℥iv
Spt. Chloroformi ℥ii
aq. ad ℥viii
ft

Liq:: Tablespoonful thrice a day before meals.

If the urine has a foul odour, I know of no drug better suited to counteract this condition than Boracic Acid.

R. Ac. Boracis Grs. XV
Tales VI.

Liq: One in a tablespoonful of milk twice a day.

In a short time after the commencement of the Boracic Acid treatment, the urine becomes clear, bland and odourless. Whilst acting on the kidneys, we must not forget that the skin and bowels are channels through which we can benefit the patient. Turkish baths may be given at intervals; but great caution must be exercised in the prevention of subsequent chill. Diaphoretics are of great benefit especially if accompanied by the use of hot water bottles around the patient, who must be well covered with blankets.

The following mixture is often useful as a diaphoretic:

R. Potas. Citratis ℥ss
Spt. Etheris Nitr. ℥iv
Spt. Etheris Nitr ℥iv
Syr. Aurantii
Aq. ad. ℥vi

Liq: A teaspoonful every 3 hours.

Liq. Glonini has been given; but the unpleasant symptoms which it produces are against it, though tolerance of the drug can be established, and little discomfort felt, evenafter taking a dose of 4 minims.

The use of Pilocarpine and Jaborandi have been lauded by some, but owing to the depression which is apt to ensue, they have been put aside by many authorities. In only one case, have I employed Pilocarpine which acted well, producing no depression. The bowels should be freely acted upon and thus any drug, which is distinctly cholagogue in effect,

stimulating the cellular activity of the liver and peristalsis of the bowel, is of great benefit, in that, the metabolic function of the liver is increased, and the kidneys are relieved of much unnecessary labour.

R. Ext. Casc. Sag. Liq. ℥ss
Tr. Hyoscyami ℥ss
Ac. Nitro-Mur. Dil. ℥j
Aq. ad. ℥viii

Liq: Two teaspoonfuls night and morning.

I found the above combination of great service, in cases of sluggish liver and combined with Hyoscyamus, the Cascara does not cause intestinal pain which may sometimes be mistaken for labour pains.

DIET:-

The diet should be as simple as possible, and one which will give the kidneys the least possible amount of labour. Milk fulfils all the requirements necessary.

Tarnier gives the following menu for an albuminuric patient:-

1st. day - 1 litre of milk and two meals.

2nd. day - 2 litres of milk and one meal.

3rd. day - 3 litres of milk and half a meal.

4th. day - 4 litres of milk or as much milk as patient can/

can take, and no other food or drink after.

One objection to the milk diet is that patients soon tire of it, and constipation is very apt to ensue.

Riviere states that milk leaves in the bowel the least possible residue for intestinal putrefaction, such putrefaction of ordinary food forming new toxic principles, which entering the blood are important in regard to Eclampsia.

Galabin states that advantage is gained by giving a dietary which causes the kidney as little work as possible in the excretion of nitrogenous material.

"This indication is best fulfilled by a diet consisting of milk and starchy material, such as corn flour, sago etc., alone. In chronic cases, according to the modern view, it is better not to restrict the diet too much, but to give a fair amount of meat. It seems desirable, however, to be sparing in the use of beef tea or meat extracts."

In one of my cases, I carried out this modified treatment, and found it to do well. The patient does not complain of the sameness in the diet and thus does not tire so soon of it, as a purely milk diet.

Tonics are beneficial, especially those containing Iron in some form.

R. Tr. Ferri perchlor 3ij.
Tr. Strophanthi 3ij.
Glycerine ad. 3iv

Liq: A teaspoonful thrice a day.

The objection to all Ferruginous tonics is the more or less pronounced constipation, which may ensue during their administration. When combined with Glycerine, this constipating action is reduced to a minimum. If the albuminuria occurs late in a pregnancy especially in a primipara, who complains of headache, disturbances of eyesight and scanty albuminous urine, we should always be on the look out for convulsions.

Bromide of Potassium may be given with the view to prophylaxis.

Chloral Hydrate is praised and is useful, if the premonitory symptoms are pronounced.

INDUCTION of PREMATURE LABOUR.-

This is a much debated subject. The late Prof. Leishman in his "System of Midwifery," vol. I, page 269, states, "The significance of albuminuria during pregnancy has been viewed by some as of such serious import, as to warrant the induction of premature labour; but to such an opinion, in so far as ordinary cases are concerned, we are unable to subscribe. So serious, however, is the probable issue of a case in which the quantity of albumen, the degree of oedema and the general condition of the patient tend to indicate the highest grade of severity, in symptoms, that we may be quite justified in entertaining gravely the propriety of such a course."

Pajot is against the induction of labour, and abortion, considering such an action more dangerous than Eclampsia.

