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The Prophylaxis of Puerperal Eclampsia.

By DAVID HARDIE, M.D.,

*Hon. Physician Lady Lamington Hospital for Women, Consulting
Physician Lady Bowen Lying-in Hospital and Sick Children's
Hospital, Brisbane, Queensland.*

THE Government Statistician has supplied me with the following statistics for Queensland:—

		No. of Confinements in Five-yearly Periods.		Deaths from Eclampsia.		Death Rate per 100,000 Confinements.
1886-90		69,495		41		59
1891-95		72,183		27		37
1896-1900		70,290		41		58
1901-05		68,189		43		63

Taking the mortality as roughly 25 per cent., these deaths represent, approximately, 600 cases of eclampsia that have occurred in Queensland in twenty years.

The death rate in England and Wales (1881-1903) was 39 per 100,000 births.¹

It is unnecessary to summarize the various theories held regarding the etiology of eclampsia, as they may be found in any standard work on obstetrics. Neither is it within the scope of this paper to refer to the various ailments associated with pregnancy. It is sufficient for my purpose (1) to refer briefly to the signs and symptoms that give distinct warning of eclampsia, and (2) to indicate some of the measures that should be adopted to prevent the development of such symptoms, and, if possible, to avert convulsions.

I. Premonitory signs and symptoms.

With these we are all familiar—headache, vomiting, giddiness, and œdema of the ankles, while, if convulsions are impending, there are more pronounced cerebral symptoms, œdema of the face, and severe epigastric pain. We inquire about the urine, and find that it has been scanty, that it has a variable specific gravity, and that albumen is present. I have placed the urinary condition last in sequence, because it is generally discovered subsequent to the establishment of the earlier and even the later premonitory symptoms. Boxall¹ puts this point very forcibly and clearly. “Unless œdema sets in, or some other symptom . . . leads the patient to seek advice, no attention is directed to the condition, until it has persisted for a considerable time. Indeed, it not infrequently happens, that the occurrence of an eclamptic fit is the first indication of danger. Then the urine is examined and is found to be loaded with albumen. But on reading the case backwards, in the light of the history subsequently obtained, it is fairly obvious that a more or less dangerous, but obscure condition has been in existence for some time.” Playfair states, what are probably the usual experience and practice of medical men when he says, “Another important premonitory sign is œdema . . . which should at once lead to an examination of the urine.”² My plea is, that the urine of every woman should be examined regularly during the latter months of pregnancy, as if she were a possible subject for eclampsia, whether she has any premonitory symptoms or not, and whether she has, or has not, any evidence of renal insufficiency. In other words, the urinary examination, unless in exceptional cases, comes first, the premonitory symptoms come after, if they will. If everything goes well, no harm is done, while if albumen appears, we are prepared to deal with the patient’s condition in time. Perhaps in no case is it more important to be forewarned of impending danger. It matters not what theory is held as to the causation of these convulsions—whether the nephritis is primary, or secondary to toxæmic agencies of maternal or foetal origin—the fact remains, that we have in the presence of albumen in the urine, evidence, and perhaps the only evidence present, that there is something wrong.

True, albuminuria is said to be sometimes absent, though personally I have never failed to find it. It must, however, at least be conceded, that its absence is one of those rare exceptions that prove the rule. As an explanation for its absence, Berkeley says, “It has been shown that one effect of these poisons is to cause constriction of the renal arteries and anuria, which would account for those cases

where no renal change is found post mortem, and no albumen is found in the urine.”³ One of the clearest cases of absence of albumen is that reported by Handfield-Jones, where there was no albumen three hours previous to the onset of convulsions. Other cases mentioned are more doubtful, and some, at least, only show the necessity for examining the urine at shorter intervals. Longridge describes the case of a woman who had had eclampsia in her first confinement, whose urine, in her second, had been examined up to the 9th month of pregnancy with negative results, who was then sent to a home to be “under supervision,” and who when admitted to hospital, at a later date, developed convulsions, the urine being then found to become “nearly solid on boiling.”⁴ It would be of interest to know if her “supervision” included weekly or bi-monthly examination of the urine during the ninth month. Jardine, who says “he has never yet seen a case of eclampsia without albumen in the urine,”⁵ records a most interesting case of a woman who, for some months, complained of severe headache, who had no œdema, whose eyesight had been affected, and who subsequently had severe fits, and yet “there was no albumen in the urine at the time of admission, nor at any time during the stay in hospital.” He does not, however, look on this case as one of true eclampsia. Again, he relates the case of a patient at full term, whose urine was free from albumen less than a fortnight previous to the onset of convulsions but was now found to be heavily loaded with albumen. The very occasional absence of albumen does not invalidate what has been said as to the propriety of looking for it in every case of pregnancy.

