PUERPERAL ECLAMPSIA

A THESIS FOR THE DEGREE OF M.D.

By ALEXANDER GRIEVE YOUNG, M.B., C.M.

Univ. Glasgow.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

Perhaps if I exclude placenta praevia, and rupture of the uterus, the most alarming and most dangerous complication of pregnancy which can come before the medical practitioner is the condition known as puerperal eclampsia.

By Puerperal Eclampsia I do not mean any convulsive phenomena which may be present during gestation; but that special condition occurring during pregnancy, parturition, or the lying in period, which is characterised by recurrent convulsive attacks, which are intimately associated with either of these states, and which are only produced during their continuance, and which attacks are succeeded by a complete suspension of the sensorial and intellectual faculties for a variable period, which phenomena are usually accompanied with albumen in the urine: puerperal eclampsia is a rare occurrence as proved by statistics. Out of 38.306 obstetric cases Cazraux found that 79 had puerperal Eclampsia or 1 in 485 cases.

Puerperal Eclampsia may be recognised or suspected early provided you have opportunities of seeing the patient before the active symptoms begin. The fact that this condition occurs much more frequently in primipara than in multipara may explain why it is often not diagnosed until the beginning of a fit, as slight inconvenience, malaise, oedema, frequent scanty micturition, may not cause sufficient indisposition as to necessitate a consultation with the physician, these being/

being put down as accompaniments to a natural pregnancy. If the urine be examined in cases such as these and found deficient in quantity; and with a quantity of albumen in it, then puerperal Eclampsia may be suspected, but frequently no opportunity is given the practitioner to diagnose this condition early, and he is only called in when the patient is seized with convulsions.

Some writers go as far as to say that no case of Puerperal Eclampsia should come as a surprise to the accoucheur as there is generally some predisposing condition, such as oedema, scanty and albuminous urine. I think this is quite so and may be multipara are more likely to rush off to the physician on the appearance of a symptom which she may not have had in a previous pregnancy. In this way a very dangerous complication is often discovered or suspected, and a form of treatment may be prophylactic preventative, or otherwise adopted, which may secure for the patient an immunity as it were to the disease (of this I am not convinced) at any rate it may tend to lessen the severity of the attacks and thus assist the patient towards recovery by putting her under as favourable conditions as possible.

Two cases of this condition came under my care while practising in a country village in Yorkshire, and it is now my intention to give a detailed account of each case, and the treatment/

treatment I adopted.

Case I.-

Mrs. D. a primipara 25 years of age called me in on June 28th. to see her and to engage me for her approaching confinement. On asking her a few ordinary and necessary questions, she said she had always enjoyed good health, and since she became pregnant even better until the last few days, when she noticed that her feet and ankles were slightly swollen. This did not appeal to me to require particular treatment, as it is quite common to find slight oedema in normal pregnancies, however I advised her to take warm baths occasionally, gentle exercise in the open air and a gentle aperient in the form of Liquorice powder to regulate her bowels, and to let me know if this swelling did not disappear. This oedema was quite simple in its characteristics being greater in the evening and almost imperceptible in the morning. Indeed I attributed it solely to her condition without in the slightest degree suspecting any morbid lesion. I heard no more of this case until a month later when I was called again to see her and to my astonishment I found a considerable amount of oedema of the feet and legs extending half way up the calves. She said this had only developed within the last day or so, and as it was considerably less in the morning, she thought it was unnecessary to trouble me until/

until to-day when her legs felt as heavy as lead. She said she felt perfectly well except for her legs. She eat enormously but attributed this to her being pregnant. I questioned her about her urine, but all the information I obtained was to the effect that she was obliged to pass it often and only a small quantity at a time. She had not noticed any sediment or muddiness in it. My suspicions being aroused I procured a specimen of her urine that night, and on examination I found a large quantity of albumen present, indeed it almost solidified in the tube on heating. I immediately came to the conclusion that there was some abnormal condition here, and my suspicions naturally turned towards Puerperal Eclampsia. This occurred on the 17th. July, and as she expected to be confined towards the end of August, I thought a course of prophylactic treatment indicated should my prognosis turn out correct. I accordingly visited her next morning and explained to her that the swelling in her legs might be reduced with advantage to her without inconveniencing her much. I ordered her to take warm baths daily, to pay particular attention to her bowels, regulate her diet, avoiding flesh meat and partaking principally of light and starchy foods as milk, arrowroot, tapioca, fish etc. and vegetables freely. As she was passing urine in small quantities I put her on the following mild diuretic:-

R.

Liq. potassi Acct. ℥p.

Tinct. Hyocyami ℥i.

Sp. Ethesis Nit ℥iip

Sp. Chloroformi ℥p.

Infusi Buchu ad ℥vi

Sig. ℥p ter in die ex aquam P.C.

In addition to this mixture I ordered a Guy's pill be taken every evening on going to bed. This I consider is an excellent example of the necessity of examining the urine in pregnant women. However slight the oedema, an examination of the urine is indicated, and the result will go far to strengthen or allay existing suspicions. I now decided that this was a case warranting careful attention and consequently I visited daily for a week, and found my treatment resulted in the oedema becoming less marked, and the urine more copious, but which on examination still contained albumen, not however in so marked a degree. The patient herself said she felt much better, that her legs were not so heavy and that she could move about much more easily. I told her to continue the medicine and take a dose of castor oil ℥p. to ℥i. twice a week instead of the Liquorice powder and a warm bath daily to keep the skin in good condition with regard to its physiological action. This was on the 27th. July/

