A Thesis on
Some cases of Puerperal Eclampsia
with special reference to the
pathology and treatment of that
disease,
by Geo. Cabrow Sandford
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April 20th 1896.
I. A Report of 3 cases of Puerperal Eclampsia which came under my care when House Surgeon to the Royal Maternity Hospital, Edinburgh.

First Case.

Mrs. McKay, multipara, married in 1887: has given birth to three living children between that time & 1890: she had an abortion (3 months) in 1891: a miscarriage (7 months) in 1892 & another miscarriage (8½ months) at the end of that year: in 1893 another living child was born and towards the end of that year she again became pregnant & at the end of this pregnancy she had an attack of eclampsia which I am about to relate. Patient says that she had fits in 1892 when her medical attendant induced labour at the 7th month & she was delivered of a still-born child: the urine was then examined by the doctor but patient does not know whether albumen was found although she says that the doctor suspected kidney disease: in the two succeeding pregnancies no fits occurred though one miscarriage was nearly at full term & a living child was born at full term in 1893.
Patient has always been of temperate habits & enjoyed good health: till the onset of labour this time she had been particularly well. There is no history of any exciting cause to account for the onset of labour. The pains came on about 10 p.m. on May the 10th and at 3 a.m. on May the 11th she had her first fit: after this she had a fit every hour till admission at 4-30 p.m. on that day: the nurse then emptied the bladder by the catheter & one ounce of high-coloured urine containing a good deal of albumen was drawn off. The temperature on admission was 102°4 & the pulse 100.

Patient had fits at 4-45, 5-5 & 5-10 p.m. Three minims of Croton oil were then given but were probably ejected during the next fit which occurred a few minutes later. Another fit occurred at 5-35 p.m. Pulse then 140. I injected ½ a grain of pilocarpin hypodermically at 5-43 p.m., the effect was not satisfactory as although diaphoresis commenced soon after the patient was almost suffocated by frothy secretions in the air passages.
Since admission patient had been under chloroform but in spite of this fits constantly recurred. On vaginal examination at 5 p.m. the os was found to be scarcely at all dilated. To aid diaphoresis patient had been placed between blankets & surrounded by hot water bottles. At 5-45 p.m. two minims more of eroto oil were given: twenty grains of chloral hydrate were given about 6 p.m. & another fit occurred about this time: chloroform was administered till the termination of labour at 6-50 p.m. Patient was not conscious from the time of admission till the afternoon of May the 12th. During the fits patient became cyanosed & rolled her eyes upwards, the pupils were dilated and great facial contortions took place, and violent movements of the limbs she foamed at the mouth and at times seemed nearly choked by frothy expectoration there was some opisthotonos, the arms were rigid at first, but clonic spasms occurred later: the eyebrows were elevated & the angles of the mouth depressed during the fits giving the
patient a horrible expression: the breathing was rapid and stertorous during the fits.

There was no oedema of the face, legs or other part of the body: but she was very thin and anaemic. It was thought advisable to terminate labour as soon as possible: the membranes were ruptured at 6-20 p.m. & the foetus which was lying transversely was delivered by pordalic version; it was still-born & seemed to be about 6 months, it was a female weighing 1 lb 2 oz, and it was 12 inches long: the placenta was expelled at 6-50 p.m. A vaginal douche was administered after delivery. At 7-30 p.m. the temperature was 103° & the pulse 125: at 8-30 they had fallen to 101°& 120 respectively. Patient passed a motion at 9 p.m. and a nutrient enema of brandy & egg was then given. Owing to the feeble condition of the pulse several hypodermic injections of ether were given during the evening. At 10 p.m. a hot-air bath was given and at 10-30 p.m. the temperature had fallen to 100°8.
Two ounces of urine were drawn off
then & still showed a large amount
of albumen: patient now perspired
freely & seemed rather better. On
May 12th she had occasional hypodermic
injections of ether and one of 1/100 Gr
grain of Digitalis: chloral and
bromide of potash were given per
rectum. At 7:30 a.m. the temperature
was 100° & the pulse 140. Nutrient
suppositories were given every 2
hours. At 8:30 a.m. another hot
air bath was given & seemed to
irritate the patient, whilst in it
she had another fit which lasted
about a minute: chloroform was
then administered & again whenever
she had a hot air bath. The temper-
ature at 9 a.m. had again risen
to 102° & the pulse was 145. Twenty
grains of chloral hydrate were
given during the next few days
six times daily. Consciousness returned
about 1-30 p.m. on the 12th when the
patient asked for milk: a mixture
containing tincture of digitalis and
acetate of potash was given 4
times a day for ten days & a large
amount of urine was passed with less albumen.
On the 13th the temperature remained about 100° all day & the pulse was 115. May 14th Pulse 104 and temperature 98°6: ten grains of phenaestin were given at noon in addition to the regular doses of chloral and diuretics: patient complained of being muddled and cried a great deal. May 15th. Very moisy to-day, seems to be out of her mind: pulse 106, temperature 100°. May 16th. Bowels opened by Henry's solution: chloral given only at night. Temperature 98° & pulse 104°. A large amount of urine has been passed during the last few days & the albumen has rapidly decreased & to-day there is only a trace present. Patient does not know where she is & imagines she is in prison, wants to know what she has done to be taken there, her memory is much impaired: she is quiet and drowsy. May 17th Temperature 102° in the morning & 99°6 in the evening. May 18th. Normal temperature to-day & till discharge: chloral discontinued on May 31st. In June the 2nd patient went home.
no albumen in her urine now: pulse 76 + temperature 98.6: she was very weak & could only walk when supported. I saw her some 3 weeks later when her mental condition was much better: her memory with regard to her illness from the time of her first fit was completely gone.

Remarks.

The exciting cause of the convulsions in this case was probably the onset of labour. Braxton Hicks does not think that uterine contraction alone causes convulsions, but owing to the peculiar irritability of the nervous centres in pregnancy & the blood-poisoning caused by renal inadequacy it seems that uterine "pains" may in some cases be the exciting cause. Colman records three cases (one of which was fatal) in which the exciting cause of the convulsions was digital stretching of the os: in one case fits could be produced at will by this means; in none of these cases was albumen present in the urine.

A case somewhat similar to this last is recorded by Corby, the convulsions being caused by irritation from the uterus being bound down by adhesions and not able to expand in proportion to the growth of the foetus.

Another point worthy of notice in the case I have recorded is the fact that the patient had convulsions in a previous pregnancy. Hall-Davis says that as a rule repetition of convulsions in a subsequent pregnancy does not occur: Dewees, however, records their occurrence in the first, third, and fifth labours of one of his patients. Bauclercou, Capuron, Perfect, Portal and others also give cases of its recurrence in subsequent labours.

The temperature in my case seems to have been distinctly affected by the fits: Herman has not found this to be the case in his own experience but Galabin thinks that convulsions have a tendency to raise the temperature of uraemia to lower it. Bournville publishes several cases in support of this view.

The fact that a convolution was
provoked by the irritation of a
hot-air bath is in favour of
Barnes's theory that any peripheral
irritation may bring on an attack
so no interference should be
undertaken unless the patient is
under the influence of chloroform.
Although the patient was certainly
of unsound mind during a few
days of her convalescence her
mind was not permanently affected.
This is in accord with what Sir
J. Simpson says — viz. "Puerperal
convulsions are often attended with
some temporary degree of incoherence
delirium or stupefaction but they
rarely as far as I have seen end
in true and established puerperal
insanity". But Churchill, Gooch, Red,
Merriman, Esquirol and others record
cases in which mania followed
puerperal convulsions. Ingleby in his
essay on Puerperal Convulsions, says:
"I am acquainted with several cases of
puerperal convulsions which were succeeded
by puerperal mania: the transition might probably
be the result of the large bleedings which were
necessary to subdue the primary disease.
The loss of memory which occurred in my patient is not unusual after an attack of eclampsia. Ramboldt\(^1\) records several cases of this kind, one of his patients had forgotten how to write & had to learn like a child again. Another of his patients could not remember what had happened during the six days previous to her confinement although she had been going about as usual during that time. Bidot\(^2\) of Marseilles reports the case of a woman delivered at term after eleven months of marriage. Eclampsia occurred before labour. She recovered but lost all memory of what had occurred during her married life, the memory of her life as a girl remaining perfect. Charpentier relates a case of amnesia, lasting 12 months, in a primipara, after eclampsia. Caleb Rose\(^4\) records a case somewhat similar to the one I had charge of. Touchard in the Journal de médecine for Feb. 5, 1892 also records two cases of post-eclamptic amnesia.

1. Medical Times & Gazette I, 1863 p. 158
4. " " II, 1852 p. 61
Second Case.
Phemie Heil, primipara, aged 16, unmarried, admitted on Saturday, May the 26th at 11-50 p.m. said to be about 7 months pregnant. On admission, the patient, who was a healthy and well-developed girl of dark complexion, complained that she had felt a gush of blood from the vagina about an hour and a half previously: no labour pains were present but she had a slight pain in her left side; she thought about 6 or 8 ounces of blood had been discharged for the first few hours after admission she continued to lose a small quantity of blood. On vaginal examination I found that the os would admit one finger easily and that the membranes were bulging of the head presenting. On May the 27th no progress occurred and the bleeding had stopped, three fifteen minims doses of Tincture of Opium were given during the day. The cervix was in the same condition on May the 28th at 9 a.m. and no pains had occurred since admission: about 11-30 a.m. the
nurse in charge observed that the patient was in a convulsion which lasted about one minute but left the patient unconscious. Chloroform was administered at once and a hot air-bath was given: the patient swallowed three minims of Croton oil. Free perspiration now occurred but as no evacuation of the bowels had taken place at 1 p.m. the dose of Croton oil was repeated but without effect. Two twenty grain doses of chloral hydrate were given by the mouth but as the second dose was vomited the same amount was given per rectum at 1-30 p.m. a twenty grain dose given by the mouth at 3 p.m. was also ejected so another dose was given per rectum at 3-30 p.m. Directly after the fit occurred two ounces of urine were drawn off & the urine was found to be nearly solid with albumen on applying the heat & nitric acid tests: four ounces were drawn off at 4 p.m. an enema was then given which was soon followed by an evacuation of the bowels.
At 4-15 p.m. it was thought best to hasten the termination of the labour and as the os remained thin & hard three ounces of glycerine were injected into the uterus, the patient being well under chloroform: the effect of the injection of glycerine was remarkable as in an hour and a half the os was found to be well-dilated; the membranes were ruptured at 5-53 p.m. & the foreeps applied, after gentle traction the child was delivered at 6-25 p.m.

The child's head was in the right occipito posterior position: rotation did not occur & the occiput was delivered over the perineum without injury to the latter. The child seemed to be under seven months and only weighed 2 lb. 13 oz. it died eleven hours after birth. The placenta was expelled five minutes after the birth of the child & on examination it was found to be depressed in one cotyledon by the presence of a firm clot: the slight bleeding which occurred prior to delivery probably being thus accounted for.
No convulsions occurred during delivery and patient soon recovered consciousness; during the next day she passed about 50 ounces of urine containing 9 or 10 grains to the ounce. Compound jalap powder was given at night for some days and she was put on acetate of potash and tincture of digitalis. In June, the 1st, the albumen had almost disappeared. Previous to this attack patient had always enjoyed good health. She now sleeps well if she went out on June the 8th, only a trace of albumen being present in the urine. Her temperature had been normal except shortly after the fit when it was 102°, 10 hours later 99°.

Remarks.

This was a mild case of eclampsia and contrasts favourably with the preceding one which was not treated until the patient had gone through more than a dozen convulsions. The rapid delivery here accomplished without any bad result, in a primigravida, seems to speak favourably for this mode of treatment in puerperal convulsions occurring before or during labour.
Third Case

Mrs Milne aged 23, primipara: was admitted on June the 5th expecting to be confined within a fortnight or three weeks: she was suffering from anaemia, the legs & feet being very much swollen, the face was pale & puffy: the labia majora were enormously distended with dropsical fluid: she complained of headache & flashes of light before the eyes: she had been passing very little urine recently and a specimen then obtained showed a copious deposit of albumen when tested: we found it necessary to aspirate the labia that evening and a large quantity (about a pint) of fluid was drawn off: two minims of eroton oil were given at once and she was ordered 30 grains of acetate of potash & 10 minims of tincture of digitalis every 4 hours.