Tarnier states that if the milk treatment is begun early and faithfully carried out, eclampsia does not take place. Winckel is against abortion or induction of labour in connection with puerperal albuminuria or Eclampsia. The interruption of pregnancy in cases where suitable treatment has failed and where Eclampsia or death is inevitable is favoured by Auvard.

Galabin favours the operation in cases where there is much oedema at the close of a pregnancy or where the albumen is persistent in spite of treatment, especially is this the case in primiparae, the child being viable. His reason being that if labour is induced, the patient has a better chance of escaping Eclampsia than if the renal condition were allowed to go on from bad to worse.

In the "American Journal of Obstetrics" (1889) Partridge states that in all cases where there is reasonable probability that Chronic nephritis exists, no matter how slight the degree, pregnancy should be interrupted, just as early as gestation is known to exist."

Spiegelberg is opposed to it, while Hofmeier on the other hand favours such an operation.

Barker (American Journal of Obstet. 1878, vol. XI, p.449) thinks that induction of labour should only be resorted to,

when treatment has been thoroughly and perseveringly tried without success, for the removal of symptoms of so grave a character, that their continuance would result indeath.

CASES:-

On 13th. August 1895, when acting as locum tenens for Dr. Syme, Kilmalcolm, I was called at 8 a.m. to visit Mrs. H. Dr. S. had previously talked to me about the case, so that I was prepared for it.

On arriving at my patient's bedside, I found that the birth had taken place some time before my arrival. The child was still lying in the discharges, no attempt having been made to sever the cord, and the placenta still unexpelled

Severing the Cord, I gave the child to an attendant and proceeded to expel the placenta by Credè's method, but without success. I then inserted my hand into the uterus and found the placenta firmly adherent to the uterine wall.

Recognising the serious case I had in hand, I sent off for Dr. Whiteford, Greenock. In the interval, I administered ergot, but without any effect.

The patient was very anaemic and the whole body extremely oedematous. There was considerable haemorrhage, from a portion of the uterine wall, from which the placenta was partly separated.

As the patient showed signs of collapse I injected per rectum fully a pint of saline solution; there was slight recovery but for a short time only. The patient died about 3 hours after my arrival and before assistance arrived, in the personof Dr. W.

The catheter was passed into the bladder and some urine

withdrawn. It was found to be highly albuminous, so much so that on boiling, it became solid. Two years previous to this pregnancy, the patient had been very ill during her confinement. The placenta had been adherent and Drs. S. and W. had curetted the uterus, removing the placenta in small pieces. At that time also, there had beenconsiderable oedema and albuminuria. I firmly believe that in this case, there had been a placentitis, resulting in adhesion of the placenta to the uterine wall, and also giving rise to a toxic material, which caused the albuminuria.

CASE 2.-

In Sept. 1898, I was called to see a young woman, who stated that she was in labour. This however was not the case. The history given me was that she had been suffering from swelling of lower limbs and body generally for some weeks. So much was this the case, that walking was almost impossible. Examination of the urine shewed it to be highly albuminous and scanty, of a strange "fishy" odour. The patient was a primipara, and 30 years of age.

Fearing the onset of eclampsia, I ordered her to drink large quantities of warm milk and water and to live on milk diet. Free action of the bowels was brought about/castor by oil enemata. Diaphoresis ensued after the applicationof hot water bottles to the feet and limbs, aided by hot fomentations.

The oedema lessened somewhat by the rest in bed and treatment. Fully a month passed before delivery took place, and for a week previous to the birth, there had been almost total suppression of urine. Whatever urine was passed, was examined, and found to be albuminous in a marked degree. Tube casts were also found, but not very many. During the week before delivery, the oedema had increased so much that, the body, up to the cervical region was much swollen. A mixture containing Liq. Glonini, Strophanthus and Digitalis was given, but could not be taken as fearful headaches were caused by the Liq. Glonini.

On the 10th. October she was delivered of a fine, healthy female child. The mother was not allowed up for 14 days, during which she was kept on milk diet only. At the end of the fortnight she was allowed for a little while sitting up. She complained of a sharp pain in the right knee, and was put to bed; hot fomentations were applied over the popliteal space with a little benefit. In two hours from the onset of the pain, the limb became more swollen. The oedema increased until the limb had the appearance of Phlegmasia Dolens. Hot fomentations were kept on the limb for 3 hours at a time, and then an interval of an hour, when they were again applied, the limb being slightly elevated.

At the end of four weeks from delivery, the oedema had almost entirely disappeared, though the urine contained albumen for 6 months, when it vanished.

The mother made, ultimately, a complete recovery.

Case III.-

On 3rd. Aug. 1900 I was called to see Mrs. Y. who was a multipara. Labour had been going on for some hours, but "little progress" was being made. She was very oedematous the labia majora being very much so.

