which took him every few minutes, and so completely prevented any refreshing sleep that his weakness had begun to be alarming. He had been treated for this symptom with a variety of sedative and antispasmodic remedies old and new, but the attacks had resisted every effort to suppress them. Finding the tongue excessively foul and the stomach considerably dilated, I advised a good aperient dose of rhubarb and heavy carbonate of magnesia, made up into a draught with tincture of cardamoms, spirits of chloroform and peppermint water, to be given without loss of time. This was done, and I heard later that the hiccough had ceased immediately that the draught had been taken, and that the patient had eventually made a good recovery—in fact, he lived for some years afterwards. I have used the same treatment in many cases of a similar kind for patients of all ages and cannot remember a single instance in which it has failed to relieve.

The action of turpentine upon the mucous membranes is utilized in the treatment of hepatic concretions. A dose of 10 or 15 drops given two or three times a day after food I have found not only to relieve the catarrh of the bile ducts but also to have a solvent action upon the gall stones—if I am correct in drawing that conclusion from the long period of relief which I have known to follow a course of the remedy. Again, in pyelitis arising from the irritation of retained gravel in the pelvis of the kidney, turpentine given in the same dose is very useful in checking the inflammation, although it has no solvent action upon the concretions. In these doses it is most conveniently prescribed in capsule.

The curative value of turpentine when given by the mouth may often be supplemented and enforced by its internal use in enema. In cases of threadworms the vermifugal action of the remedy is strikingly manifested, and it has also a salutary influence upon the mucous irritation which is a constant accompaniment of ascarides if these be numerous. For a child of 7 or 8 years of age $\frac{1}{2}$ oz. of the terebinthinate oil may be used for the injection at bedtime diluted with 10 oz. of barley water. The late Dr. Elliotson used to treat obstinate cases of amenorrhoea in young women with the same injection repeated once or twice a day, and maintained that its action was attended with almost invariable success. It has been employed, too, in cases of catalepsy. One ounce of the oil well mixed with a pint of thin gruel and thrown slowly into the bowel acts as an energetic stimulant to the torpid nervous system; and the injection, if followed by vigorous frictions along the spine with a strong irritating liniment, is said to be attended with very satisfactory results. In flatulent colic a terebinthinate enema, if given hot, is a welcome addition to the use of remedies by the mouth and will often bring a severe attack to an immediate close. In the seizures of this kind which have been before referred to as common in infants the derangement is a cause of so much suffering that nothing which tends to shorten the attack should be neglected; and the injection of a drachm of the oil in 5 or 6 oz. of hot barley water thrown slowly into the bowel will often put an end to the distress and send the child into a quiet sleep. Employed in this manner the remedy is a diffusible stimulant of extreme excellence, and in taking stock of the various curative expedients at our command in such a case to omit it from the list would be voluntarily to abandon one of the most trustworthy of our resources.

THE French Medical Congress will be held this year at Geneva on September 3rd, 4th, and 5th, under the presidency of Professor Ad. d'Espine, of Geneva. The subjects proposed for discussion are: (1) The clinical forms of arterio-sclerosis, to be introduced by Dr. Huchard, of Paris, and Professor Jacquet, of Bale. (2) The pathology of neurasthenic states, to be introduced by Professor Dubois, of Berne, and Dr. Jean Lépine, of Lyons. (3) The treatment of biliary lithiasis, to be introduced by Professor Gilbert and Dr. Carnot, of Paris, and Dr. Mongour, of Bordeaux. On September 6th there will be an excursion to Evian at the invitation of the Société des Bains of that town. A circular tour through Switzerland is also being organized. Doctors of any nationality can enrol themselves as members of the congress, but French is the only language in which communications will be received or discussions allowed. The General Secretary of the Congress is Professor A. Mayor, 6, Rue Adhémar-Fabri, Geneva.

An Address ON PUERPERAL ECLAMPSIA, WITH SPECIAL REFERENCE TO ITS TREATMENT WITH NITRO-GLYCERINE.

By JUSTIN M. MCCARTHY, M.D.,

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PERHAPS there is no more trying—indeed, one might almost say, no more terrible—way of spending a night than in a struggle with death for the life of a patient in the throes of puerperal eclampsia.