Patient said that the labia had been swollen for four days and had caused her great discomfort. On June the 6th the labia were much reduced in size and the patient was able to lie on her
side; during the next few days she passed a larger quantity of urine & the oedema of the legs became less & she did not suffer so much from headache. Two drachms of syrup of chloral were given each night & caused the patient to sleep well. In June the 13th however this improvement stopped & patient became restless & quarrelous: the urine was less in amount & diminished each day whilst the albumen increased. The labia began to swell again slightly & she was troubled with a cough, for which an expectorant mixture was ordered. Compound jalap powder was given frequently to keep the bowels in action. A hot air bath was given daily & caused free perspiration. As the patient seemed to be getting gradually worse & the amount of urine passed was getting very small it was thought best to induce labour & so at 4 p.m. on June the 18th two ounces of glycerine were injected into the uterus. As the os dilated slowly
at first and at 5-30 p.m. some more glycerine was injected and a small Barnes's bag was introduced to aid in dilating the os: this was kept in for half an hour and removed when the os was about the size of a five shilling piece: at 6-30 p.m. the patient became conscious after the chloroform which had been administered since the operation was commenced, a few pains came on at intervals of about a quarter of an hour. At 8-15 p.m. the patient was observed to be in a convulsion which lasted about a minute & a half: chloroform was immediately administered and whilst the patient was under its influence I ruptured the membranes in order that the evacuation of the liquor amnii might relieve the arterial tension to some extent: the os was now fairly well dilated & I continued dilatation with my fingers for a quarter of an hour when the os was large enough to admit forceps: these were applied & the head was slowly delivered, the forceps being removed and reapplied from time to time: the child was born at 10 p.m. but
it was not breathing and all attempts at resuscitation failed; it was a well-developed full term male child. On coming round the patient was very noisy and restless; twenty grains of chloral were given at 12 midnight and 10 grains at 5-30 a.m. on the 19th with 20 grains of bromide of potash but in spite of these the patient did not sleep and she shouted a great deal all the morning. The placenta was expelled about half an hour after the birth of the child and there was not much haemorrhage but a good deal of oozing occurred during the next day and on the second and following days several large clots were passed; ergotin and ergot were given as the uterus did not seem to be firmly contracted. The temperature went up to 99.6 on the 21st and three grains of esculin were given for a bad headache. On the 22nd a large firm conical mass was passed, about 5 inches long by two broad; the end which appeared first had some soft blood clot attached to it but the other end was firm & tough & on section it looked like a carious mole.
After this was passed the uterus remained firm and contracted. The patient now seemed much better and the albumen was diminished to a trace. Twenty grains of chloral were given at night for a week after delivery and on the 30th of June the patient went out looking very anaemic but otherwise pretty well, no albumen present in the urine and no dropsy of any part. She called three weeks later and said that she was gradually getting stronger.

Below is given the daily quantity of urine passed by the patient:

<table>
<thead>
<tr>
<th>Date</th>
<th>Quantity</th>
</tr>
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<tbody>
<tr>
<td>June 6th</td>
<td>15 1/2 ounces</td>
</tr>
<tr>
<td>7th</td>
<td>40</td>
</tr>
<tr>
<td>8th</td>
<td>40</td>
</tr>
<tr>
<td>9th</td>
<td>89</td>
</tr>
<tr>
<td>10th</td>
<td>45</td>
</tr>
<tr>
<td>11th</td>
<td>44</td>
</tr>
<tr>
<td>June 12th</td>
<td>38 ounces</td>
</tr>
<tr>
<td>13th</td>
<td>32</td>
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<td>14th</td>
<td>31</td>
</tr>
<tr>
<td>15th</td>
<td>25</td>
</tr>
<tr>
<td>16th</td>
<td>29</td>
</tr>
<tr>
<td>17th</td>
<td>22 1/2</td>
</tr>
</tbody>
</table>

June 18th - Labour induced.

<table>
<thead>
<tr>
<th>Date</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>June 19th</td>
<td>33 ounces</td>
</tr>
<tr>
<td>20th</td>
<td>79</td>
</tr>
<tr>
<td>21st</td>
<td>85</td>
</tr>
<tr>
<td>22nd</td>
<td>74</td>
</tr>
<tr>
<td>23rd</td>
<td>99</td>
</tr>
<tr>
<td>24th</td>
<td>113</td>
</tr>
<tr>
<td>June 25th</td>
<td>88 ounces</td>
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<td>26th</td>
<td>49</td>
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<td>27th</td>
<td>55</td>
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<tr>
<td>28th</td>
<td>44</td>
</tr>
<tr>
<td>29th</td>
<td></td>
</tr>
<tr>
<td>30th</td>
<td>Patient discharged</td>
</tr>
</tbody>
</table>
Remarks.

In this case the most noteworthy point is the result of induction of premature labour; some authorities disapprove of any operative interference unless fits occur while others recommend prophylactic treatment at first and if this does not prove satisfactory, induction of labour: for my part I think that no other course was open to us in this case as the patient was certainly getting worse daily. I think that she would have escaped eclampsia entirely had she been kept continuously under chloroform until the labour was completed. In any case the child ran a great risk and in many cases death of the foetus occurs in albuminuria from the toxic effects of the maternal blood on the foetus apart from the risk it runs from uterine contractions during the fits in puerperal eclampsia.

A few weeks ago I attended a primipara aged 23 who had a very large amount of albumen in her urine, she was delivered of a still-born child (8 months) which had been dead for a day or two; this circumstance may as Barnes suggests be sufficient to account for the fact.
the patient escaped eclampsia because the blood pressure was reduced after
the death of the child. Mynlieff lays stress on the point that in cases of
albuminuria in pregnancy immediate interference is often best for the
child when viable as it may die suddenly if there is delay. Galabin
says: "If there is much oedema towards the end of pregnancy or if the
proportion of albumen is large it increases notwithstanding treatment, premature
labour may be induced; this is especially desirable in the case of
a primipara, the child being viable; there is a better chance of escaping
eclampsia if labour is brought on than if the kidney disease is left
to become aggravated." On the other
hand Swayne, Forseye Barker, Charnett,
and others speak strongly against
the induction of premature labour
saying that the risks of eclampsia
occurring in labour are much
increased by this mode of treatment.
Eclampsia Puerperalis

Synonyms: Eclampsia parturientum, dystocia convulsiva, renal epilepsy, uraemic convulsions, eclampsia gravidarum: the word "eclampsia" is derived from the Greek, ἔκλαψις, to shine forth, to burst forth violently. French eclampsie: German, ekampsie.

This disease is not of very frequent occurrence, Spiegelberg says 1 case occurs in 500 labours and Krassing agreed with this remark. Largeau found puerperal eclampsia in 79 cases out of 38,306 labours, Braun in 5-2 out of 24,000. Leishman says one case occurs in 300 labours.

During the last 16 years (from 1880 - 1895 both inclusive) 40 cases of eclampsia have been treated in the Royal Maternity Hospital, Edinburgh, as 300 labours occur there annually on an average these figures would show one case of puerperal eclampsia in 120 labours but a great many of these cases would be sent to the hospital on account of premonitory symptoms or actual convulsions.
An analysis of these 40 cases shows that 23 of the patients were married & 17 unmarried: 31 of the 40 were primiparae and 23 of these were under 23 years of age as were also two parous women, only one patient was over 40 years of age and she had been pregnant three times previously. The disease was fatal to the mother in 16 cases, 15 of these being primiparae. In 20 cases the child was born dead; 17 of these children were born of primiparous parents.

Of the fatal cases to the mothers seven out sixteen were unmarried which only gives a mortality of 7 out of 17 unmarried women who were attacked with convulsions.

Mortality

In 747 cases Johnson had 29 per cent deaths

.. 104  "  Hofmeier  .. 32  ...  ...  ...

..  73    "  Braun   ..  26  ...  ...

Spiegelberg says one out of every 3 or 4 patients attacked die.
Leishman says the mortality is about 30 per cent in eclampsia.
Galabin says that 30 per cent of the mothers die. In Guy's Charity, the mortality was 50 per cent in cases which began before the onset of labour, 25 per cent in those which began during labour, and only 8 per cent in those which began after delivery, the total mortality being 25 per cent up to 1875; in the ten years following, however, the total mortality was only 9 per cent.

Lohlein's statistics give a mortality of 40.5 per cent out of 83 cases which began before the onset of labour.
Lusk says that the fatal cases in New York City from 1867 to 1875 amounted to one in 500 deliveries.

Johnston & Sinclair of Dublin give figures showing a mortality of one in five.
Zweifel of Leipzig lost 28 cases out of 129; Krasing lost 9 out of 16 cases of eclampsia.
In the Royal Maternity Charity of London Ramsbotham had a mortality of 1 in 14 in the eastern district whilst in the western district Hall
Davis had a mortality of 1 in 11. Fordyce Barker gives 32 per cent as the mortality before and during labour and 22 per cent if the onset occurs after labour.
Swayne lost 11 out of 36 cases: six out of eleven coming on before labour & 5 out of 20 coming on during labour but none out of 5 in which the convulsions occurred after delivery.

Infantile mortality.

Galabin says that 50 per cent. of the children die. I have attended 3 cases of eclampsia & one case of albuminuria in pregnancy and in all four cases the result was fatal to the child though the mother recovered in each case.

Hall Davis says that more than half the children die, partly on account of the continuous and violent contraction of the uterus on the child's body & also because of the poisonous effect of the mother's blood which is impregnated with urea.

Spiegelberg says that probably half the children perish during or soon after birth: he says that
the foetus dies from asphyxia. Largausce mentions cases in which convulsions have attacked the child in utero; after birth the child is sometimes attacked with convulsions even when apparently healthy at birth.

Some suggestions as to Causation.

The theories as to the causation of eclampsia are very numerous, before discussing those most generally accepted I will mention some theories recently put forward.

1. Parker describes a class of cases due to intestinal putrefaction, constipation, absorption of ptomaines etc. Féré ascribes puerperal convulsions to a neuropathic pre-disposition.

2. Raikes thinks the lime-salts ingested in the drinking water favour the formation of toxic materials in the blood which combined with the nervous condition peculiar to pregnancy give rise to eclampsia.

3. Butte thinks that more importance should be attached to the hepatic than the renal lesions.

Vinay names as predisposing causes of the affection, age, first childbirth, twin births, prolonged labour, vertex presentations, contraction of the pelvis, hysteria, the season of the year, epidemic factors and contagion; and as determining causes lesions of the kidney & liver, compression of the waters and microbial agents.

Andral, Dugès, Smellie, Ramsbotham Simpson & others believed that the season of the year was important & that convulsions were more prevalent in hot weather: in going over the last 40 cases treated in the Edinburgh Maternity Hospital I find that 7 cases occurred in the first quarter of the year + 12 in the second, + again in the third + 14 in the last so that here the evidence points rather to their more frequent occurrence in winter.

Eichler says that the following theories as to causation are too hypothetical: 1. Guesserow's, Bamberger's and Moericke's which ascribes the renal lesion to a dilated state of the abdominal veins.

2. Halberstam's and Haischlen's attributing the disease to compression of the ureters.
3. Leyden's that the essential cause is an increased intra-abdominal pressure leading to renal anaemia.
4. Schroder's that the fits are due to renal anaemia as a result of spasmodic contraction of arteries.

Bouchard has proved the lessened toxicity of the urine of eclamptics and considers that eclampsia is due to an auto-intoxication.

