

Thesis
ON
PUERPERAL ECLAMPSIA
WITH
Notes of five cases;
and one case of
Non-puerperal convulsions.
by
J. L. Mackenzie.



Of the various manifestations of disease with which the practitioner of Medicine has to deal there are few which make greater demands upon him than the Convulsions associated with pregnancy and the puerperium, and included under the term Puerperal Eclampsia. Whatever theory of its causation may be adopted, the condition is one which forms a clinical group of such definiteness that it may be separated from diseases of the Urinary System on the one hand and of the Nervous System on the other for the study of its symptoms, pathology and treatment.

Amongst its prodromata we find the Dropsy of renal disease and much to call to mind the milder forms of epilepsy; in the fully developed disease a peculiar combination of Uræmic symptoms with violent evidence of involvement of the Nervous System; and of its sequelæ effects upon the nervous system, even in its higher centres, which disease of the kidneys alone would not produce and urinary changes with which no other form of convulsions is associated. As illustrating these and other characters of Puerperal Eclampsia six cases which have occurred in my practice within the past three years are described, five of true eclampsia and one, which, though not related to pregnancy, possesses some points of interest bearing upon the subject of this thesis.

Much has been written about Puerperal Eclampsia

and much more remains to be done and we agree with the remark of Dr. Collins¹, that, "there are few cases requiring" "more prompt and decided practice than puerperal convulsions" "and the extent of the experience of most individuals is not" "sufficient to enable them to draw satisfactory conclusions" "from what they have themselves seen, therefore every" "contribution is valuable" which seems to justify the discussion of such a debatable condition, even by those who have to deal with the disease under circumstances which do not always conduce to exact observation.

The disease is defined by various authors in different terms but perhaps that given by Spiegelberg represents the modern opinions as to its limitations as well as any; "the name is applied to the epileptiform (i.e. tonic and especially" "clonic) general convulsions, associated with loss of consciousness" "during the attacks and with coma after them, which appear" "during, and are due to, the puerperal processes".

Spiegelberg's definition is at least convenient when it is taken to exclude, as he does in the text, hysteria, apoplexy, poisoning and true epilepsy and when we further extend the idea of "puerperal" to the whole period during which convulsions arising in connection with pregnancy and confinement, may exist.

But even the term "eclampsia" does not pass unchallenged for Nothnagel² would reserve the term for cases "which exist

1. Collins - Practical Treatise on Midwifery 1836 p. 236

2. Art: Eclampsia - Ziemssen's Cyclopaedia Vol. VII p. 302.

"independently of positive organic disease", a rare form of convulsion in connection with child-bearing.

The classification of the various forms of the disease by the older authors implied an error in pathology, in so far at least as they included cases of convulsions not necessarily associated with pregnancy. Before discussing the question of the causation and pathology of the disease it may be well to refer to certain points in its clinical history.

The frequency of the disease is variously estimated, thus -

Charpentier estimates it at 1 in 354 confinements

Cazeaux " " " 1 " 485 "

Spiegelberg " " " 1 " 500 "

It occurs much more frequently in primiparae than in multiparae. Löhlein says 85 per cent of cases occur in the former, and Winckel 77 per cent. Lantos gives 78 per cent as occurring in the primiparae. The convulsions may occur as early as the sixth week of pregnancy (Cazeaux²) or as late as eight weeks after delivery (Simpson) but the disease usually develops during the last months of pregnancy "from the seventh to the ninth month, particularly a few days before labour." (Charpentier⁵).

In its relation to labour Wiegert's statistics are given by Charpentier⁶. Of 455 cases the onset of the convulsions was

Before 109 . During 236 . After 110.

1 Quoted by Parvin. Amer. System of Gynec. and Obstet. Vol. II p. 72

2 Bil. Med Jour. 1889 (Synopsis) Jan. 12th.

3 Cazeaux and Tarnier's Obstetrics (7th ed) p. 791 . 4 Select Obstet. works. p. 300.

5 Cyclopaedia of Obstet. Vol II p. 94

6 " " " " " p. 93

The premonitory symptoms are by no means constant either in character or in intensity, but "it rarely comes unheralded" and of the symptoms the most frequent perhaps are headache, backache, temporary amblyopia, even amaurosis, oedema especially of the face and hands particularly in the morning. Barnes² also mentions "difficulty in articulation, difficulty in finding words to express the thoughts". He also mentions "aphasia as a not uncommon forerunner of convulsion". Winckel³ amongst the foregoing and other symptoms mentions pain in the epigastrium, and Lusk⁴ and other authors refer to loss of memory, a symptom which in three of my cases has been very striking. A condition to which no reference has been made so far as my knowledge goes, and which is described in Case I is the group of symptoms closely resembling in most particulars an abortive epileptic seizure. In this case the patient had such an attack four weeks before her confinement and although she had no puerperal convulsions, it can scarcely be doubted that this and the other symptoms present pointed to coming eclampsia which was cut short. Any of the foregoing symptoms should lead to an examination of the urine when the most important evidence of all may be detected, albuminuria. But none of the foregoing prodromata may be present. Barker⁵ mentions a case

which was under the most favourable conditions for

1 Parvin.	Amer. System of Obstet. Vol II. p. 723	5 Fordyce Barker.
2 Barnes.	Obstet. Med. and Surg. (1884) p. 388	The puerperal diseases (1876) p. 88.
3 Winckel.	Text book of Obstet. (trans) p. 593	
4 Lusk	Science & Art of Midwifery p. 526	

observation, and in which the patient had no warning beforehand either subjective or objective.

The phenomena of the convulsion itself resemble in almost all respects those of an attack of epilepsy in its most violent form. The paroxysm is fully described in all text books, but, on several points my experience has not confirmed the usual statements.

1. Pulse during the attack -

"At first ¹ hard and strong, now becomes rapid and feeble."

"The ² pulse is small, soft generally, frequent often intermitting."

"If ³ strong and full at commencement, rapidly accelerated,"

"and becomes extremely feeble towards the end of the paroxysm."

My experience has been that during even an attack not of the most violent character it is quite impossible to feel the pulse, and that this is true for several seconds after the cessation of the convulsion. This has been confirmed to me, on more than one occasion, by another medical man holding the opposite wrist.

2. Temperature. In only one case in my practice has the temperature been taken with the object of testing the views of Winckel⁴ on temperature as affected by the paroxysms. Winckel claims priority to Bourneville⁵ in his observations on this matter, that the temperature rises decidedly with every new attack.

1 Barnes - Obstet. Med. and Surg. p. 385

2 Spiegelberg. l.c. p. 206

3 Leishman - System of Midwifery. p. 148

4 Winckel. l.c. page. 593

5 archives de Toxicologie
tome II. (quoted by Barrin
p. 75).

⁴ferman analyses twelve cases on this and other disputed points and concludes that the fits have no direct effect upon the temperature. In the case observed by me (case V) the temperature seemed to be distinctly affected by the convulsions or by some other condition present only at the time of the paroxysms. The patient had evidently had a fit about twenty-five minutes before my arrival, and then the sequence of convulsions and elevations was - 4 - 30 a.m. Temp. 99.0

4 - 50 " Convulsion. " 100.4 after convulsion.

5 - 20 " " " 101.2 after convulsion.

There were no more convulsions and the temperature never reached a higher point than immediately after the last convulsion.

3. Duration. The greatest possible diversity of opinion seems to exist on this apparently simple point.

Barnes says "from two or three to twenty minutes" for the clonic stage alone. Lust² "rarely exceeds a" "minute and in the majority of cases lasts from ten to" "thirty seconds". Winckel³ quotes an observation by Bar on the cutus of the right biceps during an eclamptic attack. This observation was taken during a convulsion which lasted 77 seconds. Three stages are recognised in

this observation - 1st a series of oscillations
2nd the tonic stage,
3rd the clonic stage.

1. l.c. p385.

2. l.c. p52^m.

3. l.c. p592.

4 Brit. Med. Jour. July 11th 1891. p. 72

Of the convulsions observed by me the longest has been 73 seconds and included in this period was the stage of "oscillation" which is quite recognisable to the touch before the tonic spasm develops.

Spiegelberg asserts that statements regarding the prolongation of convulsions much beyond a minute must be based on error because of the incompatibility with life of prolonged involvement of the respiratory muscles.

H ² Coma. Barker says the coma varies "in character, profoundness and duration in a ratio" "proportionate to the intensity and severity of the" "convulsive attack". Darwin says "the coma deepens" "with each convulsion", and this is in accordance with one's experience. It is evidently, however, by Spiegelberg ⁴ considered exceptional for this deepening of coma to occur because he remarks he has seen it "more than once". No case has come under my observation which did not fully confirm Barker's observation, although in cases of slight severity the tendency to coma may be indistinct and transitory.

Of the sequelae of eclampsia mentioned by various authors few have come under my notice. Lusk ⁵ refers to the risk of post-partum hemorrhage, and of inflammation, and in various text books these conditions are referred to.

1 l.c. p. 207

2 l.c. p. 86

4 l.c. p. 207

5 l.c. p. 527

Denman states of inflammation that in the early days of his practice it was almost always present, but later it seldom occurred, and this change he attributes to "the present practice of liberal bleeding". Spigelberg¹ attributes the proclivity to hæmorrhage to the "albuminuria and non-aerated state of the blood". In general terms he considers the eclamptic patient more susceptible to the ordinary diseases of the lying-in woman, and in this he is confirmed by Barnes. Leve² refers to the Morbus Brightii as a predisposing cause to post-partum hæmorrhage. The other conditions to which he refers are chiefly connected with the nervous system. Paraplegia (case V) occurs in many cases associated with albuminuria (Barnes) and Fordyce Barker⁴ gives a good prognosis in such paresis or paralysis arising from a reflex cause. Amanosis and amblyopia, unless when persistent albuminuria is present, usually disappear, and of this Barker relates some striking instances.

Insanity was considered by Simpson⁵ to be rarely associated with puerperal eclampsia but frequently with albuminuria of a transitory nature.

Manton⁶ says of America "insanity as a sequel to puerperal convulsions is of such exceedingly rare occurrence" "in this country as hardly to deserve consideration in " "this connection."

1 l.c. p. 209

2 l.c. Vol. I. p. 389

3 Quins Hosp: Reports. 1842. Vol. VII, pp. 330-335

4 l.c. p. 73. 5 Med Times & Gazette. 1860. Vol. II

6 Amer: Journal of Obstet: 1845. p. 904.

Spiegelberg quotes Seydel and Fitz¹ as considering it of great importance. Fordyce Barker², after making special observations on the point, says puerperal mania is not more frequent in women with albuminuria than in those without. Of the purely psychical sequelae of the disease perhaps the most constant is loss of memory. Spiegelberg³ says "sometimes the whole period of gestation" "although most frequently only the act of parturition" "seems to have faded entirely from memory, and is" "only gradually recollected." In case III observed by me this is very distinct, and in this, as in other cases, the patient has always commenced to recall the more remote events first. The time at which the memory is best in such cases is after sleep. Probably the most serious sequel of eclampsia is chronic kidney disease. Of this Barnes⁴ says although in perhaps the majority of cases the kidneys are left intact, they may be permanently affected, and no sooner does a new pregnancy occur than the disease is exacerbated and the condition becomes more and more confirmed.

Spiegelberg⁵ says it is the exception for kidney mischief to remain. Dr. Galabin has known a case of eclampsia in which albuminuria remained after the first pregnancy for two years, but did not reappear in connection with subsequent gestations.

1 l.c. p. 209.

5 l.c. p. 209.

2 l.c. p. 179.

6. Obstet. Trans. Vol XXIX. p. 549

3 l.c. p. 209.

4 l.c. p. 394.

Pathology. The modern pathology of eclampsia received its foundation in the observations of Leve¹ and Sir James Simps²on who probably about the same time, although priority in publication belongs to the former, discovered the presence of albuminuria in the case of a great majority of women who suffered from Puerperal convulsions. In ten cases, the urine of which Leve¹ examined, the presence of albumen was demonstrated in nine, and the tenth was discovered to have been a case of acute meningitis. Previous to these observations a pathology of the disease seems scarcely to have been adopted, for Collins³ says in 1836, "I conceive we are quite ignorant of what the cause may be", and Ramsbottom (1841) gives it as his opinion "that it" "most frequently originates from some deranged" "state of the uterus itself, probably in its nervous system" "and consists in some irritation propagated from" "that organ to the brain".

