P U E R P E R A L E C L A M P S I A

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PUERPERAL ECLAMPSIA.

Puerperal Eclampsia is a term used to denote a condition or symptom-complex presented by pregnant women, of which convulsions are the most prominent manifestation. The term "eclampsia parturientium" would be more correct, as this condition may present itself either in the latter months of pregnancy, during labour, or in the puerperium.

"Its history dates back to the time of Hippo - crates, as he mentions convulsions of pregnant women, and knew that they most often occurred in women who had headache and a tendency to sleep (coma). The word"Eclampsia" means to flash or shine out, and was introduced by Boissier de Savages in 1760 and Gahler in 1776." (De Lee)

Puerperal Eclampsia is a disorder of pregnancy, for pregnancy is the essential factor in its causation. None but pregnant women suffer from this disease and its manifold consequences, and it is directly due to some disturbance of the normal course of pregnancy. The name eclampsia is given to this disease on account of the convulsions forming the chief feature or

predominant symptom of the condition, although there are many other striking symptoms that help to define the malady. But there are many diseases that may occur in pregnant women which have convulsions as their predominant symptom and other symptoms closely resembling those presented by eclampsia, e.g.,uraemia, meningitis, epilepsy and hysteria, and it is necessary to differentiate between them, a matter which in some cases is most difficult.

It is generally believed that puerperal eclampsia is due to toxins in the blood, and it is therefore called a toxaemia. Many theories have been advanced as to the manner in which this toxaemia arises, and the most important of these will be given later; but it is undoubtedly true that the disease of puerperal eclampsia is caused directly by an intoxication of the blood and its effect upon the various organs of the body - especially the liver and kidneys - and not due to any disorders or diseases which may accompany pregnancy.

Eclampsia and albuminuria are so closely associated that eclampsia may be defined as "albuminuria of pregnancy". Very few cases (and they are the exceptions) of eclampsia occur without the presence of albumen in the urine in more or less large quantities,

and it will be shown that the physiological breakdown of various organs of the body producing this albuminuria may be one of the chief causes of the toxaemia or puerperal eclampsia.

Albumen may be present in the urine owing to some pre-existing renal disease, or it may be present temporarily owing to transient causes, such as fatigue or dyspepsia; but the albuminuria of pregnancy which is liable to end in eclampsia seldom manifests itself earlier than the sixth month of pregnancy, and this is so marked that the urine on boiling may become solid. This albuminuria is accompanied by other urinary changes, and frequently by anaemia and anasarca. It often leads to the death of the foetus "in utero" and consequent premature labour, sometimes to retinitis and the occurence of convulsions. The amount of albumen present in the urine is not in itself a true index of the liability to eclampsia, for cases have occurred in which the urine has been loaded with albumen and yet have terminated without convulsions. Casts occur in the urine, which are hyaline and granular; red and white blood corpuscles are also often found. The urine is much less when there is anasarca, and if eclampsia occurs, it is very scanty and sometimes is suppressed. In eclampsia the amount of urea

excreted is much lessened, and the amount of ammonia considerably increased. This is the outcome of a modification of nitrogen metabolism in the system, which is one of the causes of toxaemia. The normal ammonia-coefficient of nitrogen excretion is 3%, and a rise in this coefficient is a sure indication of toxaemia; that of urea is 87%, and this may fall very considerably. There is also a change in the carbohydrate metabolism, which has some bearing upon the increased ammonia-coefficient. It has been shown that two acid substances, viz., diacetic acid and oxybutyric acid, accumulate in the blood and constitute the condition termed "acidosis." The excess of acid in the blood is partly neutralised by the alkaline salts of the blood serum, and whereas the alkaline base of the blood salts is mainly ammonia, the result is that the ensuing combination of ammonia with diacetic and oxybutyric acids, excreted through the kidneys, helps to raise the ammonia-coefficient. Changes such as these in the general metabolism of the body promote a state of toxaemia.

Clinical Features.