Barnes' considers puerperal convulsions are due to accumulated irritability of the nervous centres called into action by a stimulus usually conveyed from the uterus when a peculiar form of blood poisoning exists as in albuminuria or from other causes.

There is no doubt that the pregnant woman is much more sensitive to stimuli of various kinds, whether emotional or derived from diseased organs, than the non-pregnant female.

Barnes thinks that nature provides, against the period of parturition, a special supply of nerve force by an increased development of the spinal cord.

Pathological Anatomy

In fatal cases of eclampsia, generally only an early stage of tubal nephritis is found and some observers have not detected anything more than congestion. 'Braun', however, found evidence of nephritis in every one of 7 cases in which the kidneys were examined microscopically; the appearances to the naked eye were the same in other cases not examined microscopically. Of 4 fatal cases recorded by Bouverelle, parenchymatous nephritis was found in one and in the remaining three the appearance of acute tubal nephritis; namely an opaque, swollen, yellowish-white cortical substance, and deeply congested pyramids.

Ange Macdonald 2 arguing from the autopsies of two cases, considered the renal condition to be a degeneration rather than an inflammation. The epithelial cells in some tubes being converted into a colloid material which plugs both these and other tubes. Certain cerebral conditions such as anaemia of the brain substance congestion of the meninges, and sometimes

small extravasations of blood in the brain substance have been described. None of these are proved to be the essential cause of the convulsions and it is probable that the small extravasations may have been the result rather than the cause.

From post-mortem examination of 4 cases of eclampsia Erlich1 thinks that eclampsia during and after labour is caused by embolism of the renal arteries: he says that some puerperal lesion is the starting point of the embolism: the great objection to this theory is the difficulty of explaining cases of eclampsia occurring before labour commences. Erlich1 found the cerebral meninges were anaemic and oedematous, whereas Angus Macdonald2 says that in his cases the meninges were congested or the venous sinuses were engorged.

Imbert Gourbeyre3 considers puerperal eclampsia is nothing else than puerperal Bright's disease in which convulsions occur and as Bright's disease sometimes occurs without albuminuria so may eclampsia.

Massen from a study of post-mortem lesions in eclampsia found that all the organs were affected in this disease and that the appearances of an acute intoxication were present.

Lejden regards the condition of the kidneys in eclampsia as distinct from all others: he has found them in a state of fatty infiltration due to prolonged arterial anaemia.

Stumpf has suggested that acetone or an allied body irritates the kidneys in its elimination as causes nephritis: he found acetone and also sugar in the urine of eclamptics frequently.

Schmick found necrotic foci in the liver in 15 cases which he divides into haemorrhagic and anaemic necroses: haemorrhages of the brain were found in the majority of cases.

Mohamed considered that eclampsia was due to the rupture of minute blood vessels within the substance of the brain but it is more probable that the small extravasations found are the result rather than the cause of the convulsions; his experiments show the effect of increased arterial tension on the renal circulation.

Causation of Albuminuria

It is universally admitted that albuminuria is present in the vast majority of cases of eclampsia: we will now consider the reasons for the occurrence of this symptom in pregnancy.

1. The first theory put forward was that pressure upon the renal veins by the gravid uterus produced passive congestion of the kidneys: this was Lever's view, he called attention to the fact that out of 10 cases of eclampsia the urine was albuminous in nine: Rambottoman refers to this point as being important & says that his own experience of eclampsia has been similar but he does not attempt to explain the phenomenon.

It may be as well to mention here that Hamilton in 1800 & also Denuet in 1801 had previously recorded the fact that puerperal convulsions were preceded by albuminuria. The latter observer considering it one of the essential causes but Lever explained the pathology of the disease as regards many cases.

The most important objection to this theory of Lever's is that albuminuria is sometimes present in the early part of pregnancy before pressure on the renal veins can exist. Bartels in Ziemssen's Cyclopaedia maintains that even when the uterus is fully enlarged at the end of pregnancy, pressure on the renal veins is a physical impossibility.

King recently in the Lancet argued that in the latter months of pregnancy the renal vessels & ureters may be pressed upon owing to malposition of the foetal head. He says the proper position of the foetal head is in the iliac fossa not on the brim of the pelvis.

2. The second theory - that of Halbertoma - supposes the uterus to press not on the renal veins but upon the ureters. In consequence of this the kidneys will have to secrete against a higher pressure. This cause may operate whilst the uterus is still in the pelvis. Fibroids are known by pressure on the ureter to produce dilatation above the point of pressure; & there is some
direct evidence that this pressure by the pregnant uterus is a true cause for Lohlein found that in 8 out of 32 fatal cases of puerperal eclampsia dilatation of one or both ureters was discovered post-mortem. Nash of Bedford has recently recorded a case of this kind.

An important ground for the conclusion that mechanical pressure in one or both of the above ways is often an element in the case is the fact that albuminuric or eclampsia are much more common in primiparae, the tension of whose abdominal walls is greater than in multiparae. Rather opposed to the mechanical pressure theory is a case reported by Kidd of 35 attacks of eclampsia in a woman only 4 months pregnant: the abortion + recovered; there was never at any time suppression of the urine.

3. A third cause suggested is that increased work is thrown on the kidneys by the uterus having to excrete the waste products from the foetus + enlarged uterus. If the kidneys

1. Wiener medizinische wochenschrift. Vienna Sept 17 1891
are weak and barely equal to the normal work this additional strain may be enough to upset them: but as puerperal eclampsia is more common in young and previously healthy women I think it is rather out of our way to assume a debilitated condition of the kidneys before pregnancy to explain a condition met with in pregnancy, so that this cause though possible in a few is not probable in most cases.

4. A fourth cause may be the increased arterial tension which Galabin and others maintain is always present in pregnancy. This will increase any tendency which may exist to exudation or diapedesis of leucocytes into the kidney tissue. The increased arterial tension is associated with the normal hypertrophy of the heart in pregnancy described by Larcher in 1857.

5. The last possible cause is one suggested by Tyler Smith namely that of a reflex influence starting from the uterus and disturbing the circulation or secretion of the kidneys just as the circulation...
secretion of the mammary gland always
of the salivary gland sometimes
are disturbed in pregnancy.
With regard to the relation of albuminuria
to eclampsia it may be as well to
mention the investigation of Santos,
of Budapest, into the question
of what percentage of pregnant
women suffer from albuminuria.
He found the urine albuminious in
60 per cent of 600 newly-delivered
women, 70 per cent of 268 primi-
parae, 50 per cent of 332 multiparae.
Santos believes that albuminuria
occurring in pregnancy is the
result of a reflex irritation of the
sympathetic and renal nerves, due
to the irritation of the uterine nerves
incident upon the enlargement, and
later to the contraction and retraction
of the uterus. In his opinion albumi-

nuria is a physiological condition
in pregnancy and in fact is of
diagnostic value. Eclampsia he regards
as an acute peripheral epilepsy
having its origin in the uterus.
Palmer found from the examination of
the urine of pregnant women in the
Cincinnati Hospital that albumen
1, British Medical Journal Jan 12, 1889.
was present in 50 per cent of the cases.

Parkes, after a most careful examination of the urine of 30 puerperal women found albumen in every case and he believes that every woman directly after labour has albumen in her urine. It also that this albuminuria is due to a renal affection and not or at least not solely to a genito-urinary catarrh.

Perhaps it would be better if more attention was paid to the quantity of urea which is being excreted by the patient instead of laying so much stress on the presence of albumen in the urine.

For to quote Lusk: "unnecessary stress is laid on the presence or absence of albumen. It is the renal insufficiency not the albuminuria which causes uremia and convulsions." Spiegelberg draws attention to the fact that the quantity of urea present in the urine of a patient prior to the onset of the convulsions may be much diminished.

Herman's writing on puerperal eclampsia in 1889 says "It is of course no new view that the fits of eclampsia are ureamic i.e. due to the deficient excretion of urea; but this view has not been universally admitted and so far as I know it has been supported only by arguments based on our knowledge of kidney disease generally & not demonstrated by observations on the amount of urea excreted by puerperal eclamptics." He then relates two cases in which he carefully examined the urine both before & after the fits. Since then he has published 5 other cases & in all of these the urea was found to be present in very much smaller quantity than normal just before & just after the onset of the first fits but after delivery & when the fits had ceased the quantity rapidly increased. The specific gravity before the fits was high & rapidly fell afterwards. In the process of recovery first more water passed through the kidneys and the specific gravity of the urine fell and then the excretion of urea became re-established.

Since then Herran has had 5 other cases in all of which the urea was less than normal before the fits & rapidly increased after they ceased. In one of his cases the urea was only .7 per cent before the fits & two days after it was 2.3 per cent. These 12 cases showing the same point are pretty strong presumptive evidence in favour of the diminution of urea before the attack and increase afterwards being a constant occurrence.

The quantity of albumen in eclampsia is generally considerable often amounting to one third or a quarter of the bulk of the urine, after the application of heat & nitric acid; in some cases it becomes absolutely solid on the application of heat.

There are some cases of eclampsia in which no albumen is present in the urine from first to last or in which none is present at the onset of the convulsions but appears shortly afterwards, at first in small quantities and afterwards in profusion; these cases will be discussed later, they have been especially considered by Braxton Hicks, 1. Lancet II. 1891 p. 74 also Obst. Transactions Vol. 32.
Herman has ascertained in his cases of eclampsia whether the precipitate of albumen consisted mostly of serum-albumen (as in Bright's disease) or of paraglobulin: out of 9 cases in which the amounts were separately ascertained he found that in 5 the albumen was nearly all paraglobulin, in the 4 remaining cases however he only found small quantities of paraglobulin. However Champneys & Whipham recently investigated a case at St. George's Hospital and they found that serum-albumen was present to the extent of 75 per cent whilst only a small quantity of paraglobulin was to be found.

If continued observation on this point should show that in cases of simple eclampsia the albumen is always paraglobulin it may be an important addition to our knowledge of the subject & the pathology of the disease. For Ralfe & Maguire state that the circumstances in which paraglobulin - the most diffusible kind of albumen - is found in the urine are such as to warrant the belief that its presence
is due rather to altered pressure in the vessels than to changes in the renal cells: and this together with the preponderating frequency of eclampsia in first pregnancies and towards the end of pregnancy would go to show how important a factor increased pressure is in the production of the disease.

The presence of albumen in the urine in cases of eclampsia was long ago pointed out by Lecler in this country and by Rayet in France but Braxton Hicks showed that in some cases the albuminuric follows and does not precede the convulsions of which it therefore might be supposed to be the consequence rather than the cause. Blot, Litzman, Petit and Hypolite met with albuminuria in 20 per cent of pregnant women which is however says Playfair far above the estimate of other authors. Fordyce Barker says it occurs in 4 per cent while Hofmeier found it only in 2.74 per cent.

In 41 cases of albuminuria in pregnancy Blot met with eclampsia only 7 times: Seyffert met with only two cases of eclampsia out of 70 cases

of women with Bright's disease becoming pregnant: Hofmeier in 46 cases of women with chronic nephritis becoming pregnant met with eclampsia in 15.

**Caution of Eclampsia**

   
   This theory was held by the older British and American obstetricians. Denuce taught that puerperal convulsions were epileptic, apoplectic or hysterical. He held that a strong premonitory symptom was a "determination of blood to the head."

   Meigs asserted that the disease was caused by long continued or violent determination of blood to the head by the rapid revolution of the blood excited by pregnancy or labour or by too intense perception of the pains of labour.

   Hodge held that convulsions in a large proportion of cases arise from a congestion of the blood-vessels of the brain or from an actual effusion of serum or blood into its substance or cavities.

   But cerebral congestion is now thought to be the consequence rather than the cause of eclampsia.
2. Acute Cerebral Anaemia according to the Traube-Rosenstein theory.

In support of this theory are:

(1) the haemorrhagic condition of the blood in pregnancy.

(2) the intensification of this condition when there is albuminuria.

Accompanying these are:

(1) increased arterial tension.

(2) hypertrophy of the heart which favours the former.