Complaint of pains in the small of the back was made. I examined some of her urine and found it highly albuminous. There was also great headache and "flashes of light before the eyes."

The os uteri was only slightly dilated, and I made up my mind to wait.

Meanwhile I gave a rectal injection of hot water and castor oil, which produced a liquid stool.

The urine was scanty, so I gave got drinks of gruel, milk and water, and placed her in a hot pack.

Diaphoresis was profuse. About 5 hours after my arrival, the patient was delivered of a female child, which however lived for a few hours only.

The mother's recovery was slow, but with careful nursing, became complete.

Case IV.-

M.T. aet. 25 yrs. gave birth to an illegitimate child on 22 Decr. 1900. On my arrival, I noticed that there was

great oedema of lower limbs, while the body showed signs of swelling as well. The face also was puffy. It was with difficulty I could get the patient to understand what was said to her. There was evidently some mental depression.

The urine was scanty, high coloured and albuminous, though less than in my former cases.

She was placed on milk diet alone. Although great care was taken of the perinoeum, there was laceration back into the rectum.

As soon as she recovered somewhat, I advised her to go to hospital to have the laceration repaired. As she left the district suddenly, her after history I cannot tell.

PUERPERAL ECLAMPSIA.-

Closely associated with Puerperal Albuminuria is that condition known as Puerperal Eclampsia.

Bailly defines Puerperal Eclampsia as "an acute disease, occurring in women in pregnancy, in labour, or in childhood; often sudden in its onset, rapid in its progress, characterised by convulsions, with loss of sensation and of consciousness ending in Coma". (Noveau Dictionnaire de Medicine tome XII).

Most cases of Puerperal Eclampsia shew the presence of albumen in the urine, but fortunately every case of Puerperal Albuminuria is not associated with eclampsia.

Eclampsia may occur during the latter months of pregnancy or during or after parturition.

With the exception of Uterine Haemorrhage there is not, in the whole realm of obstetrics, a condition more to be dreaded than Eclampsia.

The attack comes on in many cases suddenly and without warning, and is attended with considerable danger to mother and child. Its aetiology is by no means clear, and though much has been written upon the subject, still a good deal remains to be cleared up. No doubt exists of some relation between Puerperal Albuminuria and Eclampsia. Opinions differ as to the frequency with which Eclampsia occurs in

Obstetric Practice. Galabin in his "Manual of Midwifery" states that Eclampsia is not of very common occurrence, its frequency varying in different countries, whilst others again state that it is not rare. (American Text Book of Obstetrics).

Frequency.-

Auvard in"Traite pratique d'Accouchements" gives the proportion as 3 - 1000.

In "Grundriss der Geburtshulfe" 1881, the proportion is given by Martin & Kaltenbach as 1 - 500.

The statistics of Guy's Hospital give the proportion as 1 - 842.

Vinay gives the proportion as 1 - 250 or 260. (Traite des Maladies de la Grossesse) 1894.

The disease when it does occur is more common in Primiparae and plural births than in Multiparae and single births.

The statistics of Guy's Charity show that 60% of these cases were in Primiparae (Galabin).

Statistics vary as to the period when eclampsia may occur.

Pajot's statistics shew 100 during labour, 60 before and 40 after, while Goldberg in "Centralblatt für Gynäkologie"

1891 gives 56.34 during labour, 21.07 before, and 22.59 after.

In the London Obstetrical Society's Transactions vol. 33. 1892, Herman's statistics are 2 during labour, 9 before and 1 after. Bailly, in the "Nouveau Dictionnaire de Medicine et de Chirurgie pratique" tome XII, states that the order of frequency is, pregnancy, labour and the lying-in-period.

In a case of Eclampsia which came under my notice, the eclamptic seizure took place 3 weeks before labour.

The late Prof. Leishman in his "System of Midwifery" Vol. II, gives his opinion thus, "As however it is also noticed for the first time, during the last weeks of pregnancy, in a large number of cases, and as labour is often an immediate result of a convulsize seizure, it cannot be an easy matter to determine what is its relative frequency with regard to the 3 periods of pregnancy, labour and child bed."

Prof. Leishman agreed with Bailly in his conclusions as to frequency of order, viz: Pregnancy, Labour and after delivery. Parvin states that Eclampsia is less frequent in child bed than in pregnancy or labour, and that the attack usually occurs a few hours after delivery but the interval may be several days or even some weeks. ("Science and Art of Obstetrics," Parvin.) Too much reliance evidently cannot be placed on statistics.

In cases where eclampsia occurs during pregnancy it is most common in the period from 7 to 9 months. In the "British Medical Journal" of 22 Dec. 1900, a case of Eclampsia at 6th. month with delivery at full time is given. This, however, seems to have been a case of doubtful nature.