The chief feature, then, of this disease is the presence of convulsions, and the eclampsia attack or convulsion may occur suddenly, without warning, but this is not the rule. Almost always there are premonitory symptoms, such as headache, giddiness, nausea, sickness, pain in the epigastrium, spots before the eyes, bright lights, dimness of vision, singing in the ears, etc.,etc. These may appear from a few hours to several weeks before the actual attack, and therefore serve as a warning to the accoucheur. If such are noticed, an examination may reveal the presence of oedema of the feet and eye lids, or more or less marked anasarca, pasty skin, high tensioned pulse, exaggerated reflexes, small amount of urine passed, having a high specific gravity and containing albumen and a smaller amount of urea than normal. If such symptoms are present, the patient is evidently suffering from a toxaemia, and therefore it is possible that convulsions will occur, unless the toxaemic state be immediately improved. In most cases there is a definite rise in blood-pressure preceding the occurrence of fits. A rise above 150 m.m. Hg. in a pregnant woman is a grave warning that eclampsia may supervene, even if the albuminuria is not great.

When eclampsia sets in, the blood-pressure may rise to 180 or 200 m.m. Hg., or more.

Should an eclamptic attack occur, the whole body is thrown into a tonic spasm towards one side, with the eyes turned up and the pupils dilated. The jaw drops and is pulled laterally, and later the mouth opens and closes violently, sometimes severely injuring the tongue, as this organ is generally protruded. Twitchings of the muscles of the face commence in a few seconds, and extend to the limbs and the body, the spasm now becoming clonic in character. These jerkings of the body and limbs may be so great as to throw the person off the bed or against the wall, inflicting severe injury. Foam appears at the mouth, the face is more or less cyanosed, and breathing is completely stopped, the chest being rigid. Gradually the spasm dies down, the convulsive movements becoming slower and weaker, until the patient is quiet; breathing returns, but is stertorous, the cyanosis passes away, and the heart is seen to be beating violently against the chest wall. The patient lies still, and the respirations become gradually more regular and quiet. In favourable cases, she wakes up after a while and appears bewildered, but in bad cases coma sets in, often followed by death. If the patient recovers from the first attack, other attacks may

follow at variable intervals, which, if frequent and many, make the chance of recovery very small, especially if the temperature remains high and the pulse increases and becomes weak and uncountable. But it may truly be said that if the case is treated immediately, medically or surgically, according to circumstances, the attacks will not recur and the patient recovers. Such, then, is an outline of a case of puerperal eclampsia which may present itself to any practitioner, with or without warning.

It is appropriate at this stage to describe the cause or causes of this disease, and in this I have been interested for several years, but have had only little, though interesting, experience in 25 years' practice. From that little experience of actual cases attended by me, I have learned to be always prepared for the possibility of eclampsia arising, and I have therefore been careful to watch the general health - with repeated examinations of the urine- of patients who engage me to attend them, especially during the last 2 to 3 months of pregnancy. I am convinced that owing to this care and watchful treatment, accompanied often by the active treatment of keeping the organs of the body in good functional order, many of my cases have passed through their parturient period normally, which would not have been the case if such cure and treatment had

not been given. Two of the three cases I have personally attended occurred before I adopted this premonitory treatment, but I must say that they were cases in which I least expected the disease to appear, as they both appeared to be most healthy and strong women. In my opinion, it is most advisable that every practitioner should take the precaution of making several observations of the health of his patients during the last three months of pregnancy, and, if necessary, administering the treatment that is deemed advisable. In the case of the failure of one's treatment to aid the organs in their function of elimination, one would still be ready to act in the interest of the mother as soon as it was thought best, even before the appearance of an eclamptic attack. If such a method were adopted, I feel sure that puerperal eclampsia would be rare.

Etiology.

It is generally agreed that puerperal eclampsia is due to toxins being present in the blood, but there are various theories advanced as to the causation of the toxins. According to recent writers, there are two possible sources of the toxaemia in pregnancy:-

i. Maternal,

ii. Chorionic or placental.

The maternal theory is a very strong one, for if it is not the actual and direct cause of the toxaemia, it nevertheless must be an indirect cause, in promoting the retention and preventing the elimination of the toxins in the system.

In health the waste products of the body are disposed of in two ways, viz., by elimination and by transformation. By elimination they are disposed of directly by excretion through the kidneys, skin and intestines. Transformation into harmless substances is effected by the liver chiefly, aided by the spleen and other glands. In normal pregnancy, an excess of waste products may enter the maternal blood, but the organs of excretion and transformation are healthy enough to cope with it without any serious result. But when a physiological breakdown of the functions of these organs occurs, the general metabolism of the body is interfered with and upset, which is bound to entail serious consequences, and

these consequences are much more serious in the pregnant woman than in the non-gravid condition, so that an accumulation of toxic waste products in the blood is the result. This poisoning of the blood, or toxaemia, affects the organs themselves, and pathological changes occur in them which render their function still less adequate. Perhaps the earliest and most prominent result of this auto-intoxication is that which affects urinary secretion, for the urine, almost without exception, is found to be loaded with albumen. Some authorities therefore maintain that the blood-poisoning which results from improper metabolism is the direct cause of the disease called puerperal eclampsia.