These conditions result in a temporary hyperaemia of the brain which is rapidly succeeded by serous effusion into the cerebral tissues, resulting in pressure on its minute vessels and consequent anaemia.

Convulsions are the consequence of anaemia of the bulb and coma of anaemia of the encephalon. This theory agrees with Kussmaul and Foren's and Brown-Séguard's ideas on the relation of cerebral anaemia to convulsions. It also would explain how the occurrence of labour should intensify the convulsions since during the height of the pains the tension of the cerebral arterial system is necessarily greatly increased.
Hecker answers this theory thus:—

1. "The hydramnios which is a necessary condition for the theory is so common in pregnancy that if it had an important influence, eclampsia ought to be of much more frequent occurrence; and on the other hand, as Spiegelberg says, this hydramnios is not always present in eclamptic women—they are indeed frequently young and vigorous.

2. The increased arterial tension may occur in labour but is not found in pregnancy or the lying-in state."

This is a point at least open to contest for Galabin and others maintain that in pregnancy arterial tension is always increased.

3. "The anaemia and oedema of the brain are much more naturally explained as secondary to the attacks, than regarded as primary and the cause of the convulsions. It is frequently observed that consciousness—a correlate of an intact brain—is lost to a greater degree the more frequently the attacks occur."

3. Cerebro-spinal Anaemia with Congestion of the Meninges. Angus MacDonald made two careful post-mortem examinations of patients who died from eclampsia and found in both of them intense anaemia of the deeper portions of the brain, especially of the basal ganglia and the cord, with congestion of the meninges and engorgement of the venous sinuses on the inner aspect of the cranium and spinal canal. He found no oedema of the brain. The theory then proposed by him was that eclampsia is caused by irritation of the vaso-motor centre in consequence of an anaemic condition of the blood produced by the retention in it of toxic material, this over-stimulation resulting in anaemia of the deeper seated nerve centres and consequent convulsions. Moxon\(^2\) has shown that this theory of convulsions resulting from cerebral anaemia from vaso-motor spasm is untenable. Simpson\(^3\) criticised the above cases reported by MacDonald, and said that he had based another theory upon morbid anatomy of one case.

2. Croomian Lecture 1881.
4. Retention in the blood of some toxic agent and a poisoning of the Nerve-centres thereby. The question then is—What is this toxic material?

(a) Urea. Braun started this theory in 1857. Against this, although the blood of the patient suffering from eclampsia contains an excess of urea there is no eclampsia in the cholera patient whose blood is loaded with this substance. Claude Bernard, Sir P. Richardson and others injected urea into the blood of living animals and failed to produce the phenomena of puerperal convulsions.

(b) Carbonate of ammonium. This was Freireich's idea, that the urea was decomposed in the blood into carbonate of ammonium; he made some experiments to prove this. Spiegelberg, some years later experimented upon dogs and maintained the accuracy of Freireich's views.

Hammond of Maryland made experiments with a contrary result. From later experiments Spiegelberg now says that he can only regard anaemia as one of the rarest causes of the convulsions.

(c) The poison is not urea alone but also various excretories such as kreatin + kreatinin + etc. The salts of potash have also been suggested by some observers, hence the name uroæmic, instead of uræmic, has been proposed. With regard to this, however, as Tagge says, the term "uroæmic" is generally now taken to mean the poisoning of the nerve centres by materials accumulated in the blood, as the result of defective excretion by the kidneys. But it is still uncertain whether the poisonous action is excited by one substance in particular or by all of them together.

(d) Among the most recent suggestions as to the toxic agent in the blood of eclamptics is that made by Doléris and Butte: they found soluble toxic ptomaines in the blood of eclamptic patients which they believe may be the cause of the disease at least in some cases. They argue from this discovery that the exclusive idea of renal insufficiency from congestion, spasm, compression, old lesions of the kidney etc may be put aside & the purely nervous theory also
They suggest that eclamptic like epileptic attacks do not have a single etiology.

(2) In 1887, Blanc discovered in the urine of eclamptic women a micro-organism which inoculated into rabbits produced convulsions. More recently Blanc has discovered a peculiar slender bacillus one or two micro-millimeters in length which inoculated into rabbits produces convulsive phenomena and later on acute nephritis. The blood and fluids of the inoculated animals have also the power of producing convulsions in other rabbits. From these experiments he concludes that there are some cases of puerperal convulsions due to an organism which causes infective nephritis, and this appears to generate a convulsive poison.

Cordes considers the eclamptic bacillus as the only cause of eclampsia since it is not found in other diseases and since eclampsia does not occur without it. Döderlein, however, from examination in 8 cases comes to the conclusion that the bacillus observed by the former is not the cause of the disease; his results are in accordance with those of Hoffmeister and Hugler.

Chambrelent studied three cases finding the blood serum to be much more toxic than in the normal state: by inoculation of the blood & urine in various media he got microbes but exceptionally these he thinks were derived from the atmosphere of the room.

2. Combemale & Cé of Lille have arrived at the following conclusions:

(1) Staphylococci are the direct cause of puerperal eclampsia. Other micro-organisms have been found by various authors but the staphylococci is found in cases where the eclamptic attacks follow labour, the eclampsia being considered as a manifestation of puerperal infection.

(2) The soluble products of the staphylococcus are eclamptic substances: without bringing to bear any personal experience on this subject, the experimental & clinical researches of other writers show that the toxins produced by the staphylococcus are the same as those contained in the blood of infected lying-in women.

Rapin & Monnier examined the blood of 4 women with eclampsia and in three of the cases found a special bacillus which they were able to isolate & cultivate; inoculated into animals it causes symptoms similar to those of eclampsia.

1. Chirhardt & Favre of Paris trace eclampsia to nephritis set up by the presence of germs in the placenta. In the placenta of 19 cases out of 20 of albuminuria these observers found white infarcts. All the patients except two were subject to leucorrhoea and endometritis when not pregnant.

2. Colonies of bacteria were found in the infarcts and when some germs taken from the placenta of patients who had suffered from eclampsia as well as albuminuria were injected into the veins of rabbits & guinea-pigs parenchymatous nephritis was set up. Thus the morbid changes in the decidua which cause placental infarcts are induced by the same agency that induces the nephritis of pregnancy. This agency is the presence of germs in the placenta which germs produce in non-pregnant patients leucorrhoea and other symptoms.

of chronic endometritis. The focus of
infection being the uterine mucous
membrane that structure requires
attention + treatment.
Jenkins publishes a case which he
explains by Favre's theory that puerperal
 eclampsia is due to the action on
the cerebrum of substances formed
in the evolution of a local infective
process.
Until the experiments mentioned in
the two last paragraphs (e) + (f)
are confirmed by others I don't
think they can be generally accepted.
However the view that eclampsia is due
to a retention in the blood of some
toxic agent + consequent poisoning
of the nerve-centres by it is the
most probable theory + failure of the
kidneys seems to be the cause of
this retention in the blood: beyond
this our present knowledge of the
subject does not permit us to
say anything definite but the
disease may some time be proved
to be due to a poison derived
from the eclamptic bacillus as
has been lately suggested.

5. Reflex Nervous Impulses as the Cause:

There are 3 theories under this heading:

(1) That which attributes the convulsions to cerebro-spinal disturbance from periphereral stimulation and quite independent of the kidney.

(2) That which suggests that the convulsions are uraemic i.e. due to retained waste products, but the renal disturbance which gives rise to this uraemia itself results from some reflex irritation, vasomotor spasm of the small renal vessels, with consequent degenerative changes in the kidney.

(3) That which proposes to explain these cases in which there is no albuminuria from first to last by considering them as acute epileptic attacks, the area of distribution of the sciatic nerve being the epileptogenic zone.

To take this (3) theory first:—

Spiegelberg says of the attacks in these cases that they may be grouped in the same category as the epileptic attacks which can be artificially induced. Further, there are many facts which show that the nerves of the generative organs actually correspond with very important areas of irritability.
which may act as so-called epilepto-
genic zones.
It would then be merely necessary in
any case of eclamptiform paroxysms
to demonstrate some unusually great
irritation in the pelvis, though the
possible existence of individual proclivity
or of special irritability would also
need consideration.
He mentions a case of such convulsions
in which the source of irritation
probably lay in an enormously
distended bladder, after the emptying
of which the fits ceased. La Motté’s
classical case was precisely similar.
Taking next the first theory (1) that
puerperal convulsions are reflexe
actions excited by cerebro-spinal or
medullary irritation of uterine origin
and transmitted through the ganglionic
cells in which reflex nerves terminate,
this is traced back by More Madden
to Laurence Joubert of Montpellier in
the middle of the 16th century. He
asserted that the cause the cause
was irritation only by removal
of this cause could the convulsions
be arrested.
Tyler Smith was, I think, the earliest
English writers to adopt this theory; he 
said in 1844, "The true puerperal convulsion 
can only occur when the spinal 
marrow has been acted upon by 
an excited condition of an important 
class of incident nerves namely those 
passing from the uterine organs to 
the spinal centre, such excitement 
depending on pregnancy, labour 
& the puerperal state. While the spinal 
marrow remains under the influence 
of either of these stimuli convulsions 
may arise from two series of causes, 
these acting primarily on the spinal 
marrow or "centric" causes & secondly, 
those affecting the extremities of its 
incident nerves - causes of "eccentric" 
or "peripheral" origin.
Frankenheimer of Jena has demonstrated 
& illustrated a direct connection between 
the nerves of the uterus & the renal 
ganglia. He believes that the sudden 
occurrence of the eclamptic attack 
following an external source of 
irritation (e.g. pressure of the fetal 
head on the cervix, per vaginam 
examination etc.) and from emotional 
causes goes to prove that the nervous 
system is not the vascular system. 1. See also Medical Times & Gazette Vol. II. 1852 p. 63.
is the starting point of puerperal convulsions.
The latest adherent to this theory is Pajot, who declines to admit the dependence of eclampsia on albuminuria. He believes that the cause is reflex, culminating in a cerebrospinal centre in close proximity to the disorganized centre which presides over the existence of albuminuria. He thinks albuminuria predisposes to attacks but does not act as an exciting cause.
The warmest advocate for the second (2) the reflex nervous or uremic theory is Spiegelberg. He says "We must cling to the central idea that the disease is of the nature of uremic poisoning due to an inadequate secretory activity of the kidneys - a view which is not only plausible in virtue of its simplicity but which explains all symptoms and harmonizes with all our experience."
When in women who have not suffered for some time previously from Bright's disease, eclampsia more or less rapidly develops, Spiegelberg says it is with great probability caused by an acute
affection of the renal vessels this vaso-
motor spasm being caused in a reflex
manner by irritation of the uterine nerves.
It is well known that in the worst
cases the bladder may be found for
a time entirely empty & in all
cases the quantity secreted is
exceedingly small.

That in a number of cases the suppression
of urine does occur suddenly is well
shown by Hofmeier, quoted by Lush,
who reported 104 cases in which he
had found the kidney symptoms
develop suddenly.

This sudden suspension of the urinary
secretion can only result from
disturbances of the renal secretion.
A rapidly developed affection of the
vessels would leave no post-mortem
changes & would in a case of
recovery, disappear as quickly as
it had come.

If the renal troubles were due to
pressure of the gravid uterus on
the renal veins & ureters, renal
engorgement & other post-mortem
changes should be found in all cases,
whereas, although some authorities
describe such changes, others—
Spiegelberg & Lothheim particularly have found the organs often pale and bloodless.

The precise change in the vessels is, either that the vessel walls are altered in such a way as to interfere with the process of diffusion and this view would seem most probable in those cases in which prodromal symptoms are present, or the disturbance might consist in vasomotor spasm of the renal vessels.