July, and I did not see her until August 5th. when I was hastily summoned, the messenger saying that the patient was in labour. I arrived at 6.30 p.m. and on enquiry from the nurse I found that she had started in labour about three quarters of an hour previous to my arrival. Being very interested in this case, I decided to examine the abdomen by palpation and I found a cranial presentation, at least I made the groove (of the neck) between the hard presenting part (the head) and the flat trunk of the child. I could get movement laterally of the head showing that it had not yet engaged. Knowing my patient was a woman of highly nervous temperament I refrained from making an examination per vaginum and decided to wait some time and find out the nature of the labour. She had light pains every half hour for two hours, after which time they suddenly became much stronger and with a less interval between. During this time I kept her moving slowly about the bedroom and when tired I made her sit in a chair to facilitate the engagement of the head. The pains coming on more rapidly and stronger I explained to her the necessity of an examination per vaginum and having got her into bed I examined her both during and after a pain. I diagnosed a cranial presentation through an os dilated to the size of rather less than a shilling. The vaginal canal was quite normal and well lubricated and the/

the membranes were intact. I examined her abdomen again by palpation and found the movement much restricted showing that the head was descending in the pelvis and about to engage. After my examination she had a few pains and then they suddenly seemed to leave her. I waited half an hour after this and as she had no pains I left and called again in two hours to find that labour had not returned, and she was comfortably seated enjoying a cup of tea. I told the nurse to send for me on the first appearance of labour. Before leaving I got a specimen of urine just passed. I examined it on getting home and found the quantity of albumen it contained much greater than on any previous examination. I called in next morning and found my patient quite bright and attending to some light domestic duty. This case would certainly have deceived me had I not made several examinations of the urine. I was called to see her on the morning of August 12th. She had not got out of bed so I saw her there. She said she felt her legs very heavy, and she was dreadfully tired. I noticed that she was very pale and uneasy, and on closer observation I noticed that peculiar oscillating movement of the eyeball which has been described as Nystagmus, but which I have never seen associated with this condition. I shall have something to say regarding this symptom later. She insisted on getting up and on doing so/

so I observed that her gait was quite unsteady, and she complained of being dizzy. She had a light breakfast and almost immediately after she complained of a most violent headache, at first intermittent, but latterly persistent, and which she localised over the frontal region. This eventually compelled her to lie down, and I gave her three powders containing the following:-

Rx Quininae Sulph. gr ii
Potassii bromidi gr x
Antipyrini gr v. fiat pulv.
Mitte tales tres.

Sig: one every 3 hours, and directed the nurse to apply cold cloths (ued) to her head. This seemed to relieve her somewhat as she came down to dinner, but only partook of a cup tea & a morsel of toast and then lay down again but could not rest for this excrutiating headache, and directed the nurse to apply a leech to either temple and wait till I returned. I was here called away to another confinement, and did not get back for some time. I found her almost prostrated with this headache. I tried other drugs such as Bromide of Soda with phenacetine and even injected Coccaine hypodermically into the affected part, but with no effect, and at 5.45 exactly a week after she had been in labour to the hour, she was/

was thrown into a violent Eclamptic seizure.

Just prior to the convulsive movements I noticed this peculiar oscillating movement of the eyeball, this occurred in both eyes. This lasted about half a minute, and was succeeded by distinct squint, the patient said she could not see, her gaze became fixed for a few seconds, the eyelids twitched, then the eyeballs rolled about in their sockets, the muscles of the face and then of the neck twitched convulsively, and the mouth was pulled to one side, I think the left, the face falling on the shoulder of the same side. The convulsions rapidly became general extending to the arms trunk and inferior extremities, (the whole body being perfectly rigid, the thumbs were flexed on the palms of the hands, respiration was irregular and sighing, finally the diaphragm was fixed and patient lay with all her muscles contracted, her face became livid and cyanosed and froth appeared at the mouth. Almost simultaneously with renewed twitching of the whole body, clonic spasms, respiration was gradually restored, the colour of the face returned, and patient lay in a condition of deep coma. The fit from the beginning of the tonic contractions until the comatose period would be about two and half minutes. I spoke to her and pinched her but could get no response. She was quite unconscious and was not sensible to pain. When the coma passed off she was quite/

quite stupid and dazed, and exclaimed my head, endeavouring to put her hand up to it. She did not get it the length of her head before she was seized with a second convulsion which seemed more severe than the first one. I put part of a towel in her mouth and waited on the comatose period when I examined her per vaginum, and found the os dilated sufficiently to admit the tips of three fairly large fingers, the head well down and the membranes bulging considerably. I waited a few minutes to see if she regained consciousness but she did not and was again thrown into another violent convulsion more severe and longer than either of its predecessors. I here took the pulse and found it to be very rapid and weak, scarcely perceptible, but it improved during the succeeding coma. This induced me to take the temperature which I did in the vectum, but it only registered 103.2. I was considering what to do when she went into her fourth convulsion. I saw at once that if I did not interfere and that soon there would be no necessity. I at once decided on my line of treatment, namely, Chloroform and instruments, remembering that the os was almost sufficiently dilated. While preparing my instruments she was seized with her fifth convulsion. This one was even worse than the former ones. Judging by the severity of the seizure I thought the coma would be correspondingly deep and so it proved to be. I had

a/

a small sharp-pointed knife in my hand with which I was removing a small part of my finger nail. I pricked the fleshy part of her arm without evincing any symptom of sensation, just at the beginning of the coma. From this I concluded that all reflex action peripheral and otherwise was under control. This suggested to me the application of the forceps without the production of complete anaesthesia, which procedure was subsequently ~~proved~~ adopted. I had the patient gently turned on her left side and put in the position most suitable for the application of the forceps. I examined her per vaginum and found the Os quite sufficiently dilated to allow the forceps to be applied. I removed the water from her bladder by means of a soft gum elastic male catheter, and ruptured the membranes and applied the long forceps without evincing the slightest emotion on the part of the patient. She was deeply comatose with slow deep respirations eye quite insensible to light or touch, pulse very weak. I commenced delivery and proceeded on ordinary lines until I got the head just escaping the perineum when I stopped for about half a minute to dilate the perineum. I thought this a mistake at the time but subsequent events proved it was not. I delivered the head and not a moment too soon as evidence of another fit showed itself. I rotated the head to insure the internal rotation or engagement of the shoulders. There was