It certainly does not follow that albuminuria necessarily leads to eclampsia, or that the actual amount present is of much prognostic significance, as the “albuminuria and severity of the eclampsia are not co-related in any constant manner.”⁶ Indeed, it is believed by Gutbrod that when, in eclampsia, “the albumen is small in amount, the eclamptic seizure is a very severe one,”⁷ from which he draws the conclusion, that the poison is sometimes eliminated not so much by the kidneys as by the skin.

As regards the urinary examination itself, it is specially needful, when albumen is present, to ascertain the approximate quantity of urine passed in 24 hours. This is valuable in its relation to the specific gravity and amount of urea and urates excreted, for while the specific gravity may be apparently high enough, it is really low for a concentrated urine. The higher it is, the more hopeful the outlook, as it indicates a larger excretion of urinary salts. The presence of blood and granular casts is of course of great importance.

So far I have referred only to the urinary test, because albumen is not only almost invariably present in eclampsia, but usually appears before any other sign or symptom. The next most trustworthy signs, in point of time and frequency, are œdema of the ankles and headache, and, if strong objection be made to urinary examination, it is an easy matter to request the patient to inform us if either or both of these should show themselves.

II. *Preventive Measures.*

Much may be done during the earlier months of pregnancy, by taking outdoor exercise, and by general attention to the hygiene of the home. The bowels should be moved daily. A glass of cold water night and morning, in addition to the ordinary fluids taken during the day, will help to ensure this and will promote urinary secretion. Cold bathing every morning should be encouraged during the summer months at least, but towards the end of pregnancy warm baths are to be preferred. The diet should be plain and nutritious, and stimulants should be avoided. In the last two or three months of pregnancy, the amount of exercise taken will depend on circumstances, but should never reach the point of fatigue. In no case, at this late period, should more than one nitrogenous meal be taken in the day.

Should there be any indication of renal insufficiency or other premonitory symptoms, the preventive measures to be adopted depend upon the time when the patient's condition is first observed, or in other words, upon whether the symptoms are those that *may* ultimately lead to eclampsia, or those that certainly *will* if the patient is not relieved; my contention being that if the former be attended to, the latter will seldom occur. In other words, I look on the difference between early or subacute toxæmia, and the toxæmia immediately preceding convulsions, as a difference only in degree of the poison.

(a) *In the early stage. (Subacute Toxæmia).*

Until we arrive at a better understanding of the exact pathological condition with which we have to deal, the measures adopted must necessarily be more or less empirical. We may, however, accept as a working hypothesis (whether there is any special toxin in the system or not), that there is defective metabolism of nitrogenous elements, brought about by degenerative changes in the liver, and perhaps also by deficiency of thyroid secretion, and that there is lessened urinary elimination of waste products due to parenchy-

matous nephritis. No matter then what the real origin of the disease may be, our object, so far as we know at present, must be of a two-fold nature—to reduce the intake of the nitrogenous foods, and to assist the elimination of toxin and effete matters by the kidneys and bowels; for it must be remembered that “health depends on a proper adjustment between the production and excretion of toxic substances, an increase of the former, or diminution of the latter, leading to toxæmia.”³ In this connection, Hahl¹⁵ has shown that in the last month of pregnancy the urinary excretion of nitrogen does not balance the amount absorbed. The difference is greatest four weeks before full term, less during the next two weeks, and greater again as labour is approaching. The curious point is, that, according to other observers,¹⁵ the nitrogen balance is maintained better by the primipara than by the multipara. If that be so, some other factor must be introduced to account for the greater frequency of eclampsia in the former.