The question then arose, and is still in the region of theory, what causes the albuminuria? Various answers are given to this. The opinion has been expressed⁴ by writers, of whom Lyalab⁵ quotes Blot, Petit and Hippolite, that albumen is present in about 20 per cent of pregnant women, at one time or another. Lant⁶os

estimates it at 18 per cent in pregnancy, and 60 per

1 Quin's Hosp. Reports. 1842.

2 Lond. & Edin. Journal of Med. Science. Nov. 1843. p. 1015

3 Pract. Treatise on Midwifery - 1836. p. 201

4 Princ. & Pract. of Obstet. Med. & Surg. 1841 p. 558.

S. B.M.J. Oct. 30. 1880
page. 697

6 B.M.J. Synopsis
1889. Jan. 12th.

cent in newly delivered women. These statements are not confirmed by English observers.

Hicks observed fifty cases of pregnancy. In only one was there albumen and it was traced to old kidney disease.

Lever (Hicks states) observed a similar number without finding it in any.

Galabin observed forty-three with a trace only in one.

Galabin thinks the discrepancy may arise on account of the very small quantities found by the French observers being due to cystitis, as no casts are mentioned by them.

Lever maintained that the albuminuria was due to direct pressure upon the renal vessels by the pregnant uterus, and, in this view he is supported by Caseau and many others. Leishman³ considers it certainly accounts for some cases.

Habberton⁴'s view is that the kidneys become affected by pressure on the ureters by the gravid uterus, that they (the ureters) are stretched by the upward growth of the uterus, and variously flexed and compressed until their permeability is more or less lost. In this view he is supported by Löhlein⁵, and, in this country, by Dr. Nash. Dr. King⁷ attributes the pressure to the descent, in primiparæ, of the fetal head into the pelvic cavity

before its proper time, so causing pressure on the blood

1. Obstet. Soc. Trans: Vol. VIII, p. 323

2. l. c. p. 697.

3. Sys. of Mid. p. 782

4. Trans. Intern. Med. Cong. 1881 v. 11, p. 347

5. Deutsche Med. Zeitung 1883. vide Barnes

6. Lancet 1892 vol. 11, p. 477

7. Trans. Intern. Med. Cong. 1887 p. 475.

vessels of the pelvis. He holds that the normal position for the head until the approach of labour is "poised on" "an iliac fossa", and that irrational dress has much to do with the displacement.

Certain arguments in common may be used in support of the theory of local pressure. It is shown that the great majority of women who suffer from eclampsia are primiparae, and that owing to the greater rigidity of the abdominal parietes the pressure backwards of the uterus is greater; the greater relative frequency of eclampsia in twin pregnancies in which the uterus is enlarged; the great rarity of cases of eclampsia in other than vertex presentations, and the evidence of morbid anatomy. On the latter Dr. Dickinson is quoted as saying the morbid changes in the kidney are "such as obstructive or venous" "congestion would be apt to produce". For the further support of Halbersta^m's theory are the observations of Löhlein² who found in thirty-two necropsies of eclamptic women that twenty-five per cent had one or both ureters dilated, whereas in deaths from other causes only three per cent had this condition.

Winckel has found dilated ureters in many post-mortem examinations of pregnant women without eclampsia.

1 Pathology and Treatment of Albuminuria. 2nd ed. p. 117

2 l.c. (Barnes) Vol 1. p. 397

Various arguments may be advanced against the pressure theories and it is certain they do not account for the albuminuria in all cases. Barnes points out (1) the pressure is not adequate, the uterus rising out of the pelvis and diverging from the spinal column, (2) albumen may make its appearance in the third or fourth month before the uterus can press upon the kidneys or their vessels, (3) the albumen may disappear under non-mechanical treatment, (4) the evidence from the greater frequency arising from the greater distension by twins may be due to the greater nervous or vascular tension arising from the double demand or to the larger amount of effete material thrown into the circulation. Such cases as those described by Briston Hicks² in which the albumen did not appear till after the emptying of the uterus would also seem inexplicable on any theory of pressure unless we assume a prealbuminuric stage of considerable duration. Moreover, if the pressure theories were correct albuminuria ought to be much more frequent than it has been shown to be in pregnant women.

But without external pressure on the urinary organs or their vessels some writers attribute the albuminuria to increased intra vascular pressure.

In support of this is the physiological fact accepted

1 l.c. - p 394

2 Obstet. Trans: Vol. VIII p. 323

by all writers, of the higher arterial tension in pregnancy as a predisposing cause. Beyond the purely physiological cause reflex stimuli are considered to play an important part, thus, Hicks^{1d} in four cases found the albumen did not appear until after the convulsions. Tyler Smith propounded the theory that the albuminuria "may depend upon" "sympathetic irritation of the kidney by the gravid uterus". Santos² holds the same views. On this point the evidence of epilepsy is ambiguous. Powers³ reports forty-three attacks in twenty-three patients, and found a trace of albumen only in one. whereas Nothnagel⁴ quotes Hoppert as holding it is invariably present transiently after every fully developed attack, as well as hyaline casts. The nonpuerperal case of convulsions (Case VI) quoted by me resembles more closely eclampsia than the single epileptic seizure, and, in the urine of this patient albumen was temporarily, but very distinctly present but I recognised no casts. It is certain however that albumen in the urine is present in a very large proportion of cases before the incidence of the convulsions. Other observers hold that the albuminuria arises in association with lesions connected with the kidneys themselves produced in various ways. Pre-existing

renal disease here must be taken into account,

1. Barnes. Vol 1. p 403.

2. B. M. J. Jan. 12. 1898

3. Powers Epilepsy. p. 107

1d. Obstet. Trans. Vol VIII. p 223.

4. Niemöller's. Cyclop. Art. Epilep. Vol XIV

p 226

although numerous instances of patients with chronic Bright's disease passing through pregnancy safely are recorded. Sufferer (quoted by Lusk) mentions seventy cases of women, suffering from Bright's disease, who became pregnant and only two of these had convulsions. Lusk also quotes Holmes who reports forty-six such cases of whom one third had eclampsia. Albuminuria may also be the result of excessive work thrown on to the kidney during pregnancy. Whether or not there is this actual increase of normal constituents under normal conditions seems doubtful for Winckel considers there is no great variation in the quantity of urea excreted. Herman^{2(a)} quotes Barthelemon, Chalmet and Quinquand who give the varying estimates of from 158.9 grs to 570 grs per diem. Herman suggests that a diminished excretion of water insufficient to wash away the soluble constituents, would so far as the renal epithelium is concerned be equivalent to an excess of these constituents. But apart from the excess of normal work for the kidneys, the disturbance of their function as expressed by albuminuria may be produced by various other agents. Thus, various other writers attest the presence of acetone in the urine of eclamptic patients. Stumpf (quoted by Winckel³) was uniformly successful in finding acetone

1. Lusk. p. 531

2 Text Book of Midwifery. (Eng. Ed.) p. 77

3 l.c. p. 590

2(a) Obs. Trans. Vol. 29. p. 517. p. 538

in the urine of eclamptic patients, whose breath smells strongly of acetone. He always found sugar when he had sufficient urine to examine, and he therefore, arguing from the part acetone and allied bodies play in diabetic coma, concluded, that under certain abnormal conditions of decomposition a non-nitrogenous substance, toxic in its effects, perhaps acetone, may be formed, which is so irritating to the kidney during excretion as to be able to produce nephritis. He does not consider this theory explains all cases but those in which icterus is present, as the same substance produces destruction of the liver cells, going on to yellow atrophy of the liver. This same substance would, by irritation of the brain produce convulsions. Hergott considers the kidney lesion may be similar to that producing uraemia in Bright's Disease, or that it may result from a microbe acting directly on the kidney, or by means of its products. Doléris and Poney maintain that amongst five eclamptics they found two with nephritis due to micrococci in which the infection of the blood increased and diminished *pari passu* with the eclamptic attacks. Winckel² evidently doubts the accuracy of these observations, and says all other researches along that line have proved negative.

1 Abstract Amer. Journal Obstet. Vol XXVIII p. 1144

2 l.c. p 590.

POST MORTEM. Unfortunately the post-mortem changes observed in fatal cases of puerperal eclampsia are not less numerous than the theories by which the renal incompetence is sought to be explained. Herman says "the" "most varied conditions are found post-mortem in eclampsia." He specifies Acute Nephritis, Large white Kidney, Granular Kidney, Dilated ureters and pelvis, or no perceptible naked eye change whatever. Schroeder² says "the changes" "may be from a congested condition up to the highest" "degree of parenchymatous nephritis".

³Spiegelberg says the necropsy, generally speaking reveals nothing that can throw light on the disease except a (usually) moderate degree of renal mischief, and even the kidneys may appear to be healthy with the exception of trivial insignificant changes.

⁴Carleaux and Tarnier say the kidneys have almost universally presented the anatomical characters of Nephritis. Certain observations have, however, been made in which changes peculiar to the disease have been recognised. St. Angus Macdonald recorded a condition in which disintegration of the renal cells takes place similar to what we find in the liver cells in acute yellow atrophy of the liver, or in phosphorus poisoning. ⁵Cutler records a case presenting the same features, as also does ⁶Herman.

1. Obstet. Trans. Vol. XXIX - p. 553.

2. Man. of Midwif. (Eng. Ed.) p. 318

3. L.C. p. 210.

4. Carleaux & Tarnier Obstet. 7th Ed. p. 809

5. Trans. Obs. Soc. Vol. XXXVI

pp. 176-187

6. " " " Vol. XXXIII

p. 328.

The question arises, how after such characteristic evidence of renal disease (incompetence) can the comparative absence of changes in the kidney be accounted for? Barnes¹ mentions Gubler's theory of super-albuminosis, by which the kidneys are supposed to act as a filter, removing the excess of albumen contained in the blood of pregnant women, and Spigelberg² attributes the renal incompetency to an alteration in the renal circulation - at the terminations of the vessels - either owing to some change in the walls of the vessels or to spasm. Until much more is known about the action of toxins, this question may have to remain unanswered. An examination of the urine has so far led to no definite distinction as between the various theories as to the production of albuminuria. Herman³, in view of Ralfe's⁴ opinion that paraglobulin is indicative of pressure rather than epithelial change, whereas serum-albumen is due to the reverse condition, has conducted some investigations which are not conclusive, for although he found serum-albumen to predominate in Chronic Bright's Disease in pregnancy (according to the theory) the cases of eclampsia gave contradictory results; in four cases very little paraglobulin was present, and in five the precipitate largely consisted of it.

1. l.c. p. 399.

2. l.c. pp. 213-214.

3. Obstet. Trans: Vol. XXI, p. 539

4. Diseases of Kidneys, p. 107.

The nature of the casts is also an unreliable guide for
whereas Herman's cases were always hyaline or granular,
Lyon in recording thirty-six collected by him describes
blood casts in large numbers. Santos found casts only
in four out of twenty-three cases.

The post-mortem examination of other organs from the bodies
of women who have died from eclampsia does not
throw much light on any common cause for the disease.

Brain - The brain is described by Spiegelberg² as
occasionally having apoplectic extravasations or their
remnants, as well as hyperaemia of the cranial cavity
and capillary extravasations, but it is more usual to
meet with cerebral anaemia with serous transudation
into the brain and especially its membranes.

Winkler³ describes the same conditions but mentions
aneurismal dilatations on the small arteries of the
brain. Macdonald found (quoted by Lusk - p. 354)
the meninges congested and the ventricles filled
with serum, with marked anaemia of the deeper
brain-structures.

Anaemia and oedema of the brain with congestion
of the meninges seems to be a condition on which
most observers are agreed. Cazeaux⁴ considers all the
cerebral lesions secondary. In consideration of

recent developments in some views as to the causation

1. Amer. Jour. Obstet. Vol XXVIII. p. 18

2 l.c. p. 210

3 l.c. p. 591

4 l.c. p. 808.

of eclampsia, more general observations in morbid anatomy are required, especially with reference to the liver. Leishman regards some cases as cholamic and in Prof. Simpson's case the liver was diseased.

The observations of Dubroffen are quoted; that he found icterus in sixteen out of thirty-seven cases of fatal eclampsia.

Predisposing Causes. - Of these the chief is albuminuria. Cascaux³ considered this "or rather the disease of which it is a symptom" the "only known predisposing cause", although he admitted the possibility of albuminuria being present without eclampsia. The relations of albuminuria to eclampsia are discussed later on, but that condition (renal incompetence) which it represents, it can hardly be doubted stands in causal relation to the disease in a very large number of cases.