SYMPTOMS:-

In some cases the convulsive seizure comes on suddenly and without any warning. In most cases, there are symptoms, which seem to indicate beforehand that there is danger ahead. These premonitory symptoms are various. Leishman states that one of the most important of the premonitory symptoms is oedema of the ankles, feet and labia majora. The oedema may appear some weeks before the seizure.

Parvin and Galabin give prominence to the intense headache, visual disturbance, and epigastric pain. One thing about the oedema should be noticed, and it is this, that in a case of pregnancy, should the labia majora be markedly oedematous, there is likely renal trouble present, more especially is this the case when there is a complaint of lumbago at the same time. Other premonitory signs are nausea, vomiting, restlessness, mental irritability or depression, vertigo, tinnitus aurium, insomnia.

Most authorities on the subject agree in placing as foremost of the premonitory symptoms, the intense headache, visual disturbance and epigastric pain. In a case of mine these three symptoms were very marked. Regarding the frequency of these three premonitory signs, Chaussier places the headache first and epigastric pain last.

The headache, which is usually frontal, may be of the nature of a hemicrania, or may be entirely frontal, seldom, if ever, is it occipital.

The headache may vary in character from being a dull ache to that of acute pain just before the seizure.

Prolonged headache may be associated with mental disturbances.

Usually the headache is intermittent, the intermissions gradually become shorter until, a short time before the seizure the headache is continuous.

In most cases there is disturbance of vision. This may vary from a dimness of eyesight to actual blindness.

The blindness may be due to implication of half of one or both retinae. Sometimes there is double vision, the result of convergent strabismus. The epigastric pain is not always in evidence, but when it is so, it is very acute, becoming more so as the seizure approaches.

The foregoing symptoms occurring in a case of pregnancy with accompanying albuminuria, and general oedema, almost positively forebode an attack of eclampsia, more especially,

if microscopic examination of the urineshows that changes are going on in the kidneys as seen in the presence of tube casts. In these cases the urine is scanty, highly coloured and of thick consistency.

PHENOMENA of SEIZURE.-

Once seen, the phenomena of the eclamptic seizure, cannot be forgotten. The seizure is remarkable for its sudden onset, sometimes so sudden that no warning is given. In some cases there may be a distinct aura. In a case of mine, the patient invariably just before the fit said, "This is awful". No sooner were these words uttered than the convulsions began.

The seizure closely resembles that of an epileptic attack, except, that there is no cry. The dentition of children sometimes produces convulsive seizures similar to eclampsia.

The patient just before the onset seems to be in a brown study; almost at once there is twitching of the facial muscles, the eyeballs roll in their sockets and may oscillate from side to side and turning upwards, give the face a terrible appearance. The mouth is drawn to one or other side, and the tongue may be pushed out between the teeth, and in this way, it is sometimes badly cut, unless precautions are taken. The pupils are dilated and do not respond to external stimuli.

The head moves from side to side in a jerky manner, but finally turns towards the right side. The face becomes swollen and cyanosed, and frothy saliva sometimes tinged with blood is seen on the lips. The condition of the face now indicates Asphyxia. There is a general tonic contraction of the whole muscular system causing rigidity of trunk and limbs, with subsequent noisy and hissing respiration caused by the implication of the laryngeal muscles. There is absolute loss of consciousness and sensation. Clonic convulsions follow the tonic stage at a variable interval. These convulsions begin in the face, then the trunk and limbs, there are rapid and violent contractions of the jaws. Respiration gradually becomes more natural, though stertorous The frothy and possibly blood-tinged saliva may be blown out with each expiration. The clonic stage may last from 30 seconds, after onset of tonic stage to 5 mins. As a rule after the fit has passed off, the patient remains in a comatose state, which may last from a few minutes to several hours. When she does awake, it is with a dazed bewildered look. She cannot understand what has happened and may complain of a headache. Fortunate the patient who has only one attack; but often there are more than one. Sometimes the subsequent attacks come on so quickly, that there is no return to consciousness. The patient in these cases, remains in a comatose state which is only disturbed by the

renewal of the convulsions. Contraction of the pupil, in cases of Puerperal Eclampsia always indicates recurrence of convulsions. As a rule the temperature, in such cases, increases and may reach a high figure.

"This effect on the temperature appears to be evidence that the comatose state of the eclamptic patient is not simply due to congestion of the brain, produced by interference with the circulation but indicates actual injury to the nerve centres caused by the eclamptic explosion". (Galabin.)