The chorionic or placental theory is one which attempts to prove the actual origin of the toxins which poison the maternal blood and eventually produce the disease. Certain elements of the ovum, and not of the foetus (which some writers maintain) are the cause of this toxaemia. Portions of the chorionic epithelium which under normal conditions are detached and enter the maternal blood have been proved by Abderhalden's test to be of a parasitic and toxic nature. It is reasonably thought probable that the antibodies fail to deal effectively with these synctial masses, and hence the presence of syncytium in the blood in abnormal quantity or altered quality. Schmorl

and others have shown that in eclampsia pulmonary emboli containing small chorionic villi and masses of syncytium are sometimes found. These post-mortem appearances support this placental theory, that the maternal blood is altered in its quality by the entrance of bodies from the placenta and chorion. (Eden)

It has been suggested, and the theory is very probably true, that puerperal eclampsia is a manifestation of anaphylaxis evoked by these bodies of placental origin. The entrance of syncytium into the maternal blood is mainly found in the early months of pregnancy, and apparently does no harm whatever, or it may be rendered harmless and non-poisonous by the sufficiency of antibodies in the blood to overcome their effect. Later on there is a renewed active penetration of the maternal vessels by the syncytium, and this, acting upon the combination of previous masses of syncytium with certain elements in the blood, is sufficient to set up a direct toxaemia which is profound in its virility. Many attempts have been made to isolate from the eclamptic placenta toxic bodies capable of reproducing the symptoms in animals, but so far they have not succeeded. This, however, is not sufficient to discredit the theory, which is supported by much indirect evidence, and which reasonably explains the main

features of the disease.

The chief objection, however, to this theory is that in a considerable proportion of cases eclampsia begins after labour. In these cases it is feasible to assume a continuance or exacerbation of the toxaemia after labour - all the more so since it is believed that the condition of toxaemia in eclampsia is a complex one, and such severe degenerative changes have been wrought in most of the important organs of the body, which have produced formidable changes in metabolism, so that it is natural to believe that a severe condition of toxaemia is maintained, even after the source of the toxic bodies which produced these changes has been removed.

There are several other theories as to the causation of eclampsia, but they have not been nearly so generally accepted as the two I have mentioned.

One of the earliest theories was that eclampsia was due to chronic renal disease, and that its symptoms were those of uraemia; but it has been proved by post-mortem examination that the kidneys, in death from eclampsia, did not present the same signs of disease as did those in death from uraemia following chronic renal disease.

Many believed that eclampsia was due to the

dilatation of the ureter owing to the pressure of the gravid uterus, and this producing dilatation of the pelvis of the kidney. This theory has been disproved by showing that ureteral dilatation often occurs in normal pregnancy.

It has also been advanced that the cause of the disease was bacterial infection, but hitherto no organism has been isolated.

Some believe that the placenta itself produces the toxins, and the placenta has often been minutely examined to prove this, but without result.

The most advanced theory, and the theory that is most likely to be true, is that the eclamptic toxins which enter the maternal blood are derived from the foetal membranes. These have a deleterious effect upon the maternal system, and are aided in their poisonous work by other toxins derived from the disorganized functions of the liver, kidneys and other viscera, and from areas of tissue in those organs which have undergone necrosis. The combination of these sources of poisoning produces an acute toxaemia.

The foetus is subjected to the same toxaemia

as the mother, the source of it being the same, viz.,the placenta. In my experience, and in that of many writers, the foetus has been observed to be convulsed, both in utero and after delivery, the convulsions of mother and child being often synchronous. That the same poisonous bodies from the placenta cause the same toxaemia of child and mother is proved by the fact that,by post-mortem examination,changes in the organs of the child have been found to be similar to those changes in the organs of the mother - especially when the child has died of convulsions. But often the foetus dies before term, owing to the toxaemia set up by impaired metabolism and consequent albuminuria of the mother.

Pathological Condition.