a/

a slight convulsive movement on the part of the patient and a male child was born. My next procedure was to ligature the cord which I did loosely and allowed the blood to escape first from the mother until about half a pint had escaped, when I ligatured securely and allowed about one ounce to escape from the child, but I think on the part of the child it did no good. The mother continued for fully a quarter of an hour in this comatose state without any indication of the placenta coming away. I was undecided as to whether I should assist nature or not, but having succeeded so well in the first part of my treatment I decided on assisting in the expulsion of the placenta. I did so by placing my hand flatly but firmly on the abdomen over the uterus. The effect was almost instantaneous, there was a slight convulsion probably due to peripheral irritation produced by my hand on the abdomen, and the placenta was shot (not merely expelled) out over the edge of the bed. Such then was the actien or operative treatment I adopted in my first case of Puerperal Eclampsia. After having carefully arranged her, without douching, for I consider a naturally expelled placenta a much better remover of all forms of septic matter, if such be present, than any artificial means we have got, indeed the careful management of the third stage of labour is in my opinion the key to successful Accouchment. After having got her/

her carefully washed and dressed there were still indications of spasmodic action. I could not think that there was anything left in the uterus in the shape of clots, for the discharge was quite normal, perhaps freer than I should have expected; so I gave her 30 grains of Chloral and fifteen grains of Bromide of Potassium per rectum. I may state that her bowels were completely evacuated during the convulsions (tonic). Her pulse was a good deal slower now, but still very weak. The temperature in the axilla was 102.6. Her skin was very hot and dry so I injected 1/6 of a grain of pilocarpin hypodermically. Some writers object to this mode of treatment on the grounds that the salivation it causes may get into the trachea and larynx and cause asphyxia. With ordinary care I dont think there is any danger of this complication and most writers will admit that with the skin in a good healthy condition, there is much more likelihood of getting rid of effete matter. Be that as it may in a few minutes my patient settled down, quite free from any twitchings into a profound coma with the pulse steady, respirations slow, deep and regular. In a few minutes a nice perspiration broke out on her, and I decided to watch her progress for an hour. Here I may state that the delivery with the instruments was far from easy. I attempted as I almost invariably do unless contra indicated to imitate nature/

nature as much as possible by exerting traction, then resting a little. This alternative pulling and resting I thought would be sure to start the convulsions but the coma as suspected was far too severe. When I say the delivery was not easy, I mean that although the os was well dilated, the vaginal canal and especially the perineum in a primipara require a considerable time to dilate, so that after exerting a good deal of traction to get the head to engage the perineum, I found that considerable care was necessary to avoid laceration of it. At the expiration of an hour my patient evinced symptoms of uneasiness and I deemed it advisable to repeat the injection of Chloral and Bromide per rectum in half quantity. This I did and at the same time removed considerably more than a pint of thick muddy urine from the bladder, which on subsequent examination was found loaded with albumen. This condition of her bladder may have been the cause of her uneasiness by exerting reflex irritation. However after the injection and removing the contents of the bladder, she soon fell into a good sound sleep. I called next morning Aug. 13th. and she was still semi-comatose. I endeavoured to arouse her but could not. Sensation however had returned. I gave her an enema of weak beef tea, took her pulse which was 84 beats per minute and the temperature was 102.6 in the axilla. I visited her again/

again in the evening and found very much the same with pulse between 80 and 90 per minute and temperature 103.2 in axilla. I had at this period a very unfavourable opinion of this case. I gave her another enema of beef tea and saw her on the morning of Aug. 14th. to find a great improvement. The temperature fell to 100.6 and the pulse to 80 beats per minute and much stronger than previously. She was sleeping when I went in but on my speaking to her she awakened up, and asked where she was, had she been asleep. Her memory was quite a blank but this she has quite recovered. Next morning her temperature registered 99°F. and her pulse quite normal. She made an excellent recovery after this. For a month after this she was very weak, the oedema having gone from her legs these were more like skeletons than anything else, but with the aid of Tonics of Iron, Quinine and Tinct. Nucs. Vom. she is all right again and as well as ever she was. This is a peculiar point about this condition, the recovery is very rapid, as both my cases show. I withdrew the urine from her bladder on the morning of the second day, just before she became completely conscious and an examination showed it to contain much less albumen than the previous examination. I examined it again at the end of the first week and found very little albumen present, and at the end of the second week it was practically gone. The child which

was/

was a normal one in every respect was still born, and this probably favoured the recovery of the mother. Whether its being still was the result of the convulsions or of the rapid delivery I cannot say, but I am strongly of the opinion that it died in utero just previous to delivery and most likely due to the convulsions.

I will here mention the phenomena which particularly struck me as being different from all previous records of these cases, and which I consider have an important bearing on the Aetiology and treatment of this disease.

I. The fact that labour ("practically a normal labour") commenced exactly a week previous to delivery. The relation and effect of this early labour on the subsequent treatment of the case.

II. The peculiar oscillating movements of the eyeballs prior to their fixation, (Nystagmus).

III. The severity of the convulsions and the extremeness of the coma, thereby allowing

IV. Application of the forceps and delivery without the aid of Anaesthetics ($CHCl_3$).

I will give my opinions of these phenomena after consideration of the following case:-

Case/

Case II.-

Mrs. P. a multipara, age 31, having had five previous labours, all of which she asserts were natural ones. I was called to her about mid-day on August 21st. This patient was a peculiar one, you could not exactly say she was insane but before my advent to this practice, she was considered not quite right and I decidedly say that although not quite insane she was decidedly a person of weak intellect. I had no knowledge of her condition until I was called to attend her labour, and as she afterwards said I would not have been there at all if complications had not ensued. I mention this condition of her intellect as having a connection with the relationship (if any) between puerperal Eclampsia and Puerperal Mania. I think it is a question whether puerperal Eclampsia may not be a predisposing cause of puerperal Insanity, or not, but I will leave the discussion of this until later.