Having been brought into contact with 15 such cases, it seemed that the classification and tabulation of the notes relating to them, considered in the light of information gained from the writings of those who have made a special study of this subject in recent years, would well form the subject of a paper, which might possibly be of benefit to others who may yet have to go through a similar ordeal, be of some service in the mitigation of suffering, if not in saving life, and at the same time call forth a fruitful discussion.

CASES.

Apology is offered for the incompleteness of the records, the reason being that the cases occurred in country practice, with a minimum of help, so that one's time was almost entirely taken up in rendering the necessary attendance.

In Table A we have a list of these cases with their leading characteristics, 15 of them being puerperal eclampsia, 2 epileptic, and 1 due to tuberculous meningitis during which labour took place—these last 3 being convulsions during labour, and so puerperal eclampsia, but still not of the type that presents itself to one's mind when thinking of true puerperal eclampsia. One of these, No. 18, was having a fair labour, having previously been confined of twins; the head was on the perineum, when she had a most violent epileptic fit. Her face became livid, there was much frothing at the mouth, and the breathing was stertorous. Only a partially-trained midwife was present, so that the difficulty of applying forceps, which step seemed absolutely necessary, was very great. This, however, was fortunately accomplished without injury and the patient delivered of a living boy, who, with his mother, eventually did well. This patient had had true epileptic fits on many occasions. Another point in connexion with the case was the marked post-epileptic stupor which followed and lasted some hours. Such a condition might have an important medico-legal bearing on cases of infanticide.

Coming to the true puerperal eclampsia, they are Nos. 1 to 15 in Table A. They occurred at different times from February, 1878, to December, 1906. One interesting item is that the two cases, Nos. 3 and 4, both of which were fatal, occurred within twenty-four hours. This is possibly a unique experience in the obstetric woes of a general practitioner. The 13 are taken out of some 2,800 cases attended in my part of the practice from 1875 to the present time—a proportion of 1 in 215.4. This is admittedly an excessive proportion, Galabin giving the general proportion for Europe as about 1 in 500 deliveries. It should be further noted that, of the 15 cases in question, only 8 occurred among those for whom attendance had been arranged, and that with regard to the other 7 medical aid was in the first instance summoned for convulsions. The periods of occurrence are shown in Table B.

These are so erratic that it would be quite possible for a practitioner with a small obstetric practice to have a large average of such cases, whilst another with an extensive practice might have none.

There was no possibility of infection in the two cases occurring within twenty-four hours. One sent for help in the evening, but the child was born before the arrival of the doctor. The third stage was attended to, and the patient comfortably settled for the night, when the second patient sent for help because of convulsions. Next morning No. 1 patient sent for help, as a convulsion had taken place. This came on about twelve hours after labour, and the patient had not been seen since visiting No. 2.

TABLE A.—Cases of Puerperal Convulsions.

No.	I. Name.	II. Age.	III. Married or Single.	IV. Engaged or Not.	V. Primipara or Multipara.	VI. First Convulsion Before, During, After Labour.	VII. Result.		VIII. Nitro-glycerine Used.	
							Mother.	Child.	Internally.	Hypodermically.
1	S. C. Feb. 26, 1878	19	S.	No	P.	Before	D.	D.	No	No
2	L. April 23, 1881	39	M.	Yes	M. 2	After	R.	L.	No	No
3	M. A. F. Jan. 20, 1882	21	S.	No	P.	Before	D.	D.	No	No
4	D. Jan. 20, 1882	30	M.	Yes	P.	After	D.	D.	No	No
5	B. Oct. 14, 1882	20	M.	Yes	P.	Before	R.	L.	Yes	No
6	S. June 4, 1886	35	S.	No	M. 3	After	R.	L.	No	No
7	W. May 26th, 1880	33	M.	Yes	M. 3	During	D.	D.	Yes	No
8	O. June 19th, 1895	40	M.	No	M. 2	Before	R.	L.	Yes	Yes
9	S. Oct. 6, 1895	19	S.	No	P.	During	R.	L.	Yes	Yes
10	G. Dec. 27, 1901	38	M.	Yes	P.	After	R.	L.	Yes	Yes
11	J. May 11, 1902	31	M.	Yes	P.	During	R.	L.	Yes	Yes
12	B. Dec. 23, 1905	35	M.	Yes	P.	After	R.	L.	Yes	Yes
13	H. Jan. 9, 1906	26	M.	Yes	M. 2	After	R.	L.	Yes	Yes
In consultation with partner (Dr. Thornton).										
14	J. Dec. 16, 1903	27	S.	No	P.	During	D.	L.	No	Yes
15	J. Mar. 25, 1904	32	M.	No	P.	During	R.	L.	Yes	Yes
Convulsions not true puerperal. Two epileptics and 1 meningitis (tuberculous).										
16	A. Feb. 5, 1900	—	M.	Yes	M. 6	After	R.	L.	—	—
17	M. Nov. 28, 1903	25	M.	No	M. 2	Before	R.	L.	Yes	No
18	G. July 14, 1906	—	M.	Yes	M. 2	During	D.	L.	—	—