The pathological condition of the various organs produced by eclampsia is as follows:-

Brain. There is flattening and some oedema of the convolutions, sometimes with anaemia and sometimes with congestion. Small or large haemorrhages or areas of cerebral softening, with thrombosis, are generally to be found.

Liver. Changes in this organ are almost invariably found, and they are of so great an importance that they are regarded by some writers as lesions which are characteristic of the disease of eclampsia. Haemorrhages beneath the capsule and on the cut surface can be seen by the naked eye; they may be small and numerous, or by running into each other may be seen as large areas.

On examination by the microscope, the following changes are found:-

i. Degeneration of hepatic cells, beginning at the periphery of the hepatic lobule and advancing in some areas to total cell destruction.

ii. Interstitial haemorrhages, sometimes diffused or slight in extent, or forming large haemorrhages with compression and alteration of the liver substance. Deposits of fibrin are also found.

iii. Thrombosis of vessels - mostly small ones, but occasionally large ones are affected.

iv. Degenerative Changes are found in the endothelial lining of the capillaries, and it is probable that this accounts for the interstitial haemorrhages.

Kidney. Definite changes are always found in the kidneys in cases of death from eclampsia. These are without doubt secondary either to the general toxaemia or to the disease of the liver. The changes consist of degeneration of the cortex. The kidney is generally enlarged, with the cortex swollen and pale, the pallor being due to vaso-motor spasm of the cortical arterioles. With the microscope it can be observed that there is cloudy swelling, with granular and fatty degeneration of the epithelial cells of the convoluted tubules. Thrombosis is often noticed in the capillaries of the glomeruli, and small interstitial haemorrhages and patches of necrosis are seen in the cortex. Dilatation of the ureters - especially the right - is frequently found, but this has no significance.

Heart. The muscle is fatty, with small haemorrhages, necroses and thrombi, and easily tears. Fatty heart is often found in those patients to whom large doses of chloroform and chloral have been administered.

The Foetus. Some writers have found changes in the child which correspond very closely with those in the mother, especially when the child died of convulsions.

Treatment.

Before describing the treatment of puerperal eclampsia, we should bear in mind the important treatment of pregnant women presenting signs and symptoms that lead us to believe that eclampsia will probably, or even possibly, occur before parturition is accomplished. This has been previously referred to, and I am certain that too much stress cannot be laid upon the importance of it, as doubtless many lives of mothers and children can be saved by a general treatment of the pregnant woman during the last 8 to 10 weeks of pregnancy, though this is not a treatment of eclampsia in a strict sense, but a treatment of a condition - albuminuria most frequently - which is liable to cause the disease of eclampsia, or considerably help in its causation. It may therefore be correctly styled the prophylactic treatment of eclampsia.

The treatment of eclampsia begins directly the first convulsion occurs, which definitely demonstrates what disease is actually present. It may be

(1) Surgical,

(2) Medical,

(3) Medical and Surgical,

or, in other words, there are three general plans of treatment.

(1) Surgical:- After the first convulsion, put the patient into a deep sleep and deliver at once.(Dührssen)

(2) Medical:- Put the patient to bed, protect from injury, light, noise, give narcotics, chloral and morphia, stimulate the emunctories, bleed, etc.,etc., and await the natural termination of labour. (Stroganoff)

(3) Medical and Surgical:- Those who adopt this method use every medical means in their power to combat the disease, and only resort to surgical means when they see the patient going from bad to worse.

Much may be said in praise of each method, and accoucheurs are divided in their opinion as to which is best. Experience goes to prove that the sooner the uterus is emptied the better, as the convulsions are not so frequent or so strong, and sometimes even do not occur, after the child has been separated from the mother. (Carl Braun) Dührssen and Olshausen showed that in 93.75% and 85% respectively such a result was found. It has been shown that the mortality is very much lower in cases of rapid delivery after the first convulsion than otherwise.(Peterson,Freund, and Stoeckel) But it must be borne in mind that such immediate surgical treatment can only be given

in special circumstances, such as residence in hospital, surgical home, or the houses of the wealthy. What of the majority - the vast majority - of pregnant women, who have to be attended wherever they happen to live or exist? It is not always possible or expedient to remove a patient to the nearest hospital (if there is one) directly the dread disease manifests itself.