When I was called to this case she was sitting up in bed with a most vacant expression, having as the attendant informed me just recovered from a fit. I asked her if she had any pain, but elicited no reply. I noticed this oscillating movement of the eyes present here, she put her hands to her frontal bones and fell back on the bed with all the symptoms of an Eclamptic seizure. The characters of this/

this seizure were very similar to those of the last case only the forearms were much pronated and opisthotonos was present in a marked degree. This fit lasted altogether two and a half minutes, during which time I questioned the attendant as to her previous history, if she had suffered from Oedema, scanty urine, headache, dizziness or loss of vision, previous to the first seizure. Her answer was in the affirmative. She had complained of severe headache over frontal region, loss of vision, and pain in the epigastrium (at least she said severe pain in the belly). The headache had been present for days previous to delivery, and her memory during that period, gone. It had not been good previous to this. There was no history of Oedema with the exception of a slight swelling about the ankles at night. I quite concluded that this was another case of Puerperal Eclampsia and I resolved if possible and everything favourable to it to pursue the same treatment I adopted in my previous case. And this case was a multipara I expected to be able to do so with less inconvenience to the mother and with greater safety. During the comatose period following her second seizure I examined her abdomen by palpation but could not make out any presenting part at all, indeed I turned round to the attendant and enquired if she was at her full time; (which I afterwards found out she was). I examined/

examined her per vaginum and found the Os well dilated, about the size of a four shilling piece and very thin and the membranes bulging considerably.

Wondering at the state of affairs present I asked if she had been in labour before the fits came on, but as this patient was of a very reticent nature no one knew. Afterwards I learned from the patient herself, that she had what she called thin pains in her belly for two days previous to her illness. This condition of the parturient canal made me think of the treatment adopted in my former case, and I thought it should be easier of accomplishment in a multipara. I waited to see if the fits got more severe. I had not long to wait as the next seizure was much more severe than its predecessor, and I determined if all things were favourable to assist nature during the period of coma.

I withdrew the water from her bladder and kept it for examination. I used a gum elastic male catheter. The coma appeared quite deep enough to proceed, but on my endeavour to rupture the membranes she was thrown into a much more violent seizure than any she had yet had. I had to wait on the succeeding coma, and as it seemed to be extremely deep I tested her with a lighted match and found the eye reflex gone and when I pinched her and pricked her with a needle there was no response, showing that the terminal ends of the sensitive/

sensitive nerves were under control. I ruptured the membranes and applied the long forceps without producing the slightest emotion on the patient, and in the same manner as in my first case, but a little more rapidly, and delivered her of a female child. I allowed only a little blood to escape from the mother and none from the child and then ligatured securely, and proceeded to watch the mother.

I waited for quarter of an hour during which time the mother lay quite comatose with regular long deep respirations and with what I call a fairly strong pulse. Let me here state that some writers say heart disease or weak action of the heart predisposes to this condition. In both of my cases there was no indication either of weak or diseased heart I then placed my hand on the abdomen over the uterus. This organ at once violently contracted and expelled the placenta, and almost simultaneously with its expulsion another seizure. This I think conclusively proves my remark in the first case that peripheral irritation may undoubtedly and does cause convulsions, and I think that this is particularly so after the second stage of labour is over or during the puerperium. It is excellently exemplified here as whenever I put my hand on the uterus it contracted, and by the contraction of the uterus another fit occurred, thus suggesting that indirectly peripheral irritation, and directly reflexly from the uterus produced/

produced a convulsion.

After carefully arranging her the necessary movements in bandaging her seemed to excite slight convulsive movements. I gave her an injection per rectum of thirty grains of Chloral and fifteen grains of Bromide of Potassium.

She had no more seizures after the administration of this during my stay with her of about one and a half hours. When I called at seven in the evening she was quite conscious, but wondering how she had had a baby without feeling it. My examination of the urine in this case showed a very different result from that of my previous case, Albumen being only found in very small amount, and a subsequent examination a week after showed a complete absence of albumen. I would have examined this urine more frequently, but the patient objected to the use of the catheter and specimens passed otherwise were too much mixed with blood and clot for ordinary examination.

I must now remark on the child. It was a mature female child but the most diminutive baby I ever delivered at full term. It was almost asphyxiated at birth but after a few plunges into hot and cold water alternately it came to all right. I unfortunately did not have it weighed.

The mother told me afterwards that her five previous births were all remarked on for their smallness, and I had ample/

ample evidence in believing her, as I subsequently attended three of her children through an attack of Scarlet Fever and they were all abnormally small, but strong. This phenomenally small child almost negatives the theory on causation of this disease put forward by Goodfellow I think, in which he states that pressure on the abdominal and venal vessels, assisted by resistent abdominal walls producing congestion of the kidney and surrounding organs, so alters them and the kidney especially, as to prevent the elimination of effete matter and so cause a predisposition to this condition of Puerperal Eclampsia.

This case did not require a second injection of Chloral and Bromide of Potassium until the evening of the second day when the temperature rose rapidly to 103.6 in the axilla, and she had another fit, the discharge was very scanty scarcely staining the linen and with that characteristic stain so suspicious of sepsis, and smelling strongly. I examined her per vaginam and found a large clot in the uterus. This I removed with my finger, and douched out the vagina and uterus with a mild Creolin solution. I gave her a second dose of Chloral and Bromide of Potassium by the rectum, after which the temperature fell to 99°F. the discharge returned, and continued normal for the rest of the puerperium.

I kept her on very light diet chiefly beef tea. She did/

did not nurse the child as the slightest attempt to do so threatened further convulsion, showing the remarkably strong predisposition of this woman to reflex irritation. Under the usual regimen after the first week this patient made an excellent recovery being out of doors within a month of delivery.

Knowing of her weak intellect and her strong tendency to reflex stimulation I kept her on a mixture composed of the following for six months, with unqualified success:-

℞ Potassii bromidi ʒiii
Sinct. nucis vom. ʒ½.
Sinct. aurantii ʒi
Syr. aurantii ʒvi
Aquam ad ʒvi misce.