Should the seizure be prolonged, labour comes on sooner or later, in fact, if the eclampsia begins during pregnancy, the tendency is to the expulsion of the child, which may take place suddenly. This sudden expulsion of the uterine contents may be partly due to the asphyxia caused by the convulsions. Often the child is dead born. According to Hall Davis, of 36 children born during eclampsia 10 were still-born. The asphyxia and prolonged uterine contractions may be the cause of the child's death. At the same time, it must be remembered, that death may result from the ending of pregnancy before the child is viable.

Regarding the mother, the result of an attack of eclampsia may be death, or partial or total recovery. Death may result from the asphyxia caused by the oedema and congestion of the lungs.

In "Ueber Eklampsie", Sammlung Klinischer Vorträge No.39,

Olshausen gives 5 deaths from Cerebral Apoplexy, 2 from haematoma of pia mater, and 5 from hyperaemia of brain and meninges.

Playfair in his "Science and Practice of Midwifery" Vol. II, states that there is present in those cases which have been examined post-mortem, an anaemic condition of the brain. Pneumonia may be a cause of death in fatal cases.

Recovery may be partial or complete. Mental disturbances may result from eclampsia, and may take the form of mental defect or insanity.

The albuminuria generally disappears but it may take weeks, even months ere all traces have vanished, and sometimes incurable renal disease results.

There may be permanent hemiplegia and sometimes there is blindness due to albuminuric retinitis.

ETIOLOGY:-

The actual cause of eclampsia is not yet clearly settled. We know that primiparity plays an important part in predisposing to eclampsia, and sometimes puerperal convulsions take place in plural pregnancies. This tends to show that increased work is thrown on the maternal organs of elimination. Another predisposing cause may be found in the condition of the nervous system of many patients. Those who are easily irritated may possibly have a greater tendency to eclampsia than one who is not so susceptible.

It is stated that unmarried women are more liable to eclampsia, thus shewing that mental condition may predispose to eclampsia. (American Text Book of Obstetrics Vol. II).

EXCITING CAUSES vary and may be very slight, especially when there is a predisposition towards eclampsia. Touching the os uteri, uterine contractions, movements of the child, may be sufficient to bring on the paroxysm in one predisposed to puerperal convulsions.

ESSENTIAL CAUSES.-

Braun and Frerichs held that as a result of Chronic Nephritis there was a retention of urea in the blood which caused a toxaemia resulting in puerperal convulsions. This theory was latterly modified by Frerichs who stated that the urea was decomposed into carbonate of Ammonia, and injections of this substance into the tissues of the lower animals produced convulsions similar to those of eclampsia. All albuminurics of pregnancy are not eclamptics. Albuminuria has appeared in some cases after the convulsions so that retention of urinary matters could not have been the exciting cause in such cases.

Braxton Hicks states that Albuminuria and convulsions may appear simultaneously and may be explained thus:-

1. The Convulsions are the cause of the Nephritis.
2. The convulsions and nephritis are produced by the same

cause.

3. The congested state of the venous system brought about by the spasm of the epiglottis, causes the kidney complication.

Dührssen believes that the retention of creatin and creatinin is the cause of eclampsia.

Cerebral anaemia according to the Traube-Rossenstein Theory is the cause of the convulsions. This theory does not explain those cases in which post-mortem examination has revealed hyperaemia of the brain and its membranes.

M'Donald states in "Heart disease during Pregnancy" that the vaso-motor centres in the medulla oblongata are acted on by some poisonous matter or other which should have been removed by the kidneys, the result of this retention being a capillary spasm in the brain causing anaemia of the nerve centres and resulting convulsions. It is now almost universally held, that, in the blood of a puerperal eclamptic, there are circulating one or more toxic agents which produce the paroxysms.

Granted that the cause of Puerperal Eclampsia is a toxin or toxins circulating in the blood of a puerperal woman, how is this poisonous material produced?

Some one or more of the eliminating organs of the body must be at fault. It has been found experimentally, that the toxicity of the urine is greatly lessened in the

eclamptic, while that of the blood serum of the same subject is notably increased. (American Text-Book of Obstetrics). The non-elimination of urea by the kidneys is not the cause of the eclampsia for according to Winckel, the liver and muscles of an eclamptic do not show retention of urea, in fact, there is less urea than normally.

About 24 grammes of urea are excreted in 24 hours; in pregnancy, about 33 grammes are excreted, so that there is no retention. It has been shewn that in uraemia the temperature falls, while in eclampsia the tendency is to increase.

Various matters of a poisonous nature are formed in the human body, and thrown out by the various eliminating organs. It can be easily understood, that failure of the eliminating organs to throw out of the body the various toxins, must be fraught with great danger to life.

The production of the toxic material may be maternal; but it is possible that the foetus and placenta may be the source or sources of the poison.