We know how sensitive the secreting epithelium of the kidneys is to even brief interruptions in the blood flow: spasm of the arteries will entirely arrest secretion and if it occur repeatedly will kill the epithelium and cause the constituents of the urine to be retained in the blood. A similar influence of the vasomotor nerves is known to exist in the case of other organs. There are, moreover, other signs which indicate that such an irritation exists in eclampsia e.g. the wide & sluggish pupil at the beginning of the attack & the spasm of the cutaneous vessels.
It is not improbable that the vasomotor irritation proceeds from the uterus—indeed, the association of some special irritation of the sensory nerves of the uterus with the convulsions has been frequently pointed out: if we suppose that the vasomotor nerves of the kidneys which run in the splanchnic nerves are irritated in a reflex manner by the uterus, we explain the undoubted causal connection between uterine contractions & convulsions.

In 'Cohnheim's Pathology' that author says that the true explanation of the whole subject of eclampsia is provided by the "hypothesis of an arterial spasm of the small renal arteries of reflex origin, that in a moment gives rise to the retention of the urinary constituents in the blood."

There is much to be said in favour of these theories of reflex irritation. By them we can answer two of the three objections which have been raised to anaemia as the cause of eclampsia.
The objections to the uraemic theory are—

1. That a woman who has suffered for some time previously from Bright's disease not uncommonly escapes puerperal eclampsia.

2. That in many cases of puerperal eclampsia the pathological changes found in the kidney post-mortem are very slight or they may be absent entirely.

3. Eclampsia may occur without albuminuria at all or this may not set in until after the convulsions.

1. To the first objection we may answer, that in chronic nephritis there may be sufficient healthy renal structure for the work of elimination; it is only when a fresh inflammation takes place or when extra strain thrown on these damaged organs is more than they can stand, that renal insufficiency occurs, + eclampsia follows. Also in chronic Bright's disease the nerve-centres become in some measure tolerant of the poisoned blood.

2. The reflex vaso-motor theory answers the second objection. As stated just now, the complete +
sudden arrest of the urinary secretion such as certainly does occur in many cases of eclampsia, must depend on some disturbance of the circulation through the kidney and such a disturbance need not leave any definite traces post-mortem. At the same time though there need not be any actual post-mortem changes the vaso-motor renal spasm probably can, as Cohnheim says, give rise in a moment to the retention of the urinary constituents of the blood. This is the only theory that explains the numerous instances in which, as soon as the paroxysms subside and recovery occurs, all the renal symptoms disappear and rapid and complete restoration to health follows.

3. The first half of the third objection to the uraemic theory viz. that eclampsia may occur without albuminuria from first to last - these cases are very rare but still they do occur - can be explained by regarding such cases as acute peripheral epilepsy with the pelvic and uterine nerves as the epilepto-genie zone.
This theory is now being very generally accepted as a reasonable explanation of these cases. No explanation seems to be forthcoming for those cases in which there is no albumen at first but after a few hours albumen appears in the urine and continues for some days. These are the cases from which Braxton Hicks argued that the uraemic theory was erroneous, because at the time of the fit there was no nephritis - albuminuria being taken as the only evidence of renal change: to many other observers it is these cases which seem to have formed a considerable objection to explaining the convulsions as due to the retention of urea in the system, it not having been suggested that this could arise otherwise than by an antecedent nephritis accompanied by albuminuria.

Perhaps a consideration of the physiology of the kidney may enable one to find an explanation of these cases. Michael Foster says "We may for the present conclude that the secretion of urine does consist
of 2 separate & distinct acts—glomerular & tubular secretion. The work of the epithelium of the tubules is largely if not entirely confined to simply picking the urea out of the blood, or pushing it so to speak into the lumen of the tubules. "The work of the epithelium of the glomerular portion on the other hand is mostly concerned with the passage of water from the blood together with some of the solubile diffusible constituents of the blood: as long as the epithelium of the glomerular loop is intact & sound, there passes through neither albumen nor globulin." Next on the authority of Landouix & Stirling, Litten & Ribbert we learn that ligation of the renal artery even when it is obliterated only for 2 hours, causes necrosis of the epithelium of the uniferous tubules. If the ligation be continued for a longer time the epithelium of the glomeruli becomes greatly changed & finally the whole renal tissue dies unless the ligation be removed. Starling of Guy's Hospital though
admitting the accuracy of the above statement says that in many cases if the artery stopped is small the glomerular epithelium does not degenerate at all. The nerves of the renal plexus are believed to be controlled by a “renal centre” which lies in the floor of the fourth ventricle, in front of the origin of the vagus. To attempt to explain by physiological facts the particular cases of postpartum eclampsia we are discussing. 

First, all authorities agree that during pregnancy the irritability of the nerve-centres is greatly increased; in fact there is a sort of general hyperaesthesia: then, suppose that as a result of some peripheral or emotional disturbance a stimulus from the renal centre descends along the vaso-motor nerves to the kidney in the form of vaso-constrictor impulses what will happen? In some instances these impulses may produce such spasm of the vessels of the kidneys that total suppression of urine may occur, lasting according to Spiegelberg and Overbeek, as long as three quarters of an hour.
But suppose this reflex contraction be not so intense as to totally stop the secretion of urine, but sufficiently intense to damage in an hour or so the epithelium of the uriniferous — or urea-eliminating — tubules so lead to a retention of this substance in the system: we have here the case of the patient previously healthy apparently without albuminuria suddenly developing convulsions as a result of this retained morbid material.

Suppose then that this anaemia of the kidneys from vascular spasm goes on for a longer time, what happens? Simply that the epithelium of the glomeruli will suffer as that of the urinary tubules has already suffered and then we have albumen transuding.

At first only the urea-eliminating epithelium is damaged, later on that of the glomeruli suffers too.

This seems a possible explanation of these cases of puerperal eclampsia where the urine tested at the time of or just before the first fit is found to be free from albumen, but after a time this substance appears — in small quantities at first, in greater quantity later.
In support of this theory it would be an advantage to find that in the non-albuminous urine secreted just before the first fit the amount of urea was diminished and that as albumen appears the amount of urea increases again. But as this kind of eclampsia occurs in those apparently quite healthy it is very rare that a specimen of the urine is obtained before the first fit has occurred.

The objections which may be raised to reflex vaso-motor spasm causing uraemia & nephritis are:

1. The theory is based largely upon physiological knowledge & experiments. I think that Raynaud's disease however is a good example of pathological change from vaso-motor spasm: also Lohnhein thinks that the albumin-uria following epileptic attacks, or in tetanus or lead colic is a consequence of temporary tetanic contraction of the small renal arteries.

2. The theory assumes an exciting cause to start the reflex spasm. If the pregnant uterus be regarded as this exciting cause, why do not all pregnant women have eclampsia.
Again, will uterine irritation explain post-partum eclampsia? Also will uterine irritation explain the fact that a woman may have 9 good confinements and have eclampsia with the 10th?

Answers: - There is a great increase of nervous irritability induced by pregnancy; this is much more marked in some women than in others. 60 per cent of all eclampses are primiparae, a great many being also unmarried, which is often a source of distress. The exciting cause need not arise from the uterus; fright, etc. may be the cause: in such a case the irritation would be central not peripheral. Supposing however that the exciting cause be the uterus: the fact that all pregnant women do not have eclampsia, can, I think, be explained by the knowledge that all women are not of the same temperament; some are much more excitable than others - we can imagine that a stimulus from the uterus which in one woman would be nothing would be in another quite enough to start the trouble.
Uterine irritation will I think also explain post-partum eclampsia because as Tyson in his monograph on "Puerperal Eclampsia" says, "the sting of the lash does not cease when the whip is laid down" and because the uterus has been emptied of the main source of irritation it does not follow that everything will at once settle down to involution take place. The cervix uteri at any rate may be in a very bruised & lacerated condition.

(3) The next objection which might be raised, particularly in the case of the theory I have laid stress on, which assumes the convulsions to occur in a comparatively short time after the stimulus, is that the amount of urea & of other urinary constituents which is retained in the system from this vascular spasm is insufficient to cause convulsions in a few hours.

In answer to this and in spite of certain physiological experiments pointing in another direction, I would submit that we are not able to say anything about the dose of urinary constituents necessary to so
poison the blood of a human being that
canvulsions result. The cerebral centres
which in a case of chronic Brights
disease may tolerate a large dose
may in a fresh young girl or
in a debilitated woman behave quite
differently.
For my own part, in conclusion I
think the mistake so many writers
have made is that they have tried
to draw too hard and fast a line
with regard to the pathology of this
disease. The clinical history of the
cases undoubtedly varies and why
should not the pathology vary also?
I believe that, until we know more
on the subject, the nearest we can
get to the truth is by assuming
that there are at least four
different groups of cases of puerperal
eclampsia:
(1) Cases of undoubted chronic Brights disease
where pregnancy has thrown on the
weakened organs a strain they are
unable to bear. These are true cases of uræmia.
(2) Cases where there is no evidence
of nephritis from first to last—
the so-called cases of acute
peripheral epilepsy.
(3) By far the largest group, where the convulsions are uraemic from deficient secretory activity of the kidneys, the first change being spasmodic contraction of the small renal vessels, followed by inflammatory changes in the secreting epithelium akin to acute Bright's disease: this vaso-motor spasm being started reflexly, probably in most cases from the irritation of the uterine nerves but not necessarily so in all cases.

(4) Perhaps a subdivision of class 3, where in a patient apparently previously healthy convulsions suddenly occur and albuminuria though not present at first appears shortly afterwards. Here the convulsions are the result of uraemia from sudden suppression of urine in kidneys previously healthy. Uraemia is the first result, and albuminuria the second.
Treatment of Eclampsia

Barnes puts down four cardinal principles for our guidance:

1. Moderate & control nervous irritability.
2. Moderate vascular tension.
3. Cut off emotional and peripheral irritants or excitanes.
4. Eliminate all complicating morbid conditions.

He goes on to say that treatment should be:

(1) Prophylactic (2) Remedial (3) Restorative.

Venesction

Until recently venesction was regarded as the sheet-anchor in the treatment of blood was frequently removed in large quantities. Lever\(^2\) for instance says "moderate bleedings are of no service" and he took 122 ounces of blood from one patient who recovered!

Phillips\(^3\) was one of the earliest to show the fallacy of this treatment; after giving an account of eight cases successfully treated by chloroform without bleeding he says "That bleeding has no claim to be regarded as a remedy for puerperal convulsions and"

2. Guy's Hosp. Reports 1843. 3.
3. Ibid. 1840.
that in the majority of cases at least if seen at an early period of the attack it is unnecessary; also that bleeding is often injurious by predisposing to various puerperal ailments, by retarding convalescence and sometimes by increasing the violence of the paroxysms; that the present diminished mortality is probably chiefly due to the less free depletion which is now practised.

As long ago as 1774 Leake recognised the fact that convulsions arise from anaemia sometimes and he says that "convulsions from insatiation are much more dangerous than those from plethora as it is easier to empty than replenish the vessels." Venection was strongly recommended by Denman in all cases in which convulsions existed or were apprehended, the reason he gives for this treatment not being very sound; he says "Bleeding is known to lessen in a very effectual manner all the complaints in pregnancy which arise from uterine irritation and to a certain degree in pregnant women from most other causes."
Dewees considers bleeding our sheet-anchor in eclampsia but mentions a case in which he removed 140 ounces of blood from a patient without arresting the convulsions. Free bleeding was much advocated by Broussais who considered that the convulsions were the immediate consequence of a cerebro-spinal congestion of an active kind or even of a sanguineous effusion in the arachnoid or in the cerebral substance. Braun who considers that puerperal convulsions are almost always uræmic in character would dispense with venesection altogether. Depaul however thinks that the paller, the partial or general anaærea and the existence of albumen in the urine ought not to prevent our having recourse to free bleeding. Hodges in his book on "Puerperal Convulsions" in 1864 says "there can be no question that the judicious use of blood-letting is the most valuable remedy known" for this disease. Hughlings Jackson in 'Reynolds's System of Medicine' says "We do not know what the intra-cranial changes are
which cause fits & we have no evidence that cerebral congestion occurs before the attacks. I may say that repeated convulsions are not the signs which should make us deplete.