Again, to show how circumstances influence, and in fact dominate, the choice of the method of treatment: in thousands of cases, surgical treatment could not be carried out in the patients' homes with any hope of success, as a woman in eclampsia is most liable to sepsis. Also, very,very few medical practitioners are sufficiently skilful to undertake such an operation as Caesarean Section. Therefore, as a case arises, the best method of treatment possible must be adopted, and that is Stroganoff's method. Each case must be judged carefully, and treatment must be given according to the circumstances and conditions that prevail, for when adopting the medical treatment, it may still be advisable to deliver immediately by instrumentation, if the os is fully dilated, or to employ gentle means of dilating the os in order to effect an early delivery.

The Surgical method of treatment is therefore the best whenever it can be adopted, and in the interests of the mother it is begun as soon as possible after the first convulsion.

Caesarean Section "per abdomen" is the simplest and most rapid method of emptying the uterus, and is the method most generally chosen.

Caesarean Section "per vaginam" is an operation requiring more skill and experience, and is used in hospital when it is considered best.

The selection of the anaesthetic is a very important matter in these operations, especially so as the body is in a profound state of toxaemia and the vital organs are all more or less affected thereby, and in this condition chloroform, ether, and nitrous oxide, have a greater destructive action on the liver, kidneys and blood than in the normal state. Chloroform sometimes gives rise to symptoms of grave poisoning, when administered to persons not suffering from toxaemia; therefore in eclampsia especially it should never be used, as it is liable to produce fatty heart and acute yellow atrophy of the liver. Ether is the safest general anaesthetic for operations in this disease, but local anaesthesia with novocain supplemented with nitrous oxide and

oxygen is greatly recommended. (De Lee) Some operators prefer spinal anaesthesia for operations in eclampsia, or some form of local infiltration anaesthesia, but if this be unobtainable, they invariably rely upon the administration of ether. (Eden)

Another surgical method is that of rapidly dilating the cervix, by the hand or by various kinds of dilators. This is called delivery by "accouchement forcé", and is to be condemned, as it produces a great amount of shock and is attended by dangerous lacerations of the cervix and bruising of the parts, which of themselves greatly endanger the life of the mother through sepsis, to which in eclampsia she is peculiarly liable.

The Medical Treatment of Eclampsia.

The most important and the most urgent object to achieve is (a) to eliminate the toxins,

(b) to treat the toxic symptoms.

The object of eliminative treatment is to try, by all possible means, to eliminate the poisons in the blood and tissues, and various methods are adopted with this end in view, e.g.:

1. Venesection. Several ounces of blood may be drawn from some convenient vein, and this at once reduces the amount of poison in the body, and at the same time

reduced the blood tension. This method cannot be made use of when there is marked anaemia and anasarca.

2. Saline Transfusion. This consists in introducing one to three pints of a slightly alkaline solution, which will reduce the concentration of the toxins in the blood and thereby lessen their poisonous effect. In eclampsia the alkaline solution is used in order to neutralise the excess of acid bodies which is present in that condition. For this purpose, 30 grains of acetate of soda is added to each pint of normal saline, as recommended by Jardine. The saline solution is prepared by dissolving a teaspoonful of common salt in a pint of boiling water and cooling to a temperature of 103° - 105° F.

This method can with advantage be adopted after venesection has been carried out, for the same vein could then be used. A saline transfusion to a certain extent promotes diuresis and diaphoresis -a direct elimination of the toxins through the kidneys and the skin. The saline solution may be used subcutaneously or in the form of an enema, but the absorption of the fluid is not so rapid as by the intravenous method. But if the rectum is empty and the bowel fairly clear, the slow continuous method of the saline enema is excellent treatment. A great quantity of the saline fluid may be used when allowed to enter slowly and

under low pressure. The solution should be held a few inches higher than the rectum.

3. Diaphoresis. It is very essential to ensure free action of the skin, in order to aid the process of elimination and to reduce the blood pressure. Drugs which promote good sweating, such as pilocarpine, phenacetin, sodium salicylate, etc., are all heart depressants, and therefore should on no account be used, but external means are best in the form of the hot pack or electric heat bath.

4. Treatment of the Alimentary Canal. This is most important, as here we have a very wide area in which toxins abound, and it affords a splendid opportunity of ridding the system of much poison, and at the same time of reducing the concentration of the poisons by absorption of the alkaline solution. This method of treatment has been attended with great success in the Rotunda Hospital, and consists of gastric and entero-lavage and the administration afterwards of bicarbonate of soda. Purgations have often been employed, but they are contra-indicated in view of a possible subsequent operation. The bowel should rather be irrigated until it is clear of all foecal matter, and then 2 to 3 pints of the saline solution can be injected by means of a rubber tube 12 inches or so in length.