Spiegelberg⁴ while admitting the very rare occurrence of the disease without albuminuria is so strongly impressed with the presence of this condition that he would merely designate cases "eclampticiform" in which it was absent. Herman⁵ would not apply the term eclampsia to non-albuminous cases, he would call them fits or convulsions.

Hicks (Obstet. Trans. Vol. VIII. p. 323) discusses four cases in which no albumen appeared in the urine until after

1 l.c. p. 745.

4 Spiegelberg. p. 216.

2 Edin. Med. Journal. Vol. XXXVIII pp. 889-898.

3 l.c. p. 792.

5. Obstet. Trans. vol. 36 p. 41.

the first convulsion. He considers such cases exceptional but not rare.

Primiparity is also a well marked predisposing cause, as shown by the statistics quoted earlier in this paper.

Winckel¹ says this applies more particularly to old and very young primiparae, with rigid muscles and not too roomy pelvis. Charpenier² doubts the influence of age. Twin pregnancy but apparently not hydramnios.

Winckel³ says the proportion of twin to single pregnancies is as 11 per cent to 1.1 per cent. Spiegelberg⁴ says, and he quotes Löhlein, as to contracted pelvis being the cause.

Winckel⁵ quotes Stände to show that the size of the child's head is an important element, a "narrow pelvis and" "large foetal head in the presence of hydroemia" being a predisposing cause. Vertex presentations are regarded as a predisposing cause.

Collins (Pract. Treatise on Midwifery. 1836 p 199) says amongst the cases of eclampsia occurring in 48,579 confinements only one had an unnatural presentation. Ramsbottom (G.O.) remarks,

"a natural excessive sensitiveness of the nervous" "system may predispose to the disease", but he says "the subject is at best unsatisfactory, and" "but little understood". Some influence, apparently of the weather, has been referred to by many

writers as a predisposing cause and this is based
1. l.c. p. 591 4 l.c. p. 215 b. Prin. & Pract. Obstet. Med. & Surg.
2. l.c. p. 95 5 l.c. p. 591 1841. p. 564 - p. 568
3. l.c. p. 591

on groups of cases occurring about the same time. Barker on each of two days in one week saw three cases. This peculiarity is referred to by Spiegelberg. He finds the possible explanation in the state of the skin. Ramsbottom (F.H) quotes his father, Andral, Denman, and Smellie on this point, "when thunder is in the air," "in Spring, when a few hot days burst upon us" (Ramsbottom) "Electrical state of the air on the approach of a storm" (Andral): "More liable in certain seasons of the year than" "others" (Denman. p. 245): "Seems [in a recent group of" "cases) to have proceeded from the constitution of the" "weather," (Smellie, New Syd. Soc. Ed. Vol. II. p. 229).

Sir James Simpson remarked on the same circumstances. It is possible a bacteriological explanation may yet be found for this grouping of cases.

Previous attacks may predispose, Dewee mentions a case in which it occurred in the 1st, 3rd and 5th pregnancies (System of Midwifery. p. 502). The question of persistent albuminuria was of course not recognised at that time.

Stewart reports three cases, two occurring in sisters each six months pregnant. (Lancet. 1893. Vol. II. p. 86).

Albumen^{-uria} with convulsions and it occurring in any labour later than the first, generally results from fixed granular disease of the kidney and does not disappear after delivery. (Sir Jas Simpson, Sel. Obst. Works, 1871, p. 296)

12.
The various views which have been put forward as explaining the immediate causation of eclampsia are very numerous, and it cannot be said of any one that it is entirely complete and satisfactory.

Few of them ignore the factor of a "toxic state of the blood, whether described by the term "uraemia" or otherwise, but, of these the D'Ambr-Rosenstein theory is the best known. This theory is evidently based upon two observations, (1) the hydraemic condition of the blood in pregnancy, (2) a sudden raising of the arterial pressure. These facts being assumed, a cause for the production of the latter has to be found and Rosenstein finds this in the sudden raising of the blood pressure during labour-pains. Against this theory there is the argument that one's clinical experience goes to show many of the subjects of eclampsia (as indeed other pregnant women) are by no means hydraemic but are young women of a very robust and full-blooded type. Collins observed this and remarked "puerperal convulsions occur almost invariably in "strong, plethoric, young women". The second assumption, when we bear in mind the first, is also contradicted by the authority on whose observations it is usually described as being based, for Kussmaul and Tennek only obtained the result of

1 l.c. p. 199
2. On convulsions (New Syd. Soc.) p. 7.

convulsions in healthy rabbits, and failed in the case of those the health of which was impaired when sudden anaemia of the brain was produced. Hæcker (quoted by Spiegelberg) points out that according to the theory, the pathogenic conditions being usually present, the result ought to follow much more frequently. Moreover the opinion of Kiwisch (quoted by Spiegelberg) notwithstanding the determining cause, labour pains are frequently absent. This theory has few supporters at the present time.

(2). Eclampsia is due to cerebro-spinal congestion.

This view is quoted by Ram¹bottom as having been expressed by Andral that "an attack of convulsions" "is equally affected by an over abundant, or too sparing," "a flow of blood to the head", and Ram²bottom gives pressure on the brain as probably the most usual proximate cause. Khoss³maul says an excessive flow of arterial blood to the brain can never produce convulsions, but paralysis. The observation post-mortem of intra-cranial haemorrhages does not guide us as the question is an open one whether these are the causes or the results of the convulsions.

(3) Eclampsia is a neurosis by a reflex irritation of the nervous system. Gowers⁴ points out the presence of a powerful cause of reflex irritation, and this with

1 l.c. p 562. 2 p. 567.

3 l.c. p 20

4 Dis. Nerv. Syst. Vol. II p 716

an undue central excitability may act without toxæmia. That certain cases are produced in this way and apart from toxæmia seems highly probable.

Tyler Smith while not denying the importance of renal incompetence, indeed he regards it as the most important condition, still believed this to be the cause in some cases. He quotes his clinical experience and that of Denman as to the invasion of convulsions reasonably attributable to the irritation caused in removing the placenta which was adherent. He does not limit his region of propagation of the reflex act in these cases to the uterus, but considers the vagina of equal importance and mentions other points from which the reflex act may be generated - e.g. bladder, rectum. Clinical evidence in favour of this theory may be found in those cases of convulsions which give no evidence of renal incompetence throughout. An interesting group of three such is reported by Colman². In two of these cases, at least, no albumen appeared, and, in all three convulsions occurred; one apparently owing to very rapid descent of the head, and the other two due to manipulation of the parturient passages. The arguments against the theory are stated by Depaul³ but they lose much of their force when it is remembered that the theory is not put forward to account for all cases.

1. Manual of Obstetrics. p. 501
2. "Lancet" 1889. Vol. II p. 640.
3. Cyclopaedia of Obstet. and Gynecol. Vol. II, p. 101 (Chapman)

as a factor moreover in cases in which toxæmia is undoubtedly present it seems to me the reflex act may have a very important place and may in many cases be the immediately determining cause - being too powerful for the inhibiting power of the higher centres which have been enfeebled by the toxic action of the unpurified blood. Depaul's argument that all primiparous women should suffer from convulsions, as there is in all the irritation, does not take into account the presence of toxæmia in some, and not in other pregnant or puerperal women, nor does the absence of albuminuria settle the question because chloæmia is at least a possible condition, rather than uræmia in some such cases. In relation to the reflex theory the question of the uterine pains being the exciting cause has been discussed by various authors. Baston Hicks discusses his observations in two cases, but comes to no definite conclusion as to the pain being the cause of the convulsions. He suggests that the excess of carbonic acid in the blood resulting from the convulsions may stimulate the uterus to further contraction. Leishman (p. 754) thinks the convulsions are more likely to cause pains in this way, than the pains the convulsions, but he concludes there is a very subordinate relation between the pains and

the eclampsia. Spiegelberg (p. 208) remarks that, the toxæmia being present, the uterine contraction may furnish the actual causal stimulus for the convulsion

(4). Eclampsia is caused by general or cerebral anæmia. That by the altered state of the blood there is produced irritation of the vaso-motor nerves of the cerebral arteries, with contraction of the latter causing anæmia of the brain with convulsions. Khossmanul considers this anæmia has its effect by the nutriment being cut off and not by alteration in pressure as Burrows maintained.

(5) Eclampsia depends upon a poisoning of the blood. What is the nature of this poison? This theory is that which commands most support at the present time, but the nature of the poison is by no means beyond dispute.

Uremia. After the discovery by Levee and Sir James Simpson of albuminuria in connection with puerperal eclampsia, it was only one step further by analogy, to attribute the symptoms to a condition which was already known to exist in Bright's Disease - the insufficient excretion of urea by the kidneys.

The idea that urea is the direct toxic agent has been largely abandoned as it has been shown that even a large quantity of urea injected into the blood does not produce convulsions (C. Bernard), and further, that

1 l.c. p. 43.

in Cholera, where an enormous quantity is present in the blood, convulsions are not observed.

D. Heiman has pointed out that in favourable cases the quantity of urea in the urine rapidly increased after the convulsions had ceased, and that where the cases did not progress favourably there was not this rapid increase of urea. Probably this only means that the kidneys did not regain their competence, and his observations would therefore refer to other waste products as well as urea. When urea was shown to be innocuous the theory was put forward by Strichs that the poison was not urea but Carbonate of Ammonia. That by the presence of a ferment in the blood the urea was decomposed and carbonate of ammonia formed and the poisoning was due to the presence of Carbonate of ammonia in the blood.

This theory was combated by Hammond, Richardson and others. The latter showed carbonate of ammonia to be present in the blood and breath of the healthy, and the former made various experiments.

He removed the kidneys and injected urea into the veins and examined the breath and the blood before and after the operation. He came to the conclusion that the Carbonate of ammonia did not become increased, and that consequently there was no formation of Carbonate

1. Brit. Med. Journal. 1890. p. 133.

2. Amer. Journal 1861. p. 55.

of ammonia by the breaking up of urea in the blood.

The form in which the toxicemia explanation of eclampsia is now held by most authorities, is that the poisoning is due to the retention of all the substances which are intended to be excreted by the kidneys. What particular substance, or group of substances, amongst these is responsible for the eclamptic phenomena is still a matter of dispute.

Schöttin¹ said "extractive materials" (Creatin, creatinin, leucine etc) which are retained as well as urea are the toxic agents. Boucharo² has shown that urine from a healthy individual, if injected into the circulation is more toxic than if from one suffering from eclampsia; showing that in the latter case some poisonous element is not eliminated. Doleris found a crystalline substance in the blood of eclamptic patients which caused death when injected into dogs and rats. Hughes and Carter³ believe it to be due to an albuminous substance, easily affected by heat but not dialyzable. Tarnier⁴ says less blood from an eclamptic patient than from a person in health will kill a rabbit, and when the amount required falls as low as $\frac{3}{10}$ the patient from whom the blood is taken will not recover. A further extension of the toxicemic theory is that the renal incompetence (albuminuria) is not a primary element in the disease, but that it is secondary, a poison circulating in the blood being antecedent.

1 Quoted by Chaspenhier. Vol. ii. p. 105 4. Br. Gyn. Jour. Vol. ix. p. 358

2 Abstract Amer. Journ. Obstet. Vol. xxviii. p. 444.

3 Amer. Jour. Med. Science. 1894

Winckel says, in discussing the secondary effects of pressure on the uterus as a cause of eclampsia, "the conclusion is based" "on an assumption which is certainly fallacious in many" "cases, that the change in the kidneys or the diminished" "excretion of urine is the primary factor, it fails" "to recognise the possibility, which is not to be ignored, that" "the kidney disease may likewise be a result of the pre-
"existing poisoning". (p. 589)

Stumpf's acetone theory implies such a condition, and it has been contended for recently by Clark and Skelton. They quote Gyalabin as saying "it is a very reasonable possibility that" "the kidney disease, as well as the changes in the liver, are" "the result of the poison in the blood". They also point to the not infrequent absence of albuminuria and of post-mortem change in the kidneys in eclampsia; to the observations of Tarnier and De Sinety on the changes in the liver, who say the liver is enlarged from a peculiar fatty degeneration of its cells which begins around the intra-tubular veins and extends outwards; to which might be added those of Macdonald which, on the one hand would result from poisoning, and, on the other, would lower the eliminating power of the liver. A further form of toxæmia may be due to some specific pathogenic organism.