Trousseau disapproves of bleeding & says "We cannot admit that an attack of eclampsia is the consequence of a primary congestion when on the one hand we see that the severity of the fit is by no means proportionate to the degree of previous plethora & that on the other hand epileptiform convulsions which follow on a considerable loss of blood are as severe as those noticed under perfectly different circumstances."

Donati believes in the abstraction of blood in all cases which precede or accompany labour.

Barnes thinks that the moderate abstraction of blood in some cases e.g. where there is evidence of plethora with marked engorgement of the vessels of the face is beneficial.

Hall Davis says that in the aethenic form of eclampsia as amongst robust plethoric subjects we must resort to free

blood-letting. He says "generally one full bleeding will suffice, rarely is more than a second smaller bleeding called for": the amount of blood withdrawn must be relative to the patient's condition and the severity of the disease. He states that when Denman, Gooch and others resorted to the practice of free blood-letting the mortality from eclampsia was much reduced from what it was in the time of Smellie, William Hunter and Lowder, when more than half the patients died. Hall Davis thinks that although we must guard ourselves from taking more blood from the patient than is absolutely necessary we should not be timid in taking away what may be necessary to secure the patient's brain and spinal cord from injury. Playfair says that although the free bleeding formerly practised may have been beneficial in many cases, the improvement, however, was often only transient. He says "there are good theoretical grounds for believing that blood-letting can only be of merely temporary use and may even increase the tendency to convulsion".

He then quotes Schroeder who says, "If the theory of Traube or Rosensteini be correct, a sudden depletion of the vascular system by which the pressure is diminished must stop the attacks. From experience it is known that after venesection the quantity of blood soon becomes the same through the serum taken from all the tissues; while the quality is greatly deteriorated by the abstraction of blood. A short time after venesection we shall expect to find the former blood-pressure in the arterial system but the blood far more watery than previously. From this theoretical consideration it follows that abstraction of blood, if the above-mentioned conditions really cause convulsions, must be attended by an immediate favourable result. Under certain circumstances, the whole disease may surely be cut short by it. But, if all other conditions remain the same, the blood-pressure will after some time again reach its former height. Finally Playfair says that in a properly selected case and judiciously employed venesection is a valuable aid in the treatment.
of eclampsia, but it must only be looked upon as a temporary expedient. Leishman says that nearly all cases of eclampsia were treated by free blood-letting till a short time ago but now the plan of treatment is completely altered and perhaps in some quarters the rejection of the lancet has been too absolute: there are some cases in which it is very useful but indiscriminate bleeding was a monstrous error. Charpentier in 1873 contrasted the effects of the various remedial agents for eclampsia and gives the following statistics:

Mortality in cases treated by bleeding 35 percent

"  "  "  "  "  "  "  anæsthetics 11 percent

Cappie of Edinburgh thought that the most certain effect of blood-letting was to increase the proportion of watery elements in the circulation, it may therefore be a question whether in uræmic convulsions the relative proportion of irritating material in the blood may not also be increased by large bleedings. Swayne of Bristol regards bleeding as the most important remedy of all especially in those convulsions which have a centric origin.

Swayne's most recent communication on
the subject is recorded in the British
Medical Journal, Feb. 29, 1876. He here says
that venesection acts favourably by
reducing arterial tension, he quotes
Barnes who says he often uses it with
advantage although it is now considered
out of date. Swayne thinks that
anaesthetics are also of great service
to supplement the bleeding.
blood-letting, as a general rule, without exerting any valuable effect on the symptoms is calculated to produce irreparable mischief.”

Ramsbotham says that the lancet is highly valuable in puerperal eclampsia but “no more blood should be taken than is sufficient to produce the effect desired, whatever that quantity may be.” In the Medical Times and Gazette, Vol. I, 1862, p. 464, commenting on a case reported by Cotter in the same volume, Ramsbotham says that in his opinion the patient was saved by free blood-letting and that the reduced mortality compared with the figures of Smellie, Perfect, and Spence in last century is due to the improved treatment by venesection.

Quentin gives notes of six cases of eclampsia and points out the satisfactory result of free-bleeding in four of them, whilst two which were not treated at all proved rapidly fatal: he thinks our main reliance in the treatment of eclampsia should be on free bleeding repeated if necessary.

2. Archives de Toxologie, April 1878.
In 1842 Charpentier analysing 133 cases of eclampsia observed under Depard, made out a strong case against bleeding. He showed that the mortality was 41 per cent under a single bleeding, 54 per cent under repeated bleedings, and only 18 per cent under anaesthetics. Spiegelberg, Tanceurt Barnes, Lusk, Parvin and others all speak of bleeding still as undoubtedly the most powerful resource at command for rapidly and certainly lowering the vascular tension. Spiegelberg says "Venesection ranks first in true eclampsia: nothing else has such power of restoring to the kidneys, so sensitive to any alteration in the blood-pressure, their functional activity; few have such a sedative influence on the irritated vasomotor nerves. I therefore advise the practitioner as soon as he sees his patient, after the first fit, to extract at least seven ounces of blood from the basilic vein".

Dovejoy and Allen state that they did not lose a single case of eclampsia out of the fifteen from whom they abstracted blood.

Galabin says that venesection should be left as a last resort in cases in which all other means fail to arrest the convulsions or where it may seem desirable to relieve extreme venous congestion of the lungs.

In a discussion at the annual meeting of the British Medical Association in 1891, Ovarrd, Aust Lawrence, Edis, Robert Bell, Heywood Smith, and Robert Harvey all spoke in favour of venesection in eclampsia.

Tyson says "Unquestionably the most efficient treatment of puerperal convulsions is blood-letting, for by blood-letting we draw from the system the agent which is the most important factor in the production of uraemia."

Steele of Liverpool says his own experience induces him to believe that, notwithstanding the high authority of Trousseau, cases of puerperal convulsions are sometimes met with, in which early and copious venesection is an essential element in their successful treatment; he thinks that bleeding is remedial by relieving the oppressed nervous centres and restoring the balance of the circulation.

King of Washington says "Venesection relieves puerperal convulsions simply by lessening the force of the heart." He recommends opening the temporal artery to deplete locally, also to open a vein in the leg to relieve the distension of the lower veins & obviate congestion of the kidneys.

Elliot is in favour of the use of chloroform before all other remedies but in many cases he resorts to a moderate abstraction of blood although each year his confidence in this treatment gets less.

Donkin speaks very strongly in favour of venesection to about 40 ounces supplemented by purging & cold to the head. He says that an indication for blood-letting is the contracted state of the pupils which according to Haldane is produced by a full condition of the vessels of the brain.

Donkin says that Nature towards the end of pregnancy has made special provision for loss of blood & a woman soon recovers from a free bleeding at this time.

Murphy refers to a case recorded by Lange (Heidelberg Clinique) in which

narcosis failed and venesection was performed this also failed but when 7 ounces of defibrinated blood were injected recovery occurred, Murphy thinks this treatment would be worth trying when milder methods fail.

Tyler Smith says that the two main reasons for bleeding in eclampsia are its sedative action on the spinal marrow and its preservation of the brain from injury during the convulsions. He says "It is, I believe, in great measure from the effects of blood-letting in warding off accident from the brain that bleeding is so general in this disease. The due recognition of the distinct operation of blood-letting on the cerebral & spinal systems is of the utmost consequence. In plethoric states of the circulation it is in this disease curative in its action on the spinal marrow, preventive in its action on the brain.

Largiliere records seven cases of eclampsia all of which were successfully treated by free and repeated bleedings.

Copeman of Norwich in a lengthy paper on Puerperal Eclampsia lays down the general rule that convulsions before labour require bleeding & those after do not; whilst “convulsions during labour occupy a middle space and must be treated according as they partake more or less of the other two divisions.” Hubbard of New York says “That general bleeding is called for when headache continues after labour & is attended by flushed face, restlessness, convulsions of a tonic character & there has not been much loss of blood during labour.” Kidd however says that convulsions could not fail to be aggravated by the lancet & remarks “But the day for the lancet is gone by as for stage coaches or salivation in syphilis.

For my part I think we have an argument against venesection in physiology: Foster says “When an animal with normal blood-pressure is bled from one carotid the pressure in the other carotid sinks so long as the bleeding is going on and remains depressed for a brief period after.”

the bleeding has ceased. In a short time however it regains or nearly regains the normal height. This recovery of blood pressure after haemorrhage is witnessed so long as the loss of blood does not amount to more than 3 per cent of the body weight. Beyond that a large & frequently a sudden dangerous permanent depression is observed."

All authorities who advocate venesection do so on the ground of lowering the general blood-pressure. According to Foster to do this at all effectually 3 per cent of the total body weight has to be abstracted in the way of blood. Take an average woman as weighing nine stone i.e. 126 pounds : 3 per cent of this is roughly 4 pounds. Hence to make any real impression on the blood-pressure 64 ounces by weight of blood must be abstracted. Lever it is true advised copious venesection & as above stated once bled a patient 122 ounces but 14 ounces is about the utmost advised by the authorities in favour of bleeding & Lever did not take all his 22 ounces at once but on 2 or 3 occasions during a period of seventeen hours:
it would I think be rather too heroic
treatment to take at one time or
within a very short space of time
an amount of blood equal to 3
per cent. of the body weight.
On these grounds it seems to me that
venesection as practised for the purpose
of lowering the general blood-pressure
can be of little or no good to the patient
supposing her to be a strong and
previously healthy woman: and if she
be anaemic as many women during
pregnancy are, the loss of blood must be
actually harmful.

The above statements quoted from physiology
refer to the quantity of blood necessary
to be removed from an animal with
normal blood-pressure. It is possible that
in the case of the puerceral eclampsie, where
the blood-pressure is above the normal, the
abstraction of a small quantity of blood
may lower the pressure.

There is no doubt however that in a
few cases venesection may be an
exceedingly powerful agent for good,
in relieving an engorged right side of
the heart, for instance, but here no suggestion
of reducing the general blood-pressure arises, it is simply
a rapid means of relieving dangerous venous congestion.
Chloroform

Barnes recommends the moderating of excess of central nervous irritability by the use of chloroform. He quotes Achille Foix who explains the action of chloroform in shortening a fit in the following way: "The cessation of an attack is the consequence of the asphyxia which itself produced. The quicker the asphyxia the more quickly is its action felt upon the cords rendering it incapable of reacting. Thus the danger is averted by its very excess. Chloroform by inducing asphyxia acts in a similar way."

F tremaine relates 3 cases of eclampsia successfully treated by chloroform: in one, however, repeated bleedings were resorted to as well.

Copeman says "Convulsions dependent upon fear or alarm of an hysterical or epileptiform nature do not require bleeding but are more satisfactorily treated by opium or inhalations of chloroform." He quotes Carl Braun who says that chloroform inhalations are the best means of mitigating bringing to an end uraemic convulsions either during pregnancy, labour, or in the puerperal period.

Murphy\(^1\) speaks of the extremely beneficial effect of chloroform in eclampsia but thinks it best not to give it to the full surgical extent.

Suckling\(^2\) in recording a case of eclampsia says that chloroform had a remarkable effect in abating mental excitement and the intensity of the convulsions. Laveau, he says, strongly recommends chloroform for rigidity of the os + for persistent convulsions. Bartlett\(^3\) records a case in which the convulsions rapidly yielded to chloroform of the patient was under its influence for ten hours; no convulsions occurred after the birth of the child + the patient made a good recovery.

Byers\(^4\) after describing 4 cases of puerperal eclampsia says that for quieting the convulsions there is no remedy like chloroform: he approves of Lush's plan viz after beginning with chloroform to give per rectum half a drachm each of chloral + bromide and stop the anaesthetic when the effect of the other drugs becomes apparent.

Sir George Johnson\(^5\) says that the beneficial action of chloroform is due to the lessening

of the reflex excitability of the nervous centres and not to relaxation of the cerebral arteries.