TABLE B.

1876	1877	1878	1879	1880	1881	1882	1883	1884	1885	1886	1887	1888	1889	1890	1891	1892	1893	1894	1895	1896	1897	1898	1899	1900	1901	1902	1903	1904	1905	1906	1907

Etiology.

Columns II to VI are of interest from an etiological point of view, and must be considered together. A paper by Dr. Cotton, quoted by Dr. More Madden,¹ states that the majority of patients are under 30 years of age. In the series we are considering they are about equally divided, eight being 30 or under that age, the other seven over 30. The youngest was 19 years, and the oldest 40, covering a space of twenty-one years, so that if this number be divided into three periods, we get slightly more in the first and last periods respectively than in the middle period. This is certainly in favour of the idea that the younger and older are more susceptible to eclampsia, a point which has been emphasized by another observer. Coming to the third column, namely, married or single, the same author says: "A woman illegitimately pregnant is more susceptible to this disease than one that is married." This statement introduces a psychological element. The table of my own cases shows 9 married and 4 single. You must remember that in the series of cases from which these are taken 2,740 were married and 61 single, that is, 9 out of 2,740 married, or about 1 in 304, and 4 out of 61 single, or about 1 in 15. This fact is strongly confirmatory of the above opinion—indeed, perhaps, too much so—one, I suppose, of the freaks of statistics.

The column showing the proportion of multiparae to primiparae is replete with interest. The former are five, the latter ten in number. The ages of the five multiparae are respectively 39, 35, 33, 40, and 26. All, except the last one, have a special history.

The first, aged 39, married late in life, had lost the child of her first confinement, and was particularly anxious about the accouchement in question. She was, moreover, a chronic dyspeptic, eventually dying from malignant disease of the stomach.

The second, aged 35, was single; this, however, being her third confinement. Up to the time of labour she was working every day on the pit bank, attending to her house and family,

and, in addition to all this, sitting up many nights, nursing her father, with whom she lived, through an attack of acute bronchitis, her means of living at the same time being very restricted.

The third, aged 33, was confined of the first child since marriage, but in her young days had given birth to an illegitimate child, which fact preyed greatly on her mind.

The next, aged 40, had convulsions in her previous labours. She is one of two surviving, so far as have been traced, who at the present day have albumen in the urine. There was most likely a pre-existing kidney factor in this case. In addition to this must be added the fact that, previous to her confinement, her husband had treated her very badly.

The last, aged 26, was the only case that could give no history likely to be helpful in dealing with such an attack.

The primiparae call for some notice with regard to their ages. Whilst these range from 19 to 38, the ages of five are 19, 21, 20, 19, and 22, one is 27, and four 30, 38, 31, and 35 respectively. Now, excepting the one at 27, a woman who came home in trouble to her parent's house to be confined, and who felt her disgrace very keenly, the ages are very young or rather advanced. The authority previously quoted gives four-fifths as the proportion of primiparae, whilst these figures give two-thirds. He further adds: "Old primiparae are thought to be more liable . . . than those who are younger." As the number of young primiparae attended would surely be greater than that of old, the proportion of old that suffered would certainly be greater than that of the young. You will admit that it was practically impossible to work out the ages of nearly 3,000 cases.