Hetzgott gives this as a possibility, the organism acting as in scarlatina or diphtheria.

1 Amer. Journal of Obstet. Vol. XXXV. p. 196

2 Abstract Amer. Jour. Obst. Vol. XXVIII p. 444.

Doleris and Poncy's observations have already been mentioned in dealing with the causes of albuminuria.

As bearing on the question of something beyond uraemia being present in eclampsia, researches have been conducted by Boutville and others as to temperature in the two conditions, with the result that Boutville concludes in eclampsia there is a rise of temperature from the beginning to the end of the attack. There is a slight rise at the time of each convulsion, and that if the case is to be fatal the temperature continues to rise, but, if the progress is favourable, there is a gradual fall to the normal after the cessation of the convulsions. In Uraemia, on the other hand, whether the case be of the convulsive or coma-like type, the temperature gradually falls, and may fall as low as 93° F. This has been observed in thirty-one cases of uraemia occurring in renal disease and obstructive ureters. Dr. Oliver says - "It is more than likely it is a "multiple toxæmia. Many animal alkaloids are known "to be capable of producing convulsions uraemia "must not, therefore, be regarded as a morbid condition "depending invariably upon the operation of a single "cause; it is a toxæmia due to the circulation in the "blood it may be of one or of many poisons, no one of "which has, as yet, been definitely determined and shown "to be constantly present."

² International Clinics VOL IV (fourth series) p. 7.
1 Charpentier Vol. II. p. 107.

The theory of a poison acting before the development of renal incompetence can be recognised is one which on clinical grounds is not improbable. By it an explanation might be found for cases in which albuminuria is not present, and for the prodromata of eclampsia occurring before albuminuria. At present the method of classifying cases where albumen is not present, but yet which present all the other characteristics of eclampsia, is very unsatisfactory. To say that such cases are only "eclamptiform" does not dispose of the difficulty of their pathology, and, seeing they so closely resemble true eclampsia in all their clinical characteristics, save one, it seems likely that a common cause, acting in both cases, will yet be found. The idea of a primary toxæmia helps us towards an understanding of so called reflex cases of eclampsia, and it does not displace but merely carries further the explanation offered of the majority of cases by renal incompetence. The study of eclampsia both as to its remote, and proximate, causation has been greatly hampered by attempts to cover all cases of the disease by theories which are valuable as explaining some.

Diagnosis. A fully developed attack of puerperal eclampsia with its concomitant conditions ought seldom to present difficulties in diagnosis.

Usually the patient has reached such a stage of pregnancy as to leave no doubt on this point, and, if urine can be obtained the difficulty is still further overcome.

1) Hysteria - True eclampsia may, in its earlier stages, be mistaken for hysteria. In my experience this has occurred twice; one instance is noted in Case IV and details given. Two months ago a similar error occurred. A medical friend asked me to see a case with him, at about full term. His diagnosis was hysteria, and my opinion confirmed his. This opinion was based upon the personal and family history of the patient, both well known to me as very neurotic, and upon the character of the "fits". The latter were readily interrupted by any definite peripheral stimulation and it was possible to call the patient out of the convulsive condition by addressing her loudly and firmly. There were no true tonic or clonic convulsions.

But the case was said to have developed, a few hours later, into one of undoubted eclampsia.

D. Oliver, in reference to Bright's disease, says such a sequence of conditions may occur, and he attributes it to the action of toxins in one of their various manifestations.

(2) Epilepsy - may occur during pregnancy or the puerperium. The seizures are less frequent in their occurrence. An aura epileptica may precede the epileptic attack, but this is said to be rare in eclampsia.

Rabowjewsky, in reporting a case, states that Olhansen has been able to collect only three cases. The presence or absence of albumen is here of importance.

(3) Convulsions due to hæmorrhage. - The general condition of the patient, profound anæmia and collapse would guide us in such cases.

During the comatose state eclampsia is simulated by a number of diseases. The coma of epilepsy is neither so deep nor so persistent², the loss of memory is characterised in the same way and there is no albuminuria.

Cerebral hæmorrhage is accompanied by hemiplegia and not preceded by convulsion. In cerebral hæmorrhage occurring in the course of eclampsia the diagnosis might become extremely difficult. Albuminuria, or its absence, would partially guide us.

Alcoholism would be distinguished by the smell of the breath and of the vomited matters.

In the differential diagnosis of eclampsia Charpentier³ considers the temperature a feature of great importance.

Thus "in eclampsia the temperature rises gradually and

"rapidly from the beginning of the attack, and it continues"

1 Brit. Med. Jour. Epineur. 1895. part 1 p 75

2 Caspary. p 804; and Charpentier p 121 (Winckel doubts this. p 594)

3 l.c. p 121.

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"to rise, even after death". In uremia the temperature generally falls from the beginning. In epilepsy the temperature rises slightly, and falls again, only to rise at the beginning of a new attack. In hysteria it rises during the attack, but gradually falls to the normal when the attack is over.

Prognosis - The prognosis in eclampsia is always serious. The mortality to mothers is, according to Dohin's statistics, calculated on 747 cases, 29 per cent. Death rarely occurs during the attack but usually during the comatose stage, or, as a result of puerperal diseases following.

Lusk quotes Hofmeier's mortality percentage as 32.4 and Braun as 26. The prognosis is affected by various conditions.

1) The period at which eclampsia shows itself. Winkler³ says the prognosis is best when the first outbreak occurs in the puerperium, and this is the opinion usually held. Spiegelberg⁴ says "speaking generally the danger is "greater the earlier during pregnancy and labour the "paroxysms break out".

Löhlein⁵ (quoted by Lusk) gives his percentage thus:—

Of 83 cases occurring before, or during the first stage of labour	40 per cent died.
" 15 " " after completion of first stage	1 died
" 8 " " after labour,	1 died, due to infection.

1. Spiegelberg - l.c. p. 210 ; 2 l.c. p 528 ; 3 l.c. p 595
4 l.c. p 210
5 l.c. p 528

Wiegert's statistics, quoted by Charpentier¹ broadly agree with the foregoing. Cozeaux² agrees with Ramsbottom that attacks occurring towards the termination of labour and continuing after its completion are the most dangerous. Barker³ in 1858 showed by his collected statistics that 32 per cent of all cases which occurred before, and during, labour, died, and 22 per cent of those which occurred after delivery. He expresses the opinion that now (1876) this mortality has been diminished at least 50 per cent. Winckel says they lose only from 7 to 10 per cent of women in whom eclampsia develops after labour.

(2) The number of attacks - Charpentier's statistics show -

In 45 women who had 1 to 10 attacks	the mortality was 11.
" 31 " " " 10 to 20 " " "	" 10.
" 24 " " " 21 to 50 " " "	" 12.

Closely associated with the number of the convulsions is the degree of coma. This deepens with each convulsion. The duration of the labour must also be considered, seeing how frequently the expulsion of the child is followed by the cessation of the convulsions.

(3) The degree of renal incompetency.

Herman⁵ noted in his series of twelve cases, that the rapid increase of water with less albumen and more urea was a favourable indication. This change was apparent some hours after the convulsions. Cases which did not have

1 l.c. p. 123. } 3 l.c. p. 91. } 5. British Med. Journal. July 11th 91. p. 72
 2 l.c. p. 807. } 4. l.c. p. 123. }

this restoration died. Herman refers to two types of cases as regards prognosis; - (d) an acute type with copious albumen in the urine, and rapidly recurring "fits". In these cases if the renal action is not rapidly re-established there is a rapidly fatal termination.

In favourable cases the recovery is usually complete. Bright's Disease seldom remains in such cases.

(β) Cases with slow onset. Foetal prognosis better. More urine, less albumen than in acute disease. May pass into chronic Bright's Disease. The presence of tetinitis makes the prognosis worse. The degree of oedema does not materially affect the prognosis. (Cay² cause)

The death of the foetus in utero seems to improve the maternal prognosis. Charpentier³ mentions a case and quotes numerous others in which, the eclampsia ceasing without bringing about labour, a dead foetus has been expelled later. In other similar cases quoted, when the child has survived the eclampsia, there has been a resumption of convulsions at the time of its birth. The prognosis is seriously affected by the general condition of the mother. Cardiac and pulmonary complications in particular exercise a dangerous influence. The prognosis as concerns the foetus is very bad. The table shown by Charpentier⁴ gives a fatality of over 53 per cent. Winckel⁵ estimates it as high as

1. Obstet. Trans. Vol. XXXVI. p. 48. § 4 l.c. p. 125

2 l.c. p. 806

3 l.c. p. 96

4 5 l.c. p. 595 - 6

77 per cent. This is largely due to premature birth, and is consequently less favourable the further the onset of the eclampsia is from full term.

Besides premature birth, other causes are assigned by various authors. Cazeaux¹ and Barnes² attribute death in some cases to convulsions in utero at the same time as the mother is affected with eclampsia, or the convulsions may come on in the child after its birth.

(Barker)³: Lusk⁴ attributes the fatality to asphyxia from the accumulation of carbonic acid in the blood of the mother. Charpentier⁵ quoting Prunget's researches that the foetus dies whenever the temperature reaches $104^{\circ}2$, considers the majority of deaths to be due to the high temperature of the mother. The maternal prognosis is likewise affected by the disease which may arise post partum; haemorrhage, inflammation, renal disease, already noted as sequelae of eclampsia.

The method of treatment as affecting the prognosis.

It is customary for authors to assert that owing to improved treatment an improved prognosis has been arrived at, and probably this is true, but, when the newest treatment comes to be examined it is found that in some essential particulars it is a revision to treatment used before that which it now supercedes, which latter, in its turn, had had claimed for it

1. l.c. p 308 & 11 l.c. p 529.

2. l.c. p 408 & 5 l.c. p 125.

3. l.c. p 92 &

a similar superiority over the first. No better instance of this can be found than the question of blood-letting. Last century the practice of blood letting in eclampsia, when it was performed, was done in limited quantity. Thus, of the cases reported by Smellie, I find the average quantity taken by him was 10 ounces.

Ram²bottom (F.H.) writing in 1841 quotes various authors of last century to show that the mortality (Hunter, Lowderic) ran to over 50 per cent, and even higher. Nestor's clinical guide (1800) says eclampsia generally, though not always, proves fatal. Rambottom (1841) of his own practice says "few comparatively, under good care now terminate unfortunatly, and the favourable results are to be attributed to the extent to which bleeding, and the other evacuant means are carried". And, further "the English physicians have only recently, in comparison, carried the depleting practice to the extent now almost universally adopted".

Gooch (1829) says he has never lost a patient under convulsions, where free bleeding had been practiced.

Collins³ bled freely but showed a fatality of only 5 in 30 (= 16.5 per cent). W. Phillips⁴ in reporting eight successful cases, a series much too small to generalize from, says "it seems to me to be impossible to account for the fall in the mortality otherwise than by considering it to be due to less bleeding and more chloroform".

1. Smellie's Theory and Practice of Midwifery (New Syd. Soc.)

2. Rambottom. Princ. & Pract. Obstet. Med. & Surg. 1841 p. 582

3. Collins. Pract. Treat. on Midwifery, 1836. p. 211.

4. Guy's Hosp. Reports. 1870 p. 242.

It is doubtful if our "less" bleeding and more chloroform" treatment has lowered the mortality, and, if it has, by the showing of Collins, Gooch and Ramsbottom, the credit belongs to the chloroform.

Treatment. The principles of treatment in puerperal eclampsia are recognised by almost all writers as being towards lowering the blood pressure, controlling the nervous irritability, promoting elimination of morbid products, and, under certain conditions, emptying the uterus; but in the application of these principles it is still true, in the present state of our knowledge of the disease and of the action of various remedies, that we must be very largely guided by the circumstances of the case. The disease is of such an acute nature moreover and the opportunities for observation in general practice so comparatively rare, that individual remedies are seldom depended upon alone, and, consequently, one's opinion of the methods employed is subject to serious influences towards error in dealing with separate means of treatment.

The treatment may be divided into that which would be adopted in view of the possibility of an attack, and that which would be employed during a seizure and afterwards.

(1). Prophylactic.

Of the various preventive measures in view of premonitory symptoms of eclampsia, rest, and diet absolutely restricted to milk deserve the first place. The removal of all causes of mental anxiety and physical discomfort.