Sig. ʒ½ ter in die c aquae pot.

The only drawback in this case was her memory which did not altogether become reliable till six months after her accouchment. The facts worthy of special notice in this case are:

I. The condition of the os at the commencement of the Eclamptic seizure.

II. The occurrence of Puerperal Eclampsia in a woman of weak intellect. Its connection with Puerperal Insanity.

III. Application of forceps and delivery without the aid of anaesthetics.

IV. Comparative absence of albumen in the urine showing that the severity of Puerperal Eclampsia does not occur in direct proportion to the amount of Albumen present.

V. Epigastric pain. Was it labour.

AETIOLOGY OF PUERPERAL ECLAMPSIA.

Returning to the facts which I have drawn special attention to I will now endeavour to show their connection with either the causation or treatment of this condition.

The fact that labour commenced exactly a week before delivery and occurred without the slightest symptom in itself of Puerperal Eclampsia and continued for about six hours has I think a very important bearing both on the Aetiology and treatment of this case.

Is this not a case in which nature provides for emergency in preparing the parturient canal for the subsequent trials it is to endure or is it a proof of the theory put forward by Dr. Tyler Smith, it being taken for granted and my experience of it is such that the head is usually the presenting part in Puerperal Eclampsia. Dr. Tyler Smith says that the first attack does not come on when the head presses on the Os/

Os Uteri, or clears it, but when it dilates the perineum and partially dilates the vulva in primipara.

This may not always be the case, but the fact that all the symptoms of a normal labour may be present as was in my first case, until the labour gets so far. I think until the head threatens engagement gives some strength to the argument of Dr. Tyler Smith. In this case the pains were certainly dilating the Os and preparing the parturient canal for the second stage of labour, and had it not been so I feel sure I would have been unable to adopt the treatment I did. To this fortunate circumstance in a great measure I attribute the successful termination of this case. Two or three pains will not dilate a primiparous Os, nor prepare the parturient canal for the passage of the foetus, no more will it allow of the application of the forceps, be they ever so strongly indicated One therefore cannot overestimate the value or significance of this early labour on the parturient canal when symptoms are likely to occur which call for prompt delivery, especially when the case is a primipara.

As this case proves to a certain extent the assumption of Dr. Tyler Smith I must agree with him so far especially when the Neurotic Theory more or less substantiates it, namely, that Puerperal Eclampsia is due to heightened irritability of the nerve centres, produced or aggravated by

a/

a poison circulating in the blood or by strong reflexes from the gravid uterus (Eclampsia Reflectorica) and this latter in support of Dr. Tyler Smith's argument would be greatly increased by the pressure of the descending head on the pelvic vessels and nerves causing increased congestion and irritability and ultimately resulting in a fit.

When it is known that Puerperal Eclampsia seldom occurs in the early months of gestation, I may here express an opinion with regard to the cause in primiparous women. The uterus as is well known begins to rise out of the pelvis at the beginning of the fourth month. Here note the peculiar arrangement of the renal veins, one enters the portal vein, the other the Inferior Venae Cava. Now as gestation proceeds the gravid uterus must necessarily exert some pressure on these veins, and this will be assisted by an unyielding, rigid and non-sympathetic abdominal wall. From this arrangement of the renal veins, this pressure will affect the hepatic as well as the renal circulation, and thus be a cause of preventing the blood from reaching one source of purification. This congestion becoming greater in the kidney and liver as gestation proceeds (as evidenced by Oedema etc.) must undoubtedly be the cause of the non elimination of the poisonous products, and these poisonous products circulating in the blood irritate the nerve centres beyond control/

control, and this increased by the pressure of the head on the vessels and nerves in the pelvis may cause an Eclamptic Seizure. This commencement of labour then a week previous to delivery would seem to give some weight to Dr. Tyler Smith's argument and if we follow this up some idea of the causation of puerperal Eclampsia may be arrived at. When you take into consideration the gravid uterus pressing on the venal vessels causing this congested condition of the kidney, (the kidney of pregnancy ultimately) and necessarily preventing the elimination of poisonous products from the system aggravated by the unusual irritability of the nerve centres, normally existing in pregnant women, which may be increased reflexly from the uterus until it reaches a climax in which the nerve centres are so stimulated beyond control as to cause a fit.

Taking these phenomena together then may we not naturally conclude that each of itself is not sufficient to cause Puerperal Eclampsia, but collectively beginning with uterine pressure, any time after the fifth or sixth month of gestation, (and this I think is in primipara a very strong predisposing cause) causes congestion of kidney and liver resulting in the retention of poisonous products in the blood, or their non-elimination in the urine, latent for a time (passive at least) ultimately so stimulate the nerve centres/

centres and this probably augmented by reflex stimulation from the uterus produce Puerperal Eclampsia, remembering that in normal pregnancies we have the following conditions:-

I. Increased vascular tension.

II. Altered condition of the blood, hydroemic, anaemic etc.

III. Increased irritability of nerve centres.

Reflex stimulation from the uterus especially or otherwise must not be overlooked in the causation of this condition, for if not actually producing it, it may be the means of keeping up a continuance of it, as shown by my second case, where convulsions threatened and indeed appeared after the expulsion of the placenta, and were found to be due to a retained clot in the uterus, and later on convulsions threatened on attempts to put the child to the breast.

Other theories have been put forward as causing puerperal Eclampsia, the principal of which are:

I. Brains, Urea and possibly other urinary matters in the blood (uraemia) the result of nephritis irritate the exalted nerve centres and excite convulsions. Post mortem examinations of women who have died from Puerperal Eclampsia negative this theory.

II. Frerich's, Carbonate of Ammonia and not urea is the poison circulating in the blood, not confirmed by examination of the blood.

