On Puerperal Eclampsia with cases.

I certify that this thesis has been solely composed by myself.

Waterfield, Mackill

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Manchester
On puerperal eclampsia with cases

This disease forms one of the most alarming complications which the general practitioner is liable to meet with in his obstetric practice and as I have met with three cases during the last four years, I thought that this might form a suitable subject for my thesis; especially as my professional life since graduating has been spent in general practice in a small town without opportunity for hospital or special laboratory work.

During these four years I have attended 370 labours so that this complication has occurred in a little under 1% of them. The general proportion however, judged from statistics compiled from a great number of cases, is about 1 in 600. (373 out of 9...
228,010 cases [Hinchcliff]

There are four varieties of convulsions to be met with in pregnant women.

I. The Hystericlal

II. The Apoplectic

III. The true Epileptic. These three are identical with the same diseases in the unpregnant.

IV. The Epileptiform which is the true prepurpural convulsion in eclampsia and not to be confused with ordinary epilepsy.

The convulsions may come on I either at or after the 6th or 7th month before labour has set in, or II they may come on during labour, or III they may not occur until after parturition.

The mortality is very great both as to mother and child. In the case of the mother it is usually given as about 1 in 6 but is influenced greatly by the time of the occurrence of the attacks being very much
Greater is the chance of the convulsions coming on before or during labour than in those where they come on during the puerperium.

My own cases bear this out as the fatal case occurred during labour while the other two occurred during the puerperium and both recovered.

The disease is much more common in primiparæ than in multiparæ. Where it does occur in a multiparous woman she is probably either carrying twins or is the subject of chronic Renal disease.

My cases also bear out this statement as my three first cases were all primiparæ while the other was a multiparæ of 34 years old who died 2 years later from Chronic Bright's disease.
I will now give the history of my cases shortly and then discuss the etiology and treatment of the disease and my conclusions thereon.

Case I. A.S. aged 20 years.

Primipara - unmarried.

At 2.30 pm on January 21st 1894 this girl was seized with a fit. A doctor who was passing was called in and prescribed some medicine (probably Potassium Bromide). The fit however continued, at first with an interval of about an hour and then more rapidly until 7 pm when I was sent for.

I found that she was in the 9th month of pregnancy and having epileptic-like seizures almost constantly. The face was congested and cyanotic, eyes fixed and upturned, breathing rapid, 

The whole muscular system was in a state of violent, tonic contractions.
The paroxysm would abate & the patient lie for a few minutes in a state of profound coma & unable to be aroused. Then as fresh paroxysms would occur.

The pulse was 120 & easily compressible. I drew off about one ounce of blood-stained urine loaded with albumen.

On making a per vaginam examination I found that the os uteri only admitted the tip of the forefinger & was rigid. No bag of membranes could be felt. The presentation was vertex L.O.A. The foetal heart could be heard on auscultation but the patient’s movements & noisy breathing made auscultation difficult.

I at once rubbed one minims of litha oil on the drama of the tongue as she could not swallow, dry-cupped the kidneys, & injected 1/4 th of Pilocarpin.
I then administered ergotamine. This relieved the passiveness for the time. But whenever the administration was relaxed, they returned as before. As the os did not dilate, I injected an ounce of glycerine within the cervix. After waiting 2 hours, persevering with these measures, the patient gradually getting more and more exhausted, I decided that the only thing to be done to give the patient a chance was to forcibly dilate the os to deliver as soon as possible. I accordingly proceeded to dilate forcibly with my fingers & metal dilators. After about an hour and a half I got it sufficiently dilated to apply forceps, which I did & delivered her of a still-born male child about 12 relief. The patient was now in a state
Of deep coma, conjunctiva insensitive, marked uterine bleeding, and edema
of lungs at base, but the convulsions had ceased. Pools 130.
I put her in a blanket bath and again dry cupped the kidneys but she gradually sank until she died
at 2-10 a.m. on January 22nd two hours after delivery.
No post-mortem examination was permitted.
In this case there had been no premonitory symptoms such as swelling of legs or face, diminution of
vision, or excessive headache or sickness until the morning she was attacked when she complained
of severe headache for about two hours and then the first convulsion occurred.
This was apparently brought on by severe mental shock. The father of her child had promised
to meet her that morning but instead of doing so sent his
sister, who made some communication to the girl that was apparently a great blow to her.
Besides this, there was a history to prolonged mental anxiety all the time of pregnancy.