Chastpinter lays down certain rules amongst which exclusively milk diet is insisted upon, for every pregnant woman who has albuminuria. Jaccoud, in the same discussion, even recommends a modified milk diet for all pregnant women, and, in albuminuria, he would, in addition to strictly milk diet, give oxygen by inhalation, the quantity to be regulated by the deficiency of urea excreted. Beyond dietetic treatment, saline diuretics and cathartics, with the view of lowering vascular tension as well as removing morbid products by the emunctories should be given. The skin should be acted upon by hot packs, vapour baths, or, as are recommended by Parvin although the treatment must be difficult of application, hot baths. In such cases pilocarpin, if the use of it is well justifiable in eclampsia, would probably find its most satisfactory function. In cases in which there is much nervous tension chloral may be used with advantage as being more continuous in its action than chloroform.

If these means should fail to restore the renal function, venesection ought to be performed in quantity sufficient to have the effect of lowering the tension of the pulse. In my experience, rest, regulation of diet, and medical remedies acting on the eliminating organs (Kidneys, liver and intestines) have proved effective in warding off the ^{threatened} attack.

Under their action the quantity of urine has increased the oedema has diminished, the albumen in the urine has become less, and the subjective prodromata have been distinctly relieved. The further question of the induction of labour in threatened eclampsia is one on which there is much difference of opinion.

Spiegelberg considers threatened eclampsia never justifies it. Charpentier² rejects it. Under certain circumstances of serious involvement of the nervous system or known albuminuria in previous gestations Barnes³ seems to approve of it.

Winkel⁴ disapproves of it in all cases believing the remedies we have otherwise are as effective and more rapid and less dangerous in their action.

Lusk⁵, although admitting the weight of authority to be against him, considers as soon as grave cerebral symptoms develop there ought to be no further delay. He would use the bougie, vaginal

1. l. c. p 219.
2. l. c. p 129.
3. l. c. p 413.

4. l. c. p 616
5. l. c. p 536

Douche and Barnes' Bags. In any case, even for the introduction of a catheter, as Barnes points out, the patient should be under the influence of chloroform.

The indications of danger from eclampsia would have to be very strong indeed in a primiparous patient not at full term to justify me in undertaking the operation.

In cases I and V although what might be described as "grave cerebral symptoms" were present, though diminished by treatment, and, furthermore, although in case I the history of previous eclampsia was also known to me, the delay was justified by the results.

Postnatal treatment, if the theory of pressure on which it is based is correct, ought to be of use in such cases

(2) Remedial - when an attack of puerperal eclampsia

has developed our methods of applying certain curative agents are limited, because, after one or two convulsions, the consciousness of the patient is frequently lost to such a degree that little or nothing can be given by the mouth. The principles of treatment are the same as guide us in dealing with cases which only threaten, but we are justified in employing treatment at once which it was advisable to delay in the other instance.

In all cases of convulsions certain general precautions ought to be taken. All interference with the respiration

or circulatory functions, whether in the way of posture, pressure from clothes &c, or insufficient breathing space ought to be guarded against. The tongue should be protected against injury and consequent swelling with obstruction to respiration. The patient generally, ought, by judicious handling on the part of her attendants, to be prevented from injuring herself, and the rectum and bladder ought to be empty.

Of the active means of treatment the one regarding which the greatest difference of opinion prevails is venesection. By some authorities (Winckel, and many quoted by Charpentier²) it is never used, but it at least finds a place in the treatment of the majority of writers on the subject.

Statistics as to its value are very contradictory as is shown by the tables given by Charpentier³.

Thus in statistics from three sources

	General mortality of bleeding compared.		
	One bleeding.	Repeated bleeding.	
Clinic	45 per cent	41.3 per cent	54 per cent.
Observations	26 per cent	30.6 " "	21.6 " "
Maternity	34.7 per cent	36.3 " "	33.3 " "

Even if these statistics agreed with each other as regards the value of bleeding, free or limited, which they obviously do not, their value would be doubtful as to the general

1. l.c. p 596.

2 l.c. p 132.

3 l.c. pp 133-134.

question, because the extent of the bleeding is not stated, and further, it is by no means certain that two bleedings of say ten ounces, at an interval even comparatively short has the same effect in convulsions, as one rapid bleeding of twenty ounces.

(See case VI). Kussmaul and Tenner showed that in order to produce convulsions by anaemia of the brain, the anaemia must be produced rapidly. And when convulsions occur as the result of haemorrhage in man, the bleeding has been rapid, e.g. in post-partum haemorrhage. These observations at least indicate a possible difference in slow and rapid bleeding for remedial purposes. But even amongst the supporters of blood-letting in eclampsia very different views obtain as to the quantity. Of the writers of the first half of this century the opinion was in favour of free bleeding.

Ramsbottom (1841) recommends from 40 to 60 ounces or more in a "full stream". He further says that slow bleeding is almost useless. Collins² (1886) "prefers the arm when the blood flows copiously, and, if not, the temporal artery".

Of more recent authors Spiegelberg³ advises up to 17 ounces to be repeated if the effect is only temporary.

Lusk⁴ regards bleeding as the first step in the treatment of convulsions, and would draw from 8 to 16 ounces.

Barnes⁵ considers "venesection undoubtedly the most"

1 l.c. p 575 : 2 l.c. p 237 : 3 l.c. p 220 : 4 l.c. p 537
5. l.c. p 410

"powerful and prompt resource at command for lowering"
"the high vascular tension - a primary cause of the
"eclampsia", but he evidently seldom uses it, his practice
being based upon Schroeder's theoretical argument from
the Traube-Rosenstein view of the production of convulsions.
Barrett bleeds according to its effect upon the patient,
using his judgement as to quantity.

The limited experience which has come to me has con-
firmed me in my practice of blood-letting even to a
larger extent than is usually recommended by the authors
of the present day. My experience has been that stated
by Charpentier¹, that the pulse has improved and the
convulsions have been either abolished or reduced in
frequency and violence. (Cases I; II, VI).

²Worr mentions a fact which has struck me, that bleeding
in eclampsia "renders the patient more susceptible to the"
"action of other remedies". Barrett³ considers the practice
sedative to nervous irritation, removes the tension from
the brain, relieves the congestion of the kidneys and
lungs, takes pressure off the labouring heart, and
may supplement the action of the kidneys by
removing a portion of the excess of urica.

Boucharde⁴ says "it is certain we remove from the
"economy more excreta by bleeding than by any
"other channel, the renal tract excepted."

1. l.c. p 117: 2 l.c. p 53. 4 Auto-intoxication in Disease.
12 l.c. p 133: 3 l.c. p 117. (English Trans.) p. 133

It is not contended here that bleeding ought to be resorted to in all cases of puerperal eclampsia, but for the majority in my opinion, as a single remedy it is the most effective we possess.

Chloroform, (or ether, as some prefer) whether used alone or in conjunction with venesection, is perhaps the agent most frequently resorted to in the treatment of puerperal eclampsia, and, under certain circumstances it must have preference over all others.

In attempting to arrive at a conclusion as to the relative value of chloroform and venesection as exclusive means of treatment we are again met by contradiction from the different sources of information. Thus, Charpentier gives the percentage of mortality from two sources.

	Bleeding.	Chloroform.
Clinic	45 per cent	33.3 per cent
Maternity	34.7 " "	50 " "

Probably these statistics, like many others relating to eclampsia, are not reliable, because it is very exceptional to depend upon one remedy alone.

The method of administration of chloroform as to time in relation to the seizures, degree of anaesthesia to be produced and precedence over other remedies is remarkably variable as given by different authorities.

1 l.c. p 138

Thus, ¹Carriance considered it useful after bleeding, purgation &c, had been tried and failed.

²Winckel gives it from the first symptom of the convulsion to its termination, and then ceases, but he uses it as an adjunct to chloral.

³Sprigelberg says it is wise to give it only during the paroxysms, but still, according to the same authority "it is necessary to induce complete narcosis".

⁴Leishman says "during a fit it is proper to withhold the" "chloroform". ⁵Lusk restricts the use of it to the pains and to the period of restlessness before a fresh paroxysm except when labour is nearly complete.

⁶Barnes appears to give it only during the convulsion.

⁷Charpentier says "when once complete anaesthesia" "is produced, keep it up without regard to time."

⁸Barker considers that if the fits follow in rapid succession profound anaesthesia ought to be kept up. He attributes failure on the part of some observers in the use of chloroform to the insufficient degree of anaesthesia produced.

My experience of chloroform in two cases in which I tried it continuously and apparently to the full point of anaesthesia (although it is difficult to judge during coma) has been disappointing. (See Cases I-II)

1 l.c. p 925.

2 l.c. p 596.

3 l.c. pp 220-221

4 l.c. p 758

5 l.c. p 537

6 l.c. p 111

7 l.c. p 138

8 l.c. p 119.

On theoretical grounds one would carry the patient as rapidly through the stage of excitement as possible and continue to keep her profoundly anaesthetised, but considering the duration of Eclampsia in some cases such a prolonged anaesthesia would of itself possess dangers. Probably the method adopted by Lust is the most satisfactory. When one bears in mind the complete suspension of respiration during the tonic stage of the convulsion and the very imperfect respiratory act during the clonic stage, together with the state of the blood throughout the latter part of the seizure, changed as it is to an alarming degree with carbonic acid, the recommendation that only during the paroxysm should the chloroform be given seems to me very impracticable. My rule has been to withdraw the chloroform during the convulsion, chiefly because it is useless to a patient who is not breathing, and also because any small quantity which may be taken during the clonic stage is better held back in the state of the blood at that time.

The value of chloroform in eclampsia is however not limited to the treatment of the paroxysm. One of its most useful functions is to abolish the reflexes, and so one cause at least of the convulsions, thus admitting of operative treatment if necessary, or even

the ordinary obstetric manipulations.

Until there is more accurate knowledge of the pathology of puerperal eclampsia the relative value of bleeding and chloroform will be a matter of dispute.

M. Lénor groups cases of eclampsia into those where the evidence of heightened reflexes predominates, those in which toxæmia is more apparent, and an intermediate class, and probably our choice of venesection, of anaesthesia or both, as a means of treatment, will be guided by such grouping, based as it is on clinical observation, rather than by statistics of all cases in which the pathology is assumed to be the same.

At present it seems to me a combination of the two methods is of greater value than either applied alone.

Chloral whether given alone or in combination with other remedies is a drug of great value in many cases of eclampsia. Testut² considers there are two kinds of eclampsia; one from reflex irritation, the other due to cerebral œdema. In the former he considers chloral most valuable, but valueless in the latter.

The results obtained in some groups of cases, in which chloral alone has been used, are very remarkable.

Thus Testut, by an analysis of Charpentier's tables (Date of tables, 1872) arrives at the remarkable

percentage mortality of cases in which chloral alone

1. British Gynecol. Journal. Vol. IX. p. 358.
2. Quoted by Charpentier. Vol. II. p. 139.

has been used in treatment, of only 4 per cent, and Froger¹ gives the result of 51 cases (amongst 110) treated by chlotal alone, of which 49 recovered. Doubtless treatment of all cases by chlotal would have given a different result, and here, as in the case of venesection and chloroform, we have still doubtless much to learn as to the indication for one remedy or another. Of the authors whom I have been able to consult ²Baxter is the only one who is entirely opposed to the use of chlotal in eclampsia. He considers chlotal probably excites, rather than lowers, reflex irritability.

The method of administration varies, as does the quantity given. Barnes³ limits his practice to one dose of 20 to 30 grains. He considers it dangerous if used again within a few hours. Speigelberg⁴ gives 40 grs, and repeats at suitable intervals. Lust⁵ begins with chloroform, then gives, per rectum, 30 grs of chlotal and an equal quantity of Potassium Bromide, the chloroform to be discontinued when the enema has exerted its sedative effects, and the frequency of repeating the enema to be decided by the violence of the case.

Winckel⁶ treats his cases with chlotal alone - up to 12 grams (not grains as the others deal in) a day, per rectum. Charpentier⁷, in 60 gr doses, while not fearing 240 grs in twenty-four hours, rarely exceeds 180 grs. He quotes Delannay and Froger as having given as much as 300 grs.

1. Quoted by Charpentier. vol. II. p. 140: b l.c. p 596.
 2 l.c. p. 120: 3 l.c. p 411: 4. l.c. p. 221: 7 l.c. p 141:

My practice has been to give 20 to 40 grs in enemata every four hours in suitable cases. Patients most likely to benefit from the use of chloral are those with an excitable or neurotic temperament and in whom renal incompetence has not reached a high degree, as judged by the quantity and condition of the urine.