The first convulsion occurred before labour in 4 cases, during labour in 5, and after in 6. These figures do not appear to call for much comment.

Albuminuria occurring in pregnant women has, in my experience, been a cause of anxiety in some cases, but in not one of the few observed had it led up to puerperal eclampsia.

So far as the etiological factors are concerned in these cases, it would seem that the most marked feature is the

TABLE C.—*Urinary Analysis in Nine of the Cases of Puerperal Convulsions.*

No. of Case in Table A.	Date.	Albumen.	Sp. Gr.	Reaction.	Sugar.	Urea.	Deposit, etc.	Subsequent History.
2	April 23, 1881 Dec. 11, 1900	Present Nil	— 1010	— Acid	— Nil	— —	— —	No further pregnancy, died of malignant disease (Sept. 26, 1903).
5	Oct. 14, 1882	Coagulum on boiling in spoon	—	—	—	—	Smoky	Now living in Yorkshire, 5 children since, all alive, no further convulsions.
6	June 4, 1886 Dec. 12, 1907	— Trace	— 1015	— Acid	— Nil	— 1.5 p.c.	— —	No further family.
8	June 19, 1895 Dec. 4, 1907	Cloud 0.75 per cent. Trace	1020 1012	Acid Acid	Nil Nil	0.7 p.c. 1.2 p.c.	Granular casts No casts	No more family.
9	Oct. 6, 1895 1907	Loaded Nil	— 1016	— Acid	— Nil	— 1.6 p.c.	Scanty and smoky	Five children since, all well, no further convulsions.
10	Dec. 27, 1901 1907	0.45 per cent. Nil	1022 1022	Acid Acid	— Nil	— 2 p.c.	— —	No children since.
11	May 11, 1902 1907	Moderate amount present Nil	1016 —	Acid —	Nil —	1.6 p.c. —	— —	One child and one miscarriage since, no further convulsions.
12	Dec. 23, 1905	0.4 per cent.	1018	Alk.	—	—	—	Perfectly well, no further family.
13	Jan. 9, 1906 1907	0.4 per cent. Nil.	1024 1024	Acid Acid	Nil Nil	2.1 p.c. —	Casts —	Health good since, no further family.

disturbance of the nervous system. The vasomotor system also must have a severe strain thrown upon it in the regulation of the blood supply under such altered conditions. Dr. Cotton writes: "Emotional disturbances, arising either from grief or joy, are conducive to its development." The suggested causes, up to the present time, so far as one has been able to compile them from Galabin and Eden (amongst other authorities), are:

1. Increased abdominal pressure
 - (a) On the renal veins;
 - (b) On ureters.
2. Products of fetal metabolism discharged from maternal blood, and eventually eliminated by maternal kidneys. To this may be added the placental toxin theory.
3. Arterial strain.
4. Reflex nervous influence, starting from uterus and disturbing circulation in kidneys.
5. A special micro-organism.
6. Insufficiency of activity in the thyroid gland.
7. Hey Groves's theory of increased coagulability of the blood giving rise to minute capillary thrombi with hæmorrhagic infarctions surrounded by necrotic tissue, to be found more or less in all the organs, but especially in the kidney and liver. Carstairs Douglas,² experimenting since, finds little difference in the coagulation time.

Dr. Boxall,³ in 1905, writes:

While many ingenious theories have been advanced to explain the renal insufficiency which is prone to supervene in pregnancy, no adequate solution has yet been obtained, nor is it precisely known on what the process of fatty degeneration of the renal epithelium depends. Further, although the occurrence of eclampsia, and the presence of albumen in the urine are intimately associated, it by no means follows that either bears a causal relationship to the other.

The precise cause of the eclamptic fit is equally obscure; but it is now generally regarded as being dependent, in the main, on toxins. Marx⁴ divides the toxæmia of pregnancy into (1) due to heart disease, (2) due to acute pregnancy nephritis, (3) due to exacerbation of chronic nephritis, and (4) acute toxæmia.

Pathology.