Case II. E. M. aged 27. Primipara.
Married 4 months

This woman was confined about 8 a.m. on December 26th, 1894.
She was attended by a midwife & was reported to have had a fairly quick & easy normal labour.

However about ten A.M. she had a convulsive seizure and I was sent for. I found her in a semi-comatose condition. She only spoke when asked and while I was there she had another fit about half an hour after the first. It had the appearance of an ordinary epileptic seizure and lasted
About five minutes.

I gave her a draught of Dr. Broom.

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and a

of Dr. Phelps's, and induced

She continued to have convulsions at

She made a good recovery and

She again became pregnant a

year afterwards and I attended

her in a normal labour, except that the child was still-born

on November 10th 1896.

She has left this town but in

February 1898 I learned that she
was 6 months pregnant again so I obtained a specimen of her urine for analysis & found it free from albumin.

There was in this case also a marked history of mental worry and anxiety during the first five months of pregnancy. She is also of a markedly nervous temperament.

Case III. A. J. aged 21, Primipara.

married and weeks.

I was sent for to this case about five a.m. on January 2nd 1898. being told she was in labour. She complained of pain in the back but on per vaginal examination the os uteri was found undilated & the pains made no impression on it. I left & called again about
ten o'clock. But found no change. They sent again next morning about six a.m. and I then found the lo about the size of a pigeon, membranes unruptured and the presentation vertex L.O.A. Labour went on slowly and normally till one p.m. when she was delivered of a living male child. 
I did not see her again until next day about eleven a.m. I then found that she had been suffering from severe vomiting of bile-stained mucus since about three o'clock that morning. She also complained of severe headache especially of occipital region and behind the ears. She had passed water but the attendants had not saved it. The pulse was 80 and bounding and she seemed rather congested about the face. I ordered a mustard poultice.
to the epigastrium and another behind the neck and gave a mixture of Raldo, Bismuth, and Epsol Bicarb.

I was summoned to the patient again at four p.m. and found she had had an epileptic-like seizure (28 hours after delivery). She seemed in a dazed heavy state and only conscious if roused. The pulse was now 115 and easily compressible.

I ordered a blanket bath and gave a strong jalape powder and a draught of Chloral and Phineasum Bromide. She had no more attacks till 5 p.m. when she had another and another at 5:20.

I saw her again at ten p.m. She was still in a semi-conscious state and was only roused with difficulty but she had had no
more fits.
I drew off with the catheter about eight ounces of urine and found it loaded with albumen.
Next morning she was still dull and heavy but otherwise better. Pulse 110. temperature 101°. She had also passed a fair amount of urine.
She made an uninterrupted recovery, except for a good deal of headache, and was able to be up on the 14th day.
In this case the albumen has remained very persistent and on the last examination on April 12th 1898 there was still about one quarter of a half.

In this case there is a history of great oedema of legs and oedema under eyes. She had only been sick twice during pregnancy.
the oedema of the legs had
after delivery.

There was also in this case a marked history of severe mental strain and anxiety during the first six months of pregnancy as she put it to me "my husband was not doing right by me and I did not know what was to become of me till a month before we were married" namely when she was six months pregnant.

I have included the next case in the present series, although there was no eclampsia, because I think it is of interest in this connection. It is a case of albuminuria: Retinitis treated by the induction of premature labour.
Case IV 7th Oct. 1894 aged 34.2 yrs.
youngest child aged 8 yrs.

I was consulted by this woman in the beginning of February 1895.
She was 4 1/2 months pregnant and complained of her eyesight failing her. I examined her urine and found it contained a large amount of albumen.