Morphia - The question of the use of morphia in eclampsia is one which has given rise to much controversy on theoretical grounds, but Barker, who has great confidence in the use of opiates, considers the objection to them chimerical, in so far at least as they are applied to eclampsia. Sprigelberg² speaks of it with confidence, although he leaves the question as to preference between chloral and morphia open. Just³ uses morphia (from $\frac{1}{6}$ th to $\frac{1}{4}$ th gr) hypodermically, repeating in an hour if necessary.

No doubt its action has approximately the same limits as to the group of cases which it is likely to benefit, as chloral and other narcotics, and experience shows that the risk of fatal narcosis in uterina is not great.

Pilocarpin has been, and still is, used with the double object of producing rebolic action and diaphoresis, but chiefly the latter. It seems to increase uterine contraction when that has already been set up, but does not, according to Charpentier induce labour primarily. As a diaphoretic it is rapid but not safe.

1 l.c. p. 119; 3 l.c. p. 537;
2 l.c. p. 221;

Barker, in six cases in which he tried it, had to dis-continue it in all on account of pulmonary complications.

Sprigelberg quotes others to the same effect, and confirms their opinion by his own experience. Phillips reports thirty-nine cases in which it was used, and seven of them died.

Its use was followed by acute pulmonary edema.

I have used it twice, once without apparent result, and once with disastrous consequences by the production of acute edema.

Vitætrium Viride is a drug which, though not much used by British or European obstetricians, is largely used in America, and apparently with good results. Barker

who does not seem to use it in eclampsia, but in phlebitis and other diseases, speaks of it very highly as a vascular sedative. He considers the danger from cardiac failure more apparent than real during its use, and says the recovery from such a threatening condition is very rapid.

Numerous American authors recommend the drug in puerperal eclampsia, as being preferable to any other remedial agent. D. ³Oatman says, in so far as he has employed it, it has been uniformly and speedily successful.

The antidote to cardiac depression is alcohol which is said to act surely and quickly. The fall of the pulse below forty to the minute is an indication to stimulate.

In addition to the above, which might be called specific remedies, the general treatment discussed under the

1. l.c. p. 222:

3. Trans: Intern: Med: Congress. 1889. Vol II. p. 470

2. l.c. p. 296:

leading ptophylaxis, ought to be employed.

Jalap and Calomel ought to be given if the patient is conscious, and, if not, elatetium or croton oil.

Various drugs are, or have been, used, aconite, belladonna nitrate of amyl, antipyrine, and many others, but they can hardly be classed as recognised treatment of eclampsia. Counter-irritants, as blisters and sinapisms, have been abandoned, and cold water to the abdomen is not used. Not can pressure of the cathods be recognised as practicable treatment.

Postural treatment - The postural treatment of eclampsia having as its basis the pressure theory of renal incompetence has been recommended by various authors - King, Hewitt², Routh.

Obstetric treatment - all observers are agreed that eclampsia, in many cases, is cut short by the emptying^P of the uterus, and it naturally follows that, where the risks to the mother are not greater by actual interference than from eclampsia, obstetric operative treatment ought to be resorted to. The point at which we pass to less risk from interference than from inaction is a matter of dispute. Lusk³ remarks the question is "one which cannot be settled by the hap-hazard experience of individuals", although he personally, has found delay disastrous, and practices induction

1 Trans. Inter. Med. Congress 1887. p. 475: 3 l.c. p. 538:

2 Obstet. Trans. vol XXIX p. 550:

of labour, and after the completion of the first stage he would make careful use of the forceps.

Winckel never induces premature labour and describes Löhlein's practice of the reverse treatment as obsolete. He says, moreover, it is quite unwarrantable to hasten labour in eclampsia before the second stage is reached.

Charpentier² is also entirely opposed to the artificial induction of labour in eclampsia, and gives as his reasons. —

- (1) eclampsia is only a symptom of a general disease which the emptying of the uterus cannot eradicate at once,
 - (2) convulsions do not always cease after labour but may even be produced then,
 - (3) eclampsia is an acute disease, rapid in its course, and the time required for the induction of labour may exceed the duration of the eclampsia,
 - (4) labour, coming on in eclampsia, as it usually does, progresses rapidly, and allows generally of some interference without danger to the mother,
 - (5) any irritation in, or around, the uterus may induce convulsion, much more so will the processes required in the induction of labour. He adds "if we discard"
- "the artificial induction of labour, with how much greater reason should we reject forced delivery."
- "Of this there cannot be question in any case."

In support of his views he gives some figures; of 478 cases, in 278 the attacks were continued or

produced after delivery. "We do not then believe in
"the absolute efficiency of labour in puerperal eclampsia";
"therefore we discard the induction of labour".

Barnes would induce labour in all cases where
convulsions have set in, and even in cases of definite
and serious premonitory symptoms, especially
where there is a history of albuminuria in previous
gestations, or Bright's disease. He disapproves of delivery
before the cervix is "fairly² dilated". Spitzelberg
disapproves of interference. He says "there can be no question"
"of obstetrical interference, i.e. the induction of premature"
"labour". And "forced delivery is entirely forbidden"
"under these circumstances, i.e. while the cervix uteri"
"is still totally unprepared". Cazeaux and Tarnier³
think artificial labour ought only to be used when
copious bleeding and allied measures have failed, and that
"during pregnancy, however severe the convulsive attack"
"may be, forcible delivery ought not to be attempted".

Barker⁴ approves of the induction of premature labour when
the convulsions continue and the coma deepens, and other
means have failed. He does not express an opinion on
forcible delivery. W. Tweedy⁵ says labour is never induced
for eclampsia at the Rotunda Hospital, because the opinion
is held that it adds enormously to its severity.

From these opinions we gather that authorities are

1 l.c. p 413: 3 l.c. pp 820 and 821:

2 l.c. p 225: 4 l.c. p 121: 5. British Med. Jour: 1891 Vol II. p 11422:

nearly evenly divided on the question of the induction of premature labour, but they are practically unanimous against forcible delivery. St. Haultain has practiced rapid dilatation, sufficient to pass the blades of the forceps, and then forcible delivery. His results are excellent, but, as Swayne² points out, the number is too small to draw any conclusion from. Green³ records a series of 36 cases, treated at the Boston Lying-in Hospital, which does not encourage us even towards artificial induction of labour. Of 13 cases of eclampsia occurring before labour, three were not interfered with and all the mothers recovered (children non-viable), and, of the remaining ten, in which labour was artificially induced, six mothers died.

When the first stage is complete, and the os uteri quite dilatable, the recognised treatment is to produce deep anaesthesia, and deliver by forceps, version, embryotomia, or according to ^{obstetric} rules as for any case requiring such assistance.

1. Edin. Med. Journal. Vol. XXXVII. pp 126-129:
2. Brit. Med. Journal: 1896 Vol 1. p 523:
3. Amer. Journ. Obstet: ~~1899~~ Vol. XXVIII p. 18:

In post-partum eclampsia all causes of reflex irritation should be sought for and removed, eg. blood clots in the uterus. Chloral or morphia, given with regard to the effect upon the convulsions should be used, and with the addition of chloroform if necessary.

Venesection may be used even in such cases with marked benefit (case I).

The sequelae of eclampsia must be treated according to their nature.

Cases.

Case I.

Mrs Ska-ba - primipara, aged 23.

Family History:-

All four grandparents lived to be over eighty years of age. Father and mother living, and healthy.

Eleven brothers and sisters alive, one still-born, no deaths.

Married sisters have had about twelve children, without any history of eclampsia.

Personal History

Patient is of very good physique, and of healthy appearance. She was subject to "fainting attacks with" "sobbing" for some years. Three years ago she suffered from an attack of sub-acute rheumatism, which left no cardiac affection. No other illnesses - no scarlatina diphtheria, or other condition likely to be associated with nephritis. Menstruation began at 13, and, after two periods, ceased till 17. From then until her marriage at 21 she was irregular, the periods varying from three to six weeks.

After marriage the fainting attacks disappeared, and, at the end of the third month she had a miscarriage.

Her second pregnancy occurred more than a year after marriage. For the first six months she had

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good health, no vomiting or neuralgia.

About the early part of the seventh month she observed swelling of the face, hands, and lower limbs, and also that she passed too little urine.

She suffered from intense headaches and attacks of partial failure of vision which lasted for half an hour or longer. Her memory "became a blank" for similar periods. On June 29th 1895 at 3 a.m. labour commenced and was completed at 2 p.m. the same day.

The presentation was vertex, and the position L.O.A.

The labour was uncomplicated and the third stage was complete fifteen minutes after the birth of a healthy female child. The secundines seemed healthy and there was no excess of hemorrhage.

At 8 p.m. the patient appeared very slow in her mental processes, and complained of intense frontal headache and "dreadful sleeplessness".

A strong saline purgative was given, and Chloral hyd: grs ~~xxx~~ an hour later, but no sleep was obtained.

At about 2-30 a.m. (over twelve hours after delivery) the first convulsion occurred, and, from then until 4 p.m. there were twenty-eight seizures.

From 3-30 a.m. till 8 o'clock chloroform was almost continuously administered. The convulsions, an hour apart at first, increased in frequency until

the intervals were not more than twenty minutes. Chloroform was abandoned and chloral (grs xxx per rectum every two hours) substituted. Pilocarpin (gr $\frac{1}{16}$) was given hypodermically twice but without effect on the convulsions, and even without producing marked diaphoresis. At 1 o'clock venesection was performed and twenty ounces of blood abstracted. Ten minutes after the blood-letting a convulsion occurred, which proved to be the last, except for a much milder attack over two hours later.

The patient was in a condition of general oedema of the subcutaneous tissues, and the lungs were also acutely oedematous. She remained comatose for about thirty-six hours. Urine was obtained, by the catheter, twelve hours after the cessation of the convulsions and, on boiling, it became nearly solid.

Hyaline casts were present, and free blood corpuscles (possibly from the lochia) but no blood casts were observed. The patient remained under my care for five weeks, during which time her vision, greatly impaired at first, became natural; her memory, which seemed to have been largely obliterated for events which had occurred throughout her pregnancy, greatly improved, and the albumen disappeared, rapidly at first, although it persisted for three

weeks in small quantity. The recollection of events which occurred within some weeks previous to the eclampsia was never fully regained. The urine was examined at intervals for four months but without albumen being recognised again.

On October 19th 1897 the same patient engaged me to attend her in her next confinement, which was calculated to be due about 11th January 1898.

Considering her previous history, it was requested that urine be sent for examination every week.

At the date of my engagement the patient was in good health. She had had sickness during the early months of her pregnancy, but no impairment of vision, headache, or attacks of loss of memory.

The specimens of urine examined were all free from albumen and of satisfactory specific gravity until the 11th of December. This urine deposited, on boiling, about $\frac{1}{8}$ th of its bulk. An examination of the patient showed distinct oedema of the hands, face and lower limbs. The patient stated that the swelling had come on within the previous three days, each day having been worse than that which preceded it. A week before (on the 7th) the patient had been in a state of semi-consciousness for about an hour.

This condition was preceded by headache and faintness

of vision. With difficulty, a neighbour informed me, the patient was able to go next door to her house, where she remained for about an hour, apparently unable to speak and "in a dazed condition". She then returned home, and slept soundly for two hours. The patient herself has no recollection of this visit to her neighbour's house. There was some palor, no sobbing, sighing or crying. Until my visit, a week after the attack, there had been no similar seizure, but almost every day the patient had suffered from severe frontal headache, with temporary failure of vision. The attacks of headache also occurred in the night but otherwise sleep was easily obtained. After such attacks the patient felt drowsy.

Rest in bed was advised and was continued for two days. The bowels and kidneys were freely acted upon by salines, and the patient put upon a strictly milk diet. The edema diminished somewhat, and the quantity of urine increased markedly; the attacks of headache and loss of vision were less frequent and less severe, but the quantity of albumen remained about the same until confinement. Labour began at 6-30 a.m., on December 31st, and was complete at 12 o'clock, noon; two weeks before the estimated time. There was no abnormal condition

in the labour. The child, a male, was healthy, and apparently mature. Eight hours after the labour the patient had not slept, and complained of frontal headache; otherwise her state was satisfactory. Chloral hydrate and Potass: Brom: of each grs XXX in a draught were given, and the patient slept for several hours. Next morning the pulse was 76, temp: 99.0; urine. S. G. 1018, acid; albumen about $\frac{1}{8}$. Throughout the puerperium no unhealthy symptoms occurred. The albumen had disappeared in ten days and subsequent examinations at intervals of a week showed no recurrence. Lactation was successful. The child of the former pregnancy was fed by hand and, notwithstanding healthy surroundings and careful feeding, developed a very unusual degree of deformity due to ticks. The recovery from this deformity was spontaneous and, of the limbs at least, and so far as could be observed of the trunk, complete.