III./

III. Traube Rosenstein, Arterial tension is increased because (a) The left heart is hypertrophird and (b) blood in pregnancy is hydraemic especially where albumen exists. Result of increased arterial tension equals oedema of brain substance. Result of serum pressing on capillaries cause anaemia of brain, which is said to be the cause of the convulsions, but I. Clinical signs of Cerebral pressure from Oedema of brain are absent, II. Oedema of brain not found post mortem (Angus McDonald).

IV. Angus McDonald's. the convulsions are due to a poison of some kind acting on the Vaso motor centres in the Medulla and so causing spasm of the brain capillaries and consequently anaemia of the brain.

V. Blanc's. He claims to have discovered a special bacillus in the urine but not in the blood and thinks it causes both the convulsions and albuminuria (not confirmed).

VI. Reflex irritation generally from the uterus or foetus acting on a very excitable nervous system. This may be put down as a predisposing cause but certainly not as a directly exciting cause, at least before delivery. Some writers say this is the cause in those cases where albuminuria is absent. I do not agree with them as I do/

do not think this sufficient per se to cause puerperal Eclampsia and moreover I found this condition very well marked in a case with distinct albuminuria.

VII. Stumph says Acetone is the poison causing Eclampsia, circulating in the blood causing in its passage through the liver destruction of the parenchyma of that organ, through the kidneys nephritis and through the brain convulsions and coma.

Before leaving the subject of the Aetiology of Puerperal Eclampsia let me remark on the oscillating movements of the eyeballs, and which I have called Nystagmus noticed at the commencement of the first seizure. As this symptom was distinctly present before the fixation of the eyeball (squint) I came to the conclusion that it was due to a slight stimulation of the nerve centres and it gave place to the fixation of the eyeball and subsequent squint and twitching of the muscles of the face as the result of frequent and stronger stimulation.

Nystagmus is derived from (Greek) (to nod in sleep) meaning extreme drowsiness. It is a spasmodic involuntary jerking movement of the eyes, which does not interfere with but accompanies the normal movements of the eyes (Fick). Without entering into detail about this symptom I may say that it is usually associated with defective/

defective sight from youth (neither of my cases had impaired sight, on the contrary both had excellent sight) or with lesions of the Cerebro Spinal system, and this last fact made me conclude that it might go to strengthen the theory of Nervous irritation in the causation of Eclampsia. It is I think another proof that there is more than one influence at work in the production of Puerperal Eclampsia. The severity of the seizures and their resulting coma I think are undoubtedly the direct result of the stimulation of the nerve centres and that the degree of severity of the fits vary in direct proportion to the stimulation received, and this stimulation is regulated by the amount of poison circulating in the blood.

Albuminuria, its relation to Puerperal Eclampsia. Puerperal Eclampsia is a condition intimately connected with pregnancy, labour or the lying in period characterized by convulsive seizures of a tonic and clonic nature, followed by a variable period of coma, and usually associated with albumen in the urine. This I think is the general conception of Puerperal Eclampsia.

It has been proved that albuminuria occurs to a marked degree in the great majority of cases, and it has also been proved that in a few cases of this condition no albuminuria exists. Then again in many normal pregnancies albumen is found/

found in the urine in small quantities without any pathological significance, or no Puerperal Eclampsia result. It is therefore evident although albuminuria may be a common precedent or accompaniment to this condition it is not an absolute factor in the Clinical history of this disease. Most of us however agree with the opinions of Frerich, Simpson, Leishman, Shroeder, Playfair and others, who in their researches proved that albuminuria was almost always a precedent and accompaniment to Puerperal Eclampsia. My cases go farther to prove this but they also prove that albuminuria exists in a variable degree in this condition. In my first case the albuminuria was extreme before labour set in and though considerably reduced by prophylactic treatment Eclampsia occurred. In the second case, the albuminuria just before labour was not very marked, Eclampsia also occurred. This goes far to prove that although the albuminuria may give you an idea of the existing kidney disturbance it does not give you a direct indication of the severity of the approaching convulsions as proved in my second case, where the convulsions were very severe although the albuminuria present would not alone of itself have excited suspicion. The peripheral and uterine reflex irritations were in my opinion the strong predisposing factors in the production of puerperal Eclampsia in this case,/

case, with certainly a functional disturbance in the kidney, probably here a slight condition of the kidney of pregnancy.

The condition known as the kidney of pregnancy lends a very favourable view to the explanation of Albuminuria in Puerperal Eclampsia, if it does not explain its appearance altogether. It certainly calls for some attention, because it is well known in puerperal Eclampsia that after delivery in recovery the albuminuria rapidly disappears, thus proving that it is in reality a temporary arrestment of, or impediment to the functional activity of the kidney, and not a grave morbid lesion. I think therefore that it may be admitted that albuminuria is an almost universal accompaniment to Puerperal Eclampsia whether it may be caused by a slight or an exaggerated condition of the kidney of pregnancy, I cannot definitely affirm, but the view is certainly enhanced by the results of post mortem examinations in those cases.

Puerperal Eclampsia its connection with Puerperal Insanity.- The occurrence of Puerperal Eclampsia in Case II in a patient of weak intellect suggests the possibility of some connection between this condition and Puerperal Insanity, and I venture to assert that if careless or indifferent attention be given during the Puerperium or any untoward accident occurred preventing the disappearance of albumen or the complete restoration of the kidney to its functional/

functional activity, it is not unreasonable to think that Puerperal Eclampsia may result. The kidney as a rule after delivery in Puerperal Eclampsia returns to its normal state but in some cases it may not, and a condition of Chronic Nephritis result. This I think with its albuminuria might predispose to Puerperal Insanity, especially when the conditions were present in a very nervous and irritable patient and one in which the tendency was to Insanity.

Simpson's papers published in 1857 certainly went far to show that there was sometimes a connection between the two diseases. If the kidney in Puerperal Eclampsia does not recover its normal function it may develop a chronic nephritis and this may explain the view of Dr. Donbim who said that all acute and dangerous forms of Puerperal Insanity were in reality cases of uraemic blood poisoning, which theory however is not now held. The opinion of Dr. Fordyce Barker is the more likely theory that mental emotions constitute the exciting cause; therefore mental emotions brought to bear on a patient like my second case of Eclampsia where all the conditions were favourable would be very likely to result in Puerperal Insanity.