In order to control the nitrogenous element in foods, there is a general consensus of opinion in favour of milk diet, inasmuch as in the amount usually given, it contains less proteid material than the ordinary full mixed diet, and is more easily assimilated. If, however, milk be taken to excess, I have found the albumen to increase in the urine. This is more likely to happen should the milk be unskimmed, showing that an excess of hydrocarbons, as well as proteid material, is objectionable. Four quarts of milk in 24 hours are about the equivalent of an ordinary mixed dietary,¹⁴ but the patient whose case we are now considering must be satisfied with perhaps half this quantity, or, it may be, only a quart a day. On one occasion, the amount of albumen present and the headache could only be controlled by reducing the quantity of milk to 30 fluid ounces a day. This was kept up for two weeks previous to confinement—the labour being natural. This result was all the more pleasing, because in the patient's first pregnancy, four years earlier, labour had to be induced for threatened eclampsia. The quantity of milk prescribed must really be regulated according to circumstances. The smaller the quantity ordered, as indicated by the severity of the symptoms, the more should it be diluted with water, to make the toxins less concentrated, and to help in their elimination. So beneficial, indeed, is water in this respect, that I have frequently allowed patients, with some albumen in the urine and even with slight œdema, who dislike milk, to take one nitrogenous meal a day, preferably in the form of fish or fowl, and with apparent benefit, provided they also take at least two pints of water a day, between meals.

Should this diet be given when albumen is present, the urine must be examined twice a week, and a trustworthy report given concerning the œdema, so as to change to milk alone, if necessary. In connection with milk diet, Herman thinks that while it may be beneficial, it is of "less importance than rest," and he himself has not "seen an account of cases treated by milk diet without rest."⁸ Rest is, in my opinion, necessary only in advanced cases. Patients are grateful for fresh fruit and green vegetables, and these are generally allowed. Farinaceous foods may also be given, unless in stubborn cases. They should be reduced in quantity if butchers' meat is allowed in the presence of albumen, as they act as "proteid sparsers."¹⁴

The use of drugs plays but a very secondary part in the prevention of eclampsia, and will continue to do so until the etiology of the disease is better understood. The most we can do, until a serum is discovered to counteract the toxin, is to assist elimination. The necessity for diuretics depends on the amount of urine passed, the citrate and acetate of potash being the best, whilst the bowels must be made to act daily. The treatment by thyroïdin is based on the supposition that there is a want of iodo-thyrin in the system, and certainly the enlarged condition of the thyroid gland, in eclamptic cases, suggests an effort on the part of nature to make the supply equal to the demand. Thyroïdin aids the metabolism of nitrogenous substances, completing the process "up to the stage of urea formation," urea being in itself a powerful diuretic.³ Whether this treatment is to be a success remains to be seen. Sometimes the results are brilliant and sometimes negative. My own experience of it is limited, and the results have been doubtful. Sturmer claims to have reduced the mortality in eclamptic fits, from 40 to 12·2 per cent. by means of it, the most marked effect being an increase in urinary secretion, the amount in one case rising from a few fluid ounces to 120 in 24 hours.⁹ Gomot relates a case, where, under an absolutely milk diet and thyroïdin, the albumen was reduced from 12 grammes to 2 grammes per litre, the labour terminating naturally.¹⁰ Probably the treatment did good, but I have seen equally good results from milk diet alone. Berkeley remarks that milk does not require so much natural secretion of iodo-thyrin, for its metabolism, as does ordinary diet.

(b) *In the late stage. (Acute Toxæmia.)*

The treatment here practically resolves itself into the treatment of eclampsia, into which I do not intend to enter, beyond referring to a few points that seem specially important. First in importance,

comes the withdrawal of food altogether for from 24 to 48 hours, water alone being given, and, where there is not much œdema, given in large quantities, to drink. Saline injections into the rectum are also given, unless the œdema is very decided, and for a similar reason, viz., to dilute the toxins and to increase the action of the skin and kidneys. Jardine claims that by the injection of saline solution—sodium acetate and chloride—under the breast, he has reduced the mortality from eclampsia, in the Glasgow Maternity Hospital, from 47 to 17 per cent.⁵ I prefer rectal injections in cases where the eclamptic seizures are only *threatening* to occur, because injections administered under the breast give considerable pain. The first injection, which may be of plain hot water, should be a very large one, introduced through a long rectal tube and allowed to come away at once; the object being to wash out the large bowel for the removal of all foreign matters, including probably some toxic substances. The saline injections following, should be given every three hours, using each time as much as the patient can retain with comfort. As regards diuretics, Zangemeister says “the poison of eclampsia certainly does not arise from imperfect action of the kidneys, nor is it eliminated by them.”¹¹ Authorities differ as to the urotoxic dose of urine in a healthy pregnant woman and in an eclamptic,³ but whether the toxin is eliminated by the kidneys or not, it is highly important to increase, if possible, the renal secretion, were it only to remove the products of imperfect metabolism, which must necessarily aggravate the symptoms, if left in the blood stream. For this reason I give the citrate and acetate of potash, of each 20 grains every four hours, as well as diuretin in 15 grain doses also four-hourly, and am satisfied they do good. Purgatives are also of very great benefit, and epsom salts, in drachm doses, every hour, until a free evacuation occurs, are the best. Purgatives are all the more necessary, in that they not only clear the blood of offending ingredients, but remove from the small intestine those toxins which are apt to develop there from defective biliary secretion. Perspiration may be encouraged by hot packs, but, if the patient is kept warm in bed, these will not be required.