NOTES. This case presents some points of interest.

First, as an unusually severe case of post-partum convulsions which did not yield to the ordinary remedies for the condition. The premonitory symptoms were well marked. But for the peculiar slowness in her mental methods, there was nothing remarkable in the condition of the patient during labour - there

appeared to be no undue reflex irritability, and otherwise but for the oedema of the face, not unusual after a continuation of labour pains, the condition of the patient was satisfactory. The treatment of the eclampsia was a striking instance of failure of sedative, and success of depleting, treatment, even in a case of post-partum convulsions where there was no evidence to show the existence of any reflex cause of irritation. The second pregnancy affords an illustration of threatened eclampsia in a patient who had been regularly examined for albuminuria for several months after the previous gestation and for some time before the later development of the prodromata of eclampsia. Sir James Simpson said recurrent eclampsia was almost always associated with persistent kidney disease. This case seems to have been an exception to the general rule. The premonitory symptoms yielded very satisfactorily to treatment, the chief agent being, I believe, strictly milk diet. Bouehard says "milk is one of the most powerful" "medicaments which we can oppose to uræmic accidents". Probably the treatment, together with the fact that labour occurred about a fortnight before the estimated time, accounted for the patient's escape from convulsions. The attack described as having occurred about the time of the outset of the albuminuria, resembles very closely an epileptic seizure, without the usual convulsion, and may have been an abortive eclamptic attack, which did not return ^{owing to} ~~During~~ treatment.

Case II . Mrs S---d - primipara - age. 26.

Family History:

Father died aged forty-nine of cancer of the stomach. Mother died, aged 52 of heart disease. One sister living and healthy. One brother died, aged 23, of Phthisis.

Personal history:

The patient has had no serious illness previous to the present, and has not had any affection of the kidneys so far as she is aware. She is a well formed, healthy woman and has had no neurotic history. Menstruation was regular every four weeks until the time of her marriage. After marriage she had two periods, and then became pregnant.

During the early months of pregnancy she suffered from morning sickness, but not severely, and otherwise her health was good. About the middle of the fifth month she began to suffer from attacks of intense headache to which she had not previously been subject. Her face, hands, and legs also began to swell. This continued until about the end of the sixth month when she, without good reason apparently, developed a "severe cold". At mid-day on the 24th of October 1895 the patient sent for me and stated that she was six months pregnant, that during the morning she had suffered from violent headache, and had had difficulty in seeing objects in the room. An examination showed her heart to be healthy

primipara

but there was some oedema of the lungs, as well as of the general surface of the body. The os uteri was that of a multipara, and showed no commencing dilatation, the cervix being that of about a six months pregnancy. There was no other indication of impending labour.

Before urine could be obtained for examination (half an hour after my visit) the patient had an eclamptic convulsion. From 12-30 till 11 p.m. nineteen convulsions, mostly of great severity, occurred, and after the first the patient was deeply comatose. Chloroform was administered continuously for three hours with a negative result so far as the frequency and violence of the convulsions was concerned. Morphine hypodermically (gr i every 2 hours), and chloral with Bromide of Potassium (of each grs xxx) were given without effect. At eleven p.m. venesection was performed, with considerable difficulty owing to the degree of anasarca, and twenty-five ounces of blood drawn. No convulsions occurred after the blood-letting.

Until about an hour after the blood-letting no labour pains could be recognised, although an examination showed shortening of the cervix and early dilatation of the os uteri. At 5-30 a.m. a still born, non-viable child was expelled by the natural powers. The patient remained unconscious for two days. The bladder was empty

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five hours after the completion of labour, but in twelve hours urine was obtained by the catheter.

It was deep brown in colour. S. G. 1030, and on boiling became nearly solid. Hyaline, granular, and blood casts were present. The albumen rapidly diminished in quantity, and in three weeks had entirely disappeared. Almost complete amniarosis existed at first, but normal vision had returned in six weeks. The patient's memory for recent events (several months) remained impaired while she was under my care. The urine was examined, at intervals of a week, for three months but albumen did not reappear.

Two years later the urine was acid, S. G. 1020, no albumen, casts, or other evidence of renal disease.

There was no increased arterial tension, nor any cardiac hypertrophy. The patient had had good health, but had not again become pregnant.

Notes: Although the number of convulsions in this case was not large comparatively, the attacks were more violent and the coma more profound, almost from the first, than in any case which has come under my observation. It would no doubt have been described as a distinctly toxic case, and, in its response to treatment this was borne out.

Sedative treatment did not appear to afford any relief, but blood-letting, carried to what according to present-day teaching would be considered an excessive degree, was followed by the complete cessation of the convulsions and the recovery of the patient.

CASE III. Mrs. F. - - - - - primipara - age 23.

Family history.

Father and mother living and healthy. Two sisters and one brother living, no deaths.

Personal history

The patient has never had any serious illness. Previous to her marriage she was under my care, more or less, for a year and a half, suffering from neurotic symptoms which varied in character. Neuralgia in the ovarian region was the most common complaint, but she also suffered from a "lump in her throat", want of strength &c, none of which symptoms had any reasonable physical explanation. Her pelvic organs were, after examination by Mr. Jessop, (Leeds), considered healthy, the urine was free from albumen on the numerous occasions on which it was examined. She was an exceptionally intelligent young woman, well developed and healthy, but for the neurotic manifestations. As there was considerable risk of her becoming a chronic invalid, I took the responsibility of advising her to get married, in the hope that the symptoms might disappear. Until then her menstruation was regular in its onset, and lasted about four days, the ovarian pain being more severe apparently at those times.

after marriage she did not menstruate, and the
 neurotic symptoms disappeared. She had vomiting
 during the first three months of pregnancy, but not severe.
 She also suffered from facial neuralgia. Full term was
 estimated to be in the first week of January 1898.
 On December 10th, I ~~was~~ called to see the patient and
 found her in good spirits, and health. Her feet were
 slightly swollen which led me to examine the urine.
 There was no albumen. The patient was not seen again
 until after the first convulsion. On the morning of the
 24th December 1897 she remained in bed, complaining
 of headache. At mid-day she could see objects in the
 room very indistinctly, then she said she was totally
 blind, and, immediately that she could not hear.
 At once she had a convulsion, which was said to
 have lasted five minutes. Another similar attack
 occurred before my arrival. Half an hour after the
 first seizure her condition was as follows: - Semi-
 -comatose, occasionally seeming to recognise those
 about her; restless, tossing from side to side; pupils
 equal and dilated; breathing slow, regular and
 stertorous; pulse 120, regular, small, but firm;
 anasarca absent except slightly of feet.
 Tongue furred, flaccid, lacerated at its edges.
 Sight at least fair - she knew me to be a doctor

but used a wrong name. No urine could be obtained. The cervix uteri was that of a nearly full term primiparous woman, but showed no signs of commencing dilatation.

Prescribed - Pulv. Jalapa Co grs XXX, Calomel. grs V and Chloral Hyd: grs XXX, Pot. Bromid: grs XX the latter to be taken every two hours.

During the ensuing two hours the patient had four convulsions but was semi-conscious in the intervals. Between 4 and 6 o'clock (the next period of two hours) eight convulsions occurred, and the patient was comatose throughout. Enema of chloral grs XL, and Pot. Brom. grs XXX was given at 6 o'clock.

From 6 to 9 o'clock there were four convulsions of less severity. At 9 o'clock a similar enema was given.

No more convulsions till 11 a.m. when a mild seizure occurred. The enema of chloral and bromide was repeated.

No further convulsions. Throughout the period of convulsions I repeatedly examined for uterine contractions but recognised none. During the succeeding sixteen hours the patient remained deeply comatose, breathing regularly, and noisily. There was no evidence of labour and the os uteri remained about the same.

At 6 a.m., 26th Dec., the labour pains having apparently begun soon after my evening visit, the head

was on the perineum, and, after the rupture of the membranes, was born by the next pain. The child looked healthy and full grown, but was dead.

The third stage was complete in ten minutes and the after-birth and membranes looked healthy. There was no excess of hemorrhage. Urine and feces had been passed in bed. Throughout the day, patient comatose.

Dec. 27th. Patient still unconscious but, towards evening restless. Urine (by catheter) of deep brown colour, S.G. 1025.

Albumen about $\frac{1}{6}$ th precipitated on boiling. Blood corpuscles (possibly from lochia) in small numbers. No casts seen. Pulse 112. Temp: 99.0.

Dec. 28th. Patient semiconscious, has taken milk freely. Apparently sees those about her, and, on firm request, puts out her tongue. Pulse 96. Temp: 99.5.

Dec. 29th. Pulse 90. Temp: 98.6. Passed urine in bed pan. Urine paler than on 27th; S.G. 1025; albumen about $\frac{1}{8}$ th.

Throughout the puerperium the patient had no further rise of temperature, the discharge remained healthy, but the albumen and loss of vision persisted. The

eyes were examined on January 15th and showed distinct albuminuric retinitis. Now (March 16th)

two and a half months after confinement there is still albuminuria, varying from a slight trace to a well marked cloud. Retinitis is distinctly observable.

From the time of her return to consciousness her memory has been very defective, and is so now. This refers to a period of about a year, chiefly antecedent to the eclampsia, and more or less to events which have occurred since. On her return to consciousness she had no recollection of having been married. When shown her wedding dress she remembered the event, but insisted she was not called Mrs F. She had no recollection of her pregnancy she frequently repeated the same question within a few seconds, or made the same remark. Until now (March) the period covered by this blank seems to have been shortening, and the loss of memory is more confined to the occurrences within the latter months of her pregnancy. Even now she says she can carry no mental picture of places, however familiar. She remembers nothing of the streets of her own town as soon as she leaves them. There appears to be no true aphasia, and, in ordinary conversation she is intelligent and cheerful.

Her disposition has not in any way changed.

Notes: This case was regarded from the first as being in some measure determined by the temperament of the patient and treated as such. It illustrates the responsibility one incurs in recommending marriage, for although the neurotic ^{symptoms} seem to have been cured the patient has passed through a condition of great danger, and has now fixed renal disease.

3-

Throughout the albumen was small in amount, was absent a week before the confinement, still retinitis and persistent albuminuria show how serious the effects upon the kidneys have been. The psychical results are very strongly marked, especially in loss of memory. Venesection was not practised in this case for the reason before noted, the probably large nervous element. In such a patient, probably the stimulus required in the form of toxemia is much less than in others, to excite the reflex act and produce convulsions, or the reflex-inhibiting centres may be more easily overcome, a condition of the nervous system having been produced by even a slight degree of toxemia resembling that found normally in children. The improvement under chloral was very satisfactory, both in the steady improvement as regards the lessening of the paroxysms and their disappearance. Possibly obstetric interference, had we known of the progressing labour, might have saved the child.

Case IV. Mary S---h - primipara - age about 24. The history of this patient could not be obtained, as her confinement and death occurred amongst those who knew little of her life. The child born was illegitimate. She was first seen at 3 p.m. on July 9th 1897. She was evidently well advanced in pregnancy - about the end of the seventh month probably. During my visit she had

several attacks of an epileptiform character, but in some respects suggestive of hysteria. There was an absence of the initial tonic spasm of the eclamptic seizure, and of any marked interference with the respiration. During the attacks the patient frequently uttered cries and sobbed.

There was no very definite commencement or termination of the attack, but a general and constant state of restlessness, and she was drowsy and slow in replying to questions. Her general condition was good, - heart and lungs healthy. There was slight swelling of the feet but apparently none of the hands and face. No urine could be obtained. In the absence of history the case was regarded as one of so-called hystero-epilepsy occurring in a pregnant woman.

Potass: Brom: grs xxx every two hours was prescribed.

On my second visit, four hours later, it was obvious the patient was suffering from true eclampsia, and that of an acute type. The seizures came on every twenty minutes, or less, and the intervals were passed in a state of deep coma.