The existence of a placentitis has been denied; but the placenta is liable to many morbid conditions, from which toxins might be produced causing eclampsia. The foetus plays an important part in the production of toxins which may cause eclampsia. In a case of Puerperal Eclampsia, if the foetus dies the eclampsia ends; as it also does when the uterus is emptied; sometimes, however, the eclampsia

continues after delivery. It may cease before delivery, and pregnancy may go on to completion.

The fact that Puerperal Eclampsia may cease before delivery or occur after it casts doubt on the placental or foetal origins of the toxins.

The Microbian theory has been rejected. The presence of a previous endometritis was assumed, but primiparae are not as a rule subject to endometritis and yet they show the great majority of Eclampsia cases.

When studying the toxin theory of causation we must not forget the increased irritability of the Nervous System in pregnancy.

EFFECT OF ECLAMPSIA ON PREGNANCY AND LABOUR.-

In cases of Puerperal Eclampsia, abortion or premature labour, usually results. The foetus dies, and may be retained in utero, to be expelled some days later.

Labour is almost certain to come on, especially if the attack is of long duration and severe. Should the convulsions appear during labour, the second stage is usually short, and birth rapid.

Eclampsia may be simulated by hysterical convulsions, and may be confounded with epilepsy, occurring in pregnancy. The face of the eclamptic during the attack becomes horribly distorted and cyanotic, not so, in hysterical

attacks. Coma is usually present in eclampsia after the convulsions have ceased; not so in hysterical convulsions which may end in laughter or tears, and sometimes with excessive urinary flow.

In eclampsia, the onset is usually sudden and without warning, whereas in epilepsy there is the characteristic cry. There is no albumen in the hysterical urine. In cases of Apoplexy, there may be convulsions, sudden coma and paralysis, but the urine is not albuminous.

PROGNOSIS.-

The prognosis depends on whether the attack occurs before or after labour. According to the statistics of Guy's Charity 50% was the mortality in those cases which began before labour, and after labour, the mortality was only 8%. Charpentier in the "Annales de Gynecologie"1893 gives the mortality in 171 cases as 12.5%. With improved treatment the mortality has been considerably lowered.

TREATMENT.-

Prophylaxis.-

Constipation must be avoided as this may be one of the maternal origins of the toxic matter. Pil. Colocynthidis Co. should be given daily. Pulv. Jalapae Co. is also given. If there be any tendency to coma 2 - 3 minims Ol. Crotonis should be mixed with a little butter and placed at the back

of the tongue. There should also be free action of the skin and kidneys. This may be attained by placing the patient in a hot bath for about 15 minutes. When she comes out of the bath the patient should be well protected from chill by warm blankets.

A glass of hot milk or gruel should also be given. Should the patient be unable to be removed from bed, for the purpose of having a hot bath, hot water bags should be placed round about her, and especially one at the lower dorsal region. The bags should be covered with flannel, to prevent blistering. By these means, very often, there is produced profuse perspiration, with increased flow of urine.

As to diet, there is nothing so good as milk. If the diet can be wholly milk, so much the better, as there is left in the intestine less detritus, which might give rise to toxic absorption. Skim milk and also butter milk are excellent diuretics. The patient should be warned as to the necessity of avoiding chills. Should the albuminuria be great nitro-glycerine in minim doses may be given thrice daily. This drug requires to be carefully watched, as it is very apt to give rise to most uncomfortable sensations in the head. One patient told me that after a single dose of a minim she felt as if her head would burst.

The patient should be kept free from all worry and noise.

During the seizure, Chloroform anaesthesia is a favourite method by which the fits are controlled.

According to Charpentier out of 63 cases, in which Chloroform was used it lessened or arrested the seizure in 48 and only one proved fatal. It should be administered whenever there are signs of the paroxysm beginning.

When combined with some other drug e.g. Chloral, Bromide of Potash or Morphine the beneficial effect is greater.

The administration of chloroform must be carefully done.

Pilocarpine has been lauded by some as being beneficial. The drug increases the secretion of saliva, bronchial and nasal mucus, which if in large quantities may cause asphyxia. It was thought that diaphoresis and salivation would purify the blood of toxic matters. Many authorities forbid the use of Pilocarpine, and amongst these are Fordyce Barker, Winckel and Kaltenbach.

Amyl Nitris has been, according to the statement of Dr. Whitla, successfully used in eclampsia. (Therapeutics q.v.)

It requires, however, to be very carefully administered, if given too often, cerebral convulsions may ensue.

Hypodermic injections of 10 minims of Tincture of Veratrum Viride may be given, but being a cardiac depressant, it is objectionable.

Venesection was at one time regarded as a Specific in the treatment of Puerperal Eclampsia. Theoretically, it is all right; but in many cases the benefit derived is only temporary. Cases in which venesection is employed should be carefully selected. In a weak anaemic patient, venesection would do more harm than good.