The question next arises, should labour be induced? It is certainly better not to induce it for at least 36 hours, because the uterine contractions of labour might precipitate the convulsions. In this connection Zangemeister says, “during labour there is diminished diuresis, the proportion of ammonia salts, phosphates and especially chlorides, is on an average lower, and the specific gravity is less,” while “the variations in intra-cranial pressure, as the result of

rhythmical uterine contractions, are conducive to the occurrence of convulsions.”¹¹ After active treatment for 36 hours or more this risk is very much lessened, and labour may then be terminated unless there are decided indications that the patient may progress favourably. This treatment is opposed to that recommended by Byrne, who says that should there be “albumen present in large quantities or albuminuria accompanied by œdema or headache . . . our duty is to terminate pregnancy at once purgation, sweating, diuretics, and drugs of all kinds are useless, so long as the cause is allowed to remain.”¹²

That much can be done towards the prevention of eclampsia, may surely be conceded. It is perfectly certain, of course, that, in spite of all our vigilance, eclampsia, because of the rapidity with which it may occasionally develop, cannot be invariably prevented, but if we examined the urine frequently during the latter months of pregnancy, it should largely become a thing of the past. Yet we read very little about it from this point of view. Occasionally notice is taken of its importance,¹³ but, as a rule, it is embedded in a paper of other interests. Boxall's introduction to the discussion on eclampsia, at last year's meeting of the British Medical Association, is one of very special value in this respect, though, with one or two exceptions, those joining in the discussion referred only to the etiology and treatment.¹ It is high time our teachers devoted more time to its prophylaxis, so that our young friends, who are about to be set at liberty to practise their art, will not have to find out for themselves, from bitter experience, that the prevention of eclampsia is better than its cure.

It is important in carrying out the examination, to annoy the patient as little as possible, and, in order to allay any apprehension on her part, to assure her that it is done in all cases. When the patient engages the medical man for her confinement, she is asked to report to him if any swelling of the feet occur, and also to send a small bottle of urine at stated periods without further notice. If all goes well she may not be seen again till labour comes on. The urinary examination should commence not later than the end of the 7th month, and should be made at least twice during the 8th month and weekly during the 9th. Should albumen be present, the examinations are made weekly (or bi-weekly if meat is allowed) during the 8th month and first half of the 9th, and afterwards bi-weekly. The frequency depends, however, on the condition of the patient and of the urine.

With the patients of medical men the way is perfectly clear and

simple, but how are we to reach those who may go direct to the lying-in hospital, or to the midwife? With regard to the former, Boxall makes the following suggestion:—"One cannot but wish that our maternity hospitals, as for instance in Paris, were provided with a special department, where women who are able to work either in the house or garden attached to the institution, are kept under observation for two or three months prior to delivery."¹ I fancy this could not be easily carried out in the case of married women who have homes to look after, but it might certainly apply to most illegitimate cases and others who have no special habitat. In many places, public homes are provided for such cases up to the time of confinement, and there is surely no reason why these homes should not be affiliated with hospitals for the purpose here indicated.

There remain those women who are attended by the midwife. The case here seems hopeless, but it should not really be so; for certainly no midwife is properly qualified if she cannot examine urine. It lies with those who teach, and train, and examine midwives, to see that the latter know, not only how to deliver a woman, but also how to recognize, in addition to the simple ailments of pregnancy, the symptoms that may lead to eclampsia, and to impress upon them a sense of the responsibility they incur when they undertake to attend women in confinement.

My own responsibility was specially brought home to me, some seven years ago, by the case of a patient who without any warning suddenly became almost blind from albuminuric retinitis. Under treatment, and the induction of labour, eclampsia was averted, but it was many months before the eyesight became normal. From this time I began the systematic examination of the urine in pregnancy and have kept it up with fair, though not with absolute regularity ever since, certainly with benefit to my patients, and with less anxiety to myself. At first these examinations were not made so often, in each case, as they are at the present time. During these years I have had one case of eclampsia and one of threatened eclampsia. The former case occurred in 1902, the patient being near full time. The urine, two weeks previously, had been free from albumen, but this only shows that after eight months it should be examined more frequently. The patient recovered. The other case has points of decided interest.