I ordered her to adhere strictly to milk diet and ordered mustard poultices across the tines and saline purgatives to be taken in the morning. I also prescribed a mixture containing Linum, Ferri Prot., and small doses of Linichia & Digitalis. She was also told to wear woollen clothing reach the skin to avoid all exposure to cold or wet.

After persevering with this treatment for a month I found
that the albumen was increasing in amount and that her
eyesight was going worse. It in
fact got so bad that she
could not distinguish people's
features in the room with
her and only recognized them
by the voice. She also was
unable to go about alone.

I therefore sent her to Manchester
to get Dr. Donald's opinion.
He sent her on to Dr. Griffiths,
an oculist, who reported "advanced
albuminuria, Retinitis in both eyes."

Accordingly Dr. Donald advised that
premature labour should be
induced immediately as the
prognosis was very grave.

I therefore on March 11th introduced
a gum-elastic bougie between
the uterine wall and the membranes
and left it in "in situ."
Labor commenced about 12 hours after and she was delivered of a still-born child very easily. No alarming symptoms developed and she made an uninterrupted recovery and was able to go about in a fortnight.

The eyesight gradually began to improve from the 3rd day after delivery and went on improving slowly till in two months she was able to see and do other delicate work comfortably.

The albumen also rapidly decreased in amount and on examining in June none could be detected. However a few months after the patient complained of sickness and headache and the albumen was found to be again present in small quantities. S.S. 1008. She kept in this state about 2 years suffering from Chronic Bright's disease. On June 4th 1897 she had an apoplectic seizure and became comatose died on June 10th 1897.
Etiology.

The etiology of Puerperal Eclampsia is still uncertain and there are different theories brought forward by various observers to account for it. One great fact is almost universally associated with it, namely, the presence of albumen in the urine.

At first sight this appears to satisfactorily explain the phenomena of the disease, were it not for the fact that in some cases the convulsions have preceded the albuminuria or else the albuminuria has only appeared simultaneously with them. Therefore the theory that the eclampsia is entirely due to the presence of arias (Braun) or Carbonate of Ammonia (Freichs) in the blood cannot hold good in these cases. Also albuminuria does not invariably
Induce eclampsia in pregnant women and in fact suffers from chronic Bright's disease who become pregnant are only rarely attacked by it. (Case IV illustrates this.)

I have also the notes of a case under the care of my brother W. M. MacMillan of Hartford.

A young woman, a primipara, consulted him for swollen legs and face. The urine was found loaded with albumen. She was put to bed and ordered milk diet and diaphoretics. Labor came on to days after and a child born normally but with no fits. The albumen was excessive for some days after but gradually disappeared in about five weeks.

Some explain the presence of the albumen as being due to pressure on the renal veins by the gravid uterus.

The special liability of primiparae is
is explained as being due to their abdominal walls being more unyielding than those of multiparae and therefore more pressure is thrown on the veins in their case.

One great objection to this theory is that in cases of great distension of the abdomen, from ovarian tumours and other causes, albumen is rarely present in the urine.

Another theory is that the cause of the Eclampsia is an "Auto-intoxication" due to the presence of an actual poison in the blood, due to the retention of some toxin that the kidneys are unable to eliminate. (Larnier, Chamberlen)

Again it has been suggested that the cause may be an irritation of the vaso-motor centres from their anaemic condition,
This anemic condition being brought about by the hydroemic state of the blood. (Angus MacDonald)

Others think it is due to oedema of the cerebral tissue or exudation into the ventricles and consequent pressure upon and anaemia of the brain. This oedema being caused by the hydroemic state of the blood plus the increased arterial tension found in pregnancy. The albuminuria would also increase the hydroemic state of the blood while the increased blood pressure during a pain would aggravate matters and explain the greater liability of patients to have a convolution with very pain. (Graube) and (Rozenstein).