5. Decapsulation of the Kidneys - an operation with the object of overcoming anuria. This operation is seldom resorted to, unless it is found that after emptying the uterus extreme anuria persists. In some cases it has proved successful.

Clifford White advocates this operation, and has had successful results in six cases in which he has attempted it. He attributes the suppression of urine in pregnancy toxaemia to pressure on the collecting tubules, due to increased tension inside the fibrous capsule of the kidney, and is of opinion that this operation may be advantageously combined with Caesarean Section in suitable cases.

Fothergill also has made use of this operation, and has had excellent results. It is an operation advocated also by Edebohls.

"Chirié collected 30 cases with a mortality of 46%, whereas Littauer reports 62 cases with 20 deaths." (Eden)

"Both theory and practice speak against this operation." (De Lee)

Treatment of the Toxic Symptoms.

The first definite symptom of eclampsia denoting the presence of toxins in the system is the onset of a convulsion. This may occur at any time, before labour has commenced, during labour, or after the birth of the child. This symptom - perhaps the most important one - must have immediate treatment, for the severity of the convulsions varies, but in all cases they are a severe strain upon the heart, and unless they are modified and controlled as far as possible, they may be the immediate cause of death. The patient must then be protected against injury, for sometimes the convulsions show frightful vehemence. She should be placed on the bed, quietly undressed, surrounded by pillows, and watched constantly by an attendant. The patient should be placed on her side, to allow of the free escape of secretions from the mouth, and to allow the swollen tongue to fall forward and aid respiration. To save the tongue from being bitten, it is best to place over it and between the teeth a folded handkerchief, a wooden peg, or the handle of a spoon covered by a handkerchief, and this should be held in position until the convulsion has ceased. As soon as possible a hypodermic injection of $\frac{1}{4}$ or $\frac{1}{6}$ gr. morphia and $\frac{1}{100}$ gr. atrop. sulph. should be administered, with a view to

quietening the very excited nervous system and con - trolling the recurrence of the convulsions. This may be repeated every 2 or 3 hours, if necessary, until quite two grains of morphia have been administered. This is without doubt the greatest aid in controlling the convulsions by allaying the irritability of the cerebro-spinal system. Some object to the use of morphia, on the ground that it is likely to stop the secretion of urine; but the secretion is already more or less impeded by a vaso-motor spasm of the arterioles of the kidney, caused by toxins in the blood. It has been conclusively proved that morphia does not act in this manner, but,on the contrary, not only arrests or modifies convulsion and spasm, but also tends to increase the flow of urine. The use of morphia has a prominent place in the medical treatment of eclampsia, and is strongly recommended by such writers as Stroganoff, Nagel and McPherson.

Next to morphia, the use of chloral hydrate in combination with bromide of potassium should be adopted, and these may be given by the mouth between the convulsions or per rectum. These narcotics should not be used repeatedly in severe cases, but only in those that return to consciousness between the convulsions. In cases where coma results, they are contra-indicated.

If labour has begun, it is often found that it is accelerated by the use of morphia, and when the patient is under its influence and has recovered from the initial convulsion, then all efforts should be used to bring about the completion of labour, by resorting to gentle means of dilatation, and if this is not required, then the gentle use of the forceps to extract the child.

Chloroform has often been used both to control the convulsions and as a general anaesthetic in operative treatment, but it is not to be recommended, on account of its deleterious action upon the heart and liver. The best general anaesthetic is undoubtedly ether. Veratrum viride has been highly recommended on account of its further action in lowering arterial tension, but it is liable to bring about a condition similar to shock.

Medical treatment is therefore the best to adopt in mild cases of eclampsia, for the majority of such cases occur a few hours after the onset of labour, and the presence of eclampsia rather tends to hasten the end of labour than otherwise, for the uterus continues to contract powerfully, and the child is very often premature; therefore in many cases the labour is rapid and easy, notwithstanding the initial and perhaps solitary convulsion. When eclampsia occurs before

the onset of labour, be it mild or severe, the outlook for good results from medical treatment alone is anything but favourable. Labour should be induced as quickly as possible, and yet not forcibly, and every form of medical treatment should be used; but surgical treatment must then be considered and decided upon early, if the patient shows signs of weakening and the toxaemia and its symptoms show no signs of lessening.