Mrs. C., pregnant for the second time, sent for me hurriedly in December, 1904, ten days before her full time. I found her in a somnolent condition, with œdematous face and legs, and complaining of a severe headache and epigastric pain, the urine being loaded

with albumen. Convulsions were evidently impending. The treatment above mentioned was carried out, and after 48 hours, during which time she improved very much in every respect, I induced labour. No convulsions developed. The point of special interest is, that, for some reason or other, the urine had not been examined up to this time, or the diet regulated in any way. This was all the more inexcusable, because in her first confinement three years before, the urine began to show traces of albumen about the 7th month and there was also some œdema. In that pregnancy she was carefully dieted, and though the albumen and œdema did not altogether disappear they were kept under control and her confinement was natural. Looking back to her first pregnancy, there is a very distinct probability, in view of what happened in her second, that by attention to the state of the urine and to diet she at that time escaped eclampsia.

Let me recapitulate, and state in less abstract terms what have been my practice and experience during the seven years I have specially watched the condition of the urine in pregnancy.

1. Up to the 8th month the requirements of pregnant women, in almost all cases, are merely hygienic. Special attention is given to the need for daily movement of the bowels.

2. From the beginning of the 8th month onwards, the urine is examined at stated periods and the diet regulated according to circumstances.

3. Even though the urine be normal and the patient perfectly well, butchers' meat is allowed but once a day from the beginning of the 8th month; otherwise there is no change in diet.

4. According to my experience albumen is present in about 12 per cent. of pregnant women. I have not estimated what proportion of these show casts in the urine. Looking on albuminuria as an indication of toxæmia of some sort, and on the absence or presence of casts as mainly showing a difference in degree of the toxicity of the blood, I have included and treated all cases without waiting for the development of active degeneration of the renal epithelium.

5. Should a trace of albumen appear during the 8th month, fish and fowl are substituted for butchers' meat and eggs, and farinaceous foods are reduced. A glass of water is given four times a day between meals. Under this restricted diet the urine is examined more frequently. It is found that the albumen in many cases does not increase. During the last two weeks of pregnancy, however, the purely nitrogenous and farinaceous elements of food

are discontinued and skimmed milk alone is given if there is still some albumen present.

6. Should the albumen increase under this diet, the patient is kept on skimmed milk and farinaceous foods alone, together with fresh fruit and vegetables. A decided reduction in albumen generally follows. In many cases the albumen disappears altogether.

7. Should the albumen increase on this diet, skimmed milk alone is given (fruit and vegetables being allowed for lunch) and continued till labour occurs. These cases are rare. The quantity of milk given depends on the condition of the urine as time goes on. The more it requires to be reduced, the more the drinking water is increased, and the more necessary is it to rest, to reduce tissue waste.

8. To again summarize these (3, 4, 5, 6, 7): in 88 per cent. of my cases of pregnancy, the diet is not altered in any way, beyond attention to the standard rule that meat is taken but once a day during the 8th and 9th months. In the remaining 12 per cent., fish and fowl take the place of butchers' meat and eggs. Should this be followed by an increase in albumen the patient is fed on milk and farinaceous foods alone, till labour occurs. Rarely, milk alone is required.

9. Should albumen be late in appearing and be attended by œdema, both purely nitrogenous and farinaceous foods are stopped at once and milk alone given. The earlier the albumen appears in pregnancy, the more time there is to observe the effect of a fuller diet on the urine.

10. Should the symptoms be those of impending convulsions, the patient is kept warm in bed, and all food, including milk, is withdrawn for 36 hours. Sips of hot water alone are given if the œdema is great. If there is not much œdema, water is given in abundance to drink, saline rectal injections being also administered. Epsom salts are given in repeated doses so as to move the bowels freely, and the citrate and acetate of potash as diuretics. At the end of this time, labour may or may not be induced, according to the condition of the patient.

11. In every case in which the urine has been carefully watched and the diet regulated, the albumen and œdema have been under control, and no advanced symptoms such as œdema of the face, persistent headache or epigastric pain have developed; in other words, the first stage has been allowed to advance to the second. In the two cases already noticed, these points were not attended to.

12. The patients willingly enter into the arrangement. They look on it as a matter of necessary routine, though sometimes they

neglect to send the samples of urine at the prescribed periods. It is no great hardship to the medical man, because the first test is for albumen and if this is absent no further test is applied.

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