Among the pathological changes found *post mortem* by different authorities, the following appear of chief importance:

Ureter.—Somewhat dilated above pelvic basin.

Kidney.—Early stage of tubal nephritis; degeneration of renal cortex; interstitial hæmorrhage; areas of necrosis.

Liver.—May be reduced in size; pale; hæmorrhage; outlines of lobules indistinct; thrombotic hepatitis with degeneration of liver cells.

Spleen.—Similar to liver.

Brain.—Anæmia; oedema; congestion of meninges; small thromboses; hæmorrhage.

Now these, as far as they go, point to changes in the small vessels generally, throughout the organs of the body, of which the kidney is only one, and in conjunction with them one calls to mind such clinical facts as visual dis-

turbance, which is recorded, and about which some oculist may be able to enlighten us. The vomiting, indeed, may be the result of a like condition of stomach, as also the glandular disturbance, especially noted lately in the thyroid.

Passing on to the clinical aspect, what is to be noticed? First, it is generally admitted that puerperal eclampsia, unaccompanied by albuminous urine, is quite exceptional. Galabin states that of all the cases which occurred in Guy's Charity during forty years up to 1875, in which the urine was examined, two only were entirely free from albumen. In one of these convulsions were produced by arachnitis, as verified by an autopsy, while in the other they followed severe *post-partum* hæmorrhage in a girl that had been seduced. The urine in our cases was albuminous in all examined. For No. 6 the report is missing. However, as there were traces of albumen in her urine when recently examined, it is more than likely that it was present during the eclampsia. The earlier specimens were tested in the house by boiling in a spoon; the later ones were sent home for methodical examination. "Urine scanty, smoky, with coagulum on boiling"—such is the commonest description.

Of the series 1 to 13, nine recovered, and in Table C. you will find what was the condition of the urine at the time of the attack, and also the result of a subsequent examination, most of them at the end of 1907. Nos. 5 and 12 had left the district, one having gone to Canada, and the other to Yorkshire, so it was impossible in these cases to ascertain the condition of the urine. They are both, however, reported to be well, one having since given birth to five children, all living and well; nor had the eclampsia reappeared in any confinement. As this report was received fifteen years after attendance for eclampsia, there could not have been much permanent damage done to the kidneys. No. 8, referred to earlier in this paper as having had a previous attack of eclampsia, still has a trace of albumen, as also No. 6. Five are marked "Nil." The urea was estimated in one case, No. 8, 7 per cent. at the time of illness. This healthy condition of the kidneys in such a large majority of the cases in later life is very remarkable, and up to the present time has not, perhaps, received the attention to which it is entitled. It clearly points to the fact that, severe as the affection may be at the time, it is temporary, and one from which in many instances perfect recovery is possible. The ages of those who had no further family should be noted, as having a bearing on this point.

The puerperal eclamptic convulsions are very similar to uræmic convulsions. Indeed, in very many instances the conditions appear to be identical so far as causation is concerned—that is, in each case the convulsions are probably brought about by a toxin the origin of which is faulty elimination on the part of the kidney. Uræmic convulsions, however, are, as a rule, due to permanent

disease of the kidneys; whereas in the majority of cases of puerperal eclampsia the disease is only temporary.

Looking at the *post-mortem* appearances of those who die in eclampsia, especially having regard to the widespread changes in the small vessels of the body, may it not be possible that we have vasomotor changes due to inhibitory nerve influence, and that the kidneys are only the prime offenders among all other organs of the body by virtue of the suspension of their usual function? That nerve influence over the kidney may lead to death, as in suppression after severe operations or accidents, is generally admitted. Of the suggested causes mentioned above, arterial strain, reflex nervous influence, insufficient activity of the thyroid gland, and Hey Groves's blood condition might be accounted for.

There remain, therefore, for consideration as accessory: the products of fetal metabolism, although it remains still for this to be demonstrated as a cause; and, lastly, a special micro-organism, which has yet to be isolated.