My personal experience of this disease has been limited to three cases, the details of which are as follows:-

Case I.

Eclampsia occurred in a young, strong and healthy-looking primipara, in whose case I had not the slightest suspicion of the disease. On being called to the case, I found that labour had well set in, the os being partly dilated and pains coming fairly frequently. She was very nervous and excitable, and whilst I was examining her "per vaginam", a convulsion occurred - not a violent one, but a convulsion much resembling an epileptic seizure. This eventually passed off, and after a period of mental dullness, the patient regained consciousness and became hysterical. I naturally

concluded that it was a case of hystero-epilepsy. I questioned the mother, who was present, but could ascertain no history whatever of epilepsy in the family, and the patient had never had a fit in her life before. In about 20 minutes another convulsion occurred, of just as mild a type as the former, and this was succeeded by another in a short space of time. Nevertheless, the patient became quite sensible, but remained very excited, between the convulsions. I gave her a dose of chloral and bromide of potassium by the mouth, and left another dose to be given in two hours' time. I left her in charge of the nurse and mother, instructing them not to leave her for a moment. In about two hours' time I was sent for, as the patient was having "fits" more frequently. I at once gave her a hypodermic injection of morphia $\frac{1}{4}$ gr., with atrop. sulph. $\frac{1}{100}$ gr., which had a wonderfully soothing effect upon her - so much so that I was able to dilate the os by the digital method without exciting the nervous system sufficiently to incur a repetition of the convulsions. The patient remained quiet for over an hour, labour pains doing their work so well that I was then able to apply the forceps and deliver her of a living child, without any further trouble.

After the expulsion of the placenta I gave her another ¼ gr. Morphia + Atrop. Sulph. 1/100 gr. Hyp. Inj. and left. Her convalescence was so normal as to be quite uninteresting. I obtained a sample of urine next day and found it contained albumen in a large quantity, and yet there was no oedema of the feet and legs or puffiness about the face, or scanty urination. I attended her again 2 years later, in quite a normal labour, and I have since known her, but she has never had a semblance of a convulsion, fit or attack of hysteria. This was my first experience of convulsions in pregnancy, and I had great difficulty in making my diagnosis; but judging from her previous and later history, I ultimately had no doubt but that it was a mild case of Eclampsia.

Case II.

In my second case again Eclampsia was quite unexpected, and fortunately was of very short duration, but whilst the convulsion lasted it was horrible in the extreme. The woman I had never seen before, as she had not engaged me, and I was called hurriedly to the case as she was in heavy labour. She was a primipara, a very heavily-built woman, apparently as strong and healthy as could be. She was screaming and shouting with labour pains, which occurred about every two minutes, throwing herself about the bed in every conceivable manner. I found the perineum distended with the descending head of the foetus, and after

firmly advising her to keep quiet and as still as possible for a few minutes longer, I proceeded to deliver her with the forceps. Directly I attempted to apply them, the patient became violently convulsed and it required all the efforts of myself, nurse, and mother to hold her down and prevent her from injuring herself and us. This convulsion was so sudden and unexpected and the woman so strong as to be violent, that I had no chance to administer morphia, $CHCl_3$ or Ether or anything, but just had to wait and see it through one way or the other. She became so livid that I thought she must surely die in the attack; but in time the clonic spasms became less and her breathing gradually became more regular, until she was quiet, stupid and drowsy. Shortly afterwards I gave her a Hyp. Inj. Morph. $\frac{1}{2}$ gr. and after waiting for its physiological effect I delivered her with instruments of a living child without any trouble whatever. She made a good recovery, and knew nothing of what had happened. I examined her urine and found it full of albumen, but this very soon disappeared altogether.

Following the experience of such cases I have since invariably made it my business to know the true condition of every patient who engages me to attend her confinement, especially during the last 2 months of pregnancy. My method is not only to examine the urine, and this several times if there is the slightest

albuminuria, but also to enquire into the general health of the patient, paying particular attention to the state of the digestive organs and the nervous system. I am positive that my prophylactic treatment has not only saved me much trouble and anxiety, but I believe it has helped to save many a life of mother and child. During my 25 years' experience as a general practitioner, I have delivered upwards of 2,300 women, and I attribute my practical freedom from preventable disease to the close investigation and treatment, if necessary, which I make beforehand.

Case III.