Chloral Hyd: and Potass: Brom: , of each grs xxx, were given, per rectum, twice, at intervals of two hours.

Until now there had been no labour pains so far as could be ascertained, and the os uteri was not dilating.

At 11 p.m. the convulsions continuing, and the condition of the patient seeming to justify it, Pilocarpin gr $\frac{1}{6}$ th was given hypodermically. a similar tabloid was left, to be

given at 3 a.m.; the chloral and bromide to be continued. The patient was seen again by me at 6 a.m. and her state, in the interval, had changed very remarkably. During the night she had developed a condition of acute oedema of the subcutaneous tissues. She was scarcely recognisable on account of the swelling of her face, and her limbs and general body-surface were in a similar state. The pulse was soft, and very rapid, the heart obviously failing, and the lungs acutely oedematous. The convulsions continued. Labour had now begun, the os uteri being dilated to about two inches. The uterine contractions were strong, regular, and at much shorter intervals than the convulsions. A premature (about 7 months) female child was expelled at 8-30 a.m. by the natural powers, and the placenta was immediately expressed. There were several convulsions after the completion of labour, and the patient died of heart failure three hours after the child was born. The treatment during the last six hours of the patient's life had been directed towards restoring the power of the heart and consisted of ether, brandy, strychnia and digitalis, given subcutaneously, and dry-cupping over the bases of the lungs.

Notes: The two facts especially worthy of remark in this case were, the difficulty in diagnosis from hysteria in the patient's earlier condition, and the, as I believe, injurious effects of pilocarpin; both I have noted in the text of my thesis.

Venesection seemed to be contra-indicated by the apparently nervous nature of the case at its commencement, and perhaps I was also influenced against even such a slight operation by the squalid surroundings amongst which the patient had to be treated. The acute oedema seemed to have followed directly upon the use of Pilocarpin, and this is in accordance with the experience of others, as formerly noted. The child might have been delivered, with safety to the mother, an hour earlier than it was, but I regarded the patient then as moribund.

Case. V. Mrs R-----m. - primipara - age 28.

Family history: - Father and mother in good health, eleven brothers and sisters living, two dead, one in childhood, the other aged twenty-three of Phthisis. Four sisters married, only one of whom has had children, - two. Her confinements have been without complications.

Personal history: - The patient has not previously had any medical attendance, but has always had good health. She is not a robust type of woman, but is of fair physique. She had previously engaged me to attend her in confinement in the first week of March 1898, but called me to see her on January 13th. She then complained of dull pain in the back (lumbal region), frontal headache, and attacks of giddiness. These symptoms had been

present for about six weeks, and for the past fortnight she had also been subject to loss of sight (partial) coming on for an hour, or less, at irregular intervals. Otherwise she said her vision was perfect. An examination showed her heart and lungs to be healthy. There was a slight degree of swelling of the feet, and possibly also of the hands, face, and general surface of the body. Pulse 76, of rather high tension. Bowels regular every day.

Urine in average quantity she considered.

Urine, acid. S.G. 1016. Deposited on boiling $\frac{1}{8}$ th of its bulk of albumen. Casts were examined for but not found.

Treatment consisted of rest in bed for a few days, diet restricted to milk only. Saline cathartics and diuretics were given. In three days the backache, headache, giddiness and loss of sight had all disappeared.

The dropsy was distinctly less, but there was no perceptible decrease in the quantity of albumen.

Patient was allowed to be up but nothing was allowed in the matter of diet except milk. The urine was examined twice a week and continued to show the same quantity of albumen. The subjective symptoms continued to be absent, or only slight, but the anasarca increased.

February 10th. Urine, acid, S.G. 1020, albumen increased to about $\frac{1}{5}$ th. She had had milk pudding, bread and tea against instructions. She declined to lie in

bed but returned to the milk diet exclusively.
February 16th. Patient was seen three times, as labour had begun. The pains were very weak and at intervals of about half an hour. Os uteri, at my evening visit, about the size of a half-crown, greatly thinned cervix, os rigid - Patient somewhat drowsy but readily conversed. Pulse 92, small, hard. Her general condition compelled me to add to the milk diet Brand's Essence.

Urine showed about $\frac{1}{6}$ th albumen.

February 17th. At 4-30 a.m. the patient was seen by me. She was said to have had a "fainting fit" at 4-5 a.m. Her condition was as follows. She was conscious, but in the same drowsy condition as on the previous day. Pulse 130: Resp: 28: pupils somewhat dilated, Temp: (axilla) 99° 0'. The pains were of the same character as on the previous evening, but the os uteri was now nearly fully dilated. 4-50. Eclamptic convulsion (50 seconds). Pulse (after convulsion) 144: Temp: 100° 4'. 5-20. Convulsion, pulse irregular; Temp: 101° 2'. An assistant now having arrived, chloroform was given, and a stillborn child delivered by forceps at 5-30. Five minutes later I delivered (by forceps) another child, living. The secundines were immediately expressed and ether given hypodermically, and brandy per rectum. There were no further convulsions, and, in an hour, the

patient was able to take beef-jelly and brandy.
During her convalescence her lungs presented some
symptoms which led to the suspicion of phthisis, and
there was partial paraplegia which, however, had
disappeared within a few days. Her temperature did
not become normal for a fortnight, and no trace of
albumen could be found at the end of that time.
Her memory is largely a blank for some days before her
confinement, but, previous to that, and afterwards,
her recollection of events &c does not seem to be impaired.
Ophthalmoscopic examination four weeks after confine-
-ment gives no evidence of retinitis.

NOTES: This case was one in which prophylactic treatment
was possible, and attended with very satisfactory results.
The twin pregnancy is to be noted. This is a case in which
interference under chloroform, even two hours earlier, would
probably have prevented the development of the convulsions,
which, however, of themselves were not serious, and did
not require special treatment. The variations in temperature
associated with the convulsions are confirmatory of Bournville's
observations.

CASE VI. M^{rs} O. --- m. - age 32.

Family history: Parents living, and the whole of a large family
of brothers and sisters.

Personal history: Patient is a fullblooded, healthy woman, who has had no medical attendance except for five natural confinements. Her children are all living and healthy. None of them have suffered from convulsions or other nervous disorder. I was called to see the patient on the 14th July 1895. She had, the previous day, returned from a fortnight's holiday at the seaside, and, but for some headache of the frontal region and vertex on the morning of her attack, had been in good health. On my arrival the patient was in a condition of violent mania, struggling with those about her and extremely restless. At intervals of about 15 minutes the mania was interrupted by violent epileptiform seizures, resembling in all respects severe attacks occurring in puerperal eclampsia, but she was not pregnant, as I ascertained from her husband, and, later from herself, that her menstruation had been regular. Even during the non-convulsive periods it was obvious the patient did not know what was going on around, but her conjunctival reflex was not abolished. It was impossible to give anything by the mouth, and enemata were almost equally difficult to administer, and, if given, were immediately expelled by the violent effort of the bowel. Hypodermically the patient was given Morphia gr i and Hyocine gr $\frac{1}{100}$, but without apparent result. The condition continued about two hours. Venesection was then

performed, the patient being held by force during one of the maniacal periods. A good flow of blood was obtained from the left median-basilic vein. When four ounces had been extracted the extreme restlessness had subsided, and when twenty ounces had been taken from her she was quite conscious and practically well. The convulsions did not return, neither did the mania. She was considerably bruised about the legs on account of her struggling, and her tongue was extensively lacerated. Her urine was not examined until next day and no albumen was present. The urine was examined frequently within the following month and no albumen discovered. The S. G. was satisfactory on these various occasions, and I could recognise no undue arterial tension. No paralysis, and very little loss of memory except for the period of the attack. She remained well until the 17th March of the present year (over two and a half years). On the morning of that day she felt uncomfortable, complaining chiefly of flatulence and frontal headache. She was able to go about her daily work. At 11 a.m. she was met by her husband who advised her to go home as she "looked haggard, and ten years" "older than when he left her to go to his work". She returned home at once, and, on reaching the

door of her house, fell forward, bruising her forehead considerably. She immediately passed into an epileptiform convulsion. In twenty minutes I saw her and found her in a state of extreme restlessness, throwing her arms and legs about, and struggling to rise from the couch on which she was.

A few minutes after my arrival she had a second convulsion, which had all the characters of a violent epileptic seizure. The tonic stage was disproportionately prolonged (about 45 seconds) with the result that the patient became very deeply cyanosed, nearly asphyxiated I should say. The total length of the convulsion was exactly 65 seconds (by my watch).

Previous to the convulsion the pupils were natural in size.

Pulse 76, of good quality and medium tension. During the convulsion it was impossible to feel the pulse at the wrist, nor was it discernible for 6 seconds (time accurately taken) after the paroxysm had subsided. The pupil during the convulsion was much contracted, but widely dilated as the attack passed off and restlessness returned. I decided to do bloodletting at once, and opened the right median basilic vein, but very little blood flowed. A vein in the opposite arm was opened with a similar result, then one on the dorsum of the foot (left) with only partial success (about 4 ounces)

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about a pint was ultimately obtained from the opposite limb, but even here the process was very slow. From the first puncture to the cessation of bleeding was over an hour, and the process was interrupted by two convulsions. Another convulsion occurred immediately after the bandaging of the last puncture, but, on its subsidence, instead of the restlessness which had always occupied the intervals, there was a period of rest. Pulse 80 regular, soft. Pupils contracted. On my raising the eyelid, to test the conjunctival reflex, the pupil suddenly widely dilated, and, at the same moment, restlessness returned. The state of restlessness thus induced continued for about twenty minutes without intermission. Chloroform was then given (about 3/4) and in twenty seconds the pupil contracted and synchronously the restlessness ceased. Chloroform was then withdrawn and for about five minutes this state of rest with contracted pupil continued. Suddenly, and at the same moment, and without any apparent cause, the pupil dilated and the restlessness returned as before. A small quantity of chloroform was again administered with the same result - contracted pupil and rest. This occurred on three successive applications of chloroform, but, after the fourth, the pupil did not dilate to the same degree, and there was less restlessness. No more chloroform

was given, and one could observe the cycle becoming less and less marked both as regards the dilatation and contraction of the pupil until it became natural in size. During this period the patient was gradually regaining consciousness. She then passed into a state of sleep. The whole period of unconsciousness was a few minutes over two hours. Four hours after the attack she passed urine highly charged with lithates, S.G. 1030. Albumen was distinctly present, forming a well marked cloud on boiling. Several slides were examined for casts but none found. On the following day the patient gave no evidence of the attack. She was quite conscious and cheerful, and wished to be allowed to get out of bed.

Urine passed twenty hours after the attack contained no albumen. There is no paralysis, and the loss of memory is limited almost entirely to the period of the attack. Her vision is good, and there is nothing abnormal to be seen by the ophthalmoscope. The urine was examined every day for a week, but there was no albumen.

Notes: Although this case is non-puerperal, it presents one or two points of interest in connection with the foregoing thesis. (1) The convulsions were, in all likelihood the cause of the transient albuminuria which followed them, and some confirmation is here given to the view of Brashton-Hicks

before referred to - that in certain cases of eclampsia the convulsions are a possible cause of the albuminuria.

(2). The value of venesection in cases of convulsions with high arterial pressure is clearly shown. On the occasion of the first attack the patient passed within ten minutes from a state of mania with violent convulsions to a condition of rest and consciousness. I attribute the partial failure on the second occasion to the unsatisfactory performance of the blood-letting, in that the flow of blood was very slow. Still great improvement was apparent after the venesection.

Lusk's remark finds an illustration here, that after bleeding, other remedies produce their results more readily. The quantity of chloroform in this case was disproportionately small to its good effects.

The group of cases here reported is much too small to admit of any generalisation being made from it, but, as I have already indicated in the reports, and throughout my thesis, the effects of free bleeding have fully justified my adoption of the practice.

Although the number of cases is small, observation at the bedside has left no doubt in my mind of the value of venesection. With the object of lowering the pressure, in the renal vessels, it ought to be practiced early.

Later, this object is still attained, though the earlier the more valuable the relief, and, in addition, the risks from

cerebral pressure, whether arising from stasis or hemorrhage, may be greatly reduced. The direct action, in the removal of toxic material is likewise a matter of extreme importance.

Much has been omitted in these reports which, under other circumstances, would doubtless have been included in a full description of eclampsia, and my explanation of the gaps is to be found in the constant calls of a general practice, and the imperfect assistance, as observers, of the average helpers at confinements in the homes of the poor.