Coming to the question of mortality, we have five mothers dying out of fifteen. Standard works state the mortality to be 40 to 50 per cent. This has recently come down to from 20 to 25 per cent. Our cases, old and new, show about 33 per cent., which agrees in the main with the more recent data. But if we divide the mortality into three classes, having respect to the use or otherwise of nitro-glycerine, as follows—(1) cases in which it was not used, (2) used only internally, (3) used hypodermically—we get the following results:

Without nitro-glycerine, 5 cases, 3 deaths.

With nitro-glycerine internally only, 2 cases, 1 death.

With nitro-glycerine hypodermically, 8 cases, 1 death.

The mortality among infants was four out of fifteen, or about 26.7 per cent. And here, by the way, it may be noted that, in selecting the treatment to be employed in any particular case, the life of the infant should have careful consideration; what effect, for instance, is a prolonged period of convulsions in the mother likely to produce on the life of the infant, and, if not on its life, on its future? In the case of a patient of the Roman Catholic persuasion, the first question would be a very important one.

Treatment.

In Table D we have a list of drugs that have been recommended, and also the number of times that they were used in our cases. This, however, is not perfect, as drugs such as opium and aperients were used in some cases and no record kept. Such a list would be somewhat confusing to any one, if presented to him, when on the point of attending his first case of puerperal eclampsia. This shows the need for determining general lines of treatment.

TABLE D.

Drugs Suggested.	No. of Cases used in.	Drugs Suggested.	No. of Cases used in.
Amyl nitrite	1	Potassium bromide	2
Aconite	—	Phenacetin and caffeine	2
Chloral hydrate	1	Potassium iodide	—
Chloroform	11	Pilocarpin (hypodermically)	1
Cocaine	—	Potassium citrate	—
Calomel	—	Sodium nitrate	—
Codeine	—	Sparteine	—
Digitalis	—	Strychnine	—
Ergotin (hypodermically)	1	Sodium salicylate	—
Guaiacol	—	Squills	—
Hot-pack	1	Strontium lactate	—
Hyoscin	—	Sodium bromide	2
Iron perchloride	2	Saline injection (rectal)	1
Morphine (hypodermically)	2	Thyroid extract	—
Nitro-glycerine (internally)	8	Veratrum viride	—
„ (hypodermically)	8	Venesection	2
Opium	—		

This does not include ordinary aperients, enemata, and uterine douches.

The stage at which the convulsions threaten or occur will indicate the nature of treatment. First, we have the premonitory stage, in which they threaten before labour occurs. Here the treatment should tend to avert toxæmia and convulsions. During this period thorough examination should be made daily. And all that care in treatment which would be given to an early case of Bright's disease, especially that relating to diet, should be carried out. Nitro-glycerine may be very useful. Marx,⁵ referring particularly to the toxæmia of pregnancy complicating nephritis, writes: "We have a large class of cases in which the continuous administration of from $\frac{1}{100}$ to $\frac{3}{100}$ of a grain of nitro-glycerine . . . gives brilliant results." This was in 1898. The first one on our list treated with nitro-glycerine and nitrite of amyl inhalations was in 1882.

Second, the period of labour itself, which may well be divided into two stages:

(a) Where the convulsions are slight and the labour slow. Here again the object should be to avert convulsions. Perhaps the subcutaneous administration of morphine and nitro-glycerine, used alternately, will at this stage be found beneficial. It is about at this time that the greatest difficulty arises in determining what course of treatment to adopt in order that both patient—namely, mother and child—may have the best possible chance. It is even possible that, with a rigid os and increasing convulsions, such difficulties might be encountered as would warrant the consideration of Caesarean section.

But when we come to (b), when convulsions are increasing and labour advancing—even if the os is dilating kindly—then all the energies should be directed to expediting labour. There has been, I know, considerable difference of opinion on this point, some even at this stage preferring morphine. But whatever be the cause of the death-dealing toxin, surely the source of its manufacture should be got rid of as speedily as possible. This is the stage where chloroform is essential and a drug of the greatest utility. It should be remembered that at this stage and the next, when the convulsions are at their worst, it is a question whether any reliance can be placed upon drugs given through the mouth. Some may never reach the stomach, and, if they do and the stomach shares the general condition of the other organs of the body, they may not be absorbed. Therefore, wherever possible, give medicine hypodermically.