Sedatives and Narcotics are of great importance in the treatment of Puerperal Eclampsia.

A rectal injection of 30-40 grs. of Chloral Hydrate, with Mucilage every 2-3 hours until 120 grs. have been given in the 24 hours, is of benefit. If chloroform be administered, whenever there is the slightest symptom of an eclamptic attack, the chloral has a better effect.

Hypodermic Injection of Morphine has been regarded favourably by some.

Spiegelberg and Clark of Oswego favour the use of Morphine. Spiegelberg advises the injection of 1½ grs. of morphine into the arm and this is to be repeated in 2 hours if the paroxysm returns, and in 8 hours if the patient is in labour. (Amer. Jour. of Obstet. 1880).

According to Smith of Melbourne no patient has died from eclampsia in the Melbourne Hospital since the Morphine treatment was introduced. (Lancet 16 July, 1881).

Of 60 cases treated by Morphine, Veit only lost 2

patients. He gives ¼ to 1/3 of a grain hypodermatically; in a few hours the injection is repeated, but less morphine is used. (Centralblatt für. Gynäkologie 1888).

Cases have been recorded lately (British Medical Jour. 24 Nov. 1900) in which good results were obtained by the hypodermic injection of Morphine with Atropia. Morphine allays the cerebro-spinal irritability and thus controls the eclamptic convulsions. Of late, Morphine seems to have been discarded by many. (Therapeutic Gazette, 15 Dec. 1900) and amongst them are Edgar, Reynolds and Norris. Nevertheless, there are many who obtain good results by the use of Morphine and are justified in using the drug, which they know has been beneficial.

During the seizures, the patient must be prevented from injuring herself. The tongue is very apt to be bitten; and a small piece of wood, half an inch thick and covered with cloth, should be inserted between the teeth at the onset of the seizure.

Where there is suppression of urine or urine of a high colour and scanty, the hypodermic injection of salt water has been recommended (Lancet 1893). A litre of sterilized warm water containing 7.5 grammes of sodium chloride was introduced into the buttocks, the skin being first of all thoroughly disinfected. The time taken was 20 minutes and the temperature of the water was 88°- 90° F. The results

were very satisfactory.

Dr. Jardine, Glasgow, has published a series of results obtained by the Intercellular injection of Saline fluid. (Lects. on Haemorrhage and Eclampsia 1899).

Believing that an unknown toxic substance in the blood is the cause of the convulsions, Dr. Jardine aims at counteracting its effect or getting rid of the toxin. He recommends the use of Chloral and Potass. Bromide, also the hypodermic injection of 10 - 15 mins. of the Tincture of Veratrum Viride.

The saline solution consisted of Potas. Bicarb. and Chloride of Sodium, in the proportion of 1 - 2. Acetate of Sodium was substituted for the Potas, Bicarb. in equal parts with the Chloride of Sodium, 60 grs. to the pint. From one to 3 pints can be used at a time. Care must be taken to use sterilised water, as well as aseptic precautions.

OPERATION:- The apparatus consists of an Aspirator trocar and cannula, tubing and glass funnel.

The puncture is made under the breast, into the abdominal wall, axilla or arm pit. A pint is run into the part, taking 4 mins. to do so. In about 15 mins. the injection has been absorbed, and then Common Salt solution is used, 60 grs. to the pint. Dr. Jardine reports that the injection produced an increase in the urea and uric acid in

the urine. Milk was the chief diet.

Of 22 cases recorded, there were 4 deaths. 1 of these was from perforating ulcer of the duodenum on the 7th. day after the eclampsia had been cured.

There were 23 children, of whom 10 were alive and 13 dead; of the living children, two had craniotomy performed.

In two of the cases, the children were macerated, and yet the urine was albuminous; a contradiction of the statement, that with the death of the child, albuminuria ceases. The cause of the persistent albuminuria in these cases may just have been the maceration resulting in the production of a toxic substance, the cause of the albuminuria. In one case which died, the post-mortem examination shewed normal kidneys while the liver was degenerated. In 2 cases there was hyperaemia of the kidneys. This proves the presence of an irritant in the blood. It was also noted that there was less urine in those cases treated with Morphine than in those treated with Chloroform, Bromide or Veratrum Viride.

It was also observed that the child's urine contained a similar amount of albumen to that found in the maternal urine.

OBSTETRIC TREATMENT.-

In this, as in the other methods of treatment, there is considerable difference of opinion. Some recommend that the uterus should be emptied of its contents, whether labour has

begun or not. There is the opposite opinion, viz:- leave the labour alone. A case was recorded by the Edinburgh Obstet. Society, on 12 Decr. 1900, in which eclampsia occurred at the 6th. month and yet there was successful labour at full time. In this case there were convulsions at intervals of 4 days and there was no albuminuria.