It seems to me however that there must be some other essential factor to explain...
Why some women should be attacked and not others.
The point I wish to emphasize from a study of these cases is that there appears to me to be a mental element in the case which has to be considered.
We know that in pregnancy there is a mental instability often and that the nervous system is already in a hypersensitive condition.
Then in each of these cases there is a distinct history of prolonged mental anxiety with in the fatal case a severe mental shock superadded an hour or two before the first convulsion came on.
Could not this prolonged strain on the nervous system, while in this hypersensitive condition, produce some change in the
Cerebral cells themselves which might cause (upon the addition of some exciting cause like labor or some other distorting factor) such discharges to nerve fiber as to produce the convulsions.

This change in the cerebral cells, acting through the vasomotor centers, might lead to entire spasm and so, causing a disturbance of the vascular balance in the kidneys, produce the albuminuria.

We know that some cases of functional albuminuria in otherwise healthy persons may be caused by some such disturbance of balance in the arterioles of the kidney. Also sometimes prolonged mental strain has been given as a predisposing cause of Bright's disease.
In connection with this theory of a mental element in the production of Eclampsia a case mentioned by Hahneman is interesting. He records a case of twin sisters being seized with convulsions after normal pregnancy and delivery. He concludes that "some congenital irritation of the cerebral cortex clearly existed in both cases."

In my cases in Case I where the severe shock immediately preceded the attack there were no symptoms or any history of premonitory symptoms of ischaemic poisoning; except the 2 hour headache after the shock, and yet the attack was fatal which favours the theory that it was entirely cerebral in origin.
In Case II and Case III, however, the premonitory symptoms were more marked especially in Case III and were strongly suggestive of haemorrhagic poisoning.

The convulsions however did not come on till after delivery and both children were alive and well. It appears therefore as if the blood cannot have been loaded with toxic elements to cause the children would have suffered. Perhaps however the blood was slightly toxic and after delivery a greater quantity of this impure blood going to the brain was sufficient to cause the convulsions.

In these two cases the lesion appears to have been traced in the kidneys and in the blood and to have
Acted on the central nervous system through them rather than directly through the cerebral cells themselves as in Case I.

I think a comparison of Case IV with these cases favours the theory of a mental element, acting on the cerebral cells and as it were making them especially susceptible, being an important factor in the production of eclampsia.

In this case the albuminuria was pronounced and appeared very early in pregnancy. It had also acted severely on the vascular and nervous systems as evidenced by the Retinitis - and yet even upon the induction of premature labour no Eclampsia
I would explain this by suggesting that the central centres were not in that specially hypersensitive state as to be thrown into excessive activity even by the very normal flood and increased arterial pressure during labour which were present in Case IV.

Treatment.

I. Prophylactics.

In all cases of pregnancy it is advisable to examine the urine but especially in this the case where there are any symptoms such as swelling of legs or face, excessive sickness or headache, or dimness of vision. In
all such cases albumen ought to be looked for.

If albumen is present treatment ought to be begun at once and persevered with.

(1) A strict milk diet should be enforced.

(2) Free phlegation with saline or calines.

(3) Encourage diaphoresis by the use of diaphorotics and

laundering of Perspiration if done is often of special value.

(4) Have woolen clothing meet the skin.

(5) Avoid all excitement, worry, or anxiety, or exertion.

II. When the eclampsia has occurred

(1) Treatment of the fit itself.

(2) In the first place we must keep the patient from injuring
herself, biting her tongue &c., by appropriate measures as in ordinary epilepsy.

(b.) Then as to medicinal treatment we ought first to try free purgation by Compound Salap Powder or if the patient is unable to swallow by rubbing one minim of cotton oil on the tongue.

(c.) Encourage free diaphoresis by blanket baths or the injection of 1/4 grain of Pilocarpine.

(d.) The administration of large doses of Chloral Hydrate and Potassium Bromide to soothe the nervous system and to lower the vascular pressure.

(e.) The use of Morphia is very
recommended strongly by some observers and some very good results have been attained from its use but I did not use it in my cases.

(f.)