Gallain ¹ says that chloroform produces a rapid lowering of excessive arterial tension and thus soothes the irritability of those nervous centres which excite the spasmodic action of the vasomotor nerves.

Jellie ² speaks very highly of chloroform followed by large doses of chloral to assist its action.

Chaillly Honoré saved 18 out of 19 cases which he treated with chloroform and Braun did not meet with a single death in 16 cases treated in the same way.

Discussing Freylich and Lehman's theory Hall Davis ³ says that the beneficial effect of chloroform is supposed to be due not only to its anaesthetic effect but also to its having the property of preventing the transformation of urea into carbonate of ammonia; this is explained thus:—Chloroform when fully administered is known to produce a tranquil presence of sugar in the urine and as sugar is known to prevent the ordinary change of urea into

2. Transactions of the Obstetrical Society 1869, Vol. XI.
carbonate of ammonia so it is inferred that its presence in the blood of the chloroformed patient serves to guard her from a repetition of these convulsions. Phillips strongly advocates the use of chloroform and says that by preventing the recurrence and checking the violence of the paroxysms it diminishes or prevents the danger from secondary congestion and exhaustion; he also lays stress on the advantage of being able to deliver with forceps or dilate the os under chloroform, if it is necessary to hasten the completion of the labour: the above theory as to the production of temporary diabetes by chloroform causing the area to be innocuous he regards as very doubtful indeed.

Strang 

regards chloroform as the best remedy for the convulsions, combined if necessary with morphine or chloral, the latter being preferable.

In the other hand Hastings Tweedy says that chloroform, pilocarpine or chloral all tend to kill in a manner similar to the eclamptic poison and they must therefore be avoided.

But it seems to me that the great indication in the management of eclampsia is to control the convulsive action by means of sedatives and first of these I would place the inhalation of chloroform. The general consensus of opinion now is in favour of chloroform and there are few practitioners who do not give it to some extent, whatever their other treatment may be.

Spiegelberg says that having lowered the blood-pressure by venesection, chloroform narcosis should not be induced too soon as the blood-pressure may be further reduced and death occur. As Barnes says: "Chloroform is both prophylactic and remedial. Used during the convulsion it certainly shortens it. By watching for premonitory twitchings and then giving it, the fit may be averted or certainly mitigated." Chloroform may be administered continuously for many hours if so time is gained for the completion of labour but I think it is better just to give a little more when there are indications of an approaching fit than to keep the patient very deeply under the anaesthetic all the time.

Chloral Hydrate

Dugardin has found chloral so satisfactory that he thinks it will replace chloroform in many cases, having the advantage over the latter of being employed where we require to keep the patient for a long time under the influence of some anaesthetic. In nervous women especially he thinks it useful as it diminishes the frequency & intensity but not the efficacy of the pains & thus shortens the labour and lessens the shock to the system.

Chouppie considers that chloral hydrate is of all means we possess the most reliable remedy for puerperal eclampsia. In 12 cases in which it alone was employed it was successful although some cases were desperate when the treatment commenced: 60 grains, he says, at least should be rapidly taken & the use of chloral prolonged after the convulsions have ceased.

Milne records a successful case of eclampsia treated with large doses of chloral hydrate. Sir James Simpson said that he believed this was the first case of puerperal eclampsia in which this drug had been employed.

Thomas, in reporting 2 cases of eclampsia treated by chloral hydrate says that recovery was due to this drug and states that Liebreich claims that chloral is a quick anaesthetic, non-excitant, leaves no bad after effects and interferes but slightly with the cardiac nervous power.

Purefoy, treated two cases successfully by hypodermic injection of this drug. Bower has used it in a similar way and found that it stopped violent convulsions in a woman after labour.

Fitzmaurice says that chloral hydrate acts powerfully in diminishing the albumen in the urine and it has frequently had a good effect in cases of Bright's disease. I may here mention that I have used this drug at the Birmingham Fever Hospital in a case of post-scarlatinal nephritis in which the urine had been albuminous for months, and after a few doses the albumen disappeared for several days and the general condition of the child was much better.

Charpentier recommends the use of chloral in eclampsia either alone or if there is marked oliguria after bleeding from 400 to 500 grammes.

Arnau'd of Marseilles uses chloral as a prophylactic and as a remedial agent in eclampsia.

Blane recommends chloral in doses from 1/40 to 2/80 grains in the 24 hours when eclampsia has developed.

F. W. Robbins advises the administration of one drachm of chloral per rectum in eclampsia to be repeated in an hour or two if necessary.

Hirst and Goodell advocate chloral hydrate as being useful in the majority of cases of eclampsia.

Lanceau approves of the administration of chloral per rectum, the drug to be given in doses from 60 to 75 grains and carried far up the bowel: a purgative enema to be given the next day. In my experience chloral hydrate has had a very beneficial effect in many cases of eclampsia by moderating nervous tension and excitement. It may be given alone but I think the combination with bromide of potassium is a distinct advantage: 20 grains of chloral with 30 grains of bromide may be given & repeated at intervals of from 4 to 6 hours.

5. Annales des maladies des organes genito-urinaires June 1893.
If the patient cannot swallow, these drugs can be administered with an equally good effect by the bowel. Playfair approves of the hypodermic method, six grains dissolved in a dram of water and injected under the skin. Fordyce Barker is opposed to the use of chloral which he thinks, excites instead of lessening reflex irritability.

W. Berry, Alford and Reynolds Wilson speak of the good results in cases of eclampsia treated by Morphine.

Hastings Tweedy says that of all drugs morphine in doses up to 1/2 grain by hypodermic injection in 24 hours is the best.

Maberley Smith says that he has had great success with morphine in eclampsia: he uses from 1/4 to 1/3 grain by hypodermic injection and he prefers morphine alone to giving it with atropia: one large dose he thinks better than several smaller.

He has collected 26 cases of puerperal convulsions treated by morphine injections and in all cases the fits were arrested and recovery occurred.

Fraser, records a case similar to the above. Bowstead has recorded cases of eclampsia successfully treated by injections of \( \frac{1}{2} \) grain of morphia, 2 minims of Fleming's tincture of aconite + 2 minims of liquor atropiae.

Seavonson records a case of eclampsia in which the labour was forced + venasection practised + 3 morphia injections given with a satisfactory result.

Alfred Grace also records 2 cases in which he used an injection of 1 grain of morphia for puerperal convulsions with excellent results.

Burton also speaks in favour of this drug in the treatment of eclampsia. R. C. Johnson records a case in which large doses of tincture of opium were used to stop puerperal convulsions in a girl of 16. Barnes says that although this drug is said to cause cerebral congestion it often acts well by allaying nervous irritability.

Denman, on the other hand, states that he has known cases of puerperal convulsions brought on by an opiate given during labour: in the second

\[ \text{Brit. Med. Journ. II. 1885 p. 396.} \quad \text{2} \quad \text{Lancet I. 1889 p. 547.} \quad \text{4} \quad \text{Brit. Med. Journ. II. 1882 p. 90.} \quad \text{3} \quad \text{Med. Times & Gaz. I. 1860 p. 376.}
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\[ \text{4} \quad \text{Brit. Med. Journ. I. 1889 p. 588.} \quad \text{5} \quad \text{Medical Press & Circular I. 1883 p. 463.} \quad \text{6} \quad \text{Lancet II. 1889 p. 14.} \]
case I have reported in this thesis about 45 minims of tincture of opium were given on account of a somewhat rigid os but I should very much doubt whether this had any connection with the convulsion which occurred shortly afterwards. Witten & Duncan have also used opium with success, the latter says it acts as a cardiac sedative & lowers the pulse. Morphia has also been spoken very favourably of by Barker, Hecker, & Clarke of Oswego: Clarke gives 1½ grains hypodermically at once: this dose he repeats in two hours if another paroxysm occurs & if the patient be in labour another dose at the end of 8 hours.

Herman recently reported 5 cases at the Obstetrical Society, London, 4 of which recovered & all were treated with morphia.

Of course morphia is open to theoretical objections in this as in other renal affections but as Stephen Mackenzie & others are now treating uraemia by morphia it seems that practice in these cases does not support theory.

1; Med. Times & Gaz. I. 1861 p. 684. 2; Med. Times & Gaz. II. 1861 p. 147. 3; Lancet I. 1889 p. 799.
Pilocarpine.

Blanquinque reports two grave cases of eclampsia successfully treated by injections of pilocarpine when venesection, chloral, & chloroform inhalations had been tried without success.

Taylor prefers pilocarpine to morphine to provoke diaphoresis & declares the latter drug to be dangerous.

Stricker has treated 10 cases of eclampsia by injections of hydrochlorate of pilocarpine saving all the patients. He considers it a reliable remedy in the disease & that it is not contra-indicated by cardiac affections.

Fourrier records a case in which bleeding & chloroform failed to control the convulsions but 1/3 grain nitrate of pilocarpine administered hypodermically had the desired effect, 5 injections were given within about 36 hours.

Hamilton of Chester records a case in which the hypodermic injection of pilocarpine caused the cessation of puerperal convulsions & seemed to bring on uterine contractions in 2 hours time as the convulsions had returned another injection was given which made the pains come on with greater strength & frequency.

Churchouse mentions a case of uraemic convulsions in which three injections of 1/4 of a grain of pilocarpine caused great improvement.

Pilocarpine has been used with success by Braun in puerperal convulsions, he records a series of cases in which the most striking results were obtained. This mode of treatment has been ably advocated by Fancourt Barnes and others, although Fordyce Barker considers its propriety is open to question.

Murphy relates three cases in which he used pilocarpine in eclampsia with success: he says that he has now used it in six cases during the last three years and it has acted so satisfactorily as to leave little to be desired.

Kirk does not speak so favourably of the drug, he records two cases in which he used it, the first one was fatal whilst in the second case he does not think the good result was due to pilocarpine.

Horrocks publishes two cases treated by the subcutaneous injection of 1/3 gr. of pilocarpine with satisfactory results; he says that this drug acts as an ecchymotic.

Horrocks says that if no sweating occurs within five minutes after the injection the drug may be looked upon as useless & a fresh solution of it must be obtained. He says that the eebolic action of pilocarpine was first pointed out by Massman of St. Petersburg in the Lancet Feb. 8, 1849; cases also of Clay & Kleinwachter in the London Medical Record of that year show that pilocarpine has an eebolic action. Horrocks says that in one case it produced tetanic uterine contraction fatal to the child.

Cullingworth of Manchester has not found pilocarpine to have the effect of a diaphoretic or an eebolic in a case of albuminuria in pregnancy although solutions of proved efficacy were used. J. Campbell has found subcutaneous injections of pilocarpine (¼ grain) prove very useful in one case when the patient was almost moribund, but as it is a dangerous drug to use on account of the salivation it produces he thinks it should be kept as a dernier ressort.

 galabin states that pilocarpine is thought by some to act favourably by diminishing arterial tension; but as it is not nearly so efficient in this way as nitrite of amyl or nitroglycerine, drugs, which in his opinion have no permanent good effect in eclampsia, he comes to the conclusion that "it must be accepted as an empirical result that pilocarpine appears to tend to prevent the recurrence of the convulsions".

I think that if we accept the uraemic theory of eclampsia the good effect of pilocarpine can be explained differently viz. by producing a copious diaphoresis & so relieving the kidneys to say nothing of removing some of the retained secretions. Donati speaks of the dangerous effects of pilocarpin on account of its depressant action on the heart & the copious secretion excited in the respiratory tract. Sänger & Spiegelberg & others mention cases in which the reflux of abundantly secreted saliva & the copious secretions of the mucous membranes lining the air passages have caused dangerous symptoms of suffocation. I have myself injected 1; Manual of Midwifery p.327. 2; London Medical Record Apr. 15. 1883.
pilocarpine (1/8 grain) in a case of eclampsia.
the result was so alarming on
account of the frothy mucus at the
entrance to the larynx that I should
hesitate before using this drug again
in eclampsia.