But my last case was that of a woman I had kept under observation and treatment for some time before her confinement, and yet even then she passed into the eclamptic state. In all probability she would have suffered from the disease much more severely had she not undergone preparatory treatment.

This was the case of a strong, well-built, healthy-looking woman, who was pregnant for the first time. She was highly excitable and nervous about her condition, and held the belief, which she frequently expressed, that she would not survive her confinement. During the last 2 months her legs began to swell, and on examination of the urine albumen was found in a considerable quantity. I ordered her regular gentle exercise, to keep warm and free from catching colds, a light diet and a mixture of Mist. Rhei. Co..

The albumen remained very abundant notwithstanding, and she began to swell in all parts of her body, and even the face and eyelids became puffy. I then put her on a good diuretic, and acted upon her liver by giving Mercury and Colocynth pills. Later I restricted her diet to that of a milk one, with the result that the albuminaria became much less. The anasarca remained the same. When I was called to attend her I found that with very slight pains at long intervals there was yet considerable dilatation of the os and that apparently labour was proceeding most satisfactorily. In about 3 hours afterwards I was called again and the patient was then in severe labour, and it appeared as if my very entrance into the room so excited and agitated her as to send her into a most violent convulsion, requiring the help of three persons to restrain the movements of the woman and keep her on the bed. As soon as possible I administered a Hyp. Inj. of Morphia $\frac{1}{2}$ gr. and Atrop. Sulph. $\frac{1}{100}$ gr., and very soon the convulsion expended itself and in a short time left the patient quiet but more or less bewildered. On examination I found labour advancing but not sufficient to warrant my use of the forceps. Strong labour pains continued at regular intervals for $\frac{3}{4}$ hour when another convulsion seized her, but this was not so strong and frightful as the first. I am of the opinion that during this convulsion I could feel the convulsion of the foetus through the abdominal

wall of the mother. Soon after this convulsion had passed away I was able to apply the forceps without risk of lacerating the cervix. I delivered her without trouble, but immediately on the birth of the child, another convulsion occurred; and synchronously the child on the bed was convulsed before I had time to separate it from the placenta. Very free haemorrhage followed, for which I was glad, as it must have rid the patient of much of the toxines. The woman eventually had two more convulsions which were much less severe, and afterwards made a slow but uneventful recovery. I attended her for the birth of a second child and this confinement was perfectly normal, and she never had the slightest albuminaria since her first pregnancy.

Prognosis.

The prognosis in Eclampsia - the disease perhaps most dreaded by accoucheurs - is always very serious for the mother and the child: and the reason for this is not only the nature of the disease itself, but the many circumstances that invariably surround the case, such as:-

(a) the severity and number of the fits;

(b) the treatment adopted;

(c) the time when eclampsia presents itself:-

(1) latter end of pregnancy;

(2) during labour, or

(3) during the puerperium;

(d) the relation of patient to other co-existing disease.
(e) her liability to sepsis.
etc.

The greater the number of fits the more serious is the prognosis. When they are prolonged, when the temperature steadily rises and when there is early and continuous coma, the outlook is very grave. Eclampsia has the highest mortality during pregnancy, less during labour, and it is generally believed to be least during the puerperium. Death may ensue from so very many causes, such as:- exhaustion, heart failure, toxaemia, embolism, cerebral haemorrhage, asphyxiation etc., or by the results of severe operations undertaken to deliver her, post-partum haemorrhage and sepsis etc., so that it is very necessary to be on one's guard in giving a prognosis. Some of the worst cases of Eclampsia have occurred after the birth of the child, but in the majority of cases there is hope of recovery once the uterus has been emptied, and if after this there is a diminution in the severity of the symptoms, there is then good reason to entertain that hope.
It is generally held that about 20% of women afflicted with eclampsia die.
The prognosis for the child is necessarily worse than for the mother, as in addition to the many causes which may result in death, the child is often sacrificed - sometimes intentionally - in the interests of the mother. About 50% of children die, either from (a) prematurity, (b) toxaemia, (c) asphyxiation due to the repeated convulsions of the mother,

(d) injuries sustained during birth, especially from forced delivery, (e) convulsions, and sometimes (f) poisoning by drugs administered to the mother. It is therefore the wisest and safest course to give a very guarded prognosis concerning both the mother and child.

Statistics concerning the mortality of mother and child in Eclampsia vary so considerably in different countries and in different hospitals, due largely to the various modes of treatment adopted, that they cannot be taken as a reliable guide.

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