All emotion however was carefully avoided in this case with the result that this woman's mental condition was improved by an attack of Puerperal Eclampsia rather than impaired/

impaired by it.

Treatment of Puerperal Eclampsia, Delivery without the aid of Anaesthetics.- From the facts made out in these cases I come to the conclusion that Puerperal Eclampsia has not always a definite clinical history, that the symptoms in each individual case are not always uniformly the same, therefore the treatment adopted should be as far as possible to remove the cause and as in Puerperal Eclampsia the cause or causes are not entirely agreed upon, it should be directed in the direction which will remove or alieviate the symptoms, which give so much anxiety to the practitioner. The treatment when possible should be divided into

(a) Prophylactic

(b) Curative

As I have had experience of those cases it is not always possible that Prophylactic treatment can be adopted. I think the commonest occurrence in cases of this kind is to have it brought under your notice by a fit, but in some cases where you have the opportunity of studying the early or suspicious symptoms of this disease namely, Oedema, scanty urine, albuminuria etc. you ought to push prophylactic treatment to the farthest in the hope of staving off the dreaded complications or alleviating them as much as possible. Thus on the first suspicion of Puerperal Eclampsia put the patient on/

on a milk diet and keep her on it. Regulate the bowels thus removing a source of reflex irritation and recommend a mild purgative such as fluid extract of senna, but have found this does not always answer the purpose in which case I give pil Colocynth Co. If the urine is scanty I give the diuretic before mentioned see page 5, and if the Oedema still continues I give a Guy's pill night and morning. A hydragogue cathartic materially assists the oedema such as Calomel grains V pulv. Jalapae ℈ ℈ gℸxv. An enema is sometimes invaluable where purgative medicines fail.

The skin should be encouraged to perform its physiological functions and hot baths I think are the best means to accomplish this. As the skin is one of the most important channels engaged in the elimination of excretary products I think careful attention to it very necessary, when Puerperal Eclampsia is suspected. Some writers say that if a patient suspected of having Puerperal Eclampsia be treated carefully and kept on milk diet the condition can be averted. I do not think so as in my first case I treated it as conscientiously as possible, and knew that all my instructions were followed to the letter, and yet Eclampsia occurred, and with a very considerable degree of severity. Now if after all your precautions you find that the active symptoms of Eclampsia set in (viz: fits) the treatment must take a very different/

different course, it is no longer palliative it is urgent and "prompt action by averting one fit may save life."

My opinion on the active treatment of this condition differs from most writers and I hope to prove my treatment worthy of consideration as by the means of it I was able to bring two cases of this condition to a successful termination.

The curative treatment of Puerperal Eclampsia therefore should have for its aim the arrest of the fits or more correctly if possible removing the cause of the fits; this has evidently given the greatest trouble to the practitioner and has led to the suggestion of the various forms of treatment, chief of which are the following:-

I. Chloral and Chloroform treatment; this consists in administering 30 grains of Chloral Hydrate per rectum on the first appearance of the fits and repeating it with discretion every two hours until the fits cease. The inhalation of chloroform is commenced at the beginning of a fit and continued till the fit ceases.

II. The other method of treatment is by morphia hypodermically as recommended by (Veit), half a grain is given hypodermically at the beginning of a fit and followed every two hours by a quarter of a grain until the fits cease. Not more than three grains should be given in twenty-four hours.

The Chloral and Chloroform treatment seems to have found/

found most favour in recent years in the treatment of Puerperal Eclampsia.

How does Chloroform act:

I. It subdues reflex irritability.

II. Cuts off perspiration.

III. Blots out memory.

IV. Prevents emotion.

Advocates of the Chloroform treatment say we must procure absolute rest, exclude light, disagreeable ideas, irritation of the skin at the acme of spinal and cerebral irritation just at the commencement of the fit. How does chloroform act here; after a fit the patient lapses into a state of coma, well chloroform by inducing asphyxia accelerates this stage and rapidly renders the nerve centres incapable of ~~f~~ reaction. This I claim is the only advantage to be gained by Chloroform. Does it facilitate or retard labour? It is well known that anaesthesia is often produced in obstetric practice to relieve very painful labours and does so without causing a cessation of labour, but does it not here by increasing the comatose condition already existing retard the progress of labour. To a certain extent then I think chloroform a very useful agent in the treatment of this disease, provided the labour is far enough advanced to introduce forceps, when it may assist considerably, but I will/

will proceed to show that it is not necessary. I am convinced that in the great majority of cases Chloroform will not terminate the labour itself, and I am inclined to think that in primipara death is likely to supervene from depression of the heart's action (heart failure) from the long administration of Chloroform.

From my knowledge then of the action of Chloroform, I do not see the necessity of accelerating the efforts of nature and at the same time increasing the risk of a most dangerous complication, viz: heart failure. Why not apply the forceps without administering Chloroform during the period of Coma, and thus remove the risk of the dangerous complication. The question of shock may here be raised. Well are forceps not commonly applied without anaesthetics without practically any shock resulting; but you may say the sensitiveness to nervous irritability is so increased here, as to contra-indicate forceps. Against this last assertion the extreme degree of coma when it lasts any length of time after a fit allows of the easy application of the forceps, and in some cases of delivery before another fit comes on. This I think I prove in my first case, when I succeeded in delivering the head and in fact the whole child before a succeeding fit expelled the placenta. Even should a fit come on after the forceps are applied and before the head/

head is born, I do not see any particular danger; provided the blades of the forceps are accurately applied to the child's head and held passive until the fit is over. The severity of my first case I think accounted for the easy application of the forceps, and although the comatose condition continued for a longer period than in my succeeding case, I think it was partly due to the large dose of Chloral and Bromide that I administered per rectum, after the third stage of labour was over and which I gave with the object of preventing the recurrence of the fits which drugs I think are invaluable in this stage for rendering the nerve centres less sensible to peripheral irritation.