Lastly, when labour is over, the treatment should aim at the restoration of the function of the kidneys and the recovery of the nervous system from the shock it has sustained.

The experience of some—that the convulsions cease with the emptying of the uterus—was not confirmed by our cases, for 6 of them were not attacked until the labour was completed, while 1 of them, No. 4, died at this stage, and another, No. 13, nearly died. Of the other 9 who were attacked before or during labour, 4 died and 5 recovered, the deaths in every instance occurring after the completion of labour.

Now here it is just possible that nitro-glycerine may prove to be one of the most useful drugs. In Case No. 5, attended in 1882, nitro-glycerine was used internally, at the first combined with amyl nitrite inhalations. This, a case of severe convulsions continuing after labour, recovered. Previously, 3 out of 4 cases had died from continued convulsions following delivery. Passing over No. 6, of which the record has been lost, we come to No. 7, where nitro-glycerine was administered internally only, and which died also of continuous convulsions after labour was completed. Cases 8 and 9 were those that impressed one most. Labour in both cases was concluded, and yet the convulsions continued. Bearing in mind the necessity of hypodermic treatment, and the manifest success of nitro-glycerine in No. 5, this drug was administered hypodermically with No. 8, commencing with 3-minim doses, increased to 4 minim, when diuresis was established, and recovery followed. In October of the same year No. 9 occurred. Here also the convulsions continued to get worse after labour was over, until things could not have been more critical. Nitro-glycerine hypodermic injections were then resorted to, and continued up to 7-minim doses. The patient recovered, and has had healthy children since; no return of eclampsia at the time of delivery, while the urine in 1907, as will be seen by

glancing at the table, was normal. In both these cases chloroform had been given every chance, before pushing nitro-glycerine. Since then nitro-glycerine has been used in every case, with only one death, No. 14, where the treatment was commenced very late, perhaps too late.

The value of nitro-glycerine in Bright's disease commenced to exercise the minds of professional men about 1879. Murrell⁶ had discovered that this drug caused great arterial relaxation. Ringer⁷ wrote: "Nitro-glycerine is employed in cases of contracted kidney, to lower arterial tension; it relieves the distressing headache of Bright's disease, and is said to increase the quantity of urine in chronic parenchymatous nephritis." The diuretic effect had further been verified by Dr. Fancourt Barnes in a case of epispadias. Then arose in my mind the similarity of condition existing in patients suffering from puerperal eclampsia and those suffering from uraemic convulsions, and thus I was led to try nitro-glycerine for the former class of patients.

As regards the use of other drugs, the usual routine treatment was tried. Venesection was used once, in addition to which one was a placenta praevia where the bleeding was necessarily free. This case died. The hot pack was tried with No. 7, but the result was not encouraging. Chloral and bromide of potassium were used internally and per rectum, but failed. Saline injections, per rectum, were used once, and should, perhaps, prove valuable in later stages, especially following the complication of haemorrhage. Veratrum viride, which has been highly recommended recently, was not used.

In conclusion, whilst the results quoted above necessarily produced a great impression on my mind, yet the suggestions are not offered in a dogmatic spirit, but rather for the purpose of encouraging further investigation in this complex subject.

Finally, I would suggest that the scope of the use of nitro-glycerine hypodermically might be extended into the physician's domain. It is quite possible that this series, with its results, may be only one of those remarkable coincidences which occur from time to time. But should the consideration of them in any way tend to a more successful treatment of these painful cases, in which the joy that should come with morning is often turned into the sorrow of darkest night, my object will have been fully attained.

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ON THE CAUSES OF INVERSION OF THE UTERUS.

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IN the *BRITISH MEDICAL JOURNAL* for April 11th there appeared a communication from Dr. George Hall, of Stockton-on-Tees, in which he related a case of inversion of the uterus after parturition. A few days subsequently I received a copy of the report of the Rotunda Hospital from the Master, Dr. Hastings Tweedy, in which mention is made of a similar case. The nearly simultaneous occurrence of these cases attracted my attention, as I had in bygone days paid some attention to the subject of inversion, and endeavoured to find an explanation for its occurrence and the causes tending to produce this rare accident, for did it follow on pulling on the funis or on pressure applied to the fundus, it would be very common instead of rare.