If these measures fail to stop the convulsions we should at once administer chloroform which has a wonderful effect in controlling them. It is of the greatest service in the treatment of eclampsia.

(g.)

Dry cupping over the kidneys may also be tried to relieve the congestion of these organs.

(h.)

A much-debated question is as to the efficacy of enucleation. This was the old routine of practice but died out
If use. It is certainly not applicable in every case but in a certain class of cases (where the patient is strong and full-blooded with much cyanosis or edema of the lungs) it seems advisable to try it.

According to certain observers it has done good in many cases (Swaine).”

Perhaps if Venesection had been performed in Case III. the convulsions would not have occurred. In that case the premonitory symptoms lasted twelve hours and there was a full bounding pulse and some congestion of the face. I feel sure that if some blood had been abstracted it would have done good and perhaps have warded off the eclampsia.
Then, in those cases where labour has not begun, there is great difference of opinion as to the advisability of leaving things to nature or of effecting forced delivery.

Those who, like Charpentier, hold that it is unjustifiable to effect forced delivery (except in cases where there is uterine inertia and delivery would otherwise be easy) point to the fact that in most cases of eclampsia or pain during on a fresh paroxysm and argue that therefore any active interference likely to irritate the uterus is apt to aggravate matters.

On the other hand, Dührsen advocates deep incisions into the cervix and Haltentomäie.
has published some remarkable results of treatment of Caesarian sections.

He records 26 serious cases 10 of which were actively treated of whom 8 survived. The other 16 were only treated medicinally and of them only one survived.

Zweifel too found that in 49 cases before 1892 treated expectantly 32.6% died while since then in 80 cases treated actively on Dührssen's principle only 15% died.

These statistics seem to show in favour of the active treatment and I cannot help thinking that perhaps if I had treated Case I by incisions into the cervix and prompt delivery she might have survived. While on the other hand I feel confident...
that if I had left things to
nature she would have died
undelivered.
However the very heroic treat-
ment of Caesarian Section
seems inapplicable to private
practice whatever it may be
in special hospitals.
Dr. Haultain\textsuperscript{14} has published
some interesting cases where
the cervix was manually
dilated & then delivery
effected with perfect without
serious recurrence of the
convulsions that had been
previously suffered.

I would now like to
make a few observations
upon the treatment adopted
in Case 17.

Barnes\textsuperscript{15} in an interesting
address says "the strins
duty of a physician when he is called to a pregnant woman in difficulty is to consider and determine how to secure the safety of both mother and child; but when both cannot be secured, the safety of the mother demands our first attention.

The first question therefore to be answered in considering Case IV is as to whether the safety of the mother was endangered by allowing the pregnancy to go on.

I think the answer is most unquestionably in the affirmative.

Mr. Beaumont records a very interesting case treated at the Bath Eye Hospital.

The case was that of a girl suffering from Actinomucose Rea-
Initis who subsequently became pregnant. Then at the 6th month she was taken ill in the night with pain in the stomach, rapidly became comatose and died the same day. This shows the extreme danger of pregnancy complicated with albuminuric Retinitis.

Mr. Simon Snell has a very good paper on the subject and concludes that, especially in cases coming on during the early months of pregnancy, the induction of premature labour for albuminuric Retinitis in pregnancy is the proper treatment.

In my case the treatment adopted seemed justified by results. I feel sure that if pregnancy...
References.

4. "Jax des Eages, Emm" February 1st 1894.
7. "hlexer der Zusammenhang von Herz und Nierenkraankkeit".
   "Gesammelte Beiträge zur Pathologie und Physiologie".
had gone on to term the patient would have lost her sight if not her life. However, after premature delivery the eyesight improved vastly and a fatal termination of the case was probably delayed a year or two. If the patient could have lived under more favourable conditions for the diseased kidneys it might have been still further postponed. Perhaps objection might be taken to the fact of inducing premature labour before the child was viable but delay seemed contraindicated by the state of the eyes and their rapidly progressive failure.

Wakefield MacGill