Galabin recommends that in mild cases, in which there are few fits, and these at long intervals, leaving little or no coma, purgatives, Chloral and Potassium Bromide, should be given; at the same time giving the patient milk diet. In severe cases, in which labour has not begun, induction of labour is favoured. Playfair recommends that if the os be undilated and labour not begun, nothing should be done to bring on labour.

Dührssen recommends the emptying of the uterus, whether the child is viable or not. (Archiv. für Gynaekologie 1892) Olshausen and Charpentier are against this method, the latter stating that premature labour should only be induced, when medicinal treatment has failed. In the Trans. of Edin: Obstet: Soc. 1890-91, 3 cases of eclampsia are recorded in which premature labour was induced. All the patients recovered while of the children, two lived. Winckel and Pajot are against the induction of premature labour, while Gooch recommends the watching of the convulsions, and allowing the uterus to take care of itself. In the face of so many different opinions, it is extremely difficult to decide.

For my part if the os was well dilated, I would rupture the membranes and use the forceps. The irritation from such a procedure would be less than that from forcible dilatation of the os. If after delivery the fits continue, then chloroform should be continued as well as Chloral and Bromide of Potass given. The patient should be carefully nursed for sometime after the fits have ceased and the temperature ought to be noted. If there is any very great rise in the temperature, cold sponging of the body should be resorted to and continued till the temperature has come down to normal. The diet should still be wholly milk, and the urine carefully examined as to quantity and also the amount of albumen. Absence of fits with decreasing albuminuria and increasing quantity of urine, point to recovery.

The pathogenesis of Eclampsia is by no means settled. It is a condition peculiar to the pregnant or puerperal state. Eclampsia has not the characters of either Apoplexy Epilepsy, or Hysteria. It depends on a toxaemia, the result of overproduction of toxins and the under-elimination of them by the maternal eliminating organs.

The source of these toxins is not definitely fixed; they may be due to food undergoing changes, in its passage along the intestine, and the consequent absorption of toxic material. It is possible that the toxins may be foetal in origin, perhaps partly maternal and partly foetal.

PUERPERAL ECLAMPSIA.-

On 20th. May 1899, I was summoned to see Mrs. D. a multipara. She stated that she was six months pregnant, but suffered from fearful headaches and vomiting. As there had been a slight indiscretion in diet, I attributed the headache to this.

However, further questioning shewed that in March 1898, she had given birth to a still-born child, after having had several convulsions. I consulted my friend and colleague Dr. Bradford, who had attended her in previous confinements, but who was unable to go to her, at the time I was called. I called next day, and advised Mrs. D. to keep her bed and also, not to worry herself. We prescribed Pepsin and Nux Vomica. The intense headache and vomiting disappeared. About a month after, I was hurriedly sent for to Mrs. D. I was informed that she had had a convulsion and was unconscious.

I sent for Dr. Swan, who was Dr. B's. assistant at the time, and on our arrival were told that Mrs. D. was just out of a convulsion, but was "sleeping".

We found our patient unconscious. Shortly after our arrival Mrs. D. exclaimed, "This is awful" and immediately the eclamptic seizure began. The muscular twitchings began in the ocular muscles, causing oscillation of the eyeballs. Soon the tonic contractions spread over the whole body. The head was twisted towards right shoulder and the thumbs

drawn across the palm of the hand. The muscular contractions were so strong, that the body was curved backwards producing the "opisthotonic" posture. Mrs. D. seemed to rest on the occiput and heels. The face became very much cyanosed with "hissing" respiration. Just as it seemed, the fatal moment had come, clonic contractions began and gradually the convulsion ceased, leaving her in a comatose condition. Urine was passed in bed, unconsciously, so that no specimen was at first obtained. The os uteri was firm and undilated. Morph. Sulph. 1/6 gr. and Atrop. Sulph. 1/100 gr. were injected into the arm every hour until the convulsions ceased. During the first hour of attendance on Mrs. D. there were 6 seizures. After the third injection of Morph. Sulph. the fits were less severe and the intervals longer. From 2 a.m. - 10 a.m. on 21 July, there were 12 seizures, and no more. The question of inducing Labour was considered, but it was agreed upon to allow labour to come on, which it did in 3 weeks after, when a macerated foetus was expelled. The urine from 21 July, which was obtained per catheter, on till the end of August, shewed albumen present. The urine however did not solidify. The patient made a rapid recovery and left the district shortly after. This case shewed the great value of the morphia. Chloroform was not tried.

Milk diet was what she had as food. She was also encouraged to take copious draughts of water.

In two previous pregnancies, there had been abortion, and also albuminuria with retinitis, which disappeared in the interval between the pregnancies.

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