Oxygen
Inhalations of oxygen have been satisfactory
in the treatment of eclampsia in some
cases recorded by Favre of Kharkov whilst
Soff of Karsan, Schunkchin of Kharkov and
Satinin of St Petersburg record cases in
which this treatment did not prove
very satisfactory.
Robin & Jaccoud recommend inhalations
of oxygen: Bompiani records 2 cases
 treated in this way, in one there was
marked benefit but in the other the
success was only partial.

Nitrite of Amyl
Barnes says that nitrite of amyl is even
more valuable than chloroform in counter-
acting puerperal convulsions: by the
use of it he thinks he has saved several
lives: its power of subduing muscular
spasm is remarkable.

Archibald Macdonald used nitrite of amyl in a case of eclampsia and found excess of uric acid in the urine afterwards: he also suggests the use of colchicum in puerperal eclampsia.

**Nitro-glycerine**

Green records a case in which nitro-glycerine was of great service in eclampsia, he regards its action in reducing arterial tension as much more lasting than that of amyl nitrite. Murrell and Riniger have suggested the use of nitrate of sodium which has properties similar to nitro-glycerine.

**Ether**

Storer has found ether of service in several cases of puerperal convulsions in which he has used it. Perron recommends the hypodermic injection of ether in eclampsia.

**Cathartics**

H. Harris' records a case in which he gave 14 or 16 drops of eroton oil: he frequently gives ten drops in eclampsia & has never known a large dose fail. Overton records 2 cases treated by suppositories of eroton oil with a favourable result. Pulv. Jalapae Co. is in drachm doses an exceedingly efficient purgative in this disorder though its action is not so rapid or so effectual as that of eroton oil. Lever used to give his cases tartar emetic in large doses & he speaks of it as a very important adjunct to bleeding.

Boyd refers to a case of his own & ten cases of Forn of Brooklyn in which Veratrum Viride was used with advantage.

American observers, B. Hart, Kirch-Kelly, Alford & Chase all speak in favour of this drug.

Jewett recommends it by hypodermic injection in doses averaging from 10 to 20 minims, the smaller dose repeated in half an hour. He says "I prefer to place the patient at once under the influence of the drug fully. The guide to the dosage is the frequency of the pulse."

experience seems to justify the statement that no convolution will occur while the patient is under veratrum sufficiently to hold the pulse below 60 to the minute. The average time required to develop the full effect of a single subcutaneous injection is 30 minutes." If necessary smaller doses may be repeated but I seldom find that the total dosage exceeds 20 to 30 minims of the fluid extract.

Personally I have had no experience of this drug nor can I find any cases recorded in this country which prove its value but from the experience of American observers it seems worth trying if other remedies are not at hand or fail.

Benzoic acid

When the wine is scantly Bahnson recommends benzoic acid, this was first suggested by Frenichs who said that it would neutralize the carbonate of ammonia in the blood.

Antipyrin

Palmer treated a case in which convulsions occurred twice during the same pregnancy successfully by antipyrin: the child

was born at full term & there were then
no fits but as the child was dead
some time before birth Barnes thinks
the reduction of blood-pressure & subsidence
of the fits was perhaps due to this
cause & not to the antipyrin.

Citrates of caffeine.

Bolton Corner¹ especially advises citrates
of caffeine where there is nervous
irritability, neuralgia, vertigo, headache
or other signs of neurosis: he has
used it with success in eclampsia not
dependent upon renal disease.

Cocaine

Abuloff of Vilna ³ has used cocaine in
eclampsia and saw disappearance both of
general convulsions & of spasmodic
closure of the os, after intra-uterine
injection of one-third of a grain of
coke in solution.

Amongst other drugs suggested for the
treatment of eclampsia are: – lactate of
strontium recommended by Minazer
& chlorate of potassium by Brown.

Diaphoresis

Hot baths to produce diaphoresis and to
relieve the kidney congestion are certainly
of use in some cases.

³. London Medical Record 1885 p. 257.
Ransom relates a case of post-partum eclampsia in a primipara successfully treated by a warm pack for six hours.

Bruns of Vienna treated 6 cases by the method of Liebermeister, only one case proving fatal. After half an hour in a hot Bath patients were packed for 2 or 3 hours & copious sweats produced; narcotics & other remedies were also combined with this treatment.

Veit approves of hot baths & blanket-packs as used by Liebermeister and Bruns: he thinks the simultaneous employment of diaphoresis & narcosis should bring the patient through.

Veit uses both morphine & chloroform in eclampsia.

Postural treatment

Graffy Hewitt & Routh believing that disturbances of renal & abdominal circulation from pressure of the gravid uterus, excercised a powerful influence in producing eclampsia, have in several cases placed the patient in the knee-elbow position, maintaining this as long & as frequently as possible.

and they say that the patients seem to
derive marvellous benefit from this position.
Löfflein has also spoken favourably of this treatment.
Bahnson lays stress on the benefit
derived from change of posture, the
convulsions being arrested by turning
the patient on to her side: he says that
Brown called attention to this in the
Practitioner of April 1846.

Compression of carotids.

Playfair has in one case tried compression
of both carotids during a paroxysm
which by producing anaemia of the
brain seemed to stop the convulsion
he suggests that this is a simple method
worthy of trial in all cases.
Anaemia of the brain stopping convulsions
is rather opposed to some authorities
who regard cerebral anaemia as their cause.

Peripheral stimulation.

Some authorities recommend peripheral
stimulation. Hall Davis speaks favourably
of cold applied to the head or dashing
cold water on the face: sinapisms
to the back of the neck or the shaved
head or to the calves or feet are also
praised: he mentions a case in which
Harvey arrested convulsions by stimulating
the sensory nerves within the nostrils.
1. Practitioner 1846 p.137. 2. Science & Practice of
Midwifery II p.316. 3. Obstetrical Transactions Vol. X.
Rivet has recently spoken in favour of friction with mustard followed by sinapisms over the entire body.

I can only say that personally I should never think of resorting to this mode of treatment as I am of the same opinion as Barnes who says that on account of the increased irritability of the skin in eclampsia this treatment is pretty sure to provoke a fit.

Salt-water injections

Porak & Bernheim report several cases showing the good effects of subcutaneous injections of salt-water.

Artificial Respiration

Millican records a case in which he used artificial respiration with success in asphyxia due to eclampsia. Murphy speaks of a similar case.

Obstetrical Treatment.

With regard to the obstetrical treatment in cases of eclampsia occurring before delivery there is great difference of opinion but now most obstetricians seem to be in favour of emptying the uterus as rapidly as possible as the fits are caused by the pregnancy in some way or other and as delivery can be accelerated under

4. Lancet II. 1882 pp. 121 & 188.
an anaesthetic this seems to me to be the best treatment; as if the patient should not go into labour at once there is a great risk of the recurrence of fits when labour does come on.

Desbonnets 1 believes that labour should be terminated as soon as possible in cases of eclampsia by the foreps anaesthesia being used.

Charpentier 2 says “Labour should be waited for & terminated naturally whenever possible”. Induced labour should be reserved for exceptional cases in which medical treatment has entirely failed. Caesarean section & forced labour especially by cervical incisions should be entirely discarded in eclampsia.

In a discussion on the above paper Tarnier, Guinot & Robin joined & according to them forced labour was not advisable in any case.

Again Swayne 3 quotes Charpentier as follows: — Premature labour is never to be induced & still less abortion. It is a deplorable procedure no matter how gently it may be performed it always results in contusions or tears of the cervix, sometimes with resulting gangrene. It is only

2. Annual of the Universal Medical Sciences II.1895. p. 66.
possible to practise it after the cervix has been softened in consequence of the preceding haemorrhage. Richardson says the only adequate measure is a thorough evacuation of the uterus by artificial premature delivery. Haultain argues that as uterine contractions appear to be a potent factor in the production of the convulsions, after the patient is put in good condition by the use of the usual remedies artificial abortion or premature labour should be induced. Simpson and Milne Murray agreed to this line of treatment. Lauder approves of inducing labour when other means fail. Fehling recommends this treatment in desperate cases.

Kaltenbach and Gussew favour Caesarean section, Chrobak, Muller and Söcklein are not strongly in favour of it while Swieciecki reports an unfavourable case treated by section as advocated by Halbertsma.

Fayette Dunlap favours the rapid emptying of the uterus in cases of eclampsia. He quotes as bearing him out in this:

advice, statistics which show that 50 per cent of all women who have eclampsia before labour perish and that about 90 per cent of the children are lost. Charpentier, Rajot & Fannier are opposed to the induction of labour while Lusk favours it. Auffrecht of Berlin says if eclampsia occur at the beginning of labour or during the pains, the labour should be hurried on as much as possible because in his experience renal disease with albuminuria is without exception the cause of eclampsia.

Fordyce Barker arguing on the premises that the chances of convulsions in labour in albuminuric patients are 1 in 5 says that they will be very much increased by inducing labour. Gooch dismisses the subject of the local treatment by saying "Attend to the convulsions & leave the labour to take care of itself." Schroeder says "No kind of obstetric manipulation is required for the safety of the mother.

delivery for the sake of the child. R. Barnes also spoke in favour of this treatment.

Merkel records a case of eclampsia in which he saved the lives of twins by incising the cervix by Dührssen's method, the mother making a good recovery.

Zweifel² whilst advocating a speedy termination of the labour in eclampsia shows from his experience that Dührssen's method does not give such good results as the method of dilatation of the os without loss of blood.

Robert 4 Fancourt Barnes say "The disease depends upon gestation: can the system bear the double strain of gestation and the disease, working as it must do with organs which have proved unequal to the task? The question is frequently answered by nature: labour comes on spontaneously."

These authors proceed to show: —

1. "That in every case in which convulsions have set in premature labour should be induced."

2. "That when there is marked albuminuria, with oedema, difficulty of breathing,

a quickened pulse with a rising temperature,
or amaurosis or any form of paralysis
and relief does not follow bleeding,
purging & sedatives, the operation
of induction of labour should not be
delayed."
3. "When the patient has had albuminuria
in previous gestations or is known to
have Bright's disease & hypertrophied
heart, again no time should be
lost."
"Conclusive arguments are based upon
the following considerations which
apply to all the foregoing three
conditions."
1. "If the case be allowed to go on, even
if the woman recover, every day
adds to the strain on the kidneys
and eyes and may lay the foundation
of permanent disease."
2. "It is not justifiable to let the
woman run the risk of losing her
life or of drifting into grave
disease under the expectation of
saving the child."
3. "Granted that the object of getting
a live child should rule our judgement
the prospect is better of getting a
live child in a future pregnancy
than by trusting to the actual pregnancy already gravely threatened.
4. The fate of the child is linked with that of the mother as too commonly if the mother perishes the child dies also.
5. "We are doubly bound to seek our motive of action in the interest of the mother."
"Finally, admitting that some women have, under our own observation and sanction, gone the full period of gestation and have been delivered of living children apparently without damage; frankly speaking, we would not, with enlarged experience, again encounter a responsibility so great."

I think the above statements coming from such authorities deserve the fullest consideration, at the same time other observers say that if there are only a few fits and these are unattended by some of any length, it may be sufficient to give purgatives, chloral and bromide, to put the patient on a milk diet and watch carefully the proportion of albumen in the urine.
Personally I think this treatment may often suffice in cases of albuminuria in pregnancy; but if there are any fits or the albumen increases whilst the quantity of urine decreases I should recommend the induction of labour. Cullingworth of Manchester gives an account of an interesting case of albuminuria on account of which he induced labour with satisfactory result: the child was born alive but not having reached the 6th month it did not live two days. The cases I have reported in the first part of this thesis are instances of the satisfactory results obtained in eclampsia from the accouchement forcée; in no case did the disease prove fatal to the mother: in the first case the child probably died before treatment commenced, in the second the child was born alive but was scarcely viable and in the third the toxic effect of the maternal blood very probably caused the death of the child before delivery: my own experience therefore leads me to advocate the speedy termination of the labour in all cases.

Lancet II. 1886 p. 534.