In 1881 I read before the Dublin Obstetrical Society a paper on the subject¹ founded on a case in which inversion had been produced by the action of the uterus in its efforts to expel a sessile tumour which grew from the very centre of the fundus, and I stated that I doubted the correctness of the view commonly held that inversion rarely occurs except immediately after delivery, inasmuch as out of five cases of inversion which had come under my own observation only two had occurred as a sequence of parturition. In the other three it had been due to the presence of sessile tumours which had grown from the centre of the fundus. In former days when dilatation of the uterus mechanically was little practised, indeed hardly known, I have removed quite a large number

of pedunculated tumours from the uterine cavity, some so large as to compel me to remove them in sections, others protruding through the os, the pedicle having elongated so as to permit this, but never have I met with a pedunculated tumour which caused inversion. I therefore laid it down as a rule that for an intrauterine tumour to cause inversion it must in the first instance spring from the very fundus, and next that it be sessile; I further laid it down that in cases of inversion following on parturition the placenta was invariably found to have been attached to the central portion of the fundus. Further, so far as my own experience went, I came to the conclusion that fundal attachment was not alone the predisposing but was the only cause of inversion occurring.

I had during my long professional life—extending as it did over more than fifty years of active practice, including in it ten years' residence in the Rotunda Hospital, first as assistant and then as Master—introduced my hand very many times in cases of retained placenta, some of them very firmly adherent, and in no single case did I find the attachment anywhere except laterally, and in none of these was there the least tendency to inversion. I have often wished to have had some independent testimony on this point, but none that I am aware has been offered; therefore, on seeing the report of the two recent cases mentioned above, I wrote asking for information as to the site of the placental attachment in each case, and have received the following replies, for which I desire to thank the writers.

Dr. George Hall writes:

The placenta in my case was attached pretty nearly to the centre of the fundus uteri.

Dr. Arthur N. Holmes, late senior assistant, Rotunda Hospital, who had charge of Dr. Tweedy's case, writes:

The patient, a primipara, delivered herself normally. About fifteen minutes after the infant's birth the patient cried out, saying the pains were returning. A few minutes after, she having cried out again, the placenta was noticed bulging through the vulva. Then, placing my hand on the abdomen, I realized the absence of the fundus above the pubes, and immediately after complete inversion of the uterus took place. I then noted that the placenta had a fundal attachment, and was adherent by its centre.

Dr. A. W. Anderson (Dalbeattie, N.B.), who published particulars of a third case in the *BRITISH MEDICAL JOURNAL* for May 2nd, writes:

The placenta in my case was centrally attached, and was comparatively easily detached.

These three cases go a long way in confirming the opinion I enunciated in the paper referred to, and which was published in 1881—"that for inversion to occur immediately after parturition the placenta must be attached nearly centrally to the fundus"—but I have as yet seen no case reported confirming the remainder of the paragraph, in which I said that for it to occur under any other circumstances it must be "in consequence of the presence of a fibrous tumour springing from the fundus." I also pointed out that such never occurred except when the tumour was sessile. Perhaps some of the readers of the *BRITISH MEDICAL JOURNAL* may have seen cases bearing on this point.

The two classes of cases in which inversion occurs being analogous, so far as the site of attachment of placenta or tumour is concerned, the question to be solved is, Why does a sessile tumour springing from the very fundus, or a placenta attached there, predispose, if not actually cause, inversion of the uterus?

Any gynaecologist who uses with frequency the uterine sound must have been struck, as I often was, by the fact that, while the patients seldom feel any pain while it is being passed through the cervical canal, if that be skillfully done, they will cry out if the point of the uterus be pressed even gently against the fundus. Again, if fangle tents be used to dilate the cervical canal—and I used them very frequently during my long years of actual practice—they were borne without causing much discomfort for hours, and remained *in situ* till removed by the operator, provided they were so short as not to touch the fundus; but they would, if too long ones were used, soon induce pain and be expelled long before they had swollen sufficiently to dilate the cervix. I think from these facts it may be inferred that not alone is the centre of the fundus the portion of the uterine wall most