The question arises here, in how many cases is application of the forceps practical I cannot answer the question generally, but in both my cases I had no difficulty. In the first case there was undoubtedly labour a week previous to delivery, and this I think considerably assisted delivery. ~~by this means~~. In the second case I have only the history of the patient, and the attendant that she had a few niggling pains a day before delivery, this might assist in the second case. I have put down as note-worthy in the second case Epigastric pain, but I rather think the patient may have mistaken this pain in the abdomen for the onset of labour. This reasoning is not unlikely at any rate as Epigastric pain is/

is recorded in Leishman as a symptom of questionable significance.

If these are not assisting causes in the dilation of the Os then the uterus must participate in the Eclamptic seizure for the fact remains that the Os quickly becomes dilated and that to the extent of facilitating the application of the forceps.

I had no trouble in the first case after the expulsion of the placenta, but in the second one abnormal symptoms developed on the second day of the puerperium, evidenced by quick pulse 120 to 130 per minute, hot dry skin, temperature 103.6, cessation of Lochia, which eventually became unmistakeable by terminating in a fit. I examined her carefully per vaginam and found a large clot over the os uteri which I broke down and removed with my finger. I douched out the uterus and vagina with a mild Creotin solution and the case went on excellently after this.

This may give rise to the question of pyrexia in connection with Puerperal Eclampsia, but in my first case the temperature never rose above 103.2 F. and on the second day it was 99°F. In the second case the cause of the pyrexia was this retained blood clot, assisted probably by some emotion as this woman was of a very excitable nature and then the temperature only rose to 103.6°F. falling quickly to normal/

normal on removal of the cause.

This does not bear out foreign writers opinions on the subject as they almost universally associate a high degree of pyrexia with Puerperal Eclampsia.

The morphia treatment I never tried, nor am I likely to, should I be called upon again to a case of Puerperal Eclampsia. My objections to it are based upon bad results, obtained from it in a case of severe pain in an Abortion (fifth successive abortion in same patient), and in which there was a suspicion of kidney disease.

Wherever there is albuminuria to any extent, there is evidence of a morbid condition, or a serious functional disturbance most likely in the kidney. Morphia is strongly contra-indicated as it increases arterial tension and causes cerebral congestion. Therefore I think it is contra-indicated in Puerperal Eclampsia. Belladonna is a drug I have a liking for, as it is good for allaying reflex and spasmodic irritation and this is often a cause of the continuance of a fit.

Pilocaspin I consider a very useful agent in assisting elimination of excretory products through the skin. I gave a hypodermix injection of a sixth of a grain of pilocaspin in my first case, with a very beneficial result as diaphoresis was produced in a short time.

Other/

Other treatments have been recommended, for example:
Accouchment force" and Caesarian Section;
Accouchment force" is to be condemned, for the necessary manipulations in the accomplishment of this operation would certainly produce more fits and would predispose strongly to "shock." even if Chloroform were used. I think Caesarian Section would be uncalled for unless in a case of deformed pelvis.

Venesection was at one time popular but I think the age of bleeding is long time past, as it only reduces the patient's strength, unless in a very plethoric patient it should never be practised.

Cases of the successful treatment of Puerperal Eclampsia by Artificial Respiration have been recorded, but I fear that the necessary manipulations would act as a reflex irritation and produce a succession of fits (unless in rare cases). It might perhaps be used in some cases with advantage after delivery.

In reviewing the treatment of this disease it is necessary to review the causation. It is therefore evident that all the causes have a tendency to produce irritation of the Cerebro-Spinal System by:

I. Reflex by when produced by irritation of the peripheral extremities of sensitive nerves.

II./

II. Directly, when produced by direct irritation of the nerve centres (from poisoned blood.)

The treatment then should be:

I. Moderate central nervous activity.

II. Remove emotions, irritants or excitants.

III. Prevent peripheral irritation.

IV. Eliminate or reduce to minimum all complications.

Shall we put an end to central nervous excitability by delivery and thus remove the exciting cause. The affirmative suggests itself strongly. It is not always possible however or wise, but if it is, and all things favourable by all means deliver. The indication is clear when Eclampsia manifests itself after the sixth month of gestation, for here we have the intense exaltation of the nervous irritability and the poisoned blood constantly keeping it up, and which we know does not disappear until the pregnancy comes to an end.

In concluding my remarks on treatment then I will quote the following rule from Dr. Tyler Smith:

In Puerperal Eclampsia we should adopt that course of treatment which seems least likely to prove a source of irritation to the mother. Thus if the fits seem induced and kept up by the pressure of the foetal head and the head within reach of the forceps, deliver (if everything else be favourable/

favourable). But if on the other hand there be reason to think that the operation necessary to complete delivery is likely of itself to form a greater source of irritation than leaving the case to nature then we should not interfere.

In concluding this paper I should like to add that these cases took place in a country village in Yorkshire, that assistance was practically out of the question, my nearest brother practitioner being five and a half miles distant. I was therefore thrown entirely on my own resources and probably I adopted a treatment which I might not have done had I had the advice of another physician. That my treatment was successful and indicated in both cases, I think is justified by the results I obtained. Although Puerperal Eclampsia is a comparatively rare condition and many men may never be called on to treat it, yet there are exceptions, and I am one who has had the fortune to meet with two cases in the short period of five years private practice, therefore I say one is never safe from the complications which may occur in obstetic practice. I strongly advise the general practitioner and more especially the country practitioner the necessity of being thoroughly acquainted with the means of combating not only this dangerous complication of pregnancy, but all conditions which may occur in obstetric practice.

Remember/

Remember that the mind may be likened to a mirror reflecting only the impressions it receives and if these impressions be the result of careful observation, appropriate treatment of each individual one will almost invariably have a